George J. Thomber

The ART of RESUSCITATION

By

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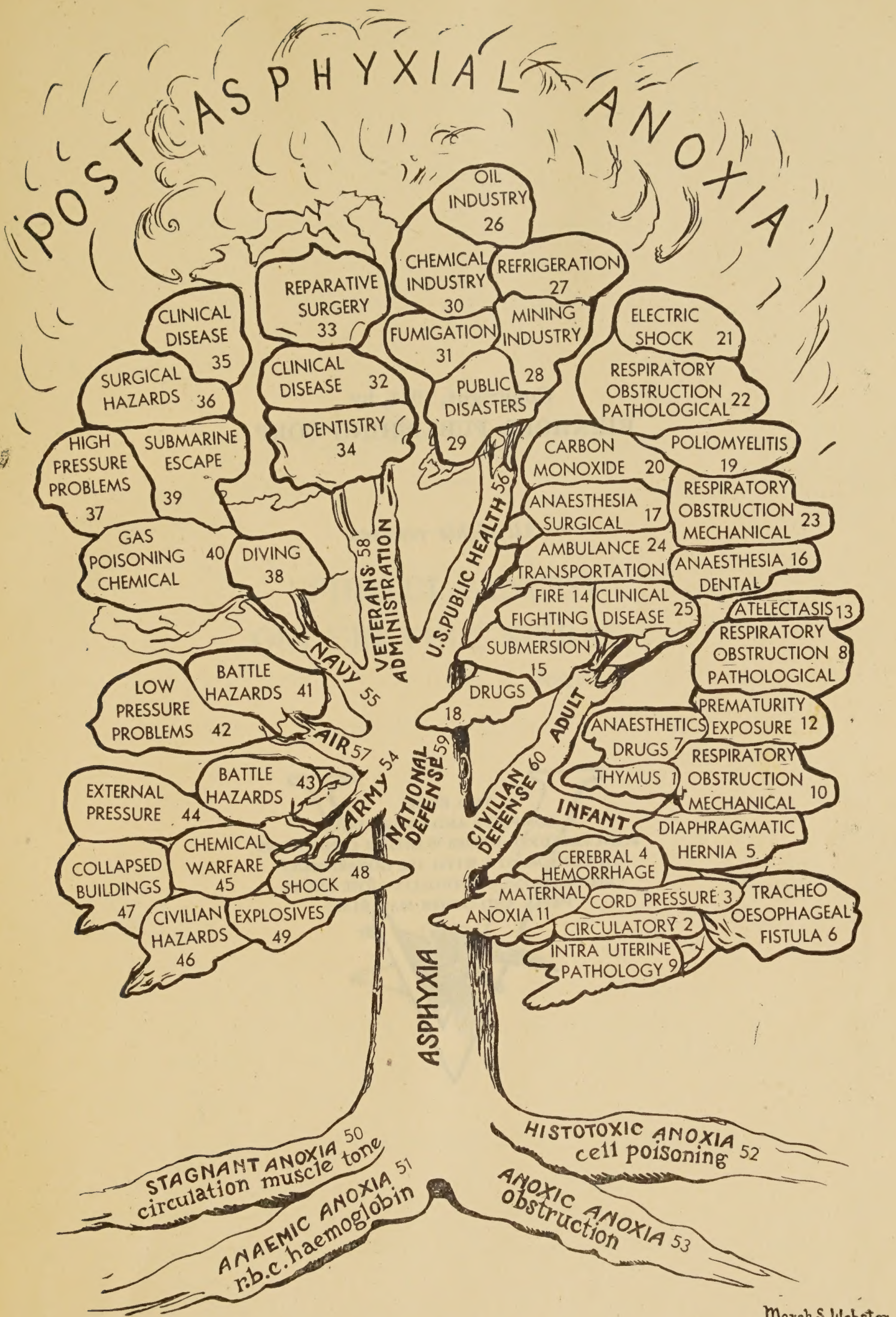

The author's conception of asphyxia, indicating its sources and suggesting an integration and a segregation of its predisposing pathological and clinical etiology.

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Foreword

The first thought that comes to my mind in reviewing your book is the great number of lives it will save. You have a happy way of presenting things that will attract many a reader, and to read is to learn. To learn from this book means to save human lives. Preventable asphyxial deaths are still appallingly frequent, and once again you have contributed enormously to the lessening of their number.

Just as I agreed with you many years ago that anaesthesiology must be recognized as an independent department of medical science and clinical work, I quite agree with you now that the time has come for medical schools and hospitals to recognize the fact that Pneumatology is a departmental load that must be taken off the shoulders of the over-burdened anaesthesiologist.

Your book merits and will certainly have wide circulation.

Philadelphia, Pa.
July 27th, 1944

Chevalier Jackson

Preface

The purpose of this book is an intensely practical one. It is an attempt, based upon more than twenty-five years of intimate experience with the unconscious patient, to tell the reader what to do when faced by an acutely asphyxiated patient about to die. Nothing in the entire field of medicine calls for more understanding and intelligence, for greater calmness and speed combined with a restrained, precise, but strong technique. The instant reward is a life saved at a cost which will vary from a ruptured spleen, fractured ribs, bronchopneumonia, lung abscess or late cerebral effects of prolonged anoxia, on the one hand, to a total absence of morbidity on the other.

The dramatic and disturbing environmental factors of the asphyxial accident are due to several causes, *i.e.*, immediate and real danger to life, limited time available for treatment, lack of familiarity with the pathologic physiology involved, ignorance of the appropriate technique to apply in the given case, traditional resistance to instrumentation which may be indicated, routine reliance upon mechanical robots, and despair when these fail.

Fifteen years ago the field of asphyxia was dark; the only discernible glow emanated from the occasional genius in experimental or clinical medicine through whose enthusiasm the pathologic physiology of asphyxia has evolved. The awakened interest of organized medicine, however, has illuminated the field. In the light which has now broken we are bewildered in the attempt to do justice to the expanding scene, for it not only embraces the customary haunts of the physician, his hospital and public-health activities in war and peace, but it has entered the skies as one of the chief medical problem of high-altitude flying, and has penetrated to the depths of the seas, in its attempts to protect the deep-sea diver and the personnel of the submarine.

In spite of the scope of the problem, which might seem universal had not clinical medicine sharply circumscribed it, we find a readily discernible common denominator. In each and every asphyxial accident the first and immediate indication is to provide oxygen. This therapy constitutes resuscitation by an appropriate technique. If resuscitation is successful, the patient lives; if it is unsuccessful, he dies—in asphyxia. Asphyxial death is therefore that type of fatal accident in which asphyxia is the dominant pathologic lesion. Other factors are contributory or yield promptly to resuscitation, *i.e.*, submersion electric shock, foreign-body obstruction, etc.

The writer freely admits that he has contributed but little which is original in the pages which follow. His task, which has become a duty, is to suggest a logical integration, from which it is hoped an acceptable unity has been evoked.

The patient in whom unconsciousness has been artificially induced (anaesthesia) presents a pathologic physiology which accidental asphyxia closely resembles. For example, a patient undergoing caesarian section may regurgitate during operation and drown herself in her own gastric contents, an asphyxiated new-born whose airway is filled with amniotic fluid and detritus, and a bather at a city beach whose lungs may be polluted by sea water, vomitus and blood present indications for treatment which are identical. Each of these three accidents requires that the obstructive foreign matter be immediately removed and oxygen placed in a position where it will be most effective. If, therefore, these problems are identical, why not deal with them as such, by coördinating personnel responsible for treatment and making the necessary equipment available.

The matter presented herein is elementary. It presents a framework upon which the experiences and the contacts of the author may readily be studied. Should this form and matter prove of interest, the entire subject, it is hoped, will attract the attention and enlist the activities of highly qualified research workers and technicians, as did the author's book, "The Art of Anaesthesia".

The subject of resuscitation has been developed quite simply as follows. It was first found necessary to define the term, since "Resuscitation", as used in England, has meant chiefly circulatory stimulation, whereas in America it implies artificial respiration.

Since, as has been well said, there is nothing new in medicine, a brief reference to the historical background is presented. This background seemed to the author to emphasize rather clearly activity directed first by the individual investigator,* and secondly by organized medical groups. The rapid acceptance and the full support by organized medicine of the movement to prevent asphyxial death has proved a continual source of surprise to the author. His personal efforts to enlist interest have invariably met with complete coöperation.

Resuscitation implies rescue—rescue from the respiratory and circulatory collapse accompanying asphyxial accidents. The author regards asphyxial death as a major medical problem, a generic pathologic entity. It has therefore become necessary to set forth the pathologic physiology of asphyxia. In preparing this matter, the investigations of the late Dr. Pol Coryllos have been freely drawn upon. To this matter were added important contributions by Dr. Herbert Chase of New York City

The pathologic physiology of asphyxia has been developed from two

* Papers appearing in the Transactions of the S. P. A. D., Inc., presented by the late Dr. James Walsh and by Dr. Henry Hall Forbes. These have been freely drawn upon as may be noted in the textual style of this section.

points of view: from that of experimental physiology—the viewpoint of the research worker, and from that of clinical medicine—the viewpoint of the man who attends the case.

After a statement of the nature of the problem to be treated, the principles of resuscitation which may be deduced are considered. These principles provide a reliable index by which manual and mechanical methods of resuscitation may be appraised. The emphasis is very properly shifted from apparatus to patient. It is clear that the importance and value of any mechanical device turns upon the degree to which it contributes to the pathological indications requiring relief. A lack of familiarity with this matter will suggest why it is that so many have nonchalantly relegated these most desperate medical emergencies to non-medical engineers and technicians.

Following a consideration of manual and mechanical methods of resuscitation, the attention of the reader is directed to the hazard involved in the transportation of the unconscious patient. The suggestions offered for improvement are not complicated; they may easily be adopted, in whole or in part.

Having prepared a basis for the considered treatment of the generic problem of asphyxia, it becomes desirable to consider in some detail the specific causes of asphyxial accidents and the indications requiring relief.

The chapter on "Asphyxia in the United States Army" is chiefly devoted to the question of war gas poisoning, a subject of much present interest. The approach to this problem has been by way of pathological physiology. An effort has been made to report just what happens to the respiratory and and circulatory systems after exposure to various poison gases. In the vast literature dealing with poison gases, the author was surprised to find relatively few references to the pathological lesions produced. When these lesions are made plain, the indications for treatment become clear. This chapter has been read by the Office of the Surgeon General.

In 1939 Ross T. McIntire, Surgeon General of the United States Navy, presented a paper entitled "Asphyxia in the Navy" at the opening of the Department of Pneumatology at the New York World's Fair (p. 17). This paper has served as a nucleus for the corresponding chapter in this book. The author wishes to take this opportunity to express his appreciation to General McIntire for his many acts of thoughtful kindness in connection with the movement to prevent asphyxial death.

Grateful acknowledgment is made at this time to the Surgeon Generals of the United States who for ten years have encouraged the prevention of asphyxial death. During this period Generals Robert U. Patterson, Reynolds and Magee of the Army, Rossiter and McIntire of the Navy, and Cummings and Parran of the U. S. Public Health Service, have extended many courtesies as members of the advisory board of the S. P. A. D.

Much interest in the subject of asphyxia of the new-born has been expressed by the State Directors of Maternal Health and Child Welfare of the Children's Bureau. Reference to this interest appears in the chapter "Asphyxia Neonatorum".

The interest of organized medicine in the problem of asphyxia is typified by the report of the Committee on Asphyxia of the American Medical Association. The author wishes to extend his thanks to Dr. John S. Lundy and Dr. Thomas Vischer, fellow-members of this Committee.

Acknowledgment is made to the United States Navy and to the United States War Departments of Publication, to the *American Journal of Surgery*, to the *Maine Medical Journal*, to *Modern Hospital*, to Ciba Pharmaceutical Products, Inc., and to Upjohn Company for permission to reproduce material appearing in this text.

In 1936 the superintendents of all the hospitals in the United States with a bed capacity of 100 or more beds were invited to act on a Committee on Asphyxia to consider its control within the hospital. Seventeen hundred hospital Superintendents received invitations; 335, representing 46 states, accepted this invitation, constituting an extraordinary institutional interest in the problem of asphyxia. The project is referred to in an appropriate reference in Chapter 28.

Thanks are also extended to the many loyal members of the Advisory Board and the Board of Directors of the S. P. A. D. in the person of Judge Oscar Ehrhorn, Dr. Lee Hurd, Dr. John F. McGrath and Dr. Robert Wilson.

Extended experience in the prevention of asphyxial death has convinced the author that the coördination of gas therapy under a comprehensive term such as Pneumatology is essential to progress in what has become a major field. The physician familiar with the administration of anaesthesia is not only the logical but the best-trained man.

Realizing the innumerable opportunities for errors in attempting to treat a field as extensive as asphyxia, the author has turned to his friends for assistance in eliminating the more conspicuous faults.

Dr. Walter Boothby has been good enough to read the chapters on the "Generic Problem" and the "Principles of Resuscitation".

Dr. Edward H. Dennen kindly reviewed the chapter "Asphyxia Neonatorum".

Dr. Don Gudakunst, Director of the National Foundation of Infantile Paralysis, rendered valuable assistance in the preparation of the chapter "Asphyxia in Polio".

Dr. Olsen of the Brooklyn Polytechnic kindly read the chapter "Asphyxia from Gases Encountered in Fires".

Dr. Roy Sayers, Director of the Bureau of Mines rewrote the chapter in connection with the work of the Bureau of Mines.

Dr. Malcolm Carr, President of the New York Academy of Dentistry, kindly reviewed the chapter "Asphyxia in Dentistry".

To Dr. Theodore Curphey, the author is indebted for much encouragement and assistance in bringing this work to completion; he is also grateful to Dr. Andrew A. Eggston for valuable suggestions and to Miss E. Cameron Turner of Greenville, Ohio, for repeated courtesies.

The author wishes to thank Dr. C. C. Sweet for his assistance in securing material for the chapter on electric shock, and more particularly for his innumerable acts of kindness to many of the author's closest friends.

If the chapters on respiratory obstruction are of particular interest it is because of the extraordinary genius and enthusiasm of Dr. Harrison Martland, Medical Examiner of Essex County, whose activities in this field are of international significance.

Since the reader may have under instruction First-aid groups for national defense, simple instructional matter which may prove of assistance in this field is provided in a chapter dealing with the Instruction of Technicians.

Through the extreme kindness of the Surgeon General's Library, a bibliography for the fields of asphyxia and resuscitation is appended.

The author is particularly fortunate in the enthusiastic reception which his publishers, the Reinhold Publishing Corporation, have extended; without this vision and understanding, this volume might not have materialized.

Finally he wishes to express his appreciation to Francis Coccoza, Jane Richardson, Patricia deW. Flagg and Dorothy Flagg for assistance in preparing this text, and to Marah S. Webster, of Farmington, Maine, for assistance in preparing the frontispiece.

The task of preventing asphyxial death begins *in utero*. Continued life may crown our efforts, or inevitable death may supervene. In order that the smoldering spark of life may never be overlooked and that its final extinction may be formally confirmed, a chapter called "The Signs of Death" supplies the eternal finis.

New York, N. Y. PALUEL J. FLAGG
July, 1944

Part I

Definition and Background

Chapter 1

Definition of Resuscitation

"Resuscitation", a pamphlet issued by the British War Office in July of 1941, devotes forty-nine pages of text to transfusion and three pages to oxygen therapy. Webster defines resuscitation as meaning to revive, or to revivify; accordingly, the British usage is quite correct. In America, however, our acceptance of the term is quite different. Resuscitation, in American medical circles, immediately suggests the prevention of asphyxial death by artificial respiration.

It would seem that, from the point of view of urgency as well as from that of the interdependence of respiration and circulation, the American usage of the term finds support in the pathological physiology requiring relief. Asphyxia kills quickly, within 5 to 8 minutes, if it is severe; shock from trauma or hemorrhage, on the other hand, is a matter of hours, or even days.

One of the best circulatory stimulants is deep, free respiration with conservation of tissue and blood carbon dioxide. Mechanically, deep inspiration empties the veins and assists the right heart. Chemically, the addition of oxygen and the conservation of carbon dioxide restores muscle tone, thereby reducing capillary stasis and raising the blood pressure. This sequence of events is a common operating-room experience, as witness the circulation of an almost pulseless patient suffering from intra-abdominal hemorrhage. A few minutes of oxygen administration with rebreathing restores the pulse and the pallid skin becomes pink, as though reacting from a transfusion. Withdraw the oxygen and the rebreathing, and the respirations are quickly reduced in rate and volume and the patient lapses into his former condition of circulatory depression.

If, however, during the period of circulatory stimulation resulting from oxygen therapy, further blood loss is prevented by hemostasis, if blood volume is restored by transfused plasma, and if the oxygen-carrying red cells are replaced by whole blood, a lasting resuscitation may be effected which does not disappear upon cessation of treatment. Immediate oxygen therapy provides the necessary emergency pick-up to enable the patient to secure the full benefit of transfusion.

Reverse the sequence of treatment by immediate transfusion, to be complemented later on by oxygen therapy, and difficulty may be anticipated. Such a shocked patient, relaxed by overventilation of carbon dioxide resulting from the frequent breathing due to pain, depressed by sedation, and having a shallow, slow respiration accompanied by cyanosis which may scarcely be discernible because of the small volume of blood, may develop circulatory deterioration to a point where oxygen therapy will be of little avail.

The contrast is sharply drawn for the sake of emphasis. It is to be noted that in actual military practice, as reported by the British War Office, it is assumed that transfusion and oxygen therapy will be administered simultaneously. An integration of the British emphasis on transfusion and the American emphasis on oxygen therapy may be calculated to yield an approach to the problem of resuscitation which will become highly effective. The experience in resuscitation "gained at Dunkirk and from the air raids in 1940" is a model of clear thinking, concise statement and simplicity. Further reference is made to this report on page 45.

The pages which follow are intended to develop the art of resuscitation as a necessity, predicated upon the present high death rate from asphyxial accidents in civil as well as in military experience (see page 32). This death rate among civilians alone is conservatively estimated as 50,000 lives a year.

A curious and unfortunate situation has accompanied the evolution of resuscitation in the United States. The layman has been so far in advance of the physician in his contact with the patient, not only physically but in his First-aid interest, that resuscitation has become identified with lay relief. This situation has frequently reached an extreme in which the surgeon has called for the First-aid squad to assist him in resuscitating a surgical patient in the operating room. The call for such lay assistance by obstetricians, especially in residential practice, is a common occurrence. This identification of resuscitation with lay services has brought about a double effect, which has served to retard a serious professional approach to the problem. On one hand the physician has come to regard resuscitation as something beneath his professional attention, and on the other the rescue squad has come to look upon the asphyxiated patient as its exclusive problem to which the physician has little if anything to contribute. To add to the confusion, manufacturers of resuscitation apparatus designed to amplify and simplify the efforts of the rescue squads have flooded the field with mechanical robots, each fortified by unimpeachable scientific research quite unintelligible to the prospective purchasers. Indeed, this scientific support for mechanical devices for resuscitation equipment has reached a point where the average physician asked to pass judgment upon their relative merits is bewildered by the claims which he is asked to adjudicate by virtue of his medical education. Such a physician finds himself in a position in which he is asked to act upon a medical problem for which his medical-school training did not fit him, for asphyxia as a major medical problem and the scientific medical means of relief attracted little attention fifteen years ago (see page 30).

Asphyxial death and the technique of resuscitation by which it may be prevented constitutes both an emergency and a long-range problem. As an emergency, it engages the attention of the armed forces and those of civilian defense. It is faced daily by the physician on hospital duty and in general practice. As a long-range problem it becomes the obligation of medical schools and military training centers. It awaits development by the United States Public Health and by the Children's Bureau. The latter, through its State Directors of Maternal Welfare and Child Health, has expressed a lively interest in a proposal to provide a special training service to prevent asphyxia of the new-born.

The following pages are offered as a guide, or outline, to indicate the direction which it is felt emergency measures may safely take to help untangle the present chaos and to prepare the public for the considered approach of the teaching centers (Chapter 29).

The importance of apparatus must give way to the time-honored leadership of pathologic physiology. *What* is to be treated is more important than *how* treatment is to be applied. A clear understanding of the indications for treatment will not only suggest a mechanical means of meeting these indications, but it will reduce this treatment to the simplest terms. It will prepare the physician to form a just appraisal of the mechanical devices which may be brought to his attention.

Anaesthesia becomes an art when safety, efficiency and comfort are achieved, simply, with a minimum of morbidity. Resuscitation becomes an art when the pathologic physiology requiring relief is clearly understood and where the indications for treatment are met promptly, precisely, and without trauma. Within the limits of their education in fundamental medicine, technical groups are now making and may continue to make a priceless contribution.

Chapter 2

Background

Historical References to the Evolution of the Art of Resuscitation

" 'Asphyxia' is almost exactly a transliteration from the Greek 'asphyxia', the Greek upsilon being replaced, as is customary, by our English 'y'. The word which has now come to mean shutting off of the breath, meant in the original Greek a stoppage of the pulse. It was noted from very early times that any incident which shut off the breathing, such as choking from the effort to swallow too large an object, or drowning, or strangling, very soon caused a stoppage of the pulse and the heart beat."

Until very recently indeed the reaction of the average medical man, from chief to ambulance surgeon, has been to react to the asphyxial emergency as though recovery depended first upon the stimulation and the support of the pulse and secondarily upon the stimulation and support of the respiration. Increasing cyanosis and labored breathing have been taken as a matter of course, circulatory stimulation being calculated to eliminate these objective signs. Even today the stertor of the unconscious patient struggling for air fails to provoke an interest in those who have the responsibility of her care. Recently the author's attention was attracted by the sound of a patient's labored breathing in an adjoining room. This patient had received avertin. She lay with her head flexed upon her chest; the physician who had administered the avertin was nowhere in sight. Her nurse gazed upon her, mildly interested by these strange sounds, but quite unaware of the progressive cyanosis accumulating in the darkened room. Simple extension of the patient's head immediately freed the airway so that the respirations became so quiet they were no longer audible. The relief was so striking that the nurse was speechless. Within the last two years, the author has seen a desperately ill patient in a Drinker respirator, his pharynx filled with mucous and vomitus which was churned back and forth through his larynx by the action of the respirator. Those in attendance were quite unaware of the fact that while they were carrying on artificial respiration they were at the same time effectively drowning the patient in his own secretions.

Because of these things which occur daily in our midst we may better appreciate the "asphyxia consciousness" of medical men who have gone before us—men who grasped the need of immediate respiratory relief and were quick to apply it. Before the suggested coördination of the causes of asphyxiation (p. 31), early historical records refer to a number of conditions frequently causing fatal asphyxia. These may be grouped as inflammations and ulcerations of the larynx, suffocation from foreign bodies too large to be swallowed, strangulation, diptheria and asphyxia of the new born.

"Among the Greeks in that golden age of medicine which began with Hippocrates it was recognized that various forms of inflammation of the larynx might shut off respiration with the production of asphyxiation. Among these diphtheria was probably the most frequent. The word has a Greek etymology but the term was not invented until about a century ago by the Spaniards or perhaps by Bretonneau. Aretius, who lived in the first century, mentions diphtheria under the term, *ulcera Syriaca* (Syrian ulcers) as if it were a rather common disease. The next mention of it comes among the Spaniards who called it *garrotillo*, that is, the little garrote or choking affection. A series of epidemics of this disease were noted and always the tendency to produce asphyxiation was set down as characteristic of it. We had epidemics of it in this country in the seventeenth century when it was spoken of as the disease of the bladders in the throat, a descriptive term for the false membrane which forms above the mucous membrane, portions of which may be removed and look not unlike the sort of tissue that makes up the bladders of animals.

"Human beings have always been familiar with the shutting off of respiration by means of choking when an attempt was made to swallow something that was too large, which got beyond the back of the gullet and yet could not be forced downwards. It was caught, moving neither up nor down. In children particularly this was sometimes the cause of death. There was a tradition in old Irish medicine that when something like this happened in a child and it began to get a little bluish in the face, this was the signal that there was a dangerous blocking of the respiratory passages. The child should be struck firmly on the back between the shoulder blades or a little below them with the open hand in the hope of causing an eructation that would bring the offending object up."

Viewing these conditions from the standpoint of resuscitation, we may trace three methods of approach through the centuries:

Tracheatomy, in which the respiration is short-circuited below the point of obstruction by an opening made in the trachea.

Intubation (**Canalization**), in which a rigid or semi-rigid tube is passed through the obstructed area to the normal trachea, the patient being allowed to breath through this or air being forced into it.

Oral insufflation, in which the operator's breath is forced through the obstructed area, to and into the trachea by mechanical pressure or by the rescuer's breath (mouth-to-mouth insufflation).

Tracheatomy

Col. Fielding H. Harrison suggests that there is not only the possibility but the probability that prehistoric man may have slit the trachea of the child or adult suffocating from croup or diphtheria as the savages of Central Africa have been known to perform abdominal section. It is said that the surgeons of Salerno, Bologna, and Paris practised tracheatomy as a surgical routine in the twelfth and thirteenth centuries. The first successful case of tracheatomy in modern times was performed for Elizabeth de Puysegur on July 1, 1825 by Bretonneau.* Trousseau was the first

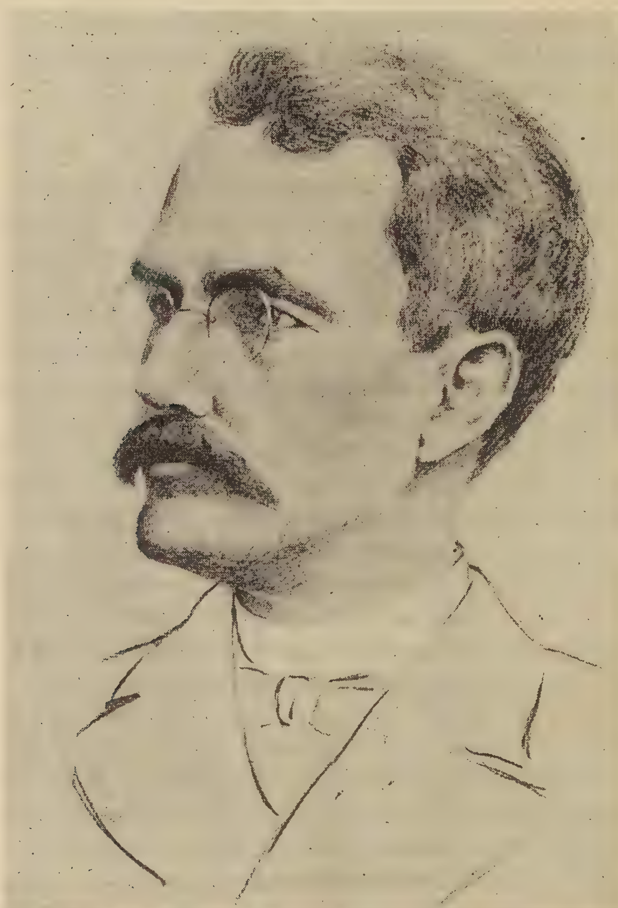

Fig. 1. Dr. Joseph O'Dwyer

Paris clinician to perform tracheatomy for diphtheria. His operation was performed on Gustave de Marcillet on November 23, 1831.† Gurlt.‡ has described the early operations for tracheatomy beginning with Aesclepiades in the second century B.C. and coming down to the sixteenth century.

* "Des Inflammation spéciales du tissu muqueux," Paris, 1826.
† *Journalle des Connaissances Medicales*, Paris, 1833–34.
‡ *Geschichte der Chirurgie*, Vol. 3.

Intubation (Canalization)

Hippocrates suggested the possibility of putting a tube into the larynx in inflammatory and mechanical conditions. In the first quarter of the nineteenth century Desault succeeded in placing a tube in the larynx. About 1820 Patissier, another French surgeon, suggested the employment of the same procedure. Owing to the difficulties involved in the contrivance and the retention of an endotracheal tube, the practice was discontinued until its development by Dr. Joseph O'Dwyer of New York City.

According to Dr. Fielding H. Garrison* of the Welch Memorial Library of Johns Hopkins University, "Intubation first introduced by Eugene Bouchut (1818–1891) and first done in Paris in connection with tracheatomy by Trousseau (1851–1859) was perfected through the conscientious labors of the self-sacrificing Joseph O'Dwyer whose name stands with those of Semmelweis and Crede as the great benefactor of infant life." O'Dwyer's efforts were directed toward avoiding the older method of tracheatomy; he attacked the problem by relieving the local laryngeal condition through the normal airway.

Dr. O'Dwyer was graduated from College of Physicians and Surgeons of Columbia University in 1866. In 1872 he was appointed to the staff of the New York Foundling Hospital. In those days a diphtheria epidemic used to have a mortality of 40–50 per cent. Dr. O'Dwyer saw and was touched with pity by the suffering of little children two to three years old, blue and struggling for air, grasping the sides of their cribs. On April 23, 1884, he first applied the "Oval tube" in a three-months old baby suffering from dyspnoea caused by diphtheritic laryngitis. The account reads: "Dr. O'Dwyer put in laryngeal tube and it afforded instant relief. Tube soon coughed out, too small. Immediately another of a larger size was put in and again afforded relief."

Dr. O'Dwyer's first public presentation of his method appeared in the *New York Medical Journal* in 1885 under the title "Intubation of the Larynx." The method was soon applied in treating an adult, as fully reported in the *Journal of Respiratory Organisms* in 1890 under the title "Intubation in a case of diphtheretic laryngitis in an adult." The *Transactions of the New York Academy of Medicine* for 1892–1893 carry a further discussion of the relative values of intubation and tracheatomy in the treatment of acute stenosis of the larynx of children; and the International Congress on Internal Medicine held in 1890 carries a current discussion of his method of intubation as appreciated abroad. Early in 1894 Dr. O'Dwyer brought the subject up to date in a paper delivered before the New York State Medical Society under the title, "The present status of intubation in the treatment of croup"; and so interested were foreign coun-

* "History of Medicine," p. 351, W. B. Saunders Co., Philadelphia.

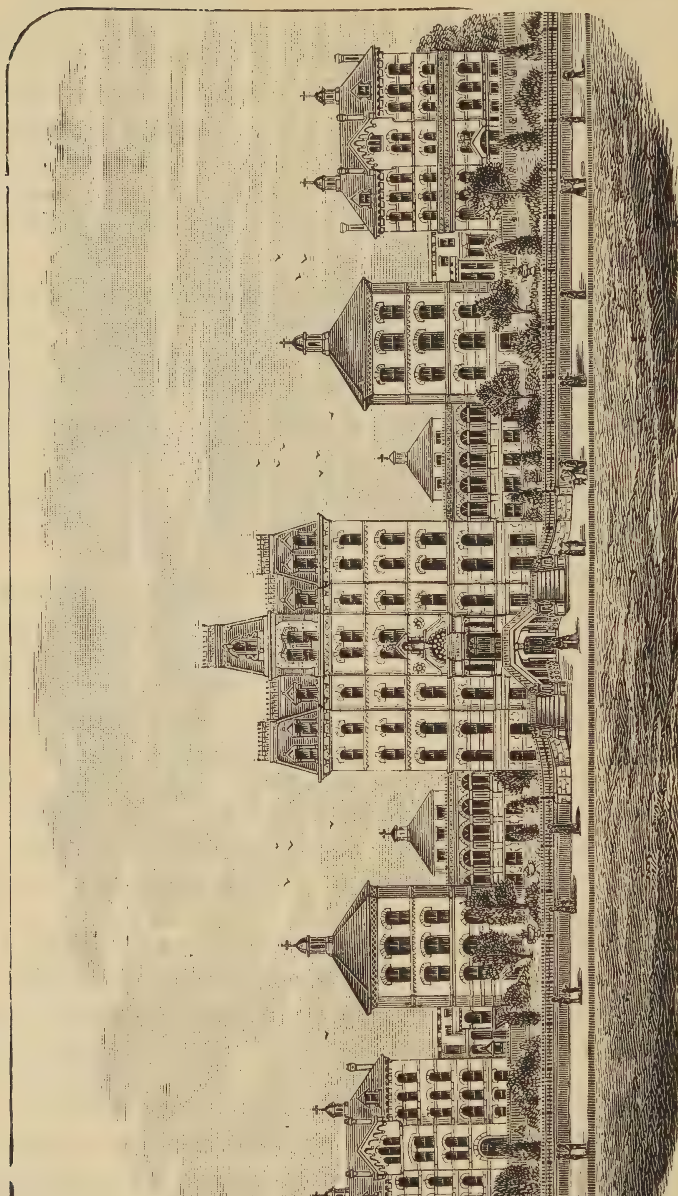

Fig. 2. New York Foundling Hospital, 1884.

tries in his findings that medical journals in Norway, France and Germany soon afterward carried special articles on the subject. In 1897 the *Archives of Pediatrics* published a valuable technical article on "Retained intubation, tubes, causes and treatment."

Courtesy Anaesthesiology

Fig. 3. Fell O'Dwyer Apparatus.

But the invention which has the widest application in relieving asphyxiation from a number of causes was first described publicly by Dr. O'Dwyer in 1892 before the American Pediatric Society. With his usual simplicity he called his talk "An improved method for performing artificial respiration". As usual he refused to take much of the credit for the development and described his apparatus as "a modification of the method devised by

Dr. George E. Fell of Buffalo." At this time he reported that the instrument had been in use in the New York Foundling Hospital for six years, for some of the time when it was still in the experimental stage. He warned that the chief danger was from over-distension of the lung and rupture of the pulmonary air vesicle. He suggested that the method might be of value in preventing blood from entering the air passages in operations in the throat. The idea was well received in many quarters, and in 1894 he was invited to talk before the British Medical Society.

This method of forcible respiration was applicable in many types of temporary dysfunction. A case cited before the British group was that of a cerebral tumor at the Presbyterian Hospital in New York City. While the patient was walking about the ward, a sudden pressure of the tumor paralyzed the respiration. Dr. O'Dwyer and Dr. Northrup were immediately called, and forcible respiration was applied for 24 hours until the pressure was removed. The patient recovered and lived for quite a period in good health under the care of Dr. Flint.

In the fall of 1909, twenty-five years after Dr. O'Dwyer's first recorded intubation with the oval tube, it was the author's privilege to receive instruction from Dr. O'Dwyer's son in this technique. Twenty-five years later, he invited Dr. O'Dwyer's son to a banquet at the Hotel Biltmore given in honor of his father in connection with the second annual conference of the S. P. A. D. in February, 1934.

The evolution of the author's experience in resuscitation may be briefly referred to as follows: Out of the field of unconsciousness artificially induced by general anaesthesia came an opportunity to study the phenomena of asphyxia. There is a close similarity between the asphyxia seen in anaesthesia and that complicating asphyxial accidents. Indeed, asphyxia *per se* rapidly induces the unconsciousness, spasticity, and flaccidity effected by anaesthetic agents. Studies directed to the clinical measuration of the oxyhemoglobin content of the blood in the early twenties, complemented by a simplified technique developed a few years later with the aid of Dr. Chevalier Jackson designed to overcome the asphyxial complications common to surgery of the head and neck, provide the clinical background for the matter hereinafter presented.

Intimate and personal experience with asphyxia neonatorum suggested the desirability of applying surgical principles to the needs of the asphyxiated baby. The application of these principles, namely, illuminated exposure of the field of operation, removal of foreign matter by suction, and the application of treatment where most effective, *i.e.*, endotracheally, yielded exactly the result anticipated. The dramatic rescue of infants who were looked upon as doomed, by residents and visiting obstetricians, was reported to the author with ever-increasing frequency. At the same time unskilled treatment applied to poorly selected cases developed a resistance to the general acceptance of the principles advocated.

These references to the background of resuscitation have dealt with the interest and the activities of the individual physician in his efforts to prevent asphyxial death. We shall now consider briefly the efforts of organized medicine.

Efforts of Organized Medicine. Repeated clinical success in the resuscitation of asphyxiated babies from 1927 to 1931 suggested the desirability of studying other types of asphyxial accidents. It occurred to the author that perhaps the many different types of accidents in which artificial respiration was indicated as the primary treatment might represent disassociated specific causes of the same generic problem. The conception that asphyxia constituted a major medical problem of important dimensions was confirmed by Drs. Walter Niles, Yandell Henderson, Alexis Carrel, W. H. Howell, Chevalier Jackson, and others. At the suggestion of Dr. Carrel, the author examined the vital statistics of New York City and found that when the numerous causes of asphyxial death are assembled, the mortality from asphyxia in the United States is in excess of 50,000 a year.

Society for Prevention of Asphyxial Death.* The *New York Times* for February 14, 1933 carried the following news item: "Doctors form Society aimed at saving 50,000 lives now needlessly lost yearly; organize to check asphyxial death; new Society announced at the meeting of the Society of Medical Jurisprudence at the New York Academy of Medicine." The new Society referred to, The Society for the Prevention of Asphyxial Death, or the S. P. A. D. as it is usually called, had commenced a movement which in the next decade was to make the American physician "asphyxia-conscious."

Organized medicine was quick and generous in welcoming the new movement. First to offer support and assistance was the Academy of Medicine through its Director, Dr. Linsley Williams. The Committee on Public Relations endorsed the new movement and assisted in the preparation of a full day conference in Hoosack Hall, supported by an exhibit and demonstrations, which took place on May 24, 1933.

The conference, the first integrated effort of its kind, brought to a focus many new points of the problem. Vital statistics were discussed by Dr. Shirley Wynn, assisted by Dr. Haven Emerson. The Medical Examiner's findings were brought out by Dr. Harrison Martland, and discussed by Dr. Thomas Gonzales. The economic aspects of asphyxia were pointed out by Albert H. Whitney and discussed by Leon Senior. First-aid methods employed by the Police Department were described by Dr. Daniel J. Donovan. The afternoon session was distinguished by papers from Chevalier Jackson (discussor, Dr. Charles Imperatori); from Dr. Yandell Henderson (discussor, Dr. Dayton J. Edwards); from Dr. Edmond

* A Membership Corporation under the Laws of the State of New York, located at 38 East 61 St., N. Y. C.

New York Times
February 14, 1933

ORGANIZE TO CHECK ASPHYXIA DEATHS

Doctors Form Society Aimed at Saving 50,000 Lives Now Needlessly Lost Yearly.

NEW TREATMENT IS USED

New York Evening Pos

February 14, 1933

DOCTORS BANDED TO CURB ASPHYXIA

Society to Use Advanced Method of Treatment—Needless Loss of Life Cited

New York Sun

February 14, 1933

FIND ASPHYXIA DEATH TOLL BIG

New York American
February 14, 1933.

ANTI-ASPHYXIAL SOCIETY FORMED

N. Y. Times
June 11, 1933

DEATH RATE HERE TAKES ANOTHER DROP

Utica, N. Y.
PRESS
May 1, 1933

For Prevention of Asphyxial Death

New York Sun
May 23, 1933

ASPHYXIAL TOLL HIGH

Physicians to Confer Over Problems Involved.

N. Y. Evening Post
May 22nd, 1933

GAS, SMOKE TOLL TWICE CAR DEATHS

State Conference Here Wednesday to Plan Drive on Asphyxial Casualties

Asphyxial deaths, or deaths from gas poisoning, inhaling smoke, elec-

Buffalo, N. Y.
NEWS
May 26, 1933

DOCTORS UNDERTAKE ASPHYXIATION DRIVE

Many of 50,000 Deaths Annually in U. S. Are Declared Preventable.

N. Y. Evening Post
May 24, 1933

WYNNE HAILS WAR ON INFANT DEATHS

N. Y. TIMES
May 25,

FIGHT ON ASPHYXIA PLANNED BY SOCIETY

N. Y. American
May 25, 1933

CRUSADE OPENS TO CUT DEATHS BY SUFFOCATION

Cincinnati, Ohio
Post
May 25, 1933

CAMPAIGN TO CHECK ASPHYXIAL DEATHS

Academy of Medicine Launches Prevention Drive

Brooklyn, N. Y.
EAGLE
May 24, 1933

Seek to Reduce Asphyxial Deaths

New York Times
May 26, 1933

Death by Suffocation. Only at first sight is there an odd touch about a Society for the Prevention of Asphyxial Death. Where is the enemy to be attacked, like the dread bacillus of tuberculosis or the unidentified agent that lurks behind cancer? Yet a moment's reflection will show that it is the magnitude of the problem which constitutes the challenge. What difference does it make whether the toll of human life is paid to a micro-organism or to human carelessness and indifference?

N. Y. Evening Post

May 1, 1933

PARLEY ON ASPHYXIATION

Fig. 4. Press reports of the organizing of the Society for the Prevention of Asphyxial Death, Inc.

B. Piper (discussor, Dr. H. J. Stander); and from Dr. Pol Coryllos (discussor, Dr. Horatio Williams).

The character of the support accorded this first conference is significant. Every invitation extended was accepted. The medical press gave it full publicity; the *Journal of the American Medical Association* ran an editorial and sixteen columns of abstract material. Lay press notices before and after the conference appeared in New York State, Ohio, Tennessee, Michigan, Pennsylvania, West Virginia and elsewhere. The movement to prevent asphyxial death was on its way. Its progress was noted by an exhibit on asphyxia at the Century of Progress at Chicago and at the Lake Keuka Medical and Surgical Association on July 13–14, 1933.

By this time Surgeon Generals Cummings, U. S. Public Health, Rossiter, U. S. Navy, and Patterson, U. S. Army, had joined the Advisory Board of the S. P. A. D. In less than a year, the Society of the State of New York (May 14, 1934) and the American Medical Association (June 12, 1934) had approved its aims and purposes.

The 128th Annual Meeting of the Medical Society of the State of New York convened at Utica on May 16, with a symposium on asphyxia, which Dr. Chevalier Jackson declared to be "Just about the most perfect presentation of the subject we have ever heard."

Increasing and broadening interest in asphyxia found expression in the following year, 1934, in a two-day conference at the Hotel Biltmore, New York City. There were morning, afternoon and evening sessions. Respected and familiar names appeared in this program. Drs. Horatio Williams, C. H. Watson, Charles Norris, Howard W. Neail, Henry Hall Forbes, James J. Walsh, Harrison Martland, Alexander O. Gettler, Wendell Phillips, General C. R. Reynolds, Col. F. L. Devereux, Drs. John D. Kernon, R. R. Sayers, Major Fox and Col. L. H. Bauer, Capt. E. W. Brown and Dr. M. H. Foster. At the closing banquet appeared Dr. W. P. Northrup and Dr. John Hartwell.

Once more the *Journal of the American Medical Association* devoted eight pages of abstracts to the papers presented. The scientific success of the program was equalled only by its economic failure. The conference was handicapped by a printer's strike which held up its mailing of 14,000 booklets and programs, so that many of these were not received until after the meeting. The famous blizzard of February, 1934 struck it fairly amidships. Dr. Chevalier Jackson wrote, "I had a farmer out with a good strong team to break the road, but he came back and reported that the horses would stop when they got into the snow up to the collar. I am sorry that this heavy storm synchronized perfectly with the morning of the meeting."

A model department of Gas Therapy was established in a suburban hospital by the Society. The Director was made municipal consultant in S. P. A. D. to the City of Yonkers, N. Y. During the year 1936, the

SECOND ANNUAL CONFERENCE
FEBRUARY 19-20, 1934
BILTMORE HOTEL, NEW YORK CITY
THE PURPOSE OF THE SECOND ANNUAL CONFERENCE IS TO CONSIDER

New York Times
February 21, 1934

INJURIES TO BRAIN FOUND IN ASPHYXIA

Federal Study Reveals Deaths From Carbon Monoxide May Be Cut by New Therapy.

TIME
March
1934

Asphyxia

Tasteless and odorless carbon monoxide crumples the coal miner, turns his body cherry red. From the exhaust pipe of his automobile comes the same deadly gas to

New York Times
February 20, 1934

ASPHYXIA TOLL PUT AT 50,000 YEARLY

Large Percentage Avoidable, Doctors Hold—Hospitals Urged to Extend Help.

S·P·A·D

SOCIETY FOR THE PREVENTION OF ASPHYXIAL DEATH, INC.

Incorporated: February 8, 1933

Aims and purposes approved by
Medical Society of New York State - May 14, 1934
American Medical Association - June 12, 1934

THE JOURNAL

OF THE

American Medical Association

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OLUME 101, No. 5 ... ICAGO, ILL. JULY 29, 19

Current Comment

ASPHYXIAL DEATH

In last week's issue of THE JOURNAL appeared the first instalment of the published proceedings of the Society for the Prevention of Asphyxial Death. The concluding instalment appears in this issue. This society was organized to focus medical attention more definitely on the importance of asphyxiation as a cause of death, particularly in relationship to carbon monoxide poisoning, drowning, electrical shock, smoke poisoning and acute alcoholism. It is estimated that more than 50,000 lives are lost every year in this way. There has been a tendency in industry and in government to attempt to handle such cases largely by the use of lay teams rendering first aid and practicing muscular and ... resuscitation long before any attempt is ... medical advice. Such aid may ... measure of first aid, ... be saved by

T INDEX

Fig. 5. The S.P.A.D. approved by the American Medical Association.

S. P. A. D. contacted the superintendents of all the hospitals in the United States with a capacity of 100 beds or more. Invitations were sent to 1700 hospital superintendents to serve on a committee to study asphyxia from the point of view of hospital administration; 335 acceptances were received. Twenty represented hospitals of 2000 beds or more. Forty-six states were represented. Invitations were also sent to all anaesthetists in the country. Of 338 appearing in the National Director, 177 responded to serve on a committee to study asphyxia from the point of view of anaesthesia. Both of these committees finally lapsed because of the impossibility of securing funds to operate them. It would be difficult to estimate the value of the creation of these committees, however, as a means of drawing the attention of these key men to the problem of asphyxia.

Fig. 6. Left to right: Dr. Nathan B. Van Etten, the author, Dr. J. Peter Houget, Surgeon-General Ross T. McIntire.

In 1938, the S. P. A. D. received an invitation from the New York World's Fair to take over the asphyxial problems in connection with the Fair. This invitation was accepted as an opportunity to organize and operate a model department of Gas Therapy or Pneumatology (Chapter 27).

The department secured and made available a modern assembly of equipment geared to the needs of the hour, proposing a working model for the integration of gases. It was opened with the following remarks: "The Board of Directors of the Society for the Prevention of Asphyxial Death welcomes you to the official opening of the Department of Pneumatology, of the Medical Department of the New York World's Fair. We commemorate on this occasion the one hundred and fiftieth anniversary of the opening of the Pneumatic Institute of Sir Thomas Beddoes, at Clifton, near Bristol, England, in the year 1789. The famous Pneumatologists

Pneumatology at the World's Fair

The New York World's Fair has created a Division of Pneumatology which will direct the therapeutic use of gases as these are employed for the relief of pain (anaesthesia), the saving of life (resuscitation), and for the treatment of clinical disease (oxygen therapy). This department will be housed in separate quarters offering adequate accommodations and completely equipped. The department will function in the double capacity of caring for the daily attendance of a quarter of a million visitors, and will serve as a practical demonstration of the most modern methods employed in this field. The various features of service may here be studied by institutional and municipal groups.

The full range of the field will be covered. This will include a system of communication, transportation of the patient on land and water, operating room treatment and emergency hospitalization. Suitable equipment will be provided to care for the following hazards: Asphyxia from gases, drugs, submersion, fire fighting, foreign body obstruction, tumors within the airway, from electrocution, strangulation, allergy, from pressure due to moving objects, collapse of buildings, from poisonous gases in manholes, and from the infrequent, but occasional, premature asphyxia neonatorum.

The Society for the Prevention of Asphyxial Death, Inc., whose pioneering activities in this field are well known, is cooperating in the organization and development of this important work.

Dr. McIntire Joins the S.P.A.D.

Ross T. McIntire, M.D., Surgeon General of the U. S. Navy, has accepted an invitation to serve on the Advisory Board of the Society for the Prevention of Asphyxial Death. He takes the place of Surgeon General Rossiter, retired.

The society, with main offices in New York City, now enjoys the cooperation of Surgeon General Reynolds of the U. S. Army, Surgeon General Parran of the U. S. Public Health Service, and Surgeon General McIntire of the U. S. Navy.

Therapeutic Use of Gases at World's Fair.—A division of pneumatology has been created by the New York World's Fair to direct the therapeutic use of gases as they are employed for the relief of pain (anesthesia), the saving of life (resuscitation) and clinical disease (oxygen therapy). The department will function in the double capacity of caring for visitors and as a practical demonstration of the most modern methods employed in this field, which may be studied by institutional and municipal groups. Suitable equipment will be provided to care for the following hazards: asphyxia from gases, drugs, submersion, fire fighting, foreign body obstruction, tumors within the airway, electrocution, strangulation, allergy, pressure due to moving objects, collapse of buildings, poisonous gases in manholes and premature asphyxia neonatorum. The Society for the Prevention of Asphyxial Death, Inc., is cooperating in the organization and development of this work.

Dr. McIntire New Surgeon General; Will Remain President's Physician

Appointment to Rank of Rear Admiral, Succeeding Rossiter, Announced at Warm Springs —Jumped Over 84 Captains

By The Associated Press.

WARM SPRINGS, Ga., Nov. 29.—Appointment of Dr. Ross T. McIntire, White House physician for five years, as surgeon general of the navy with the rank of rear admiral, was announced today by President Roosevelt.

Dr. McIntire will take over his new post Thursday but will continue, with some one else yet to be named, to guard the President's health.

The President told a press conference he would have two physicians hereafter, indicating that Dr. McIntire would be consulted frequently by the new White House physician.

M'INTIRE PRAISES STUDY OF ASPHYXIA

Surgeon General of U. S. Navy Adresses Society at Fair

Recent submarine disasters have emphasized the need of greater storage of oxygen on undersea craft, Rear Admiral Ross T. McIntire, Surgeon General of the United States Navy, last night told a meeting of the Society for the Prevention of Asphyxial death in the auditorium of the Medical and Public Health Building at the World's Fair.

The United States Navy is doing "pioneer" work in developing improved technique in oxygen therapy in order to curb deaths from asphyxiation, Dr. McIntire declared. He said the navy was proud of its performance in rescuing more than 50 per cent of the men trapped in the Squalus disaster and predicted

HOSPITALS URGED TO CURB ASPHYXIA

Dr. P. J. Flagg Says Many Who Die Under Anesthetics Could Be Saved by Modern Devices.

NEW AMBULANCE IS SHOWN

S. P. A. D.

Medical Equipment for World's Fair.—An automobile x-ray unit has been built for the medical division of the New York World's Fair. With the mobile unit the eight first aid stations will be provided with immediate x-ray service, especially in cases of suspected fractures or other internal injuries. Plates will be developed in the darkroom in the automobile, which is equipped with a dry ice device to keep the developer chemicals at the maximum level of efficiency. A special ambulance is being prepared for the work of the "division of pneumatology," which will have charge of administering gases at first aid stations and at two resuscitation centers. The ambulance will be fitted with modern equipment for care of all kinds of asphyxial accidents, such as asphyxiation from gas, submersion and fire fighting.

Fig. 7. Dr. Ross T. McIntire joins the S.P.A.D.

Priestley, Lavoisier, Sir Humphry Davy and Sir Thomas Beddoes employed gases for the treatment of clinical diseases. Half a century later

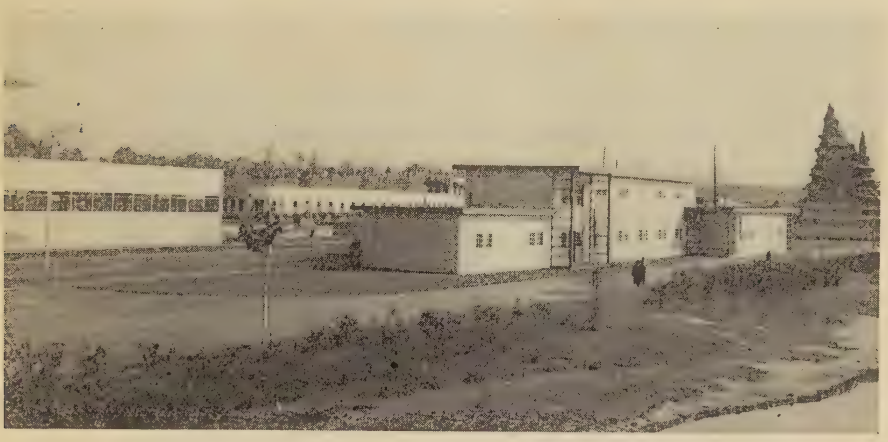


Fig. 8. Pneumatology Department at New York World's Fair 1939 (left wing).

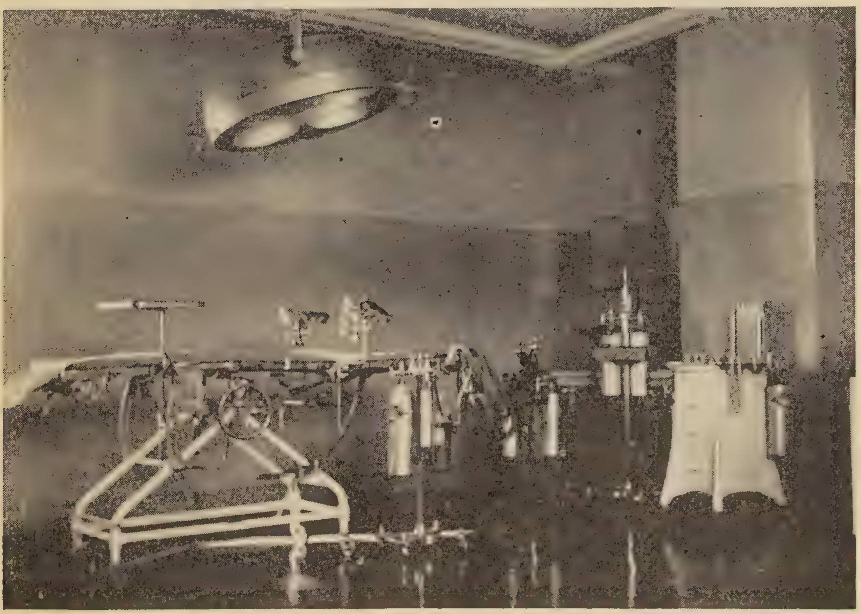


Fig. 9. Equipment set-up in department of Pneumatology at New York World's Fair.

the pneumatologists Long, Wells and Morton, revolutionized the practice of surgery by the introduction of the gases nitrous oxide and ether, for the control of pain. Oliver Wendell Holmes called this practice anaesthesia. Finally Haldane, Meltzer of the Rockefeller Institute, and others developed the research and technique for the use of gases for the saving of life, turning the attention of the profession to the necessity of modern methods of resuscitation. This evening, in the presence of Admiral Ross T. McIntire, Surgeon General of the Navy of the United States, your distinguished guest speaker, in the presence of the President of the American Medical Association, Dr. Nathan B. Van Etten, and before the Medical Director of the New York World's Fair, Dr. J. Peter Hoguet, we the Board of Directors of the S. P. A. D. offer this division, equipped with the most

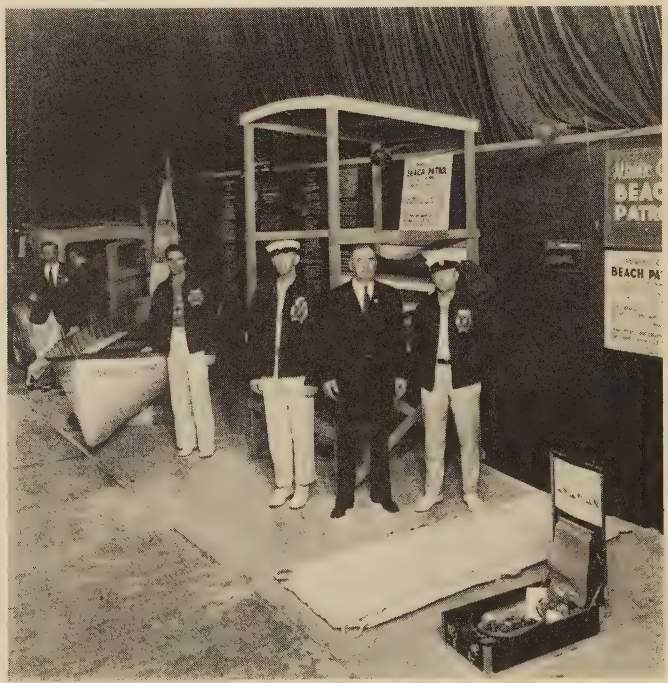


Fig. 10. Atlantic City Beach Patrol and Equipment.

modern devices for the control of pain, the saving of life and the treatment of clinical disease, for the protection of the guests and the personnel of the New York World's Fair, and with the hope that every hospital in the world of tomorrow will possess a functioning Department of Pneumatology."

The Medical Society of The County of New York. On December 20, 1941, the following press release appeared in the *New York Medical Week:* "That the Special Committee on Infant Mortality be permitted to contact the heads of Obstetric Departments of Manhattan Hospitals to inquire if these departments would be interesting in contributing to the setting up of a special training service under the auspices of the Society for the Prevention of Asphyxial Death, so that they may have present in the delivery room an individual competent in the use of resuscitation apparatus."

As a result of a suggestion of Dr. Edwin Daily, director of the Children's Bureau Washington, D. C., contact was made with the state Director of Maternal Health and Child Welfare of every state in the Union. Interested responses were received from fourteen states, a number of which indicated a keen interest in the problem.

The American Medical Association. A memorable scientific exhibit on asphyxia was held at Atlantic City in 1935. Fourteen booths were subsidized by the Scientific Exhibit of the American Medical Association, showing asphyxia due to allergy, anaesthesia, neonatorum, foreign-body obstruction, in diphtheria and croup, viewed by the medical examiner, by the internist, and by the municipal officer.

The following year saw the appointment of a Committee on Asphyxia by the Board of Trustees of the American Medical Association. In 1937, this Committee made its report, which was accepted without change by the House of Delegates.

Professional asphyxia-consciousness initiated and developed by the Society for the Prevention of Asphyxial Death during the last decade, accepted and confirmed by the American Medical Association through the last five years, has prepared the ground for a serious approach to the problem of asphyxial death, especially for its institutional and group approach, and for the development of the art of resuscitation.

Part II

Asphyxia as a Generic Problem

Chapter 3

Experimental Physiology

Asphyxia has been described by Henderson as "the process which occurs whenever it becomes difficult for the tissues to obtain the normal supply of oxygen. Any absolute deficit kills quickly. As exclusion of oxygen extinguishes a fire, so too, in man or animal a deficiency of oxygen in the blood and tissues induces death in asphyxia."

The disarming simplicity of this concise definition is the direct antithesis of the complexity presented by the physiological mechanism responsible for oxygen utilization. The problem of asphyxial death may be approached from one of two viewpoints: that of experimental physiology or that of clinical medicine.

Oxygen and carbon dioxide are the breath of life. The maintenance of balance between the two in the tissues is the chief function of respiration and circulation. Variations in tissue gas balance affect blood chemistry, which in turn influences the respiro-circulatory system to reëstablish a normal balance.

"Circulation is but a part of respiration, and both cooperate in the closest way in order to maintain and preserve constant the conditions of life in the internal environment," said Claude Bernard in his book, "Lessons in the Phenomena of Life."

"When once the fundamental fact is grasped, that the flow of blood through the body is correlated with gas pressure in the capillaries, the whole physiology of circulation appears in a new light," states Haldane. "It is neither the heart nor the nervous center which governs the circulation rate, but the tension of gases in the tissues as a whole; and they govern it with an accuracy and a delicacy comparable to that with which they govern breathing. The heart and vaso-motor system are only the executive agents which carry out the bidding of the tissues, just as the lungs and nervous system do in the case of breathing."

Tissue gas balance is thus accomplished by variations in the rate, rhythm and amplitude of the respiration, variations in rate and volume of cardiac output, variations in the caliber of the blood vessels' and variations in the oxygen and hydrogen-ion concentration of the blood. This normal tissue gas balance may be disrupted directly or indirectly. Direct causes are tissue trauma or chemical poisoning (histotoxic anoxia); disturbances in the oxygen-carrying capacity of the blood, such as reduced volume, reduced red cells, or reduced hemoglobin (anemic anoxia); and pathologic physiology of the heart or its vessels, for example; fibrillation, decompensation from myocardial insufficiency, or from shock due to trauma or exposure, resulting in smooth-muscle relaxation and capillary stasis (stagnant anoxia). Indirect causes are respiratory obstruction and dilution of respired gases (anoxic anoxia); obstructive lesions and secretions in the upper airway, trachea, glottis, oro-pharynx, naso-pharynx; spasm of the glottis, pharynx, and masseters; disturbances of the rate, rhythm and amplitude of the respiration caused by lesions of the respiratory center, the carotid sinus, the carotid or aortic bodies; pathological lesions of the lung parenchyma or inflammation of the epithelial lining; and variations in the oxygen content of the respired air.

To recapitulate, oxygen-carbon dioxide balance in the tissues is directly interrupted by histotoxic, anemic and stagnant anoxia and indirectly by anoxic anoxia.

The first three forms of anoxia present a relatively wide margin of safety by permitting well considered and deliberate stimulation and support of the respiro-circulatory mechanism. The last form, anoxic anoxia, however, even though acting indirectly through the respiration, results in a high degree of morbidity and mortality with a margin of safety frequently limited to minutes instead of hours, by reason of the sharp and rapid variations in the oxygen-carbon dioxide tension presented in the alveolae for circulatory absorption.

In order to emphasize anoxic anoxia, therefore, this last and most urgent type is now considered from a number of its mechanical and pathological viewpoints.

General Considerations

Pulmonary ventilation is a complex function which is accomplished by the rhythmic expansion of the chest, resulting in an increase and decrease of the capacity of the thorax. To this mechanical action of the chest wall are added the elasticity of the lung tissue, and the factor of *intra-pleural* pressure, which in turn is directly related to the negative (inspiratory) and to the positive (expiratory) *intrapulmonary* pressure.

Inspiration. Inspiration is an active phenomenon due to the combined activity of the inspiratory muscles, which produce elevation and abduction of the ribs and lowering of the diaphragm. The capacity of the thoracic cavity is thereby increased. The lungs expand, the *intrapulmonary* pressure decreases, and air enters. The negative *intrapleural* pressure increases.

Expiration. During expiration the chest walls collapse, the diaphragm rises, and the capacity of the chest diminishes. The lungs, obeying their elasticity, partially collapse. Intrapulmonary pressure increases, and a part of the air contained in the lungs is expelled, while the intrapleural negative pressure is reduced. Expiration is thus a passive phase of the respiratory cycle, the only active part of which is represented by the actual contraction of the bronchioli, as Lusida has recently shown. Respiration is thus carried on by muscular movements which cause rhythmic production of differential pressures between the pulmonary and the outside air.

Factors Governing Respiration. It is obvious that respiration cannot be accomplished in a normal way unless all the factors entering into its production remain normal. These factors are as follows:

(1) Normal functioning of the respiratory center.
(2) Integrity of the respiratory muscles.
(3) Integrity of the motor nerves of these muscles, including the vago-sympathetic afferent and efferent fibers.
(4) Patency of the respiratory airway.
(5) Maintenance of intra-pleural negative pressure.
(6) Integrity of the carotid sinus reflex. (In the arterial wall lie receptors which are stimulated by distention of the artery.)
(7) Integrity of the Hering-Bruer reflex (in which expansion of the alveoli cause arrest of inspiration and starts expiration, and collapse of the alveoli causes arrest of expiration and starts inspiration).
(8) Integrity of the carotid and aortic bodies (chemoceptors recording oxygen and carbon dioxide).

Respiratory Center. The respiratory center is stimulated and regulated by the nervous stimuli conveyed to it through all sensory nerves of the body, particularly through the vagi; more especially, however, it is regulated by the hydrogen-ion concentration of the blood, represented almost exclusively by the ratio $H_2CO_3/NaHCO_3$, and in last analysis by its CO_2 content acting also upon the carotid and aortic chemoceptors. Conversely, the respiratory center regulates the content of CO_2 in the blood.

The nerve cells of the respiratory center and the carotid and aortic chemoceptors require a considerable amount of oxygen to satisfy their metabolic needs. Even the slightest variations of the CO_2 content of the blood will produce marked variations in the respiratory rhythm; increase of CO_2 causes deeper respiration and hyperponea, without marked influence upon the respiratory rate; decrease of CO_2 produces slowing of the respiration and finally apnea. Decrease of the oxygen content of the arterial blood is accompanied by tachypnea, which starts a vicious circle; loss of CO_2, periodic breathing, apnea and increased anoxemia, leading to fatigue and paralysis of the respiratory center; increase of oxygen above the normal limits, on the contrary, is accompanied by no marked changes, as long as the CO_2 content remains normal.

Paralysis of the Respiratory Center. This dangerous complication may develop either suddenly or progressively. It occurs suddenly during anaesthesia, gradually slowing down and arresting the respiration due to the influence of sedatives, opium, barbituric derivatives in high doses, alcohol, etc.

In the apnea occurring suddenly during anaesthesia, differential pressure should be instituted immediately in order to maintain pulmonary ventilation until the acute anoxemia is arrested or the excess of anaesthetic is eliminated and the center has recovered its normal function. We should never forget that the time of successful resuscitation in these cases is limited to seconds. Loss of only one minute may prove fatal. When the respiratory ways are patent, mouth-to-mouth insufflation may be used. It can be applied immediately; the right amount of air is insufflated each time, the insufflated air has the right temperature and the right oxygen and CO_2 content, and it is insufflated in the right intervals, regulated by the respirations of the subject. It is the ideal emergency differential pressure method for resuscitation. Of course, this procedure cannot be kept up for long; but it gives time to proceed with intubation of the patient, by which both inspiration and expiration can be continued. Artificial respiration by manual manipulation can be combined with direct insufflation in these cases. Our principal preoccupation should be to maintain by all possible means the oxygenation of the respiratory center; the nerve cells of this center cannot resist deep anoxemia over 15 to 30 seconds without undergoing definite changes. As long as the heart beats, life can be restored; resuscitation has occurred in patients and in submersed humans and animals in whom there was no audible heartbeat, although these were still present, as shown by the movements communicated to the needle used for intracardiac injections of adrenalin.

Effect of Paralysis of the Respiratory Muscles. Pulmonary ventilation is not seriously impaired unless all intercostal muscles and the diaphragm are paralyzed at the same time. If only the first or only the second are paralyzed, respiration can still be carried on by the others. This condition is occasionally realized in infantile paralysis, in cerebral and especially bulbar pathologic or traumatic lesions; in these cases asphyxia occurs because of partial or complete suppression of the mechaincal factor of pulmonary ventilation. It is obvious that simple differential pressure cabinets, as in thoracic operations with wide openings of the chest and surgical pneumothorax, are not alone sufficient in these cases. The methods which can and should be used in these conditions are: freedom of the airway through suction, and intratracheal insufflation creating artificially induced rhythmic expansion and retraction of the lungs.

Insufflation by means of an intratracheal catheter reaching the bifurcation of the trachea was used long ago by Meltzer, Auer, Elsberg and others. Curarized dogs can survive over 24 hours by this procedure. However, excessive elimination of CO_2 and possible traumatism of the trachea by prolonged use of this method renders it improper in cases of paralysis of the motor respiratory system, which require artificial respiration of long duration.

Alveolar Gas Exchange. The regulation of oxygen and carbon dioxide content of the blood, however, depends in the last analysis upon the condition of the alveolar epithelium, through which gas exchanges occur between the alveolar air and the venous blood circulating in the alveolar capillaries. This epithelium represents an enormous surface of about 1,000 square feet; through it 300 cc of oxygen pass from the alveolar air into the blood and about as many cc of CO_2 pass from the blood into the alveolar air in one hour per kilogram of body weight. In other words 300 cc of oxygen pass into the blood in one minute in the lungs of a resting man of 60 kg. or 150 pounds; this amounts to 20 liters in one hour and 480 liters in twenty-four hours. During exertion, this amount may become ten times greater. This gives a fair idea of the tremendous activity of gas exchanges in the lungs, which seems to be accomplished, under normal conditions, without the slightest effort. On the other hand, although we need such colossal amounts of oxygen for maintenance of life, we have no reserves of it in our organisms beyond the 4,000 cc of air contained in the lungs and that contained in the hemoglobin of the red blood cells; the content of oxygen of the alveolar air being 15%, the whole reserve of oxygen in it is 600 cc, that is, the amount necessary for about two minutes. Seven or eight breaths of a neutral gas, *e.g.*, nitrogen, hydrogen or helium, bring loss of consciousness through oxygen want.

The foregoing considerations render it clear that any serious disturbance of any one of the links of the respiratory chain will lead to anoxemia, or acapnia. Death by asphyxia will follow gradually or rapidly, according to the degree of anoxemia.

Danger of Over-ventilation. It is necessary to point out that what we generally designate as asphyxia, *i.e.*, anoxic anoxia, or gasping for air accompanied by intense cyanosis, represents only one form of asphyxia, which in fact is the less dangerous. In this form there is retention of CO_2, which stimulates the respiratory center, deepens the respiratory movements, and allows the lungs to get some oxygen even through a small portion of the still available pulmonary tissue. In fact, we should not forget that, at rest, we use scarcely $\frac{1}{20}$ of our alveolar surface.

Much more serious is that asphyxia in which there is no cyanosis. This form is due to anoxemia without retention of CO_2, as in lesions of the respiratory center, over-ventilation, or in marked decrease of hemoglobin following hemorrhages. Shallow, rapid breathing is present, causing excessive elimination of volatile CO_2, leading to fatigue and definite disorganization of the respiratory center. In this type of asphyxia, there is neither gasping for air nor any marked subjective or objective signs of distress; death occurs without any of the alarming symptoms of anoxic asphyxia.

Entrance of Air into the Pleural Cavity. Intrapleural negative pressure is due to the instant absorption of any gases which may be present in the pleural cavity as a result of blood circulation in the pleural capillaries. In fact, after death and arrest of the circulation, a certain degree of pneumothorax is always present. Communication of the pleural cavity with the outside air through an opening either in the chest wall or through the lung will allow air to enter the pleura and thus create pneumothorax. If the openings of the chest wall or of the lung are small and of short duration, the collapse of the corresponding lung will be only moderate and temporary; the respiratory disturbances will be moderate and of no importance. On the contrary, when the openings are very wide, as in extensive wounds of the chest or in large surgical thoracotomies, not only will the corresponding lung be completely collapsed and incapacitated, but according to the major or minor degree of flexibility of the mediastinum, ventilation of the other lung will be more or less impaired. Roughly, in less than 50 per cent of cases, large openings of the chest can be made without any dangerous impairment of either respiration or circulation. The danger is naturally greater when both pleural cavities are opened, as this may occur in intrathoracic operations for tumors of the lung, mediastinum or esophagus, by accidental opening of the mediastinal pleura.

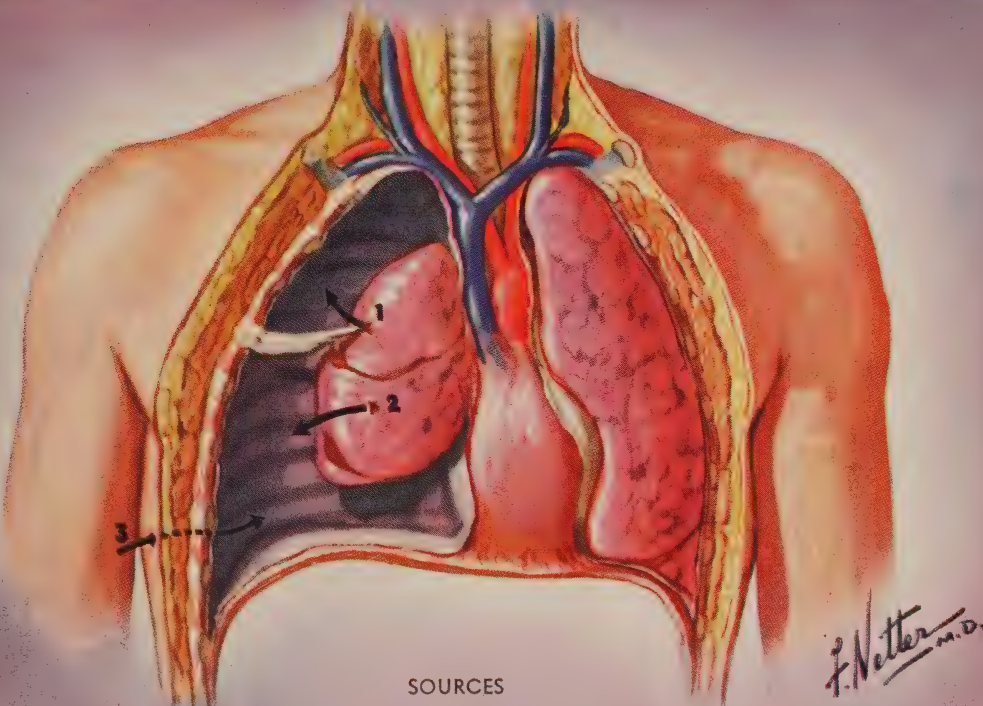
These dangers and complications following surgical pneumothorax have been the principal cause of the long delay in the development of thoracic surgery. The chest was the "No man's land" to the surgeon until 35 years ago. It can be said that the development of thoracic surgery started with the use of differential pressures, introduced by Sauerbruch, Brauer, Engelken, and others. In these methods, either the body of the patient was placed in a cabinet in which negative pressure was maintained, the head being under normal pressure outside of the cabinet, or the opposite conditions were created, that is, the head was placed in a cabinet under positive pressure, the body being outside and under normal atmospheric pressure. It is obvious that in either method, negative or positive differential pressure, the collapse of the lung is prevented, although the pleural cavity is opened wide.

The same effect can be obtained by intratracheal insufflation of air or oxygen, by applying an anaesthetic mask air-tight upon the face of the patient and raising the pressure in the mask, or by method of endobronchial balloon block.

Inflammation and Obstruction of Alveolar Tissue. In edema of the lung, of whatever origin, or in drowning, simple inhalation is sufficient only in a limited number of cases. In fact, in these cases oxygen under high partial pressure should be brought into intimate contact with the still functioning portions of the respiratory membrane; this cannot be realized by ordinary inhalation by which it is impossible to raise the partial pressure of the alveolar air above 40 to 45 per cent. We need in these cases 60, 70, 80 and 90 per cent of oxygen in order to make up the often extreme limitation of the respiratory field and to increase accordingly the amount of oxygen dissolved in the fluid filling the alveoli; this oxygen can be taken up by the alveolar blood and thus allow a sufficient degree of oxygenation.

In the experimental work on submersion carried out on animals and

PNEUMOTHORAX
CAUSES AND TYPES

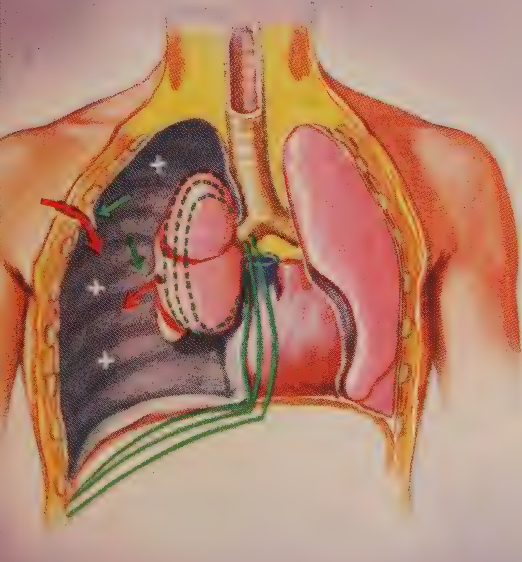


SOURCES

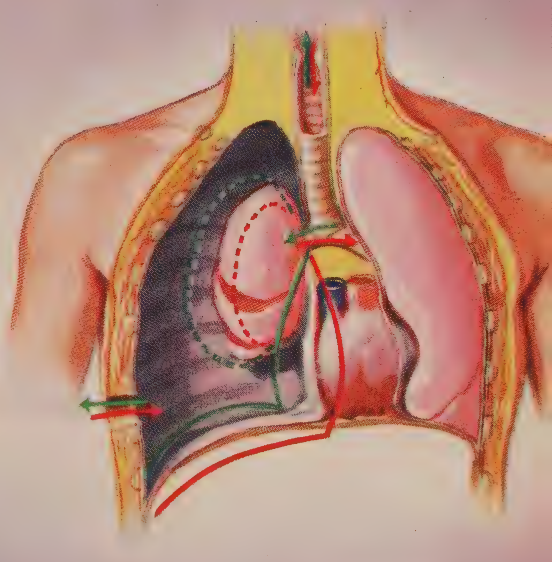
1 LUNG PUNCTURE BY RIB FRAGMENT

2 TEAR OF ADHESIONS (SPONTANEOUS PNEUMOTHORAX)

3 PENETRATING WOUND (CHEST WALL AND/OR LUNG)



VALVULAR OR TENSION PNEUMOTHORAX



OPEN OR SUCKING PNEUMOTHORAX

humans, we* were able to maintain life in dogs, the lungs of which were filled with water, by insufflating pure oxygen under positive pressure into the lungs by means of an endotracheal-catheter. Hoover has differentiated between extrinsic and intrinsic edema; in the first, in which the alveoli were filled with exudate but the alveolar membrane was healthy and permeable to gases, anoxemia was corrected by oxygen inhalations; in the second, where the alveolar membrane was infiltrated and thickened so that gas exchanges were impaired, simple oxygen inhalations could not correct anoxemia. In these cases, endotracheal oxygen insufflation by expelling and replacing nitrogen, raises the partial pressures of oxygen in the alveolar air; furthermore, it distends the alveoli and thus increases the respiratory field and, in addition, it puts in action the Hering-Bruer reflex.

A similar condition is present in the asphyxia of the newborn. When it is due to persistent atelectasis of the lung caused by obstruction of large or small bronchi, by mucous plugs, or often by amniotic fluid, as in premature respiration, intratracheal insufflation distends the lung, introduces air into the alveoli beyond the agent of obstruction and stimulates the respiratory center through the Hering-Bruer reflex. Similar complications may occur in thoracoplastic operations upon tuberculous patients, or in operations for bronchiectasis. During the operation the exudate filling the diseased lung passes by gravity into the healthy bronchi and may cause serious anoxemia during the operation. These bronchial obstructions due to internal drainage may become the origin of serious hemo-respiratory complications during the hours and days immediately following operation, because of the inability of these patients to cough and expectorate.

The pathologic physiology of histotoxic, anemic and stagnant anoxia, one or all of which may be simultaneously present, may be considered as representing sub-acute or chronic asphyxia with a margin of safety extending from hours to days. Because this form of asphyxia may be so easily overlooked, however, and because it may at any time give way to a complicating respiro-circulatory anoxic anoxia (pulmonary edema respiratory obstruction complicating unconsciousness, etc.) it is to be regarded seriously and treated promptly.

The experimental physiologist has long recognized the pathologic physiology of asphyxia. The volume of research devoted to this problem may be inferred from the foregoing summary. It is to be noted, however, that formerly emphasis on the problem of asphyxial death was placed upon causative factors and their operation. The author's contribution to the prevention of asphyxial death consisted in emphasizing the integrated effects. He guessed that a great many people were dying from a group of related accidents whose correlation had been overlooked. The astounding reaction which has resulted from this guess in the field of organized medicine suggests the desirability of referring to the original editorial which the editor of the *American Journal of Surgery*, Dr. Thurston Welton, had the foresight and the temerity to publish in December, 1932.

* Coryllos, P., Flagg, P.

Chapter 4

Clinical Medicine

Asphyxial Death: A Professional Disgrace

The death rate from asphyxia is a reproach to the practitioner of medicine: first, because of figures available from municipal vital statistics; secondly, because the medical profession has practically turned over the treatment of asphyxia to a non-medical personnel. The death rate from asphyxia drawn from vital statistics of the City of New York for 1931 with a total registered population of 7,090,086 is more than twice that for auto accidents, 18 times that for diphtheria, and almost 40 times that reported for typhoid.

The argument may be advanced that we all die of asphyxia. The test of true asphyxial death, however, readily disposes of this contention. The generally accepted immediate treatment of asphyxia neonatorum, illuminating gas, submersion, anaesthetic accidents, etc., is removal of the obstruction to the respiration and the application of artificial respiration. Prompt and efficient treatment along these lines usually results in cure. Failure to apply such treatment results in death from asphyxia.

The author has previously pointed out the fact that asphyxia is a generic condition, the specific instances of which are found in the various etiological factors listed below. The segregation of various types of asphyxia, all subject to the same essential form of treatment, is the reason for the astonishing figure disclosed.

The following figures are on file in the Bureau of Vital Statistics, City of New York:

Cause of death	Fatalities 1931	1942
Submersion	442	259
Lightning	0	1
Electric shock	25	24
Drugs	0	65
Anaesthesia	42	—
Illuminating gas	305	193
Other gases	—	51
Mechanical suffocation	33	56
Conflagration	59	85
Total	954	734

It is fair to assume that the report of drug deaths and the report of anaesthetic deaths are underestimated. Foreign-body obstruction does not appear as such.

Of the total number of stillbirths reported (5579), the following may be classed as capable of response to resuscitation properly applied*:

Cord pressure	535
Breech	140
Malposition	91
Deformed pelvis	91
Difficult labor	279
Asphyxia	282
Total	1418

If we reduce this figure by one third to allow for unavoidable deaths, we have a total of 946.

Antenatal and postnatal atelectasis results in asphyxial death, and may be treated and relieved by intratracheal insufflation under measured pressure, as is any other form of asphyxia. The figure given for atelectasis (672) is not exclusive, as it includes icterus, sclerema, etc. For the sake of being conservative, we may reduce this figure one-half, and accept a total of 336.

Pulmonary complications occurring during the first month may be regarded as due, in part, to aspiration pneumonia following birth, consequent upon a failure to remove fluid from the baby's pharynx and trachea. The figures for lobar pneumonia are 55; for broncho-pneumonia, 181; allowing one-third for pneumonia from other causes, we have a total of 158.

Premature infants dying in the first day frequently lose their lives from inefficient means of resuscitation. The total figure is 852; a 50 per cent reduction of this figure gives 426.

The totals of the various types of asphyxia are, therefore, as follows†:

General asphyxia	934
Stillbirth	946
Atelectasis	336
Pulmonary complication	158
Premature	426
Total	2800

Comparative figures from various causes of death are as follows‡:

Typhoid	77
Diphtheria	156
Auto accidents	1298
Asphyxia	2800

If it be advanced that many asphyxial deaths, such as from submersion, lightning, conflagration, etc., are quite outside the possibility of rescue, it should also be recalled that many auto deaths are in the same category.

Why, it may be asked, was this situation not known long ago? The answer lies in the segregation of the specific instances of asphyxia into a single group, responding to a common treatment. Why has the medical

* 1942 classification: Injuries at birth: intercranial and spinal hemorrhage 327, spinal injuries 46, asphyxia neonatorum 273, other injuries 131. Total 777.

† Premature for 1931, 852 for 1942, 1396 (atelectasis not listed).

‡ Typhoid 3, diphtheria 7, auto accidents 823. Little ground for optimism regarding the prevention of asphyxial death.

profession been so remiss in its care of these patients? The reason is that until within the last three years no medical technique, or armamentarium, suitable for the use of a physician was available.

The problem before us is a significant one from an economic point of view. Based upon a capital value of $5,000 for a life, we have a capital loss of $14,000,000 for New York City alone; or a quarter of a billion dollars for the United States with a population of 125,000,000. Much of this life and much of this economic value may be saved by acknowledging the problem before the nation. Approximately two-thirds of these lives are those of newborns, physically well when rescued. Would it not be worth every effort that we could make to preserve these to posterity?

Although the accuracy of the figures quoted may be questioned, the total volume of lives lost from asphyxia in the United States, namely, 50,000 a year, has been generally accepted by organized medicine. Asphyxial death constitutes a major medical problem and suggests the necessity of immediate action directed to its relief. The causes of asphyxial death published in the *American Journal of Surgery* served as a working basis in initiating the development of the problem.

The Committee on Asphyxia of the American Medical Association, however, reporting to the Board of Trustees* suggested a revision and general acceptance of the list of causes presented in its report (Chapter 20).

In an attempt to carry out the suggestion of the Committee and to revise this list proposing a group of causes of asphyxial death generally acceptable, recognized authorities in pathology, toxicology, physiology, medical jurisprudence and internal medicine were contacted. Inquiry was directed to the following group, each member an authority in his particular field:

Dr. Eugene L. Opie, Department of Pathology, Cornell University.
Dr. J. P. Baumberger, Professor of Physiology, Leland Stanford University.
Dr. W. B. Cannon, Department of Physiology, Harvard Medical School.
Dr. H. A. Christian, Physician-in-Chief, Peter Bent Brigham Hospital.
Dr. L. A. Connor, Professor of Clinical Medicine, Cornell University.
Dr. W. T. Longcope, Professor of Medicine, Johns Hopkins University.
Dr. Harrison Martland, Medical Examiner.
Dr. Yandell Henderson, Professor of Physiology, Yale University.
Dr. T. A. Gonzales, Medical Examiner.
Dr. J. W. Jobling, Professor of Pathology, Columbia University.
Dr. A. O. Gettler, Professor of Chemistry, New York University.

As a result of the inquiry undertaken, the following list of the causes of asphyxia was found generally acceptable by seven out of eight of the advisory group to which it was referred.

(1) Asphyxia neonatorum.
(2) Asphyxia from gases used industrially.

* *J. Am. Med. Assn.*, p. 1531 (May 1, 1937).

(2) (a) carbon monoxide from illuminating gas, engine exhausts and coke and coal fires.
(b) refrigerants such as ammonia, carbon dioxide and dry ice.
(c) fumes in the manufacture of chemicals.
(d) gases associated with the oil industry.
(e) gases in the mining industry.
(f) fumigation for disease; the destruction of rodents on ships and elsewhere.

(3) Asphyxia from gases in warfare.
(4) Asphyxia from drugs, hypnotics, narcotics and sedatives, including acute alcoholism.
(5) Asphyxia from disease, such as acute pulmonary conditions, asthma and cardiac decompensation.
(6) Asphyxia from developmental and mechanical abnormalities, such as neonatal atelectasis and collapse of the lung.
(7) Asphyxia from anaesthesia due to overdosage, idiosyncrasy or a failure to meet mechanical obstruction, occurring in relaxation.
(8) Asphyxia from submersion (drowning).
(9) Asphyxia from high-altitude flying.
(10) Asphyxia from fire-fighting (smoke, chemical poisoning).
(11) Asphyxia from obstruction by foreign bodies.
(a) material caught in the esophagus or inhaled.
(b) tumors or infections within or without the airway.

(12) Asphyxia from electrocution.
(13) Asphyxia from strangulation, garrotting, hanging, and broken neck.
(14) Asphyxia from allergy.
(15) Asphyxia from terminal poliomyelitis.
(16) Asphyxia from suffocation; overlying suffocation with soft materials such as pillows, etc.
(17) Asphyxia from external pressure on chest and abdomen, as caught between two moving objects (automobiles, elevators and laundry machinery).
(18) Asphyxia from collapse of buildings, earth mounds, sand, fire coal in large coal bins, landslides.
(19) Asphyxia in manholes, wells, and declivities in the ground usually from lack of oxygen and increase of CO_2 and sometimes poisonous gases.
(20) Asphyxia from polyneuritis, with facial diplegia.

The foregoing list will undoubtedly be enlarged as time goes on.

Summary

The problem of asphyxia turns upon oxygen-carbon dioxide tissue tension; as has been demonstrated, it may be viewed from the standpoint of experimental physiology or from that of clinical medicine.

Physiologically, asphyxia may be designated as histotoxic, anemic, stagnant and anoxic. The first three act slowly but are insidious; they are dangerous because they may be overlooked. Anoxic anoxia, however, is readily recognized. It is the usual cause of acute asphyxial accidents, resulting fatally. This type of anoxia has been considered in some detail. The pathologic physiology of anemic, stagnant and histotoxic anoxia are considered in the chapter dealing with circulatory stimulation, p. 42.

Clinically, viewed from the standpoint of its integrated causes, asphyxia becomes a major medical problem. As such it should receive and continues to deserve the close attention of all persons responsible for the care of asphyxial accidents.

Part III

The Principles of Resuscitation

Chapter 5

Indications for Treatment

In accordance with our approach to the generic problem of asphyxia through experimental physiology and clinical medicine, we may consider the generic problem of resuscitation from the standpoint of (a) indications for treatment presented by pathologic physiology and (b) manual and mechanical means devised to meet these indications.

In anoxic anoxia: by obstruction and by dilution of respired gases.

In histotoxic, anemic and stagnant anoxia: by chemical tissue poisoning, by reduction in quantity and quality of blood, by capillary stasis.

Anoxic Anoxia

Obstruction by Foreign Matter. Solid or fluid matter in the mouth, pharynx, glottis, trachea, bronchi, and alveolae from vomitus, blood, gauze packs, and tissue, which are due respectively to undigested meals, ruptured abscesses in the airway, operative and post-operative hemorrhage, dislodged packs in the oro- and naso-pharynx, pulmonary hemorrhage from tuberculosis; also due to aspirated fluid, drowning in salt or fresh water, amniotic fluid or to pulmonary edema. *In such cases the indicated treatment involves removal of the foreign matter.*

Obstruction by Masseters. Masseteric spasm is obvious in its obstructive effects and accompanying cyanosis. It occurs in the patient whose reflexes are active, the highly nervous, smokers, alcoholics and especially those who have received drugs of the barbiturate class (Nembutal, Luminal, etc.). It constitutes a part of the characteristic phenomena of the second stage of asphyxia, *i.e.* spasticity. As a rule, in the absence of complications, patients presenting spasm and cyanosis have a wide margin of safety. *The indications are to ignore the spasm but treat the cyanosis.*

Obstruction by Glottic Spasm. The glottis is the watch-dog of the respiratory tract (Chevalier Jackson). It is a profound reflex exhibited by relatively powerful muscles. (An endotracheal tube gripped by a glottis in spasm creates marked pressure on the tube and resists withdrawal.) This glottic reflex controls a small aperture through which all respiration must pass. The reflex is excited by contact with fluid or solid matter, sudden inhalation of vomitus, blood, sea-water, etc. Fortunately this reflex is much more active than the swallowing reflex. Consequently, in uncomplicated conditions, should active vomiting or swallowing occur in the semi-conscious, it may be safely assumed that the glottis will not tolerate the entrance of foreign matter into the trachea without severe spasm and coughing. On the other hand, unconsciousness complicated by asphyxia, sedatives, or malposition is an entirely different matter. It has been argued that because certain drugs do not in themselves depress the glottic reflex, *e.g.*, avertin, evipal, pentothal sodium, Nembutal, etc., they therefore do not constitute an asphyxial hazard. These drugs do depress the pharyngeal reflex, however, for as a rule a pharnygeal tube is readily accepted. The fact that vomitus or blood may also collect in the pharynx without causing irritation is quite overlooked. When such obstruction develops and accumulates, progressive anoxia occurs. While the barbiturates do not anaesthetize the glottis, anoxia does so promptly; and the resulting depression of the glottic reflex permits aspiration and occasionally a fatal issue.

The indications are to preserve glottic reflex activity at all costs.

As a diagnostic sign, the glottic reflex is the best measure of the patient's viability. When the activity of the glottic reflex is impaired by local anaesthesia (local tonsilectomies), in general anaesthesia, in deep asphyxia (flaccidity), by opiates or by central paralysis, its protective mechanism must be assumed by the physician. This responsibility must not be ignored. The so-called death zone of the respiratory tract, existing between the epiglottis and the vocal cords, as pointed out by Chevalier Jackson, must be eliminated. As the art of resuscitation matures, failure to observe this obligation will constitute failure to employ ordinary precautions, and will constitute malpractice.

Obstruction due to Loss of Muscle Tone. Loss of muscle tone of the airway with ensuing respiratory obstruction is commonly overlooked. It is accumulative and dangerous. The patency of the airway depends upon the tone of the muscles of the tongue, faucial pillars and muscles of the soft palate. As this muscle tone is lost by cerebral hemorrhage, or injury, in poliomyelitis, by drugs, anaesthesia, both local and general, or by asphyxia from its many causes, this patency is lost. Should such obstruction be further complicated by malposition of the head, a fatal accident may ensue.

The routine careless management of the unconscious patient is a disgrace. We see it everywhere, but particularly in transportation. Note the position of such a patient. Tradition has placed him flat on his back with the naive hope that if both lungs are left free to expand they will surely do so. The patient vomits or bleeds into his airway, filling this "well" four or five inches deep with fluid and solid matter. There are two openings at the bottom of this well—the esophagus and the glottis. When vomitus contacts the glottis the latter is thrown into spasm and closes; but in time, due to CO_2 retention and anoxia, an inspiratory effort will open it and the foreign matter is aspirated into the trachea. If the patient is almost conscious, a violent cough will occur and this matter may be expelled; but if the patient is depressed by anaesthetic or sedation, particularly in obstetrics, or if she is depressed by a progressive asphyxia from obstructing soft parts of the airway, there may be no violent cough, nor any cough at all—just a respiratory gurgle and death by drowning.

In such cases the indicated treatment is to correct the position of all unconscious patients, regardless of the cause of the unconsciousness, whether surgical or medical, and to provide free ventilation.

Constant watchful, intelligent care of all unconscious patients, especially in transportation, is essential.

Conservative use of sedatives is indicated, especially when these are long-acting and when post-operative nursing care is reduced.

Interference with the movements of the chest is entirely secondary to freedom of the airway. Where nursing care is limited, the prone position, with elevation of the forehead and the face free, will insure drainage of the pharynx.

Obstruction by Paralysis of Muscles of Respiration. Paralysis from poliomyelitis or from central causes is commonly recognized. The splinting of the muscles of respiration, especially the diaphragm, from upper abdominal pain due to surgical operations or from injury is not always recognized. Pain of this character, as well as pleural involvement, which limits the normal respiratory excursions, not infrequently result in obstructive complications from accumulating alveolar and bronchial exudate, with resulting lobar pneumonia or collapse of the lung.

Here the indications are to supplant the action of the respiratory muscles by the use of a respirator, and to control pain by sedation to the degree required for the release of free respiratory movement, avoiding the depression which follows excessive use of medication.

Obstruction by Interference with the Hering-Bruer Reflex; Intrapleural and Intrapulmonary Pressure. The pathologic physiology of the respiration referred to in the previous chapter represents the lung in motion in contradistinction to the lung at rest. The physical, rhythmic expansion and contraction of the lungs has such a direct bearing upon the circulation, particularly of the right heart, that it cannot be ignored. The collapse of the large veins of the neck at every deep inspiration may be readily observed in any open neck operation. This regular intermittent relief of the right-heart pressure, when suddenly eliminated by stoppage of the respiration, throws a heavy burden upon the right heart. So-called controlled respiration in anaesthesia, shallow respiration, or excessive CO_2 absorption involves the hazard of circulatory disturbance and interferes with the Hering-Bruer reflex. The increased rate and depth of the respiration is physiologically compensated, as in ordinary exercise.

Depression and cessation of the respiration is not physiological and is not compensated by the simple addition of oxyhemoglobin. Furthermore, a lung at rest with a fixed oxygen-CO_2 gas tension will gradually become atelectatic. If the nitrogen is displaced by an anaesthetic gas, this atelectasis is very rapid and may proceed to complete collapse, within a few minutes.

Rhythmic expansion of the lungs with the constantly varying intrapulmonary gas tension insures alveolar expansion and adequate ventilation.

Indications: Maintain rhythmic expansion of the lungs at all times, thereby supporting the heart, maintaining the patency of the alveolar spaces, and preserving the integrity of the Hering-Bruer reflex.

Anoxic Anoxia by Gas Dilution. This form of asphyxia is commonly seen in high-altitude flying or ascending high mountains. While the gases in the high atmosphere exist in identical proportions, each is diluted because of lowered atmospheric pressure. Details of this dilution will be found with other factors in the section on high-altitude flying (Chapter 10).

Dilution of atmospheric oxygen may also occur at sea level, when this gas is displaced by some other gas, as with the carbon dioxide from dry ice, in wells, mines, declivities, etc.

The indication here is to recognize the oxygen deficiency. If oxygen is not available, absolute rest and warmth must be supplied to the body.

Histotoxic, Anaemic and Stagnant Anoxia

Three factors enter into the support of the circulation: namely, tissue fluids, blood plasma and the red blood cells.

Tissue Fluids. Tissue cells and the fluids with which they are bathed, constitute the internal environment. The normal condition of this environment depends upon the maintenance of an adequate volume of fluid through which gases and nutrient balance may operate. A reduction in the normal volume of tissue fluids is called dehydration. Its recognition is basic not only in the treatment of war injuries but in civilian practice. It is briefly referred to here because of its bearing upon the problem of resuscitation.

Viewed physiologically, the purpose of dehydration of the tissues is to maintain plasma volume. When urine output drops to one pint in twenty-four hours, uremia, delirium, coma, blood viscidity, low blood pressure and anoxia result. Without water a man dies in nine days. Tissue fluid balance is maintained when the urine output is between two and three pints in twenty-four hours.

Dehydration occurs in all delayed care of accidents. It is found in crushing injuries and burns. In these cases, there is little red blood cell loss, but plasma and serum escape. Dehydration from exposure is frequently present before injury or accident. Dehydration induces stagnant anoxia which sets in motion a vicious circle (Figure 10), *i.e.*, hemo concentration, oxygen, carbon dioxide, tissue tension imbalance, capillary stagnation, right-heart embarrassment, myocardial weakness and anoxia. Ground forces in military operations are more likely to develop dehydration than the air arm of the service because of the necessary delay in treatment.

Dehydration is characterized by sunken facies, thirst, weakness, dry skin, and low urine volume. The fluid required is from six to ten pints in twenty-four hours. Where shock is present with anoxia, twelve to sixteen pints, plus the amount of fluid lost through the kidneys (four to five pints) is indicated.

Methods of Administering Fluid in Dehydration. *By Mouth:* Patients who find it difficult to drink out of a receptacle are grateful for water offered by means of a two-foot rubber tubing serving as a straw, extending into a bucket of water the level of which is below the bed level. All water should have a half teaspoonfull of salt to the pint.

Rectally: One-half strength normal saline or 0.4 per cent is recommended.

Intravenously: It is recommended that this should not exceed one pint every two hours (40 drops per minute) 0.85 per cent isotonic NaCl; 500 cc of saline should alternate with 5 per cent isotonic glucose. Rapid massive saline that is more than 500 cc may result in hemo dilution and pulmonary edema. The bases of the chest should be watched for moist rales.

Oxygen Therapy: Because of the element of stagnant anoxia, oxygen therapy with rebreathing should be used as a routine. Elimination of anoxia will raise blood pressure and help reëstablish oxygen-CO_2 tissue tension balance. When the dehydration is complicated by the loss of plasma, as in wounds and burns, this loss should be replaced by plasma or serum infusion, because saline or glucose is rapidly lost from the blood vessels to the tissue and consequently does not offer continuous support to the circulatory volume.

Loss of Plasma. When there is no tissue dehydration and moderate hemorrhage is complicated by psychic shock and pain, the condition is referred to as *primary shock*. Primary shock is characterized by a small, rapid-running pulse, low blood pressure, cold perspiration and loss of muscle tone. Such a neurovascular disturbance is ordinarily treated by the recumbent posture, the use of heat and the administration of morphine for pain. The condition precipitates stagnant anoxia. It may readily be complicated by anoxic anoxia, through the muscle relaxation which occurs following the use of morphine for sedation.

When primary shock persists for an hour with a blood pressure of one hundred or less, the condition is referred to as *secondary shock* (traumatic hemorrhage, or wound shock). The passage from primary to secondary shock is usually credited to hemorrhage. It may be precipitated however, by progressive anoxia.

It is suggested that if primary shock be promptly recognized and treated as an anoxic hazard, its development into secondary shock may be reduced. Where such primary shock is further complicated by histotoxic anoxia from gassing, from anemic anoxia, or from red cell loss, the indication for oxygen therapy is urgent.

It has been pointed out that not infrequently there is a deterioration of the patient's condition between the time of transfusion and removal to emergency surgery. It is suggested that such relapse may well find its origin in anoxia.

The respiro-circulatory support and stimulation effected by oxygen-carbon dioxide therapy is well recognized. An almost pulseless patient may be carried for a long time with oxygen-carbon dioxide, or with oxygen alone with rebreathing. This support extends to and through the operative period, during which time pain reflexes may also be enlisted to improve muscle tone. When oxygen therapy is augmented by transfused plasma and red blood cells, the effect of the oxygen therapy may be extended into the post-operative period, when it may be discontinued.

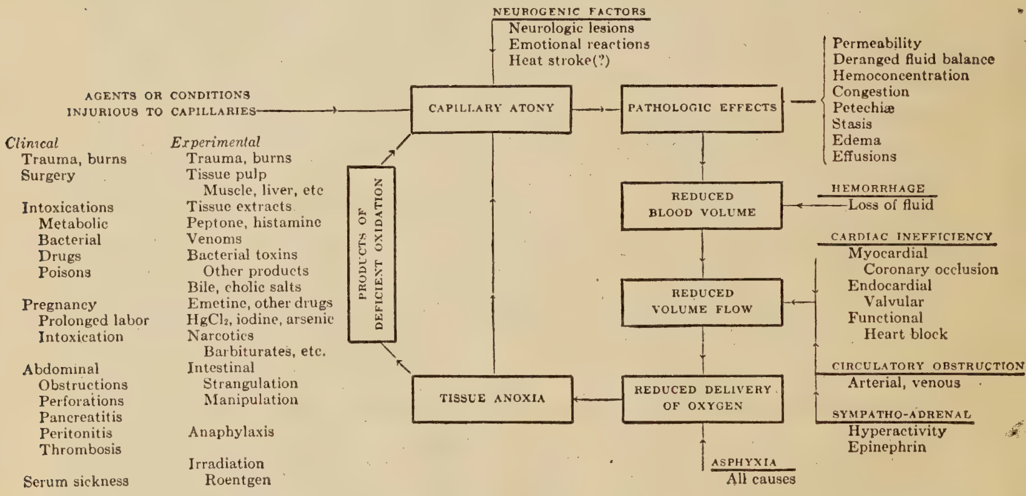


Fig. 10. Relationship of Factors in Pathogenesis of Shock. (V. H. Moon, *American Journal of Science*).

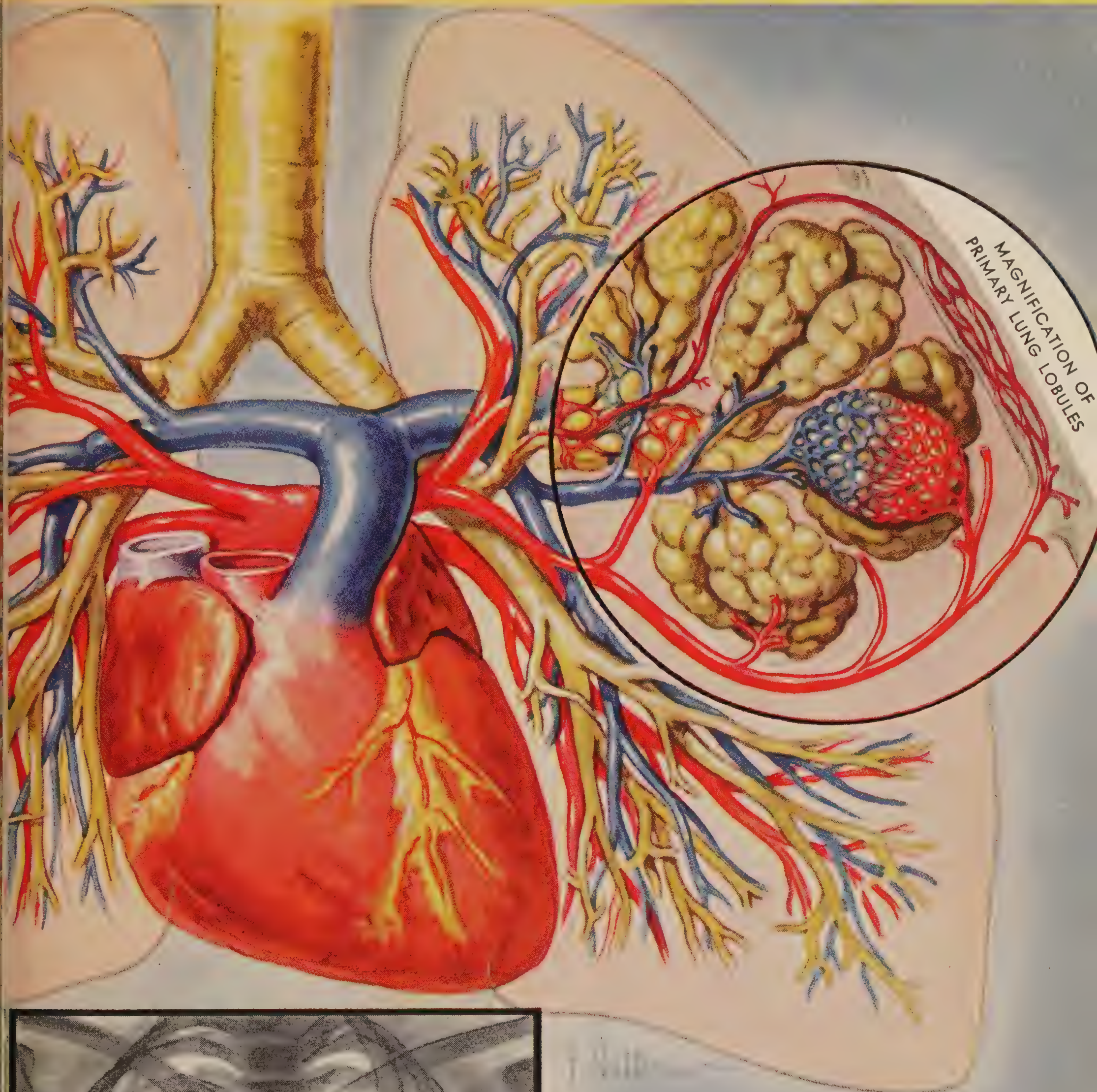
Loss of Red Blood Cells. In secondary shock from severe hemorrhage, anemic and stagnant anoxia are characteristic. If complicated by unconsciousness, loss of reflexes and malposition, anoxic anoxia may be further superimposed. Oxygen therapy is mandatory in severe secondary shock. During the administration of oxygen therapy, the effects of hemorrhage must be met by the transfusion of whole blood, for the loss is more than mere volume: it involves the oxygen-carrying capacity of the blood.

By increasing oxygen hemoglobin and plasma oxygen tension, oxygen therapy may tide over a temporary large loss of red blood cells. Such action is necessarily brief. The oxygen carrier must be returned to the circulation, if the latter is to become normal.

A few brief statements in regard to the red blood cell are here in order. The red blood cell is an oxygen carrier. Its carrying capacity is determined by the amount of free, uncontaminated hemoglobin which it contains.

PULMONARY VESSELS AND BRONCHI

Courtesy Ciba Pharmaceutical Products, Inc



Blue represents the pulmonary arteries; light-red, the pulmonary veins; dark-red, the bronchial arteries supplying the interstitial stroma and pleura.

BRONCHOGRAM AFTER INSTILLATION OF RADIOOPAQUE MEDIUM

Hemoglobin determines the color of the blood in the capillaries, the veins, the arteries, and the escaping blood. In mass, hemoglobin completely saturated with oxygen is a brilliant scarlet with a distinctly yellow tinge. Completely desaturated hemoglobin is a deep claret or maroon. Seen through the skin, this color varies from a deep purple to a greenish blue, depending upon the volume of blood circulating beneath and the transparency of the intervening medium. Normally saturated oxy-hemoglobin gives off oxygen to the plasma and thence to the tissue fluids in accordance with the degree of oxygen tension then existing in this environmental medium; but when the CO_2 tension in the red blood cell is reduced, the oxygen within it, as oxyhemoglobin, will not be fully released until the normal CO_2 tension is reëstablished (Bohr phenomenon). Anemic anoxia and tissue oxygen-CO_2 imbalance may, then, obtain in the presence of bright capillary blood. This condition has already been referred to (p. 27). It is repeated here in its relation to over-ventilation and by the excessive use of carbon dioxide absorption in the closed anaesthetic technique.

Oxygen, except when in solution in the plasma, does not reach the tissue cells. That organs may live for days and carry on their normal function without the presence of red blood cells has been demonstrated by Carrel and Lindbergh in their experiments in the culture of organs.* The technique suggests the value of a high-oxygen alveolar atmosphere extended over hours as a means of meeting massive loss of red blood cells due to hemorrhage.

The normal oxygen tension of the tissue fluids is not known.† The limit of control of the oxygen tension of the tissue fluids by raising alveolar tension is not known. Pending further research and in view of routine now in use in laboratory practice, it is suggested that high oxygen therapy be employed in all cases of serious red blood cell loss.

Transfusion. Whole blood in a 3 per cent sodium citrate solution with final concentration of between specific gravity 0.25 and 0.5 is almost as efficient as fresh whole blood, provided it is kept at a temperature of 4–6°C and given at a temperature of 40°C within fourteen days.

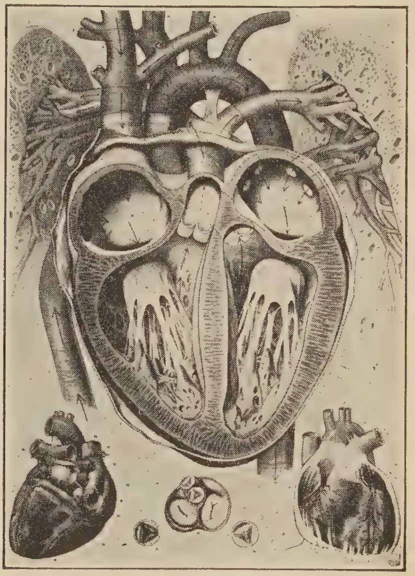
If the translucent layer (the blanket) separating the cell and plasma remains clear, or is but slightly cloudy, stored blood may be used from four to six weeks. If the blanket becomes opaque or purple, it should not be used, but should be reprocessed for the plasma it contains ($\frac{2}{5}$ volume). Centrifugal methods devised by Lindbergh may be used to advantage in securing a larger volume of available plasma. It is recommended that whenever plasma is transfused, $\frac{1}{3}$ volume of whole blood be added for the benefit secured by the red blood cells.

Plasma is blood, minus red blood cells in a 3 per cent citrate solution and glucose to specific gravity of 0.25. It should be clear golden or slightly

* "The Culture of Organs", Carrel and Lindbergh.
† Private Communication, Dr. D. Van Slyke.

orange in color. Small fibrin clots do not render it unfit for use. Stored at room temperature, it is good for 12 months. Serum is plasma without fibrinogen and without citrate dilution. Freed of the agglutinin content, it is useful as a blood volume and protein restorer. Dried serum and plasma are available for Army use. Since pyrogen-free distilled water must be carried for its dilution, however, its transportation volume is large.

If cardiac stimulants are to be added, they should be considered as complementary to the basic saline, glucose, whole-blood, and oxygen-therapy requirements of the shocked patient. Amyl nitrate has been recommended by Chevalier Jackson as a valuable cardiac stimulant. In severe terminal



Courtesy Clay-Adams Co.

Fig. 11. The Normal Heart.

asphyxia, direct massage of the heart, accompanied by endotracheal oxygen insufflation, is strongly advocated.

The *carotid sinus reflex* is a respiro-circulatory reflex which depends upon the lowering or raising of the pressure of the blood in the carotid artery. When the blood pressure increases, the respiration drops in rate and depth; when it decreases, the respiration increases in rate and depth. Thus in primary and secondary shock with blood volume loss and lowered blood pressure, the rate and depth of respiration increase.

Carotid and Aortic Bodies. These bodies act as chemoceptors or receiving stations for variations in hydrogen-ion concentration (CO_2 concentration) or in oxygen concentration. These two sensory physical and chemical systems, though close to each other anatomically, act independently.

The balance established between the *stimulation* of the respiration through the carotid sinus by low blood pressure on the one hand, and the depression of the respiration by the chemoceptors (aortic and carotid bodies) on the other, suggest the delicacy and the complexity of the respiro-circulatory control. It should serve as a warning to those who would reduce these factors to the simplicity of their own comprehension of phenomena even now but incompletely understood by those who discovered them. Where the path is plain and relatively simple, as in physics, we can and should assist by removing abnormal conditions. But in the field of biochemistry, the inexperienced still continue to rush in where they should fear to tread. It is not a matter of adding mystery to the problem, it rather is one of admitting ignorance.

Resuscitation Ward in Military Operation

The advantage of a resuscitation ward is stressed in the Army orders for July 1942, released by the British War Office. The following facts are adapted from this release.

"A properly equipped and staffed resuscitation ward insures that all patients requiring urgent treatment receive it with a minimum delay and in the best surroundings. The following reasons are advanced:

"(1) From the moment of the patient's arrival in the ward, the staff know what to do and how to go about doing it.

"(2) There is no delay in obtaining apparatus as it is all in place in the ward."

(3) Oxygen therapy can be begun at once and continued through the administration of intravenous fluid in appropriate cases.

"(4) Heating systems may fail; emergency measures can seldom embrace several wards at once, but it is simple to heat one ward and to have means of rapidly warming the very cold patients.

"(5) Response to treatment can be more acutely observed, enabling life-saving measures to be adopted at the correct time."

(6) Deterioration of transfused cases can be prevented by oxygen therapy.

"(7) It allows a proper control over the arrangements for giving intravenous fluid. The whole system of resuscitation depends on close cooperation between those in charge of the blood and other supplies of transfusion fluids and those administering them. There are times when it is difficult to obtain such fluids and a knowledge of all the working arrangements of the system is essential for the medical officer in charge of the ward to insure maximum benefit for the largest number of cases."

(8) The patient can be followed into and through his operative period by the resuscitation ward personnel, experienced in the various aspects of pneumatology (anaesthesia, resuscitation, oxygen therapy).

Summary. The foregoing facts summarized below form a guide by which the actual value of manual and mechanical methods of artificial respiration may be appraised. The indicated treatments are as follows:

(1) Remove foreign matter from airway.
(2) Eliminate or overcome the effects of masseteric or glottic spasm.
(3) Meet the complications caused by loss of muscle tone.
(4) Replace the function of respiratory muscles which are paralyzed.
(5) Protect the Hering-Bruer reflex.
(6) Maintain intra-pleural and intra-pulmonary pressures.
(7) Recognize and treat oxygen dilution in respired mixtures.
(8) Anticipate and treat dehydration.
(9) Maintain blood pressure.
(10) Maintain oxygen-carrying capacity of blood.
(11) Administer oxygen according to indications.
(12) Respect the action of the carotid and aortic chemoceptor reflexes.
(13) Administer heat to the body.
(14) Apply cardiac massage with oxygen in heart failure.

Discussion of Summary

Removal of foreign matter may be a gesture, a guess, or a carefully performed act under vision. Removal by posture, milking the airway, or rolling on a barrel is a gesture. Blind, oral or tracheal suction is a guess. Exposure of the airway, and removal of the obstruction under vision is the modern practice of medicine.

Spasm of masseters or glottis on the one hand and obstruction by loss of muscle tone of the airway on the other may neither be ignored nor overcome by remote control by pressure of gas on the face or in the mouth. Pressure of gas on the face (inhaler) may be expected to result in the escape of much of the gas into the surrounding air; in forcing fluid or foreign matter in the nose, mouth or into the relatively large esophagus and stomach; or, in company with fluid which precedes it, into the trachea. Pressure applied within the mouth (pharyngeal tube) may be expected to discharge first into the esophagus and secondly into the trachea. Saliva, vomitus, or blood, which may accidently find its way into the oropharynx at the instant of insufflation, will be carried into the stomach or the lungs.

Too much stress cannot be placed upon the correct position in all unconscious patients, their constant watchful care, especially during transportation, and conservative use of sedatives, particularly when nursing care is not available.

Unless a patient is in the terminal stage of asphyxial flaccidity, respiration will be observed if it is looked for. It may easily be overlooked, however. The correct method of stimulating such a case is to offer an atmosphere of oxygen and CO_2 under atmospheric pressure (a full mask and breathing bag), a nasal tube from which oxygen is flowing into the pharynx, or an oral tube through which oxygen is flowing into the mouth. These are strictly inhalation methods. No attempt whatever is made to force the gases into the lungs. The percentage of CO_2 used for this purpose may vary according to the experience of the administrator. Pure CO_2 mixed with air may be used with proper precautions, an occasional breath being permitted at intervals according to the response.

Whenever air or oxygen is to be blown into the lungs for resuscitation or for artificial respiration, the only safe way to perform such insufflation is by passing a tube into the larynx and applying the pressure directly. It is plain that no operator would deliberately blow a solution of vomitus contained in a bottle into the lungs. Yet this is calmly allowed to happen because the contamination is not visible. Is not this merely a desire to go through motions without regard to the effects produced, and a timidity toward simple instrumentation arising from blundering technique in experienced hands? It is well to recall that suction of the pharynx in the tonsil operation was discouraged twenty-five years ago. Metal instrumentation of the larynx and airways is still regarded with hesitation, in spite of its universal use in other fields, its inherent cleanliness, lack of friction and the precision which its use implies.

From the standpoint of common sense, simple physics, ordinary cleanliness, precision of treatment and uniformity of results, is it too much to ask that, if and when insufflation of the lungs is indicated, this be carried out by means of a clean intubation and pressure, controllable as to time and volume? Pressure masks and pharyngeal tubes are poor substitutes for knowledge of the physiological anatomy of the airway.

Objections to the simple technique required for intubation by the direct method turns almost entirely upon two factors: (1) Confusion with the difficult procedure of laryngoscopy in the conscious patient under local anaesthesia; (2) Improper selection of cases for the technique. Practice alone in a very limited number of anaesthetized operative cases or cadavers is the only answer to these objections. The sight and the tactile sense will inform where words fail.

For short periods, in the interval between respiratory failure and admittance into a negative-pressure cabinet, intubation and insufflation of oxygen for periods of five seconds to a pressure of twenty-five mm of mercury sixteen or eighteen times a minute is indicated. For long periods—hours or days—the cabinet must be employed. If the swallowing reflexes are affected, repeated suction should be employed to prevent aspiration. An airway must be maintained at all times by pharyngeal or tracheal tubes.

While insufflation of oxygen under pressure is generally recognized as a desirable or a necessary procedure, well calculated to support the Hering-Bruer reflex and to maintain intrapleural and intrapulmonary pressure, the use of suction to facilitate expiration is nowhere indicated. Coryllos defined expiration as a purely negative phase—a return to normal after expansion. While positive indications for this need are not apparent, it is said to do harm by bringing about an acapnia through over-ventilation, and by interfering with the expiratory phase induced by the Hering-Bruer reflex, the carotid sinus and the chemoceptor rhythm under the control of the carotid and aortic bodies.

Too much cannot be said of the positive effects of oxygen rebreathing as a circulatory stimulant. The simplest equipment will suffice. In an emergency, CO_2 and air used alone may tide the patient over until he can be supported by an infusion of saline, glucose, plasma or whole blood. The compensatory effect of oxygen therapy in red blood cell loss is a therapeutic measure which should be thoroughly investigated. Chilling of the surface is seldom regarded as capillary stasis, and yet how plain this stasis becomes when one is exposed to cold-water bathing! Observe the blue lips and fingernails and the pallid skin. This extreme serves to emphasize the need of surface heat for the asphyxiated in primary or secondary shock. This treatment may well be placed first instead of last.

When the heart has failed and respiration has ceased, the question of further risk to the patient seems entirely academic. Should such an accident take place during an abdominal operation, massage of the heart is easily accomplished through the diaphragm, which is relaxed by asphyxia. Coryllos has applied cardiac resuscitation successfully in experimental animals which have expired. The possibility of such resuscitation should always be borne in mind. The prone-pressure Schaefer technique applies a certain amount of cardiac massage by intermittent pressure applied posteriorly against the lower ribs pressed against the opposing hard surface on which the patient lies.

Principles of Treatment Generally Accepted

(1) Suction to airway
(2) Correct posture
(3) The use of oxygen
(4) The use of inhalation
(5) The use of pressure (insufflation)
(6) Heat to the body

Principles of Treatment under Discussion

(1) Insufflation
(2) Use of CO_2
(3) Stages of Asphyxia

(1) **Insufflation.** If an anaesthetist confronted by a patient who had ceased to breathe attempted to carry on artificial respiration by making gas pressure on the patient's face through a mask or a throat tube, he would be looked upon as unfamiliar with his field. If he attempted to popularize such a procedure, he would be unequivocably condemned. Why, then,

it may be asked, are face-mask or throat-tube attempts to insufflate the asphyxiated patient regarded with complacency and encouraged in well-regulated clinics? That such technique is simple, that it requires no training and that it is a case of this or nothing, can scarcely be regarded as valid arguments. The esophagus is easier to enter than the glottis. The opening of the esophagus presents little resistance to air. When pressure is made through a mask or throat tube, the tendency of the gas is to enter the esophagus and stomach first and the glottis second. When the stomach is distended, reflex vomiting takes place. Vomitus is easily picked up by the insufflated vapor and carried into the open glottis. This phenomenon takes place under cover of the mask, without the knowledge of the operator.

Blind Intubation. Blind intubation is a survival of the O'Dwyer intubation technique for diphtheria, the best available for many years. The advent of direct oral endoscopy introduced by Dr. Chevalier Jackson has completely supplanted the older procedure in safety and precision. A very brief experience with the two methods will suggest that the blind poking of one or two adult fingers plus a tube against the delicate tissues of a baby's pharynx, in asphyxia neonatorum, is much more likely to traumatize it than is the introduction of an infant laryngoscope permitting an unobstructed view of the field. It may be noted that the total diameter of the blade of the laryngoscope is considerably less than that of the index finger. If this technique is carried out where indicated, that is, in the flaccid patient, there is no excuse for trauma. It will be recalled that a baby has no teeth. For this reason the necessity for extreme extension of the head is obviated. In order to acquire skill and safety in oral endoscopy (examination of the mouth, pharynx, and larynx) it is recommended that the infant laryngoscope be used as a tongue depressor to examine the throat of every baby, who will permit the entrance of a finger between the gums without resistence. The trauma of such toilet is no more than is to be expected from the use of an ordinary tongue depressor. Such a routine exposure will familiarize the physician with the simple instrumentation involved and acquaint him with the orientation of the field. He will then be prepared to make the necessary exposure of the larynx for examination and intubation as required. The same principles apply in the case of the asphyxiated adult.

Degrees of Pressure. Much attention has been devoted to the pressure and the volume of the gases to be insufflated. A more important consideration, the duration of the insufflation, is seldom referred to. How many seconds insufflation is maintained is more important than how much gas is used or how great the pressure. Owing to the resistance offered by the rapidly reduced diameter of the airways, trachea, bronchi, bronchioles (representing diameters, ranging from $\frac{3}{4}$ of an inch to a fraction of a mm) a pressure of 25 mm Hg maintained for five seconds will become completely dissipated before the final radicals are reached. If, on the other hand, pressure is sustained, this will penetrate to the deeper portions of the lung and result in rupture of the alveolar air vesicles. Forceful mouth-to-mouth insufflation may develop a positive pressure of 100 mm Hg. The reason that this pressure, so often employed in mouth-to-mouth resuscitation in infant and adult, does not rupture the lung into which it is blown is that the pressure cannot be maintained for over a few seconds by the person applying it. Polak and Adams* state that injury does not occur unless intra-pulmonary pressure is above 80 mm Hg.

Instrumentation of Airway. With due respect for the tissues involved, lubricated metal instrumentation is no more traumatic than is instrumentation of any resilient material; indeed, because of the control permitted and the smaller bulk for equal strength, metal instrumentation is often less traumatic than other methods. This is particularly true of endo-tracheal tubes for anaesthesia. For a given internal diameter, a metal tube will occupy a smaller total external diameter and cause less difficulty and trauma in intubation than will a composition tube. Metal tracheatomy tubes worn for weeks are well tolerated and could not satisfactorily be replaced by rubber or composition tubes. The entire field of bronchoscopy emphasizes the value of metal instrumentation. The infant airway is delicate and demands gentle treatment, but by no means precludes the use of metal instrumentation. It may be recommended for the same reasons as is its use in the adult.

Rhythmic Suction. Coryllos stated that he considered mouth-to-mouth insufflation so useful that he set about looking for a mechanical means to carry out this principle in laboratory animals. Mouth-to-mouth insufflation, it will be recalled, is strictly an intermittent positive-pressure method without suction. There appears to be no evidence in normal physiology or in laboratory practice to suggest that suction is necessary or useful in artificial respiration.

When rhythmic insufflation and suction is directed into an open-neck bag, suction appears to reduce the respiratory lag in expiration and to allow more frequent expiration. Practically, the respiratory airways do not permit of expiration in the manner observed in the open, unobstructed bag. Insufflation in the unconscious is embarrassed by obstructive factors, especially the relaxation, already referred to in the pathologic physiology of asphyxia (p. 38). The ideal proposed, therefore, by suction insufflation not only does not work out in practice but actually interferes with the positive usefulness of insufflation which is stopped when the automatic mechanism is thrown into a premature suction phase.

(2) **Use of Carbon Dioxide and Resuscitation.** The use of carbon dioxide was popularized in this country by Dr. Yandell Henderson. The author, carrying out the principles recommended by Henderson, has used carbon dioxide in the field of anaesthesia for the last thirty years. Clini-

* Naval Medical Bulletin (Washington), **30**, 165 (1932).

cally he has found this gas to be of the greatest possible value as a respiratory stimulant.*

While the use of carbon dioxide in oxygen has become routine practice in the resuscitation of all types of asphyxial accidents throughout the English-speaking world, its use in the terminal asphyxia of the new born is questioned by Nicholson Eastman of Johns Hopkins.† "The question of the most suitable gas to introduce into the lungs of asphyxiated infants resolves itself largely into two possibilities: oxygen alone, or oxygen combined with carbon dioxide. The value of the latter gas mixture has been urged by Yandell Henderson for many years, with the result that oxygen and carbon dioxide mixtures are probably more widely employed in the United States than oxygen alone. . . . Asphyxiated babies are born with an excess of free carbon dioxide in the blood, and it would seem illogical to give them more. They also show an extremely low pH of the blood; and accordingly the use of carbon dioxide in such cases would seem not only superfluous but possibly harmful, in that it would tend to aggravate an already existing acidosis."

Yandell Henderson in his "Adventures in Respiration" (1938) challenges the entire conception of acidosis referred to by Eastman. He suggests that the acidosis of the new born be approached from the biochemical rather than by the physiochemical view-point. He prefers the term *acarbia* (lowered blood alkali) to *acidosis*, and recognizes four fundamental conditions in which such acarbia may be experimentally produced, *i.e.*, by altitude, acapnea, asphyxia, and by adding acids to the blood.

He offers experimental evidence to show that there is a vital difference between non-acidotic acarbia (altitude, acapnea and asphyxia) and true acidosis in which acids are added to the blood. He states: "The outstanding feature of our experiments with acids was that these truly acidotic animals were quickly killed by an inhalation of five to six per cent of carbon dioxide. They react, in fact, exactly as Van Slyke, Cannon, Eastman and many others claim that acarbial patients should act, but in fact do not. In the effects of inhalation of carbon dioxide, we have, therefore, a crucial test, whether in any condition of acarbia the state is acidotic or not. If it is, carbon dioxide may kill; if it is not, it tends to cure."

In view of these conflicting theories regarding the fundamental concept of acidosis, arrived at from the standpoint of animal experimentation on the one hand, and the biochemical field of asphyxia on the other, the clinician may well remain open-minded. The clinical solution to the problem should in no way be embarrassed by the adoption of a dogmatic point of view. As in other aspects of resuscitation, the time factor and an elastic response to clinical reactions should be considered. Nothing could

* See "Art of Anaesthesia," p. 302.

† "International Clinic," Vol. II, Series 46, 1936.

be more simple than the addition or removal of carbon dioxide in the insufflated mixture. The objective in resuscitation is to saturate the alveolar air with oxygen as quickly as possible. If spontaneous breathing can be deepened by CO_2 at the onset of resuscitation, this advantage may well balance the question of possible danger. The work of the observant clinician, when complementing the technical use of mechanical robots by non-medical persons, will in due time indicate the true direction that routine should follow. Approaching the problem from the clinical view-point of anaesthesia and practical resuscitation, the author has repeatedly witnessed respiro-circulatory improvement when carbon dioxide was used with oxygen. This has occurred not only in routine anaesthesia but frequently in resuscitation. He has never seen immediate or ill effects. The use or the abuse of carbon dioxide is but one factor in the art of resuscitation. Any interference with the technical administration of oxygen would seem to be of greater importance than its dilution by another gas of whose action there is so much question.

(3) **The Stages of Asphyxia.** Certain degrees or stages of asphyxia have become apparent to the experimental physiologist, as well as to the clinician. Dr. Pol Coryllos, in his experimental researches upon artificially submerged dogs, recognized four stages of asphyxiation, each lasting approximately one minute.

(1) Initial apnea. This occurs when the water inhaled first touches the larynx.

(2) Initial dysponea. This occurs when the necessity to breathe over comes the initial apnea.

(3) Terminal apnea. This takes place when the respiratory center becomes exhausted through oxygen deficiency.

(4) Apparent death. At this period, there is complete flaccidity and disappearance of the respiration. Reflexes are absent and the heartbeat may or may not be discernible.

Clinically, the author has described three stages of asphyxia. These stages have been found acceptable, particularly in the field of asphyxia neonatorum. The physical signs of these stages are quite constant regardless of the cause of asphyxia.

"*Stage of Depression.* Patient is in a stuporous or semiconscious state. He may be roused by slapping the face, friction of the lip, etc., but usually relapses following stimulation. Reflexes active, respirations shallow and quiet, pulse regular may be depressed or stimulated. Patient may retch or vomit and move extremities.

"*Stage of Spasticity.* Patient completely unconscious and cannot be roused. He is cyanosed or pallid, depending upon the condition of the circulation. Breathing is obstructed, and there is a tendency to masseteric spasm. Vomiting, and bleeding from the mouth or nose are common.

Eyes are injected; skin is cold; pharyngeal reflexes active; extremities relaxed; circulation sluggish or with the bounding pulse of anoxemia.

"*Stage of Flaccidity.* The patient is completely relaxed. Jaws separate without resistance. Pharyngeal reflexes in abeyance. Skin cold and clammy. Eyeballs fixed, reflexes gone. Laryngeal reflexes sluggish or absent. Respiration shallow and obstructed, or not demonstrable. Heart sounds are inaudible. Patient cyanosed or pallid."

Chapter 6

Methods of Resuscitation

Manual Methods

The oldest manual method is that known as the Silvester method. The first record of this is found in the *British Medical Journal* in 1858, page 576.

Hewitt, who practiced anaesthesia as an "art," describes the Silvester technique so carefully that we quote from his "Anaesthetics" (3rd Edition, pages 555–557, 1907):

"Silvester's Method of Artificial Respiration: If the patient be lying lengthwise upon a bed, he should be rapidly placed transversely, and his head allowed to hang over the side. Should he be lying upon an operating-table, similar extension of the head may be effected over the end of the table. If he be in the sitting posture at the time of the respiratory failure, he should be at once placed on the ground with his shoulders slightly raised and his head extended. Although it is here assumed that the air-passages are patent, it is best, as a routine practice, to insert a gag, apply the tongue-forceps, and make traction upon this organ in order to be quite sure that it is not obstructing breathing. As is well known, the respiration may become obstructed so inaudibly that an error in the diagnosis of the kind of respiratory failure may easily be committed. Full extension of the head and neck, as pointed out by Dr. Howard, tends to keep open a free air-way. The administrator should stand behind his patient, and grasping the arms at the elbows, should press them firmly and steadily against the sides of the chest. In the vast majority of cases this pressure will cause an expiration; but should it not do so at once, forcible pressure below the costal margins, and directly towards the diaphragm, may be brought to bear. After the arms have been steadily pressed against the sides for about a couple of seconds, they should be brought deliberately towards the administrator, so that they come into the long axis of the patient's body, on either side of his head. This procedure usually has the effect of enlarging the capacity of the chest by causing the pectoral muscles to raise the upper ribs. In this way inspiration is effected. The arms should be kept extended for about a couple of seconds, after which they may be again brought to the side. These expiratory and inspiratory movements should be repeated regularly and steadily about fifteen times per minute, careful watch being kept for any spontaneous respiration. If any signs of the latter appear, the natural movements should be supplemented by the artificial till the breathing has become thoroughly re-established. Care must be taken throughout to maintain a free air-way, and not to exert undue and unnecessary force during expiration. Ribs have been fractured, and rupture of the liver has actually occurred, from too roughly handling the patient."

He also refers to the method of "chest compression" which always comes into play as a spontaneous desire to do the obvious at once:

"Chest Compression: Rhythmic compression of the chest walls, each compression being followed by a removal of the pressure, has already been alluded to as of service in threatened respiratory arrest. As Richardson pointed out, this plan is particularly useful in young subjects, i.e., in those with elastic thoracic parietes. Indeed, in the treatment of suspended breathing in children, this simple chest compression usually answers as well as any other method of resuscitation, although, as pointed out above, the presence of mucus within the larynx may prevent the inspiratory recoil which is essential."

Fig. 12. Place. *Courtesy Bureau of Mines*

The prone-pressure Schaefer Method was introduced through an article appearing in the *Medical Chir Transactions* (*London*), **87,** 609 (1904) and again in the *Transactions of the Harvey Society* (*New York*), 223 (1907, 1908). The description of this standard technique, as it appears in the "Red Cross Handbook," is as follows:

"1. Lay the patient on his belly, one arm extended directly overhead, the other arm bent at elbow and with the face turned outward and resting on hand and forearm, so that the nose and mouth are free for breathing.

"2. Kneel straddling the patient's thighs, with your knees placed at such a distance from the hip bones as will allow you to assume the position shown in Figure 12.

"Place the palms of the hands on the small of the back with fingers resting on the ribs, the little finger just touching the lowest rib, with the thumb and fingers in a natural position and the tips of the fingers just out of sight (Fig. 12).

"3. With the arms held straight, swing forward slowly, so that the weight of your body is gradually brought to bear upon the patient. The shoulder should be directly over the heel of the hand at the end of the forward swing (Fig. 13). Do not bend your elbows. This operation should take about two seconds.

"4. Now immediately swing backward so as to remove the pressure completely (Fig. 14).*

Fig. 13. Push. *Courtesy Bureau of Mines*

Fig. 14. Rise, release, rest. *Courtesy Bureau of Mines*

"5. After two seconds swing forward again. Repeat unhurriedly twelve to fifteen times a minute the double movement of compression and release, a complete respiration in four or five seconds.

"6. Continue artificial respiration without interruption until natural breathing is restored—if necessary, four hours or longer or until a physician declares the patient dead.

* Figs. 12, 13 and 14 illustrate the rhythm of—place, push (2 sec.); rise, release (2 sec.); rest (1 sec.). (Appendix.)

"7. As soon as artificial respiration has been started and while it is being continued, an assistant should loosen any tight clothing about the patient's neck, chest or waist. Keep the patient warm. Do not give any liquids whatever by mouth until the patient is fully conscious.

"8. To avoid strain on the heart when the patient revives, he should be kept lying down and not allowed to stand or sit up. If the doctor has not arrived by the time the patient has revived, the latter should be given some stimulant such as one teaspoonful of aromatic spirits of ammonia in a small glass of water, or a hot drink of coffee or tea, etc. The patient should be kept warm.

"9. Resuscitation should be carried on at the nearest possible point to where the patient received his injuries. He should not be moved from this point until he is breathing normally, of his own volition, and then moved only in a lying position. Should it be necessary, due to extreme weather conditions, etc., to move the patient before he is breathing normally, resuscitation should be carried on during the time he is being moved.

"10. A brief return of natural respiration is not a certain indication for stopping the resuscitation. Not infrequently the patient, after a temporary recovery of respiration, stops breathing again. The patient must be watched and if natural breathing stops, artificial respiration should be resumed at once.

"11. In carrying out resuscitation it may be necessary to change the operator. This change must be made without losing the rhythm of respiration. By this procedure no confusion results at the time of change of operator and a regular rhythm is kept up."

There is probably no man in the country who has given more attention to the prone-pressure Schaefer Method and who has contributed more to its popularization through energetic, well-trained First Aid groups, than Dr. Yandell Henderson. For this reason, the following comments* deserve attention. Dr. Henderson points out that the prone-pressure Schaefer technique is the one method of artificial respiration in which all efforts are directed to producing expiration. Inspiration, the life-saving phase, occurs automatically or not at all. Before beginning the application of the treatment, the patient is put in the best inspiratory position, with both arms extended. In other words, all possible resistance to automatic inspiration from position are eliminated. The method requires less effort on the part of the operator. The tempo should be slow enough, that is, the intervals between compression should be long enough to allow for an expansive recoil (inspiration). He states that the prone-pressure Schaefer effect in ventilation is equivalent to that of normal breathing, that it is sufficient for positive effects and yet not enough to bring about over-ventilation. He points out, however, that loss of carbon dioxide from any cause throws out the elasticity of recoil and impairs the efficiency of the method. In complete flaccidity there is no expansion; no *manual* method, neither prone pressure nor any other, can induce any larger volume of lung ventilation

* *J. Am. Med. Assn.*, April 5, 1941.

than the tonic elasticity of the body at the moment permits. All inspiration or positive ventilation depends entirely upon the elastic recoil of the chest.

While these statements bring out the efficiency and the safety of Schaefer Method, they also confirm its shortcomings. These involve *loss of muscle tone* from any cause which may have taken place before the accident, such as over-ventilation from over exertion, the effects of drugs, profound asphyxia, etc. This loss of muscle tone promptly reduces the tidal volume of inspired air. Under these conditions, a vicious circle may readily develop, *i.e.*, small volume of air aspirated, increased asphyxia, more relaxation, still smaller volume of air. The second serious shortcoming of the method is that an adequate tidal volume depends upon an airway which is unobstructed by relaxed soft parts and by foreign matter, such as sea water, vomitus or blood. Within the vicious circle, oxygen is helpful and CO_2 is helpful also if there exists sufficiently free ventilation for its suction into the lungs. The complicating factors of muscle tone and respiratory obstruction tend to become cumulative in ordinary unassisted manual methods. It therefore becomes our duty to consider the possible aids which may increase the efficiency of manual methods and which may contribute to breaking the vicious circle.

Aids Available to Prone-pressure Schaefer Technique. The aids available to overcome the complicating factors of muscle tone loss and respiratory obstruction are the use of airways, nasal tubes, and inhalation of oxygen and CO_2, or of CO_2 alone.

Airways: 30 years ago Karl Connell of Roosevelt Hospital introduced the Connell tube (Fig. 15). This tube, moulded to the contour of the average oro-pharynx, provides relief to the obstruction so commonly caused by the falling back of the tongue. The relief so obtained completely eliminates the emergency use of mouth prop and tongue forceps. The early tubes manufactured were made of thin-gauge metal which presented a cutting edge and occasionally traumatized the mucous membrane of the harynx. Such pharyngeal tubes should be made of heavy-gauge metal presenting a velvet edge which when lubricated offer no irritation. Recently, rubber tubes have been introduced and these have become popular (Fig. 16). They give satisfaction, but do not present as large a lumen and consequently not so much ventilation as do metal tubes.

Suction: The universal acceptance of the principle of suction to free the airway demands that this convenience be constantly available. Every unconscious patient is entitled to immediate suction whenever this may become necessary. At this writing no well equipped operating room is without adequate suction, from a central plant, by means of a steam ejector (Fig. 18), or an electric pump (Fig. 20). Automotive vehicles provide first-class suction facilities through the intake manifold of the gasoline engine (Fig. 21). Hand suction as shown in Figs. 17 and 19, is convenient where automatic forms are not available.

AIDS TO PRONE-PRESSURE SCHAEFER

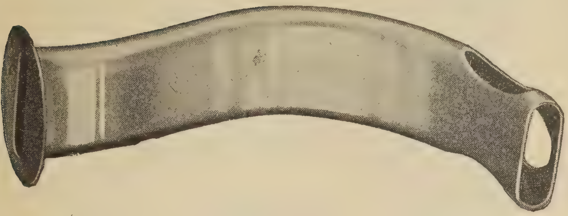


Fig. 15. The Connell tube, heavy metal with soft bevel.

Courtesy Foregger Co.

Fig. 16. Guedel rubber airway.

Courtesy Foregger Co.

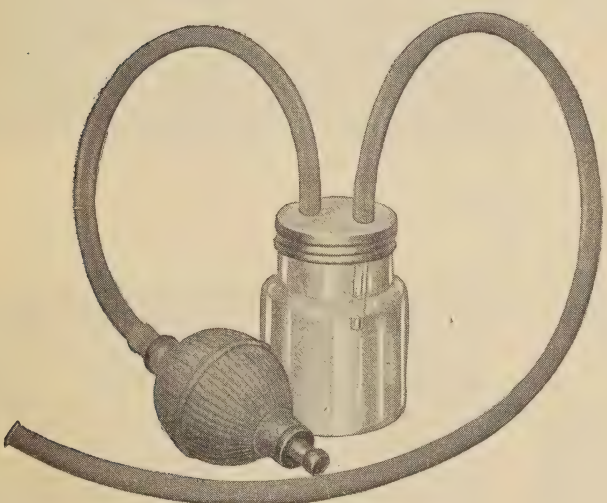


Fig. 17. The "Little Sucker," used when mechanical suction is unavailable.

Courtesy Foregger Co.

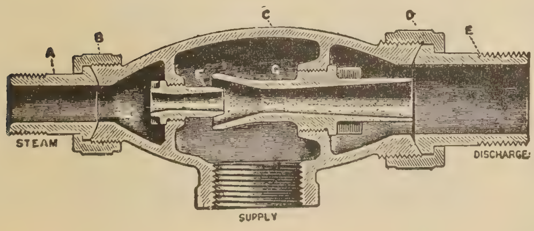
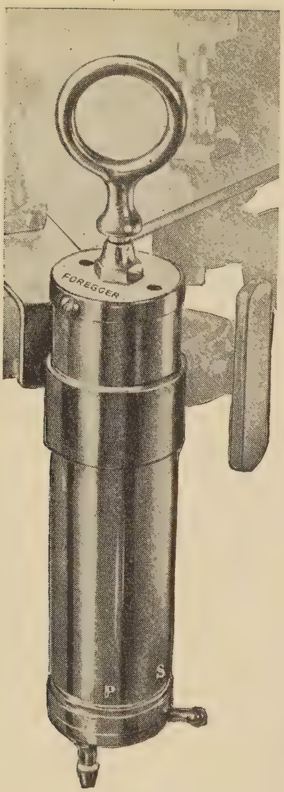


Fig. 18. Apparatus for steam suction.

From Flagg, "Art of Anesthesia."



Courtesy Foregger Co.
Fig. 19. Suction air pump.

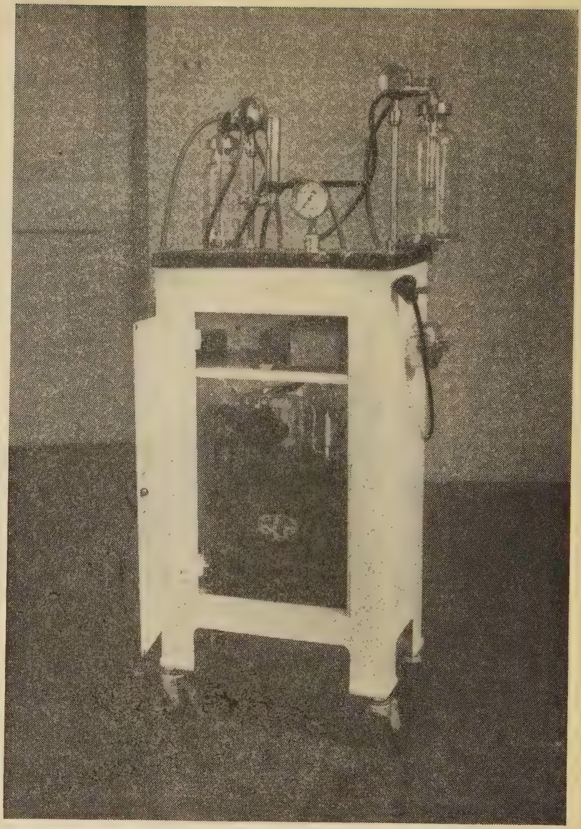


Fig. 20. Electric suction pump.

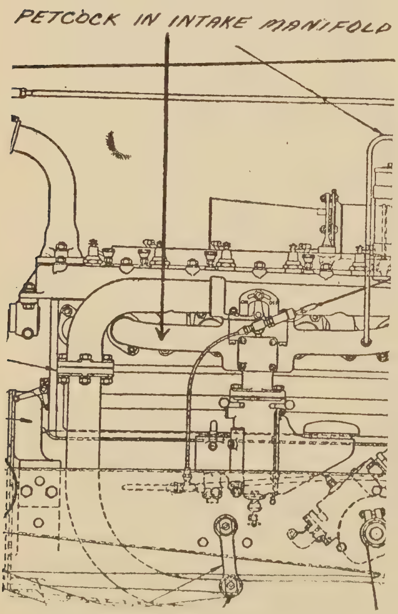


Fig. 21. Suction obtained from intake manifold.

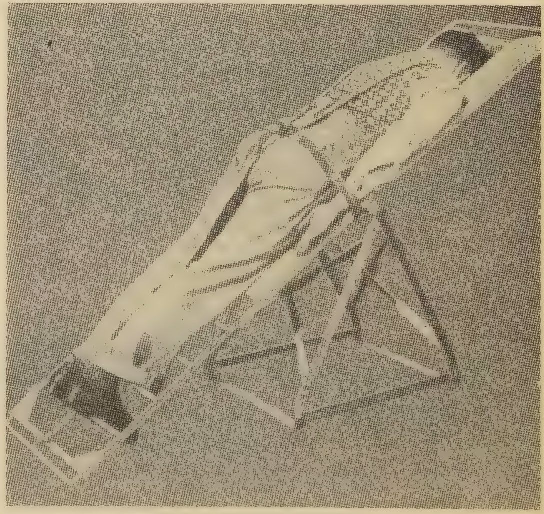


Fig. 21a. Eve's rocking method. (An excellent article by Frank C. Eve, of Hull, England, appears in *J. Am. Med. Assn.*, **124,** 964 (1944).)

Suction is probably the most important single factor in preventing morbidity and mortality in asphyxial accident. Every anaesthetizing room, resuscitation ward, obstetrical delivery room, as well as every post-operative recovery room, should be provided with strong, dependable suction. When the nursing and the operating room personnel are taught the significance of suction, the not uncommon attitude of conferring a favor in making this instantly and constantly available will gradually disappear. It should not be necessary to ask for suction: it should be present as routine equipment in perfect operating condition.

The Use of Nasal Tubes. The routine use of nasal tubes for oxygen therapy has become commonplace (Fig. 24). These tubes with multi-perforated tips permit a distributed flow of oxygen into the posterior pharynx without producing pressure. It is the author's opinion that the routine use of intra-pharyngeal oxygen by nasal tubes would be much more effective and easier to manage than is the correct use of an inhaler. The mouth remains open and exposed. Suction of fluid in the mouth, insufflation of oxygen in the pharynx and prone-pressure Schaefer constitute an excellent first aid routine, which can do no harm. The tube is lubricated by the patient's saliva and passed along the floor of the nose to a given distance marked on the tube. When strapped to the forehead with adhesive, the control is perfect.

Oxygen and Carbon Dioxide. Oxygen, when added to the Schaefer technique by the use of a nasal tube or inhaler serves to compensate for reduced tidal volume recoil of the chest due to loss of muscle tone. Carbon dioxide serves to return the muscle tone which has been lost. These two gases cut the vicious circle caused by loss of muscle tone, reduced recoil of chest, reduced ventilation, reduced ventilation anoxia, anoxia, reduced muscle tone, etc.

Modern methods of resuscitation by manual methods suggest the addition of suction, to clear the airway of foreign matter; the addition of an airway to prevent obstruction due to relaxation; and the administration of oxygen and carbon dioxide, back of the obstruction, in the naso-pharynx, where it may be picked up by Schaefer artificial respiration.

Mechanical Devices for the Prevention of Asphyxial Death

In order to appraise the value of a mechanical device, the pathological indications for treatment of asphyxia must be constantly kept in mind. For the reader's convenience these indications are repeated below.

(1) Remove foreign matter from airway.
(2) Eliminate or overcome the effects of masseteric or glottic spasm.
(3) Meet the complications caused by loss of muscle tone.
(4) Replace the function of respiratory muscles which are paralyzed.
(5) Protect the Hering-Bruer reflex.
(6) Maintain intrapleural and intrapulmonary pressures.
(7) Recognize and treat oxygen dilution in respired mixtures.

(8) Anticipate and treat dehydration.
(9) Maintain blood pressure.
(10) Maintain oxygen-carrying capacity of blood.
(11) Administer carbon dioxide according to indications.
(12) Respect the action of the carotid and aortic chemoceptor reflexes.
(13) Administer heat to the body.
(14) Apply cardiac massage with oxygen in heart failure.

Using the above indications as a guide, the reader will be able to select from the apparatus shown on the following pages that which is best adapted to his purposes.

Mechanical devices have been manufactured to provide a high percentage of oxygen and/or carbon dioxide to the asphyxiated patient by the following methods:

(1) Inhalation by oral and/or nasal inhaler.
(2) Inhalation from oro-pharynx by oral tube or from naso-pharynx by nasal catheter.
(3) Transpharyngeal insufflation.
(4) Endotracheal insufflation.
(5) Extrapulmonary differential pressures (negative pressure cabinets).
(6) High-oxygen external environment (by open or closed oxygen tents).
(7) By subcutaneous administration of oxygen.
(8) By increasing the combined oxygen capacity of the blood by ultraviolet irradiation of auto-transfused blood.

Mechanical Devices to Control Oxygen Pressure. Oxygen, either alone or mixed with carbon dioxide or helium, is sold in steel cylinders in which the gas has been compressed to a pressure varying from 1500 to 2000 pounds per square inch. These cylinders occur in various sizes and degrees of portability. Among medical distributors the contents are referred to in gallons and are delivered to the patient in liters (4 liters equals 1 gallon). In commercial circles the contents are referred to in cubic feet. Both gallons and feet are estimated on a basis of the sea-level pressure of one atmosphere. Oxygen employed for commercial purposes, welding, etc., when free from carbon monoxide, is frequently used for medical treatment.

The pressure within the tank is stepped down by single or two-stage regulators, as the case may be. Before delivery to the patient, however, the gas is passed through a gauge (flow meter) or gas bag, equipped with a pressure blow-off which prevents excessive pressure from reaching the patient. Fig. 22 (Foregger) shows a simple, one-stage, pressure reducing valve. Fig. 23 shows two-stage valves in cross-section.

When two gauges are employed the first usually designates the tank pressure or contents, the second the rate of flow to the patient, which varies from 4 to 15 liters per minute, depending upon the equipment in use. (Ordinary respiratory needs at rest are 8 to 12 liters per minute; under exertion this requirement may be greatly increased (see High Altitude,

p. 138).) Four liters per minute is, therefore, but a diluent to atmospheric air. (We are not here concerned with carbon dioxide absorption equipment by the use of which extensive rebreathing may be tolerated.) Oxygen as it issues from the cylinder is a dried gas. Delivered in volume to the patient it therefore absorbs moisture from the mucous membrane surfaces. The effect of such drying is to cause irritation and engorgment. In order to overcome the effect of local dehydration, apparatus has been devised to moisten the gas before delivery (Figs. 26 and 27).

Equipment for Inhalation by Oral and/or Nasal Inhaler. A paper bag filled with oxygen may be used as an oxygen inhaler. The inhaler of any anaesthetic machine connected to an adequate source of oxygen provides satisfactory inhalation. Inhalers which do not permit rebreathing are specially designed for use when it is desirable to remove noxious gases from the circulation, *i.e.*, carbon monoxide or anaesthetic agents of a gaseous nature. In order to avoid overventilating carbon dioxide out of the circulation by excessive oxygen without rebreathing, it is customary to use oxygen with 7 to 10 per cent CO_2 with such equipment. If rebreathing is not employed, it is of course necessary to provide the patient with his required complement of gas volume for each inspiration. In order, therefore, that the patient may never breathe against a collapsed bag due to a diminished or exhausted supply, an inspiratory valve allowing the entrance of atmospheric air is placed in the inspiratory circuit (Fig. 32). Oxygen may be administered under low positive pressure by placing resistance in the path of the expirations (Fig. 39), or forcing them to take place under water two or three cm deep (Fig. 28). (For practical purposes 25 mm of water is equal to 2 mm Hg. S.G. Hg. 13. $\frac{25}{13}$ equal about 2.)

Inhalation methods and equipment are for the express and limited purpose of supplying high oxygen concentration to the breathing patient or to the patient whose respiratory act is carried on by other than voluntary means, such as prone-pressure Schaefer or negative pressure cabinet.

As a means of stimulating the respiration, and thereby increasing the amount of atmospheric oxygen inhaled, pure CO_2 diluted with atmospheric air is sometimes used. The practice of administering a few breaths of high concentration CO_2 from a small metal container, used to charge beverages, has been employed in England (Fig. 25). Simple equipment for the use of pure CO_2 is shown in Fig. 40.

Inhalation from Oro-pharynx by Oral Tube or from Naso-pharynx by Nasal Catheter. (For use chiefly as aids to manual methods or for post-asphyxial treatment following accident or operation.) These facilities provide the great advantage of eliminating obstruction to the respiration which so frequently occurs in the mouth and the nose of the spastic patient. This technique is put into practice when the simple oral-mask inhalation is supplemented by an oral or pharyngeal tube (Figs. 15 and 29). This pharyngeal tube cannot be employed unless the patient is sufficiently re-

(*Text cont'd on page 73*)

PRESSURE REGULATORS

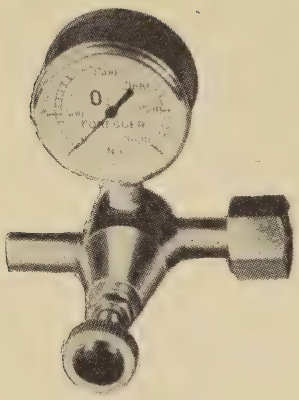


Fig. 22. Control valve with one-stage pressure gauge.

Courtesy Foregger Co.

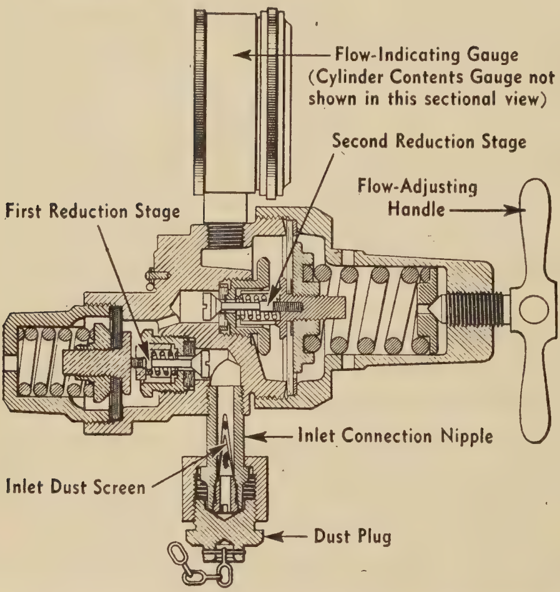


Fig. 23. Cross-section of Linde two-stage regulator.

Courtesy Linde Air Products Co.

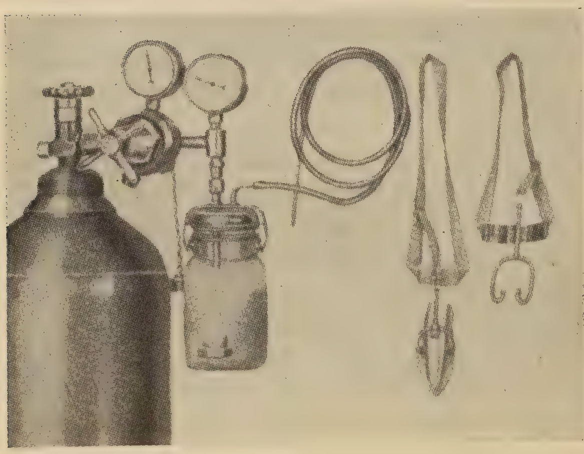


Fig. 24. Two-stage regulator and water-moistener nasal tube to be used when teeth are clenched. Oxygen from any source may be used.

Courtesy J. H. Emerson Co.

Fig. 25. Davis sparklets. (Compressed CO_2)

Fig. 26. O.B. Nasal Catheter, with apparatus to provide moistened oxygen gas. Two-bottle blower detail is shown at right.
Courtesy Ohio Chemical Co.

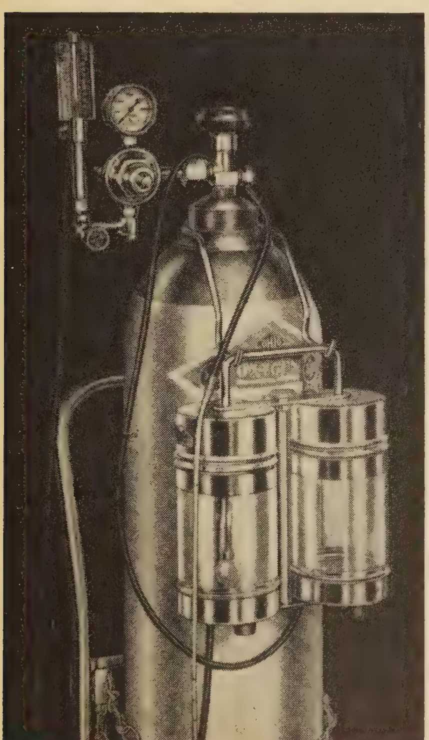
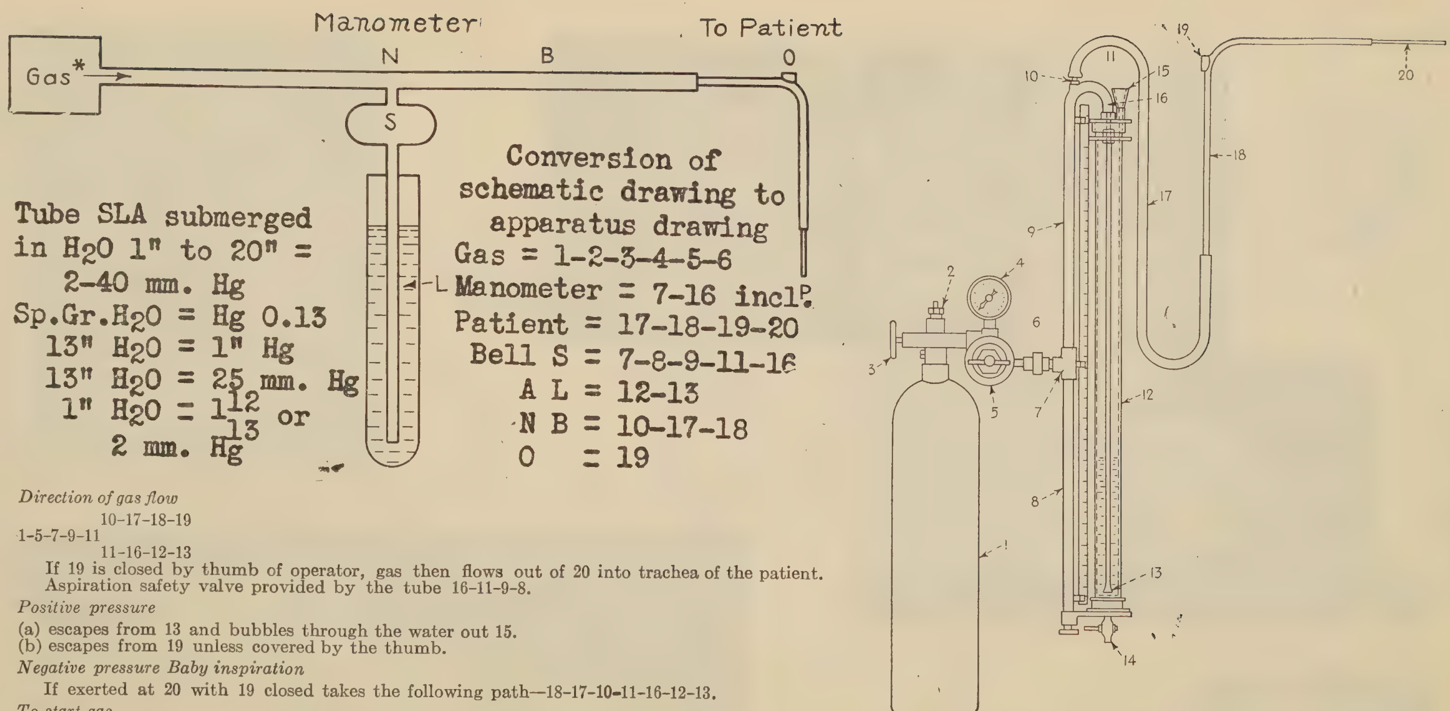


Fig. 26a. Heidebrink oropharyngeal catheter.

Courtesy American Hospital Supply Co.
Fig. 27. Pharyngeal inhalation apparatus to supply moistened oxygen gas by nasal tube.



Direction of gas flow

1-5-7-9-11 { 10-17-18-19 / 11-16-12-13 }

If 19 is closed by thumb of operator, gas then flows out of 20 into trachea of the patient. Aspiration safety valve provided by the tube 16-11-9-8.

Positive pressure

(a) escapes from 13 and bubbles through the water out 15.
(b) escapes from 19 unless covered by the thumb.

Negative pressure Baby inspiration

If exerted at 20 with 19 closed takes the following path—18-17-10-11-16-12-13.

To start gas

Turn valve 5 to right (clockwise)

To stop gas

Turn valve 5 to left (counterclock wise)

The Flagg Resuscitation Apparatus Provides

(1) Rigid support and metal connection between gas supply and manometer.
(3) Accurate water pressure attained by adding and withdrawing water instead of increasing or decreasing depth of tube (inlet 15 outlet 14)
(3) Protected submersion tube.
(4) Sufficient depth for adult 20 inches or 40 mm. Hg.
(5) Inspiratory effort (negative pressure) shows clearly in the tube 12-13.
(6) Aspiration of fluid prevented by large hollow tube 8-7-9-11.

* Cylinder of 90% O_2, 10% CO_2 at 1600 lbs. pressure. Reduced by valve to pressure varying from 2 to 40 mm. Hg.

Fig. 28. Flagg resuscitation apparatus.

Fig. 29. Emergency inhalation outfit.
Courtesy Foregger Co.

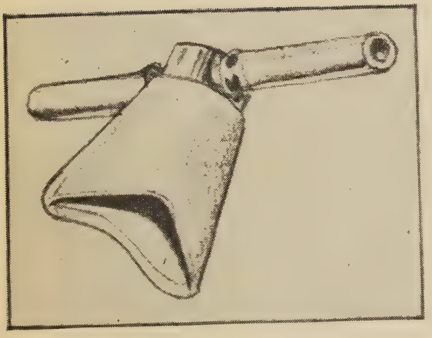
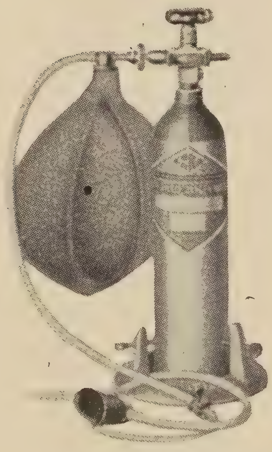


Fig. 30. Infant face inhaler of soft rubber.



Courtesy Ohio Chemical Co.
Fig. 31. Infant inhalation outfit.

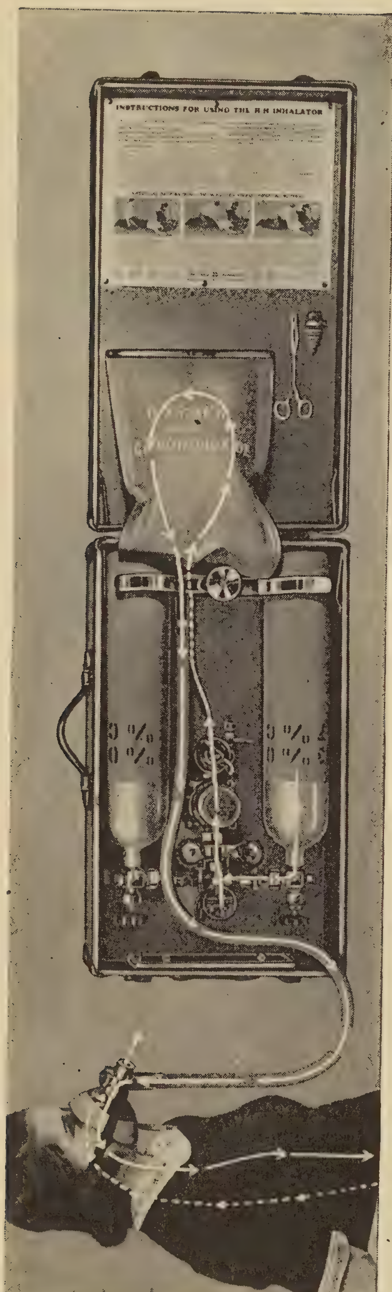


Fig. 32. The H & H Inhalator

Courtesy Mine Safety Appliances Co.

Fig. 33. H & H Inhalator used in conjunction with the Schafer prone-pressure method.

INHALATION EQUIPMENT

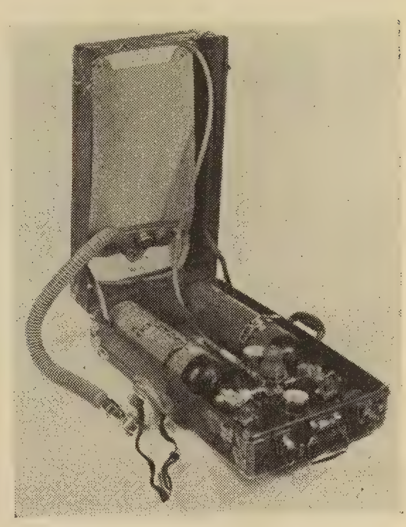
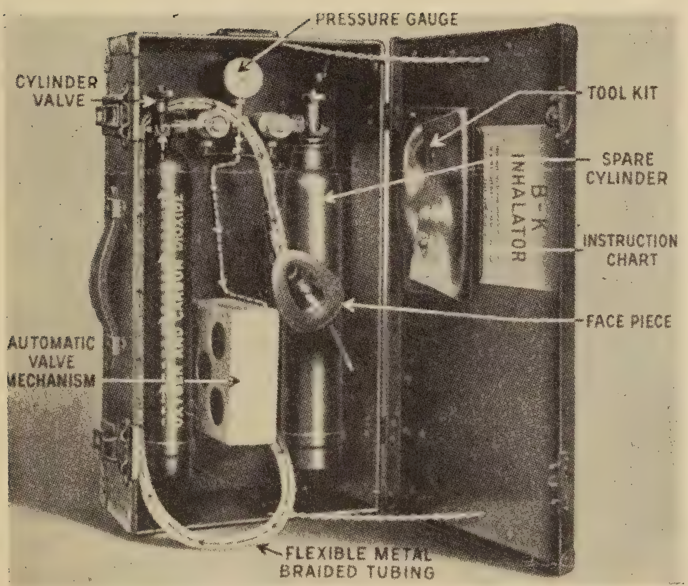
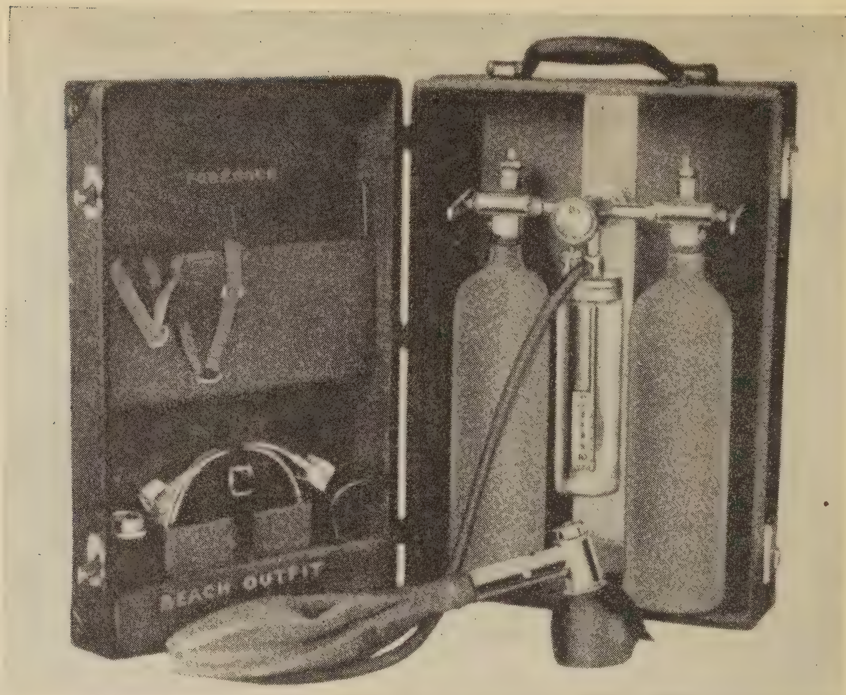


Fig. 34. Davis inhalator.
Courtesy Davis Emergency Equipment Co.



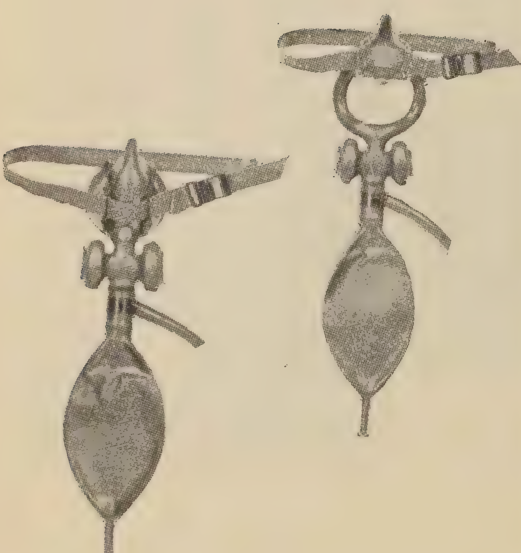
Courtesy Bishinger Koehler Mfg. Co.

Fig. 35. Inhalator showing simple flow of oxygen and carbon dioxide through the facepiece.



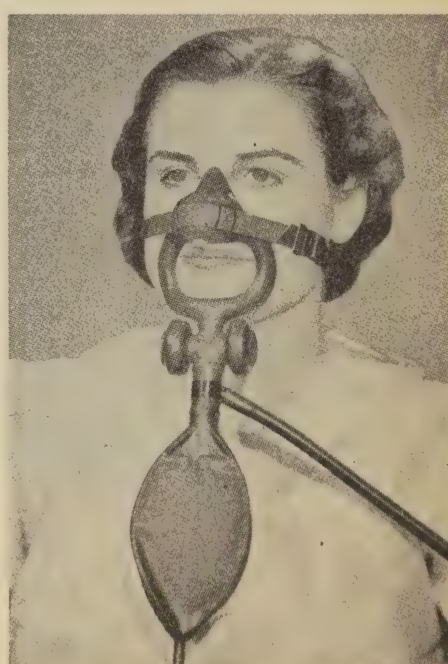
Courtesy Foregger Co.

Fig. 36. Beach Outfit for Inhalation.



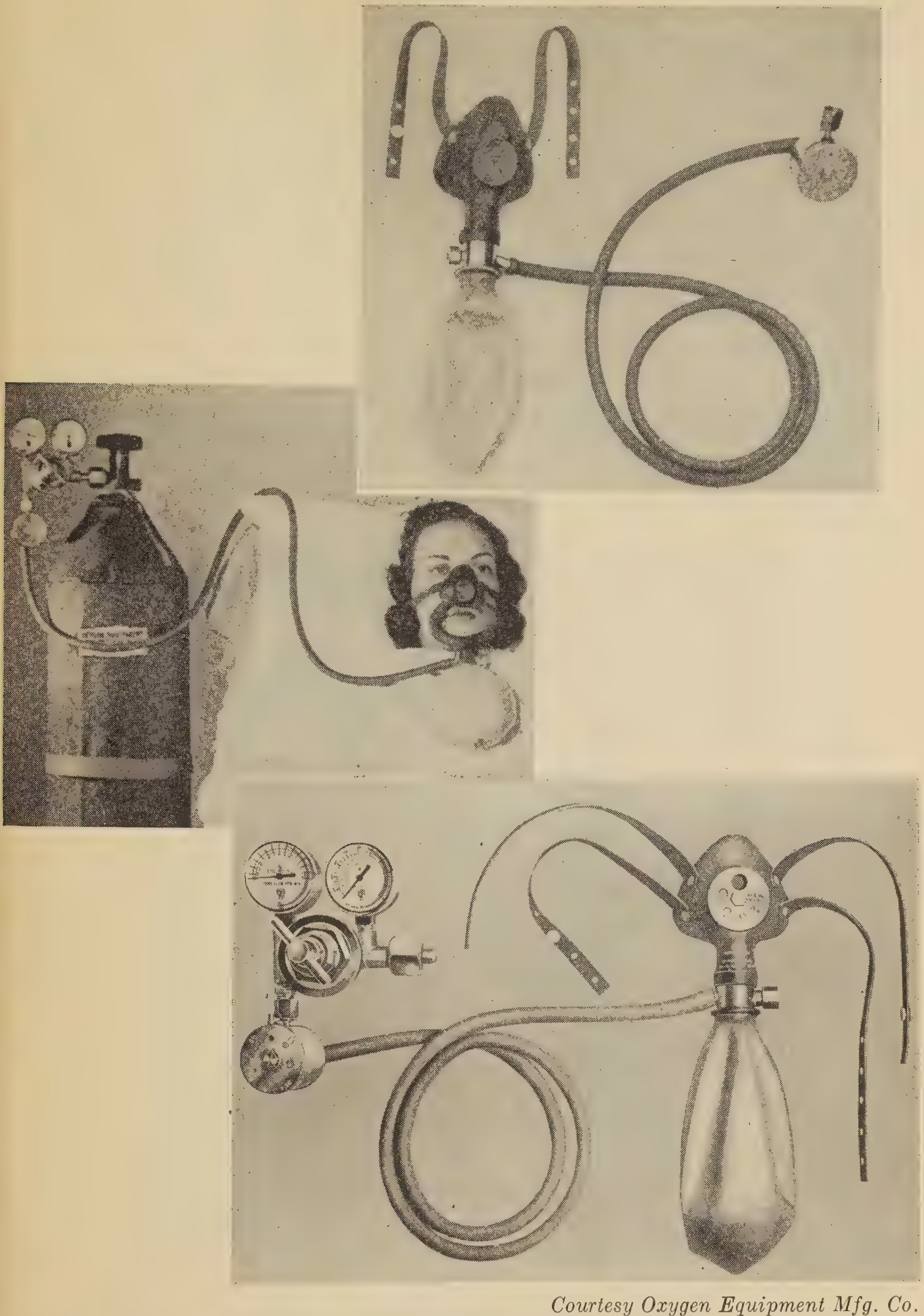
Courtesy Ohio Chemical Co.

Fig. 37. (left) Oronasal inhaler
(right) Nasal inhaler



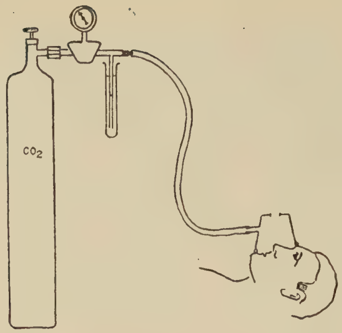
Courtesy Ohio Chemical Co.

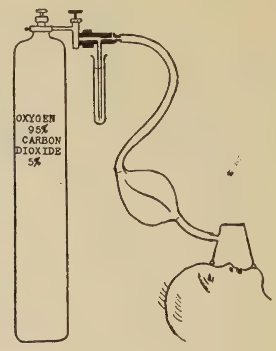
Fig. 38. B-L-B nasal inhaler for women.



Courtesy Oxygen Equipment Mfg. Co.

Fig. 39. (Top) Oro-nasal meter inhaler; (center) nasal inhaler with regulator unit; (below) positive-pressure inhaler.

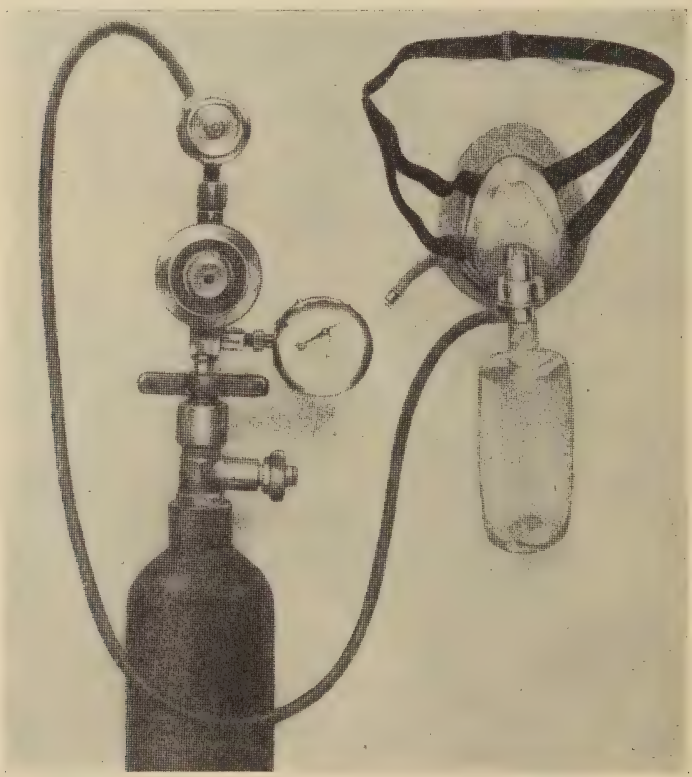




From Henderson's "Adventures in Respiration"

Fig. 40. Inhaler with open mask, supplying carbon dioxide to the inhaled air.

Fig. 41. Infant inhaler supplying carbon dioxide and oxygen.



Courtesy Oxford University Press

Fig. 42. J. S. Haldane's oxygen inhaler (Lighter Pattern). Note similarity between this and more recent inhalers.

laxed to permit separation of the teeth by the operator's fingers (mouth wedge or opener should never be used), and unless the pharyngeal reflex is sufficiently in abeyance to permit retention of the tube.

If difficulty or resistance is met to the introduction of the oral tube, the nasal catheter should be used instead. It contains multiple perforations at the tip, and may be lubricated by Vaseline or by the patient's own saliva. It should be passed along the floor of one nostril. Should nasal obstruction be present in one nostril, it is quite likely that the other nostril will be found quite free. The catheter should be passed to a depth equal to an inch less than the distance between the alae (wing) of the nose and the tragus (opening) of the ear. The tip of the catheter will then terminate just below the soft palate. The tube should be inserted with the gas flowing through it. If it is inserted before the gas is turned on, the sudden flow of gas may cause gagging. A flow of 2–3 liters a minute should be used to begin the administration; this may then be stepped up to four or five liters a minute. If not more than four to six liters of gas are given per minute, it is impossible to build up pressure in the nose or throat. Bubbling caused by gas from the nasal orifices or from the mouth is to be expected.

The nasal tube should be much more commonly employed by First Aid groups than it is at present. It is a great deal more efficient than is the inhaler, which fails to fit the face perfectly, and operates against masseteric, oral or nasal obstruction. Oxygen in the naso-pharynx will be promptly picked up by the prone-pressure Schaefer method. The technique of nasal intubation for naso-pharyngeal inhalation is more simple than for oxygen therapy for the conscious patient, for in the latter the matter of comfort and long-continued treatment is important.

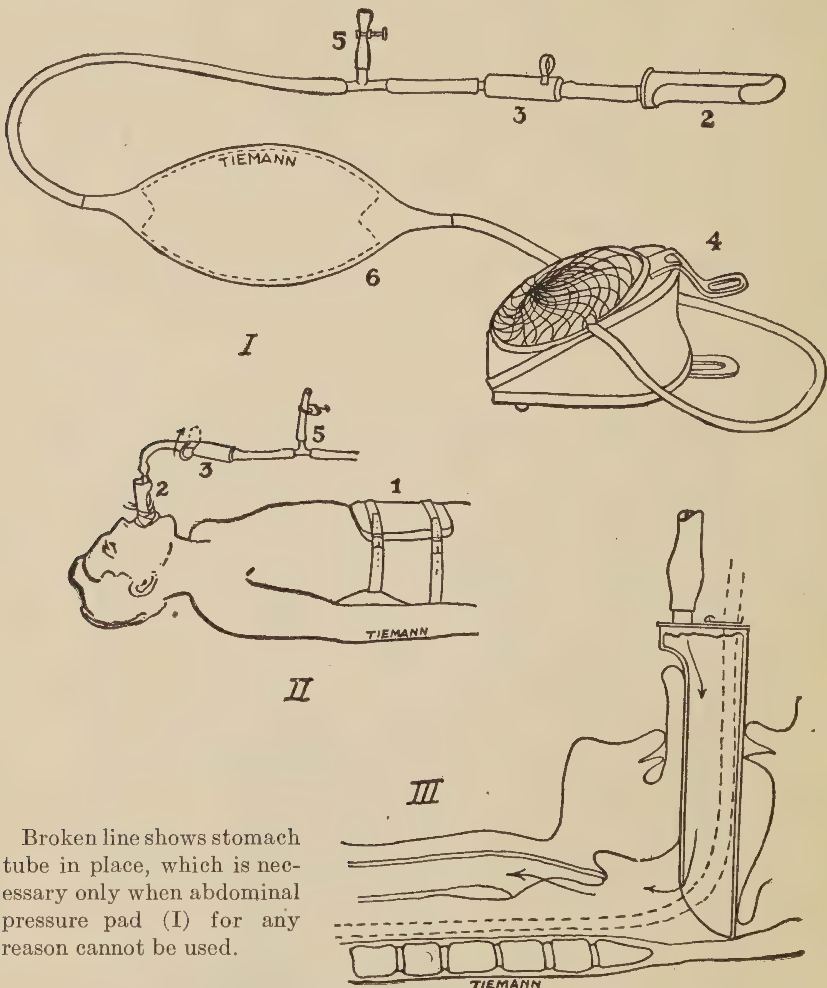
Refer to the pathological indications for services rendered by the equipment for the oro-pharyngeal and naso-pharyngeal inhalation of oxygen. (p. 61).

Transpharyngeal Insufflation. "In forcing air through the nose of an insensible patient, the tongue unless secured, is almost certain to cause obstruction, or the vocal cords may be forced together by the rushing air and act as a valve. As in paralysis of the abductor muscles, because there is no expansion of the glottis as in normal inspiration. Should the larynx become obstructed from any cause, the stomach will be inflated instead of the lungs."*

When Meltzer of the Rockefeller Institute devised his resuscitation apparatus (Fig. 43), he recognized the principles pointed out by O'Dwyer. His apparatus, the progenitor of present-day transpharyngeal insufflation apparatus, made provisions to overcome the objectionable features of the method, *i.e.*, a board with straps to be applied over the abdomen and a stomach tube to deflate the distended stomach. Furthermore the large pharyngeal tube when placed did much to pack the glottis and to prevent regurgitated gastric contents from entering the trachea.

* Joseph O'Dwyer, M.D., *Transactions American Pediatric Society*, p. 128, 1891.

Modern apparatus for transpharyngeal insufflation is handsome, durable and mechanically accurate. The engineer has done his work well. The detail men responsible for distribution, find little difficulty in popularizing a technique limited to "apply the face piece tightly, adjust the valves and let the machine do the rest."



Broken line shows stomach tube in place, which is necessary only when abdominal pressure pad (I) for any reason cannot be used.

From Flagg, "Art of Anaesthesia"

Fig. 43. Equipment for the Meltzer method of transpharyngeal insufflation.

If the principles pointed out by O'Dwyer and accepted by Meltzer are considered unworthy of attention or too difficult of application, transpharyngeal insufflation leaves nothing to be desired. If on the other hand, these principles "stand," transpharyngeal insufflation admittedly goes but part way. The dying, flaccid patient, so treated does not receive the full measure of modern medical service.

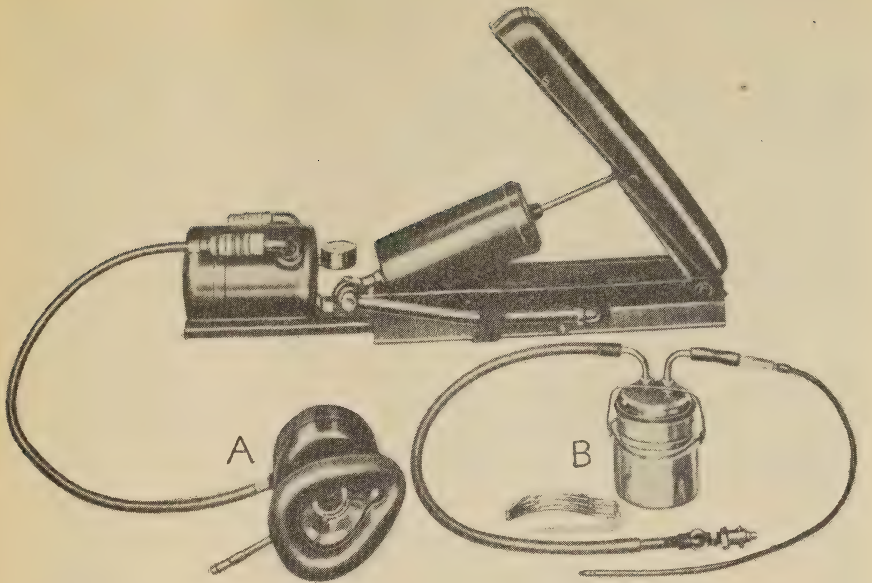
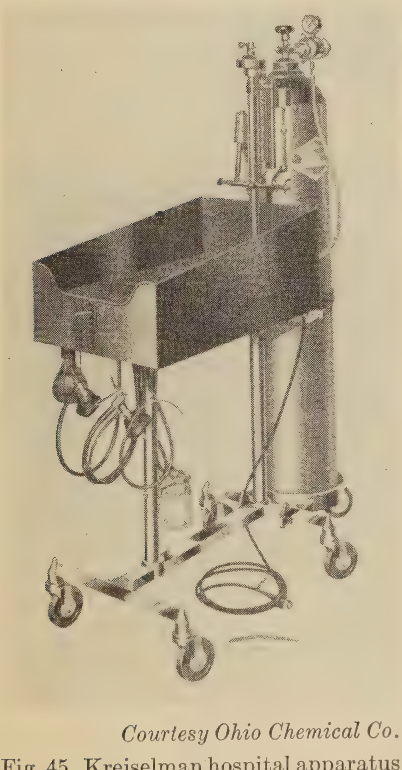
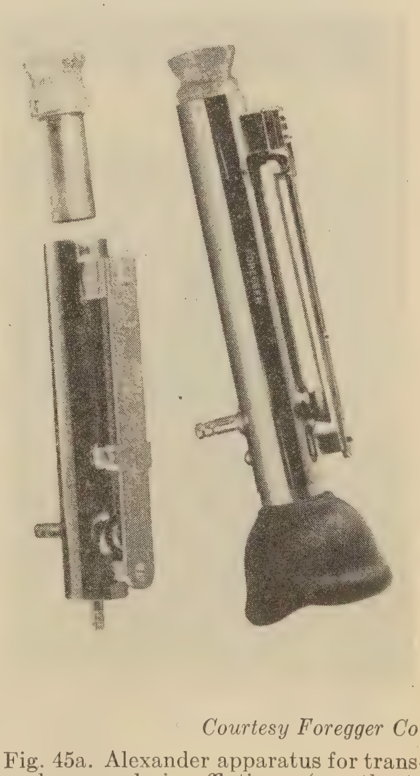


Fig. 44. Emerson resuscitation apparatus for military transpharyngeal insufflation.



Courtesy Ohio Chemical Co.

Fig. 45. Kreiselman hospital apparatus for transpharyngeal insufflation.

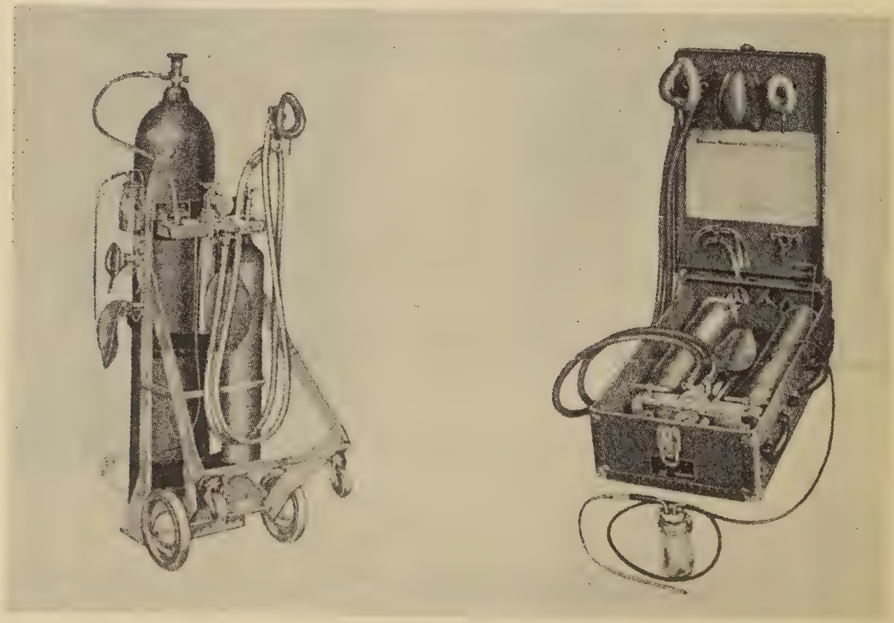


Courtesy Foregger Co.

Fig. 45a. Alexander apparatus for transpharyngeal insufflation (mouth to mouth).

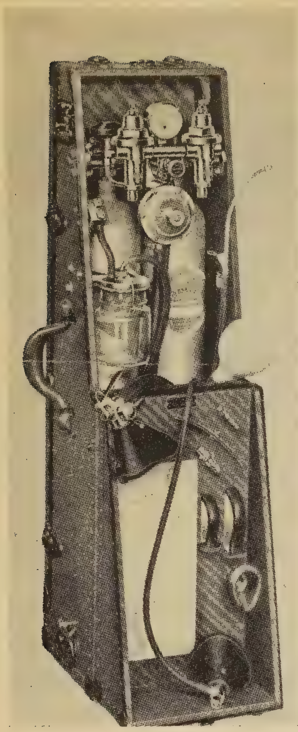
Fig. 46. Henderson's equipment for transpharyngeal insufflation of the new born.

Courtesy Ohio Chemical Co.



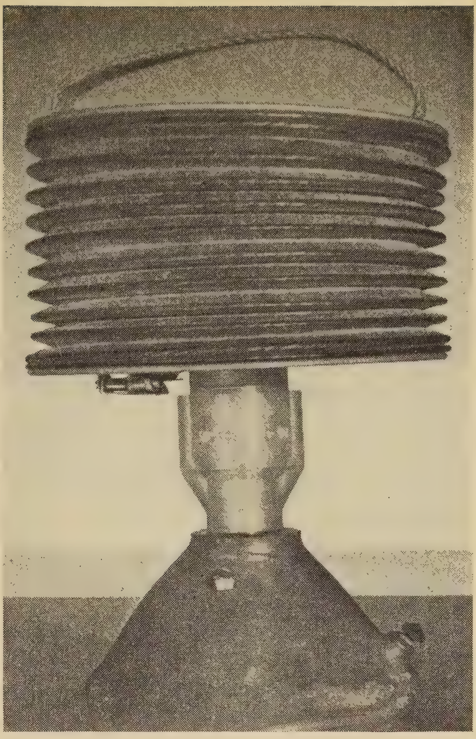
Courtesy J. H. Emerson Co.

Fig. 47. Emerson apparatus for transpharyngeal insufflation and inhalation, horizontal and portable model.



Courtesy Ohio Chemical Co.

Fig. 48. Kreiselman transpharyngeal insufflation apparatus with measured volume and pressure.



Courtesy "Anaesthesiaology"

Fig. 49. Kreiselman apparatus for transpharyngeal insufflation.

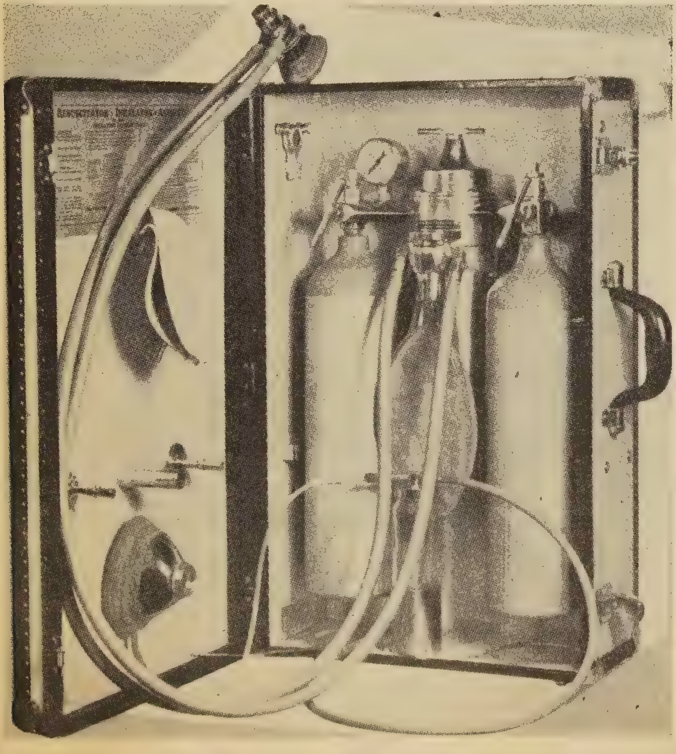


Fig. 50. Apparatus for transpharyngeal insufflation: the E. & J. Resuscitator.

Courtesy E. & J. Mfg. Co.

In the author's opinion, the technique of blind, transpharyngeal insufflation, applied by a face mask, with pressures varying from 15 to 25 mm Hg, is both hazardous and unscientific. It is hazardous in lay hands. It is not efficient in the hands of the physician.

It is to be pointed out, however, that the objections noted are professional rather than mechanical, *i.e.*, a failure to correctly diagnose present pathology and to acquire the indicated technique of treatment rather than a failure on the part of the equipment to do the work for which it is designed. When the physician and the technician have become familiar with the appearance of the upper airway and eliminate the death zone of the respiratory tract as a matter of ordinary routine, it will be found that resuscitation apparatus whose usefulness is now limited to inhalation and transpharyngeal insufflation may readily be adapted to endotracheal insufflation.

Endotracheal Insufflation. The general acceptance of endotracheal insufflation as the method of choice for the resuscitation of the flaccid patient is inevitable. The trend in this direction as reflected in medical literature is unmistakable. The basis for this positive statement is first, the author's clinical experience with the method extending over a period of the last fifteen years, and secondly, the fact that opponents of the technique offer no objection in principle but limit their objections to the need for technical skill. The same objection might be raised with equal logic to the use of the nasal, aural, gastric, urological, vaginal or rectal endoscopy! The common employment of endotracheal insufflation, however, is more than "the general acceptance." It turns upon the recognition of the stages of asphyxia and the limitation of its employment to those cases for which it is suited.

Once more, and in a very special sense, the technique must follow the indications presented by pathologic physiology. To attempt to laryngoscope and intubate a depressed or a spastic patient is to commit a triple error:

(1) Such a patient does not require intubation.
(2) Intubation under these conditions is likely to be traumatic.
(3) The technique of endotracheal intubation is thrown into disrepute because it has been mismanaged.

On the other hand, failure to intubate a breathless, flaccid patient is not only an admission of incompetence but a failure to extend to the dying patient the additional margin of safety which may save his life.

There is no substitute for technical practice; in both cadaver and anesthetized patient both are or can be made available. It would seem only reasonable that those who assume the responsibility for bringing about unconsciousness should be equally proficient in bringing about recovery, even though this may involve resuscitation; and yet how many of those who administer anaesthetics have become proficient in resuscitation routines?

If terminal asphyxia were but a phase of some condition from which the patient might be expected to recover as a matter of course, one might well speculate as to the hazard of instrumentation. But when one considers that the flaccid, asphyxiated patient is extremely likely to die, how can the physician hesitate to act immediately with precision and with all the skill that he can bring to bear upon this amazingly critical task? The theoretical approach is sound; it violates no principles, and involves no contradictions.

Two methods of intubation are in common use: the method of blind intubation by palpation, in accordance with the technique originally employed by O'Dwyer for intubation in diphtheria; and intubation under direct vision by the use of the laryngoscope, in accordance with the technique popularized by Chevalier Jackson. The first method is advocated by De Lee, the second by the author. De Lee's technique, as described in his book,* is as follows:

"*Intratracheal method of intubation and insufflation: method of De Lee.*" "The baby is wrapped in a warm blanket. He is placed with his head over the edge of the table. The head is steadied by an assistant. After sterile water has been drawn into the catheter to prove its patency, the index finger of the right hand depresses the tongue and at its base locates the tiny slit that is the opening into the larynx. The small fibre catheter is inserted with the left hand so that its tip eventually finds itself beneath the end of the finger at the small slit opening into the larynx. Then, by slight depression of the tip of the catheter with the index finger of the right hand, the catheter readily finds its way into the opening of the larynx. The position of the catheter can easily be proved by advancing the finger of the right hand slightly into the esophagus. The catheter is inserted 1 to 2 in (2.5 to 5 cm) further, and suction is made to extract mucous from the larynx and upper trachea. If the catheter becomes plugged, as it often does, it must be removed and cleaned, by blowing through it; the catheter is reinserted into the trachea and artificial respiration again started." De Lee's description of the author's technique is so good that it is reproduced herewith.†

"From a simplified method of endotracheal anaesthesia developed and described by Paluel J. Flagg of New York City, there was evolved a technique for artificial respiration of the new-born. This method, consisting in suction, laryngoscopy, intubation and insufflation of oxygen and CO_2 under controlled pressure, was an extension of the method advocated by Meltzer in 1909 and by Chevalier Jackson in 1913. The principles are

* "Principles and Practice of Obstetrics," 6 ed., p. 275.

† "Intratracheal suction and insufflation, Flagg technique," De Lee and Greenhill, Yearbook, page 238, 1938.

"Asphyxia Neonatorum," condensed from the "Pivot on which Turns the Movement to Prevent Asphyxial Death" [*Surgery, Gyn. and Obst.*, **67**, 153–162 (August, 1938)].

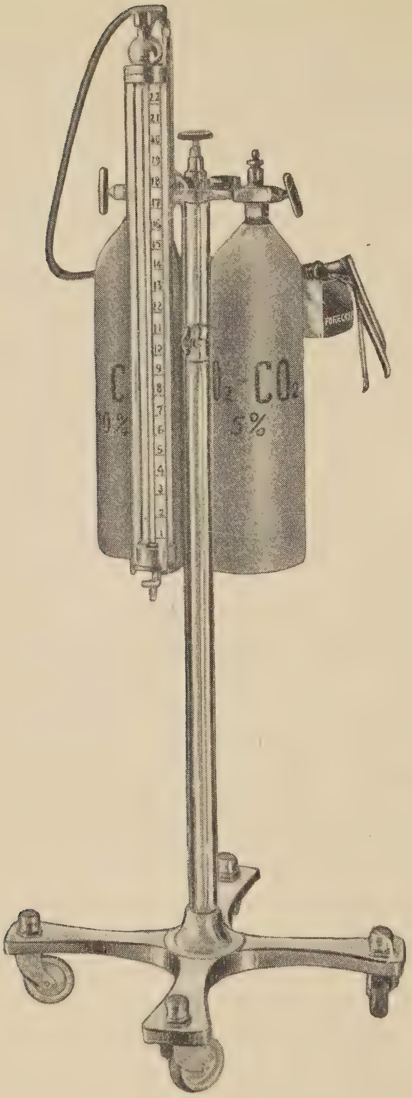


Fig. 51. Flagg apparatus for endotracheal insufflation apparatus (hospital model). (See Fig. 28, p. 66.)

From Flagg "Art of Anaesthesia"

exposure of the field (laryngoscopy under direct vision), removal of foreign matter (suction of fluid and relief of obstruction) and treatment directly to the damaged area with precision, dispatch, and absence of trauma (intubation by direct vision and insufflation of oxygen and carbon dioxide under controlled pressure).

"Routine practice in resuscitation should be carried out on a cadaver; no mutilation and post mortem signs of instrumentation interfere with subsequent autopsy findings. Intubation should not be practiced on live babies without pathological indication. Babies, to be treated for the relief of atelectasis, should be placed in the hands of experienced operators.

"An immediate prognosis of the baby's condition should be made by examining the state of the reflexes and the muscle tone. If a finger can be introduced into the mouth without resistance, exposure of the pharynx by the laryngoscope is indicated and will be non-traumatic. If on laryngoscopy there are active, swallowing reflexes and spasm of the glottis, it will be unnecessary to intubate, for the viability indicates that cerebral activity will promptly result in respiratory effort. Opening the mouth, and lifting the tongue out of the field and removing detritis in the airway provide adequate immediate treatment. If laryngoscopy reveals the pharyngeal reflex to be in abeyance and the cords inactive, the indications are introduction of a suction tube between them, endotracheal suction and then reintubation and insufflation of oxygen and carbon dioxide. In asphyxia, due to obstetric manipulation without cerebral hemorrhage or in respiratory obstruction from fluid in the airway, the reflexes return promptly; however, if asphyxia is a result of prolonged anaesthesia or of maternal medication, a longer period will elapse before the reflex returns. A vigorous circulation will promptly pick up oxygen available in the trachea and bronchi. A depressed circulation will act more slowly. Care must be exercised therefore, in providing surface heat as an initial circulatory stimulation.

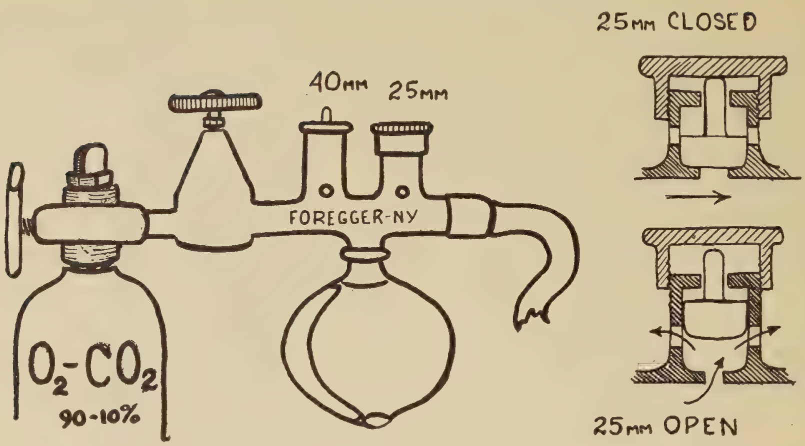
"Fluid in the pulmonary airway may be expected in Caesarian section and breech delivery. If the compression action of the uterus on the flexed chest has not functioned to squeeze out fluid contained therein, it is desirable to practice immediate aural, pharyngeal and, if relaxation permits, endotracheal suction on babies delivered by these two procedures.

"Flagg has been struck by the tolerance to instrumentation exhibited by the infant's larynx. Verified by frequent post-mortem check of the condition of the trachea and vocal cords of babies who have been repeatedly intubated and who have died of cardiac or other lesions."

Comment of editors. "Without doubt it is a scientific procedure to expose the asphyxiated baby's larynx and under the eye remove the foreign matter and then insert a catheter and inflate the lungs under controlled pressure. The objection to the method, however, rests on practical grounds. The accoucher may think it is the only successful method and may neglect to provide himself with a much simpler and much less costly ($.90) apparatus, the tracheal catheter (which will do as well). Any doctor who knows enough to put his index finger into the baby's throat and pull the epiglottis forward will be able to use the tracheal catheter successfully. He needs no assistant to steady the baby's head, and the maneuver can be done quickly before the expert laryngologist could bring the rimaglottis into view with the Flagg apparatus (cost $19.50); and when he needs to use it, he may find that the dry-cell batteries are run down."*

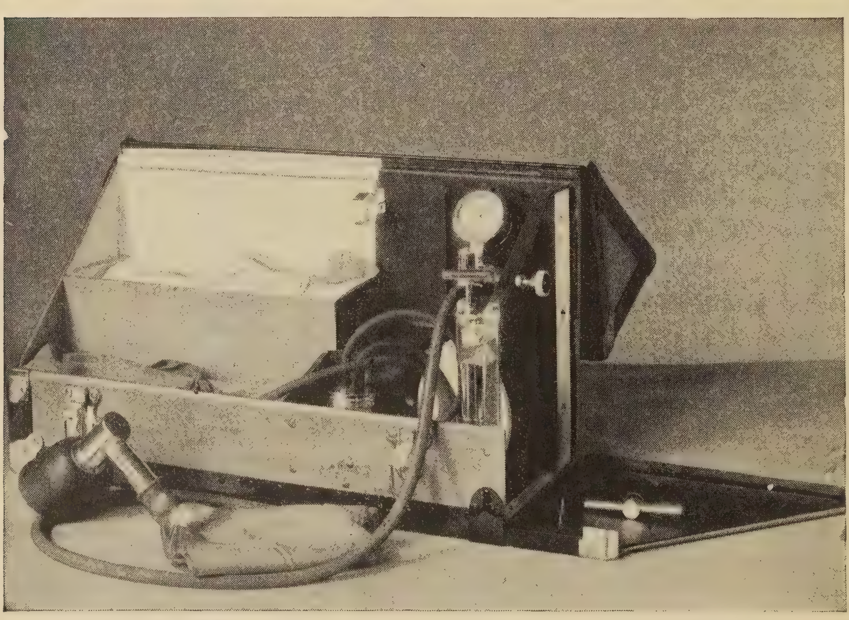
Author's Comment. "There is no objection to the accoucher providing himself with an additional catheter. A comparison of the two methods will indicate which is the more effective. The location of the infant glottic

* A laryngoscope is good for twenty years, a catheter for only a few months.



From Flagg, "Art of Anaesthesia"

Fig. 52. Portable gas supply for endotracheal insufflation, and details of weight manometers.



From Flagg "Art of Anaesthesia"

Fig. 53. Ambulance resuscitation equipment complete for inhalation, transpharyngeal, and endotracheal insufflation.

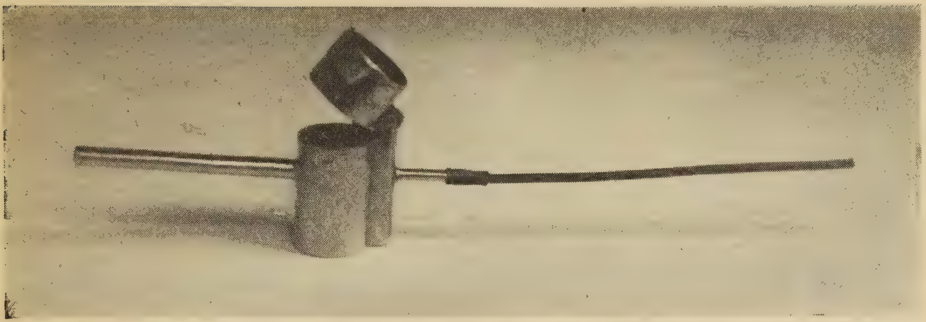
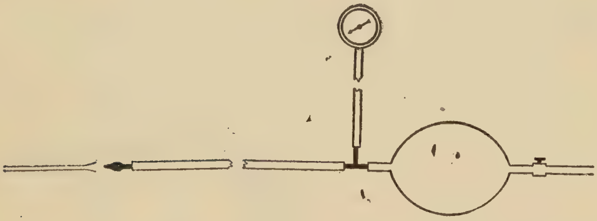


Fig. 54. Simple equipment for endotrachael insufflation of infants, showing mouth tube, weight manometer and endotracheal tube. (By Richard Torpin, M.D. Prof. Obst. Univ. Georgia.)



Courtesy The Lancet

Fig. 55. Diagram of simple equipment for endotracheal insufflation of infants. (Blaidley and Gibberd)

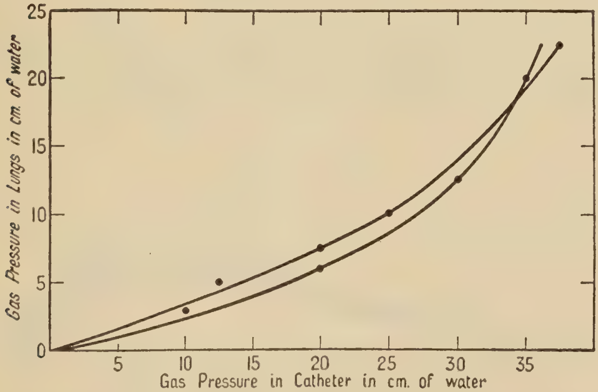


Fig. 56. The lower curve is that for a 4 lb. infant, using a No. 3 catheter; the upper curve for a 7 lb. infant, using a No. 4 catheter. (Blaidley and Gibberd)

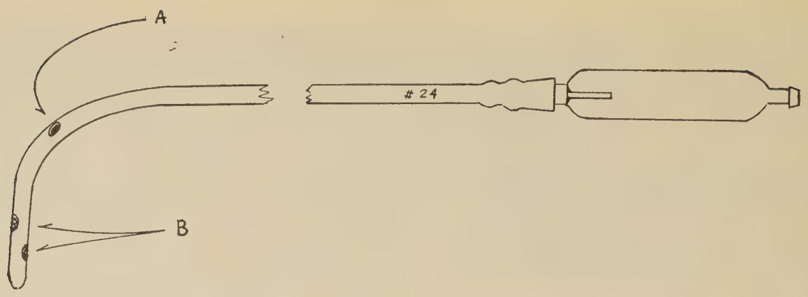


Fig. 57. Catheter with normal "eyes" closed with gauze and a new opening cut with scissors. (Courtesy of Leonard R. Thompson, American Journal of Surgery, Vol. 58, p. 140 (1942)).

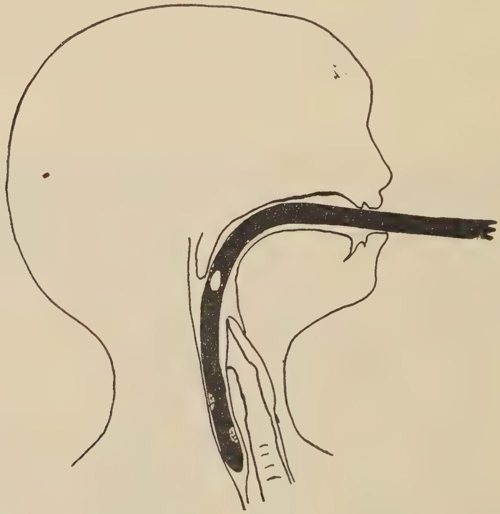


Fig. 58. Diagram shows the end of the catheter in the esophagus and the new opening in the oropharynx. (Courtesy of Leonard R. Thompson, American Journal of Surgery, Vol. 58, p. 140 (1942)). (Compare with Fig. 43, p. 74.)

Fig. 59. Infant endotracheal insufflation tube (Flagg).

aperture by palpation is similar in practice to the intubation technique of O'Dwyer. No assistant is required to hold the head, which is not extended over the table but rests upon it. A very limited practice will permit of exposure of the infant glottis within ten or fifteen seconds and intubation under vision."

Blaidley and Gibberd (*The Lancet*, March 30, 1935) devised and used a specula. "The greatest outside diameter is 1.6 cm. When we devised the apparatus and the technique described, we were ourselves unaware of Flagg's work." The technique of exposure of these operators is identical with the author's. . . . It is not necessary to pass the instrument beyond the base of the tongue in most cases, for when the tongue is pressed forward the epiglottis moves with it, and the vocal cords are exposed to view. . . . It must be remembered that the length of the trachea is short and that it is unnecessary to introduce the catheter more than an inch. (The author's insufflation tube has a shoulder which prevents more than $1\frac{1}{4}$ inches of intubation.)

While the foregoing descriptions specifically apply to the newborn infant, they have equal force in the case of the child and adult. In the latter, however, the method of blind intubation by the nasal route as advocated by Magill is occasionally used. Since, as stated by Gillespie, it does not seem to be sufficiently realized that blind intubation depends almost entirely on the sense of hearing created by active respiratory effort, and since the flaccidity of asphyxia, which indicates the use of endotracheal insufflation, eliminates the usual difficulties of direct laryngoscopy, blind intubation in the adult is of more academic than real value.

Strictly speaking, there is no apparatus for endotracheal insufflation. Such equipment is nothing more nor less than a convenient assembly of ordinary instruments for exposure, suction, and intubation, to which is added pressure safety blow-offs of appropriate design. The principles outlined may be carried out with any laryngoscope, any suction, or any suitable tracheal tube connected to any type of pressure control of the gas delivered. It is to be noted that pressure must never be permitted to build up over a period of more than five seconds. Adequate time should also be permitted to allow expiration or deflation.

The equipment shown on the following pages may very properly be added to the routine inhalation equipment now carried and employed by First-aid groups. They should be constantly available wherever negative cabinets are in use.

Laryngoscopes

The first "pocket flashlight" laryngoscope to appear in the American market was that designed by the author.* It was devised to replace the Jackson Laryngoscope for emergency work; it has been found so consistently satisfactory in the author's hands that no changes of a radical nature have been made. The presence of an acute instead of a right angle between

* *Arch. Otolaryngology*, **5**, 394 (May, 1927).

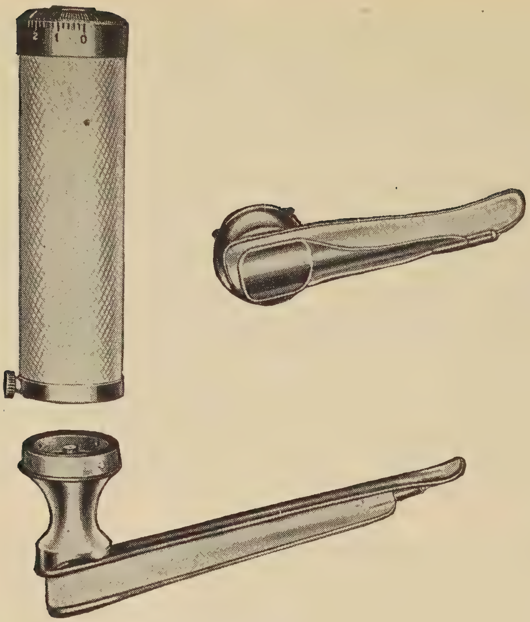


Fig. 60. The Guedel laryngoscope.
Courtesy Foregger Co.

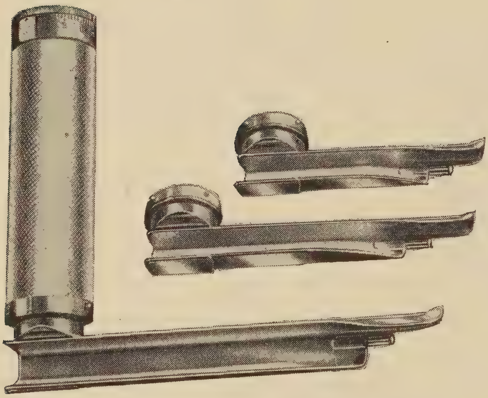
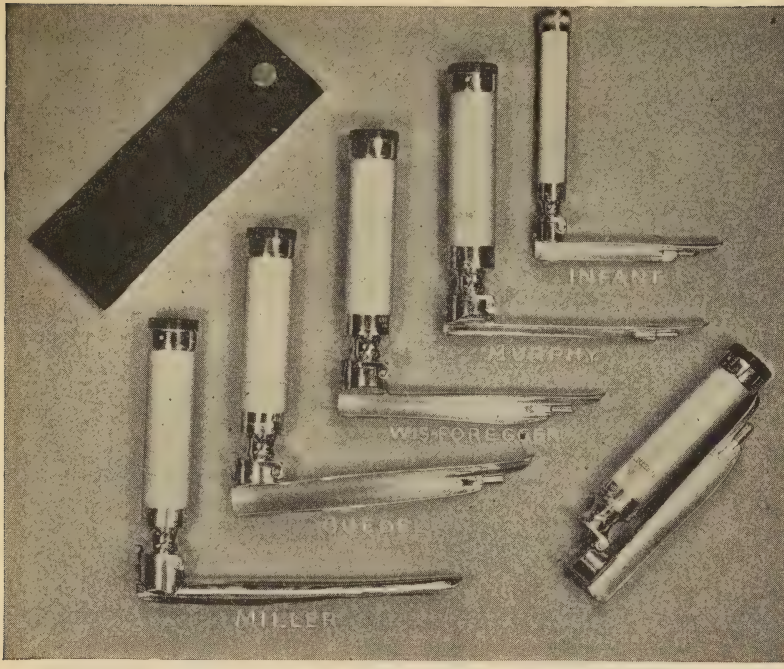


Fig. 61. The Flagg laryngoscope.
Courtesy Foregger Co.

Fig. 62. Infant folding laryngoscope.



Courtesy Foregger Co.

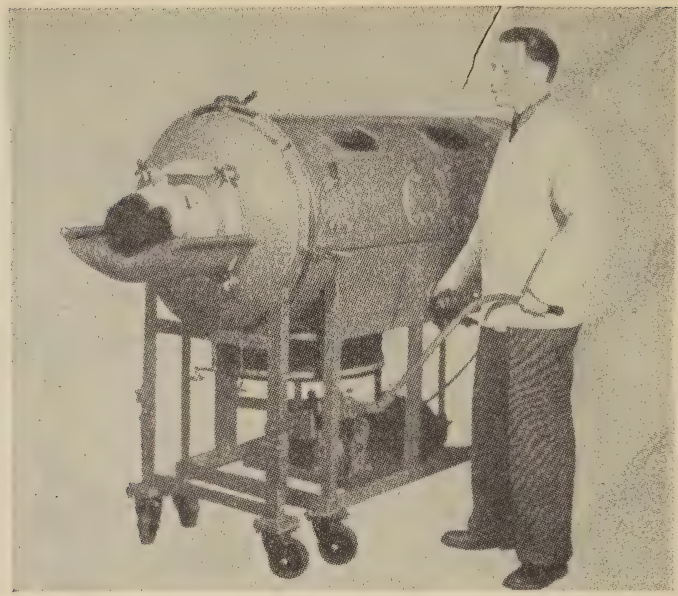
Fig. 63. Folding laryngoscopes.

the blade and the handle has seemed to be of psychological rather than practical value. Jackson in his enormous volume of peroral endoscopy has seen no advantage in such a modification. The folding laryngoscope requires a handle for each blade. Its convenience, however, has appealed to many.

Laryngoscope blades may be boiled for 5 minutes (without the light bulb) or they may be sterilized with alcohol or ether. The batteries should be carried separately. They should not be left in the handle. After a short time corrosion occurs, and it may be very difficult to extract the ruined batteries. Extra bulbs should always be available, but with ordinary care a single bulb will last for months. The light should always be tested just before laryngoscopy. A light failure when the instrument is in use is always very disturbing. The folding scope which contacts upon opening should be fully opened and checked just before use. A laryngoscope and blade may be expected to last a life time.

Extrapulmonary Differential Pressures

Under certain conditions, among which paralysis of the respiratory muscles in poliomyelitis takes first place, artificial respiration must be continued for days or for weeks. When this need arises the usual methods for performing it become traumatic. To meet the need for such prolonged artificial respiration, the negative pressure cabinet was devised. The principle involved is of the utmost simplicity; it is the exact reverse of the prone-



Courtesy Warren E. Collins Co.

Fig. 64. Drinker respirator, hand-operated.

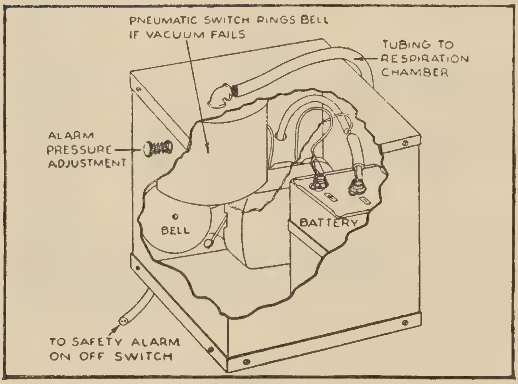


Fig. 65. Diagram of safety alarm on Drinker respirator.

Courtesy Warren E. Collins Co.

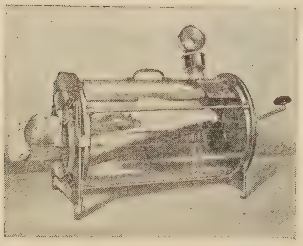
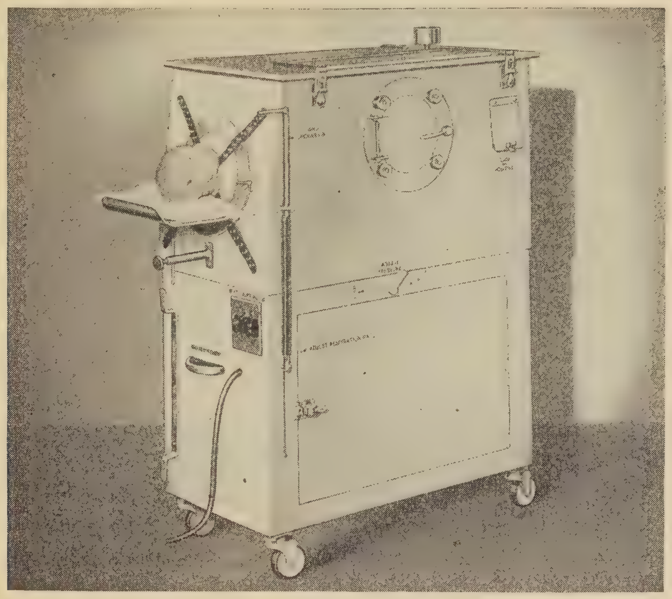


Fig. 66. Emerson respirator, transparent model.

Courtesy J. H. Emerson Co.

pressure method, which causes inspiration indirectly; which depends upon the recoil of the chest wall taking place after the operator compresses it; and which assumes that the asphyxiated, breathless patient has a chest filled with air at rest. The immediate effect of the prone-pressure method is to cause expiration. Inspiration, the vital phase, turns upon factors over which the operator has little control or information, namely, muscle tone, complete patency of the airway, and a rest period between compressions which will permit complete recoil of the chest.

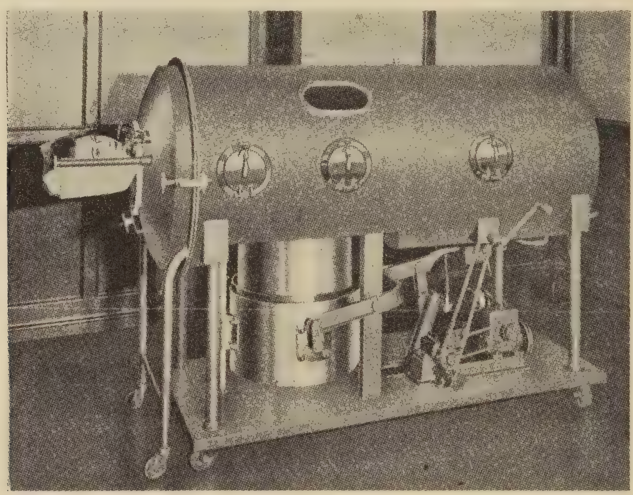


Courtesy J. H. Emerson Co.

Fig. 67. Infant model mechanical respirator.

The action of the negative pressure cabinet is direct. It causes respiration in a manner not unlike that accomplished by ordinary normal muscular efforts. This effect is produced by intermittently reducing the atmospheric pressure upon the surface of the body. The patient is placed in an air-tight box, his head remaining outside. A soft sponge-rubber collar about the neck seals this point of possible leakage of air into the box. At regular intervals a partial pressure is produced within the cabinet. As this reduced pressure takes place over the patient's body, its gas-filled cavities expand. The expansion of the cavity of the chest creates a negative pressure within it, which causes air to be sucked into it through the nose and mouth, producing inspiration. Interruption of the vacuum within the box results in collapse of the chest wall and expiration. The negative pressure cabinet therefore produces inspiration; expiration is automatic. The prone-pressure method produces expiration, inspiration being automatic.

The following important consideration is to be noted, however. By the use of the negative pressure cabinet, outside air or oxygen is added to the patient's chest capacity at rest. By the prone-pressure method the volume of the air in the chest is not increased. Its peak is at rest. Furthermore, the vital inspiratory phase produced by the negative pressure cabinet is not affected by loss of muscle tone: it will operate in complete flaccidity. On the other hand, the vital inspiratory phase will fail completely in prone pressure if muscle tone is lost. Flaccid patients who must depend only on prone pressure or upon the use of the negative pressure cabinet should be



Courtesy Columbia Steel Tank Co.

Fig. 68. Negative-pressure respirator, with liquid-sealed air pump.

placed within the cabinet with the least possible delay. Endotracheal insufflation, however, bridges the gap between the asphyxia of flaccidity and the use of the respirator, for this method is also independent of the presence of muscle tone. The widest margin of safety may be expected therefore by the routine employment of the following sequence: inhalation methods with oxygen, and CO_2 by suitable equipment in depression and spasticity, or as long as spontaneous respiration occurs, followed by endotracheal insufflation, under which treatment the patient may be transported over a period of hours*. While under endotracheal insufflation the patient is shifted to respirator *inhalation* at the proper instant. Various types of bellows have been devised to operate the respirator. Leather bellows have given way to rubber fabric and to liquid-sealed pistons.

It is clear that the suction produced by the expanding chest will not cause air to enter the lungs if the entrance is obstructed. If there is a solid obstruction, *i.e.*, soft parts of the airway, tongue, fauces, etc. the lungs will expand as do the intestines, without a change of the air within them. If the obstruction is caused by fluid vomitus or blood this fluid may be drawn into the lungs. Manufacturers recognizing these facts have recently added

* J. A. M. A., Feb. 17, 1940 (p. 602)

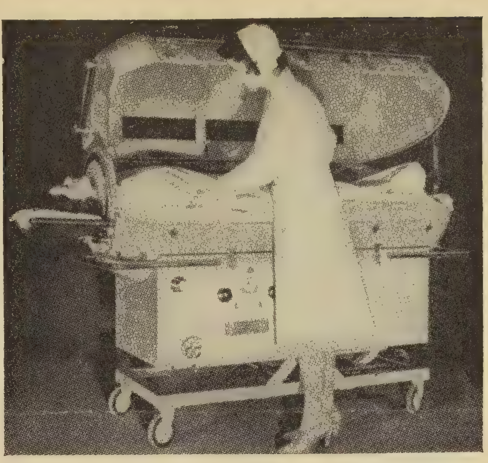


Fig. 69. Open side respirator with sealed enclosed moving parts, supplying power for additional chamber if desired.

Courtesy American Hospital Supply Co.

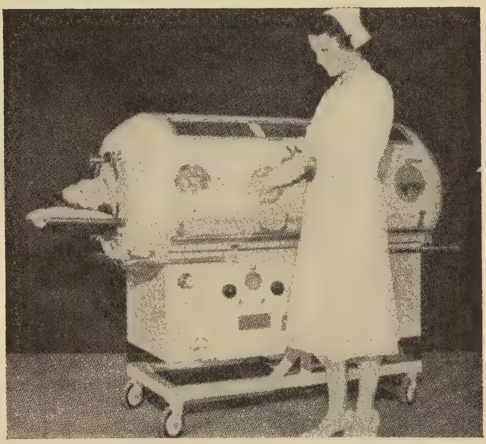


Fig. 70. Same model with the top closed.

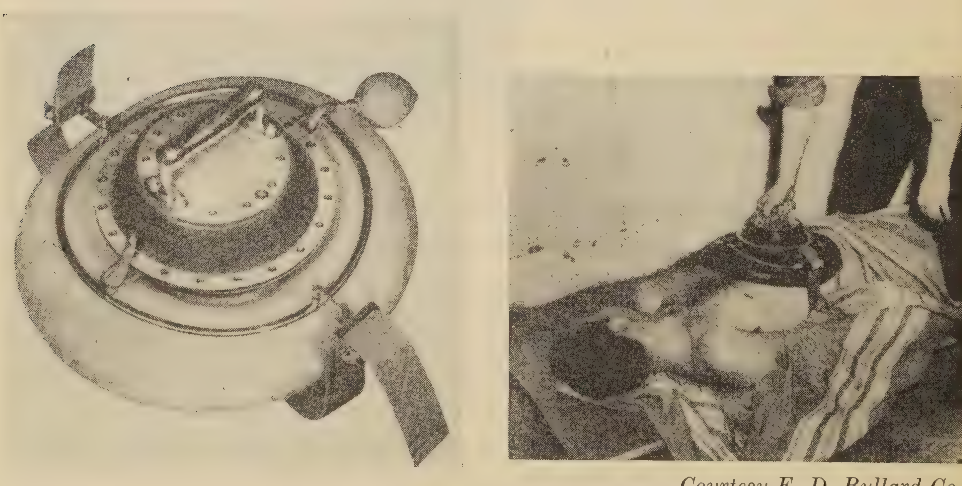
Courtesy American Hospital Supply Co.

the necessary suction to their standard equipment, as well as an automatic alarm warning attendants of air leaks in the cabinet or failure of the motor.

The negative pressure cabinet is not adapted to the needs of emergency resuscitation. To urge its routine employment in this field is to encourage morbidity and mortality. As a means of carrying on artificial respiration without trauma for days or weeks, however, this equipment is absolutely indispensable. All negative pressure cabinets should be equipped with facilities for suction, for exposure of the airway and for intubation. A trained pneumatological technician should be constantly in attendance.

Courtesy Hall Sales Co.

Fig. 71. The Teharr apparatus, for intermittent pressure applied over chest only offered as a substute for full-sized respirator.



Courtesy E. D. Bullard Co.

Fig. 72. The Rubber Lung and its application, offered as a substitute for full-sized respirator.

Equipment to Provide an Environment of Oxygen

The pleasantest method of administering oxygen, from the point of view of the patient, is to surround him with an atmosphere of oxygen at room temperature and at sea-level pressure.

This ideal is achieved by the use of the so-called oxygen tent. For very ill patients, or for those who resent the use of appliances in direct contact with the nose or mouth, the method is highly acceptable. An oxygen tent is an approximately air-tight enclosure, completely enveloping the patient as he rests in bed. The mattress and pillows on which the patient lies form the floor of the enclosure. The sides and ceiling are of rubberized fabric in which are set numerous windows of transparent, non-inflammable cellulose acetate. More recently tents have been made of Pliofilm. Tents are equipped with zipper-closed flaps permitting treatment of the patient while at the same time guarding the tent atmosphere against dilution by atmospheric air. Unfortunately, the mere addition of oxygen to the closed tent does not suffice. Shortly after the tent has been placed in position, and the sides (skirts) tucked under the mattress, the tent atmosphere becomes first uncomfortably and then unbearably hot, resembling the interior of a telephone booth with the door closed. The accumulating body heat and moisture from perspiration and exhalations must be cared for. A fan agitating the tent atmosphere relieves the situation only slightly. The solution has been met by causing the oxygenated tent atmosphere, by means of a motor equipped with a fan, to flow in and out of an air-tight box filled with ice. The ice not only cools the heated air, but causes the water vapor contained in it to precipitate on the cold surface. The level of the temperature and humidity within the tent depends directly upon the speed of the motor which controls the rate of the air circulating over the cooling ice. (Figs. 73 and 74).

Even though 8 to 15 liters of oxygen are constantly being added to the tent atmosphere, the pressure within it is never increased because of the leakage which occurs. These leaks serve to dissipate the CO_2 and body odors which tend to accumulate. Objectionable body odors may be filtered out by passing the tent atmosphere through charcoal (activated carbon); excess CO_2 may be removed by passing it over soda lime.

The principle of convection is also utilized to bring about circulation within the tent, replacing the need for a motor. This principle is based upon the fact that cold gases fall to the bottom of a container and warm gases rise. Oxygen delivered through ice is chilled; entering the tent at the bottom, it rises to the top when warmed and passes back into the ice container. (Fig 77).

Vaporized liquid oxygen circulated by a suitable fan eliminates the need of ice. The difficulty of obtaining a supply and of storing liquid oxygen, however, curtails the wide use of this method. (Fig 80).

The principle of convection is further employed in the so-called open tent. Because heat and moisture easily escape, an enclosure of reduced

Text cont'd on page 98

Courtesy Oxygen Equipment Mfg. Co., Inc.

Fig. 73. Barach Thurston Senior Oxygen Tent with Transparent Canopy.

Fig. 74. Barach Thurston Junior Oxygen Tent with Transparent Canopy.

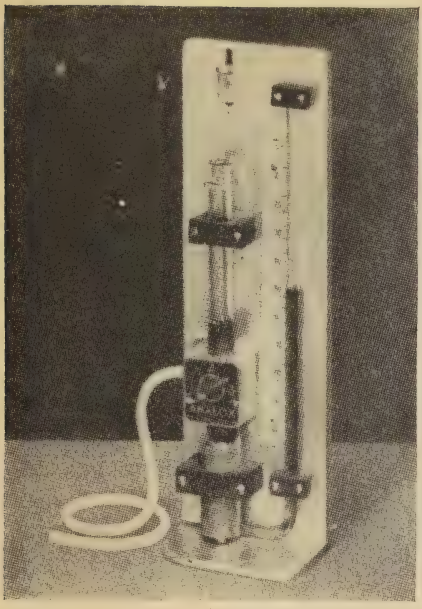
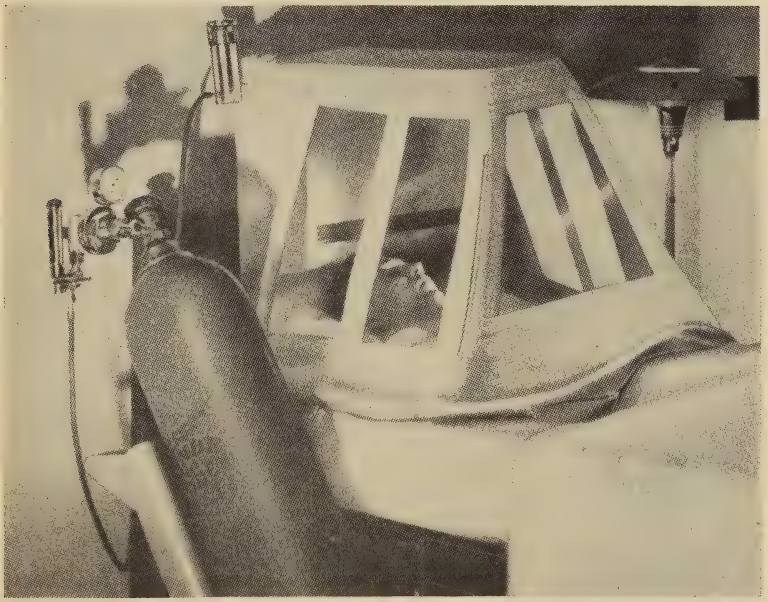


Fig. 75. Oxygen analyzer.
Courtesy American Hospital Supply Co.



Courtesy American Hospital Supply Co.

Fig. 76. Small, portable closed oxygen tent, motorless; operated by convection of oxygen.

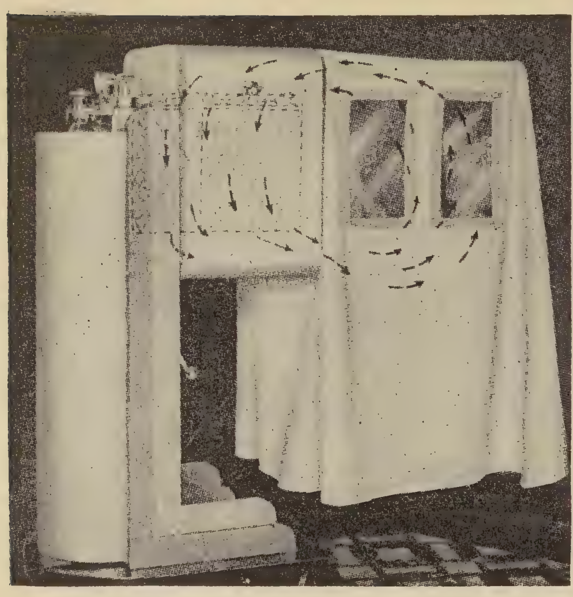
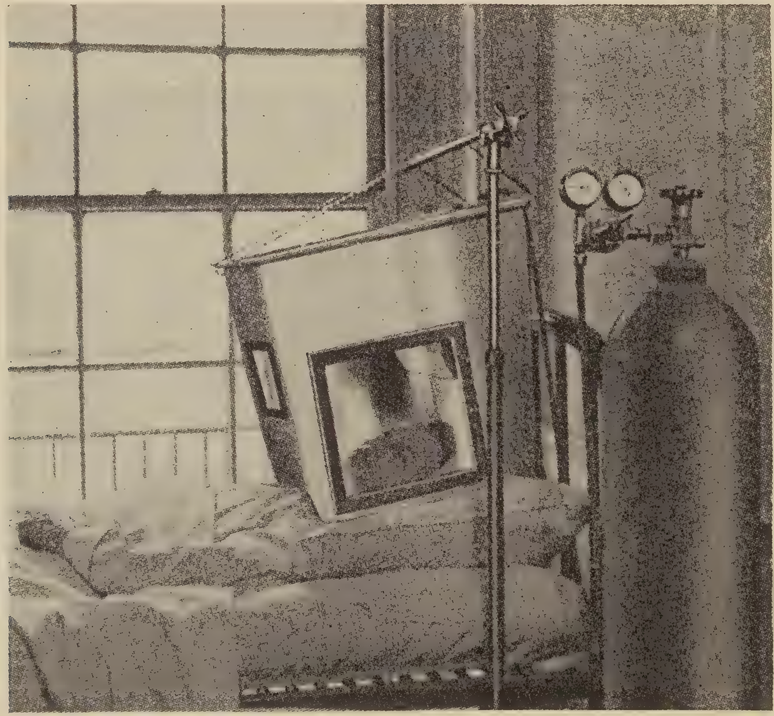


Fig. 77. Closed Oxygenaire tent, in which the oxygen circulates by convection, as shown by arrows.

Courtesy American Hospital Supply Co.



Courtesy Warren E. Collins

Fig. 78. Open-top oxygen tent.

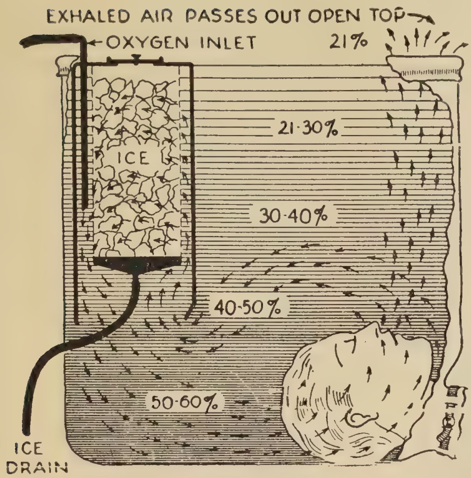


Fig. 79. Diagram of open-top oxygen tent. Note that the cooler oxygen remains at the bottom of the tent where the patient may breathe it while the warm exhalations pass out at the top.

Courtesy Warren E. Collins

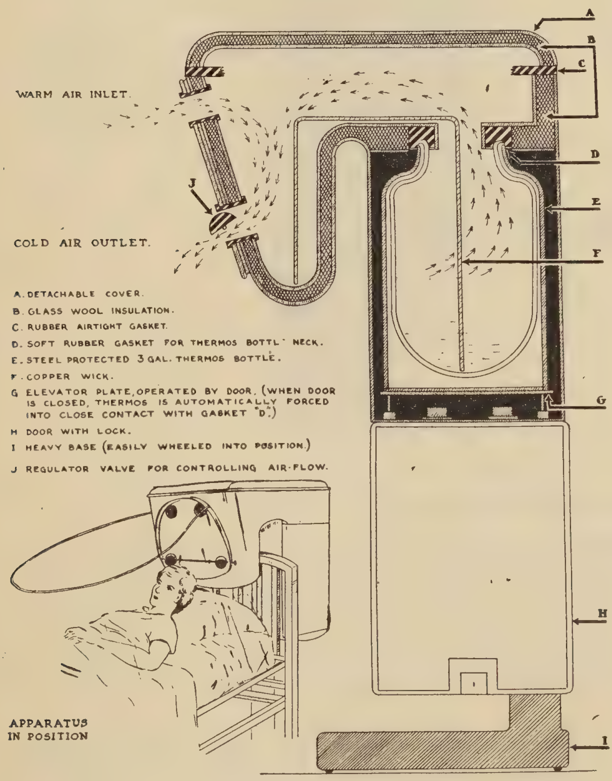


Fig. 80. Diagram of liquid oxygen tent with detachable top.

size may be employed. This method is well adapted to children and infants.

It is essential that tent atmosphere be frequently analyzed; otherwise the tent may be a liability instead of an asset. Analysis is a very simple procedure. A sample of the tent atmosphere is drawn into a glass syringe and injected into a solution of ammoniam sulfate containing copper wire gauze. The oxygen in the injected sample is absorbed by the solution, which reacts with the copper to form copper sulfate. The proportion of the total sample absorbed is indicated on a scale set like a thermometer. An analyzer is shown in Fig. 75.

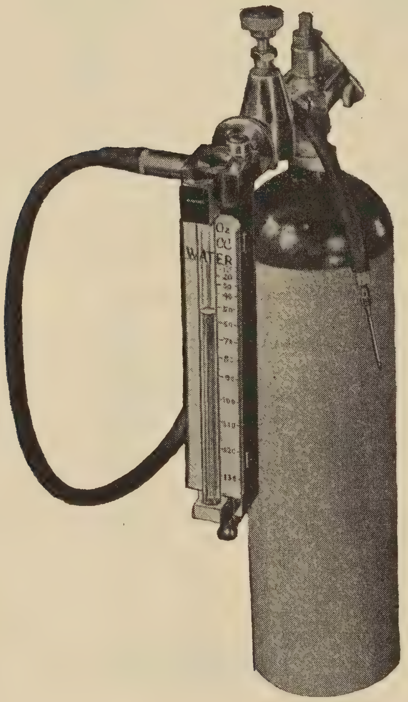


Fig. 81. Apparatus for intravenous or subcutaneous administration of oxygen.
Courtesy Foregger Co.

Subcutaneous Administration of Oxygen

In 1799 Beddoes employed oxygen for the treatment of ulcers. Bayeau* is said to have first established the use of subcutaneous administration of oxygen. Welch† injected oxygen (150–500 cc) under the skin of the outer border of the thigh on the anterior surface, three inches above the patella. Owing to the looseness of the skin in this location, injection is without discomfort.

While the impression has been created that oxygen injected subcutaneously acts directly, as does inhaled oxygen, this can scarcely be the case.

* R. Bayeau, *Compt. rend. acad. sci., Paris*, **172, 291,** 1388 (1921).
† D. C. Welch, "Subcutaneous Injections of Oxygen and CO_2," *British Medical J.*, **2,** 147 (1932).

In the first place, oxygen so injected is absorbed slowly, for the reason that the tissues in which the oxygen is injected shortly become contaminated by nitrogen and carbon dioxide gas escaping into it from the blood, plasma and tissues in an effort to establish an equilibrium in their respective gas tensions. Assuming that ordinary oxygen tissue tension is 40 mm Hg, when oxygen is injected into this tissue the tension in the injected area is raised from 40 to 760 mm Hg, minus 47 mm of water vapor, or 713 mm Hg. The CO_2 diffuses out from the tissues in a tension varying from 35 to 60 mm Hg. Therefore, immediately after injection there is about 18 times as much oxygen in the area injected as there is elsewhere in the tissues. But if after 48 hours, as appears to be the case, some of the 500 cc of oxygen injected still remains in the tissues, the rate of absorption per minute of the injected oxygen would only be about 0.18 cc (2 times 24 times 60 minutes divided into 450 cc oxygen) instead of the 250–300 cc required per minute.

However, since anoxemia is apparently relieved, as reported by numerous observers,* some other factors must operate to bring this result. Campbell and Poultron suggest the following possibilities:

(1) Reflex stimulation of the respiration.
(2) Slight mechanical injury to the tissues producing reflex effects.
(3) Slight exudate formed (necro hormones) after injection, which is absorbed into the general circulation.
(4) A psychological effect which cannot be ignored.

The chief hazard created by the method is the possibility of postponing the administration of oxygen by the respiratory route. In view of the simplicity of the technique, however, and the fact that it involves no recognized clinical hazard (save possibly aero embolism, which may be guarded against), the method may prove of value and should be employed in those cases of intrinsic pulmonary edema in which high alveolar capillary absorption is greatly embarrassed, as occurs following the inhalation of poison gases in warfare and elsewhere.

Note. I. L. Singh states that in his experiments with cats, the lungs are 15–20 times more efficient in the absorption of oxygen than is the entire subcutaneous or peritoneal surface.†

Ultraviolet Irradiation of Auto-transfused Blood

The histotoxic anoxia of chemotherapy, *i.e.*, from the cyanide ion or the sulfa drugs, certain narcotics and alcohol, in which the tissue cells are unable to make use of the abundant oxygen present, is common knowledge. The ultraviolet irradiation of auto-transfused blood as a means of overcoming histotoxic anoxia is by no means so familiar.

The startling results reported where irradiation has been employed for the treatment of patients moribund through infections have been checked

* T. S. Kirk, *British Medical J.*, 195, 196 (July, Sept., 1938).
D. DeRose, *Medical Press and Circular, London*, VXCIII, 459–460 (1912).
H. O. Howitt, *Canadian Med. Assn. J.*, (New Series), **4,** 983–5 (Nov., 1914).
F. W. Tunnicliffe, *Lancet*, 321–323 (July, Sept., 1916).
† I. L. Singh, *J. Exp. Phys.*, **22,** 193 (1932); **24,** 45 (1934).

by the utmost conservatism and the closest supervision by those responsible for the introduction of the method. This wise control, while safeguarding both patient and physician, has not been conducive to a widespread knowledge of the amazing potentialities of this new therapeutic method.

Much of the success of the method in eastern United States has been due to the wisdom, foresight, and enthusiasm of Dr. Henry Alfred Barrett of New York City. Barrett has been repeatedly impressed by the curious disappearance of marked cyanosis in patients receiving irradiation for acute infectious processes. He states that in his experience the immediate

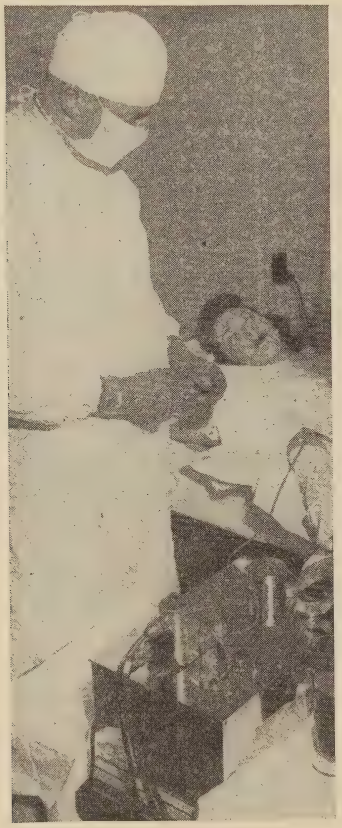


Fig. 82. Patient receiving hemo-irradiation therapy by means of the Knott Hemo-Irradiator. The water-cooled lamp which is the source of the ultra-violet energy employed in this procedure is shown connected to the hemo-irradiator by cables.
Couriesy Dr. H. A. Barrett

oxygen improvement observed is followed by a temporary relapse, which in turn is succeeded by permanent improvement. His observations are borne out by the following statement and investigations of George Miley*: "The ability of ultraviolet-irradiated blood to absorb oxygen has been studied by me, and in this work it was found that in patients with abnormally low venous oxygen values following ultraviolet blood irradiation therapy there was a marked increase in the uptake of oxygen, as shown by a definite rise toward normal venous oxygen values."

* *N. Y. State J. Med.*, **44** (Jan. 1, 1942).

Miley* found the effect of 97 ultraviolet irradiations of auto-transfused blood by the Knott technique to be as follows:

Group 1, just before irradiation and ten minutes afterward.

In 83 cases there was an average combined oxygen value of 7 per cent by volume before and 11.1 afterward, an average of 4.1 per cent by volume, or 58.6 per cent gain.

Group 2, in 14 cases just before irradiation and one-half hour afterward there was an average fall of 9.2 per cent by volume.

Group 3, in 27 cases, just before irradiation and one month after, there was an average increase of 2.9 per cent by volume, or 50 per cent over the original value.

Barrett† has suggested the desirability of investigating the value of this technique in the case of high-altitude flyers who have suffered from profound anoxia.

While at this writing treatment is carried out under hospital or office supervision, the patient suffering no constitutional reaction, further developments in technical equipment may permit the same freedom of manipulation as is now allowed by the collection of plasma for civilian blood banks.

* *Am. J. Med. Sci.*, **197**, 873 (1939).

† Personal communication.

"Inhalation Therapy," by Burgess and Saklad, J. A. M. A., Vol. 125, #7, p. 469.
Pulmonary Oedema, J. M. Carlisle, J. A. M. A., Vol. 123, Dec. 11, 1943.
Thymic Tracheostenosis, Tracheoscopy Thymectomy, Cure.
Chevalier Jackson, J. A. M. A., Vol. 58, p. 1753.

Chapter 7

Transportation of the Unconscious Patient

During the first air raids on London in 1940, the key note was speed in getting patients to the nearest hospital. The ambulance went hell-bent-for-leather, then drew up with a great show of gentleness. All that is out now. Ninety-nine out of 100 cases were shock. Realization of this came when people often died in the ambulance en route to the hospital; air-raid workers were depressed by the fact that people who had lived for hours under debris promptly died when they were "rescued." Today the A.R.P. workers are shock experts—quiet, warm blankets, hot-water bottles, morphine by the physician's orders only. Another notable change in the care of the unconscious—they are not roused—is that it is up to the physician to decide whether or not the unconscious are to be transported.*

The following are the conditions under which unconsciousness may occur in civilian practice. The indications presenting for the care of this state and the manner in which these indications may be met will now be considered.

The unconscious patient, requiring transportation, falls into one of two classes: (1) Unconsciousness due to accidents occurring in public places in which transportation is mandatory. (2) Unconsciousness of the bed-ridden patient whose removal to the hospital is indicated, but not mandatory.

Accidental unconsciousness may result from:

Acute or sub-acute asphyxiation, due to submersion, foreign-body obstruction, infections of the air-way, drug poisoning, polio, anaesthesia, etc.

Coma: uremia, diabetes, eclampsia, epilepsy.

Cerebral injuries: mania, encephalitis, Stokes-Adams disease, syphilis of the central nervous system, hemorrhage.

Traumatic: uterine, gastric, pulmonary.

Shock: from injuries or from hypoglycaemia.

Thermal: from freezing, heat stroke, high fever, or from acute injuries.

Circulatory: coronary or cerebral emboli.

In the bed-ridden, unconsciousness may result from:

Perforated gastric or duodenal ulcer, peritonitis.

* From the *Reader's Digest* for August, 1942, condensed from *Newsweek* for June 15, 1942.

Acute retention: kidney or renal lesions.

Ruptured ectopic or obstetrical complications.

Pneumonia, or other acute pulmonary lesions, including pharyngeal or laryngeal obstruction.

Cardiac decompensation from many causes.

Acute exanthemeta: including diphtheria, croup, erysipelas, or burns.

Terminal arthritic conditions.

Acute injuries of eye, ear, nose and throat.

To these general conditions, we may add the less usual cases of psychiatric patients who must be transported under full narcosis.

A total of 3 per cent of unconscious patients were admitted to the Boston City Hospital in 1933.* The conditions noted in the order of frequency made in this report of 1167 cases were as follows:

Cause	Per cent
alcoholic	59
traumatism	13
poisoning	3
cerebral vascular lesions	10
epilepsy	2.4
diabetes, meningitis, pneumonia	1.7
cardiac decompensation	1.4
syphilis of the central nervous system	06
miscellaneous	4.1

At this writing, the lack of adequate facilities for the scientific care of the unconscious patient during transit results in unnecessary morbidity and mortality. In the accidentally unconscious, this is unavoidable, and is automatically added to the existing damage caused by the accident. The bed-ridden, however, need not be deprived of the modern scientific advantages offered by the well-equipped emergency ward.

Let us consider the precise nature of this gap in medical service. In just what way is the margin of safety reduced in transit and how does the immediate morbidity and mortality take place?

We may consider the unconscious patient as a generic problem, irrespective of the specific cause of the unconsciousness. He is entitled to protection from the hazards to which his state subjects him. Accommodations to care for profound unconsciousness will necessarily include lesser degrees of this state and place us in a position to care for whatever accident we may face. As a result of unconsciousness, a patient is partially or wholly deprived of his protective reflexes, *i.e.*, swallowing, coughing, and automatic heat regulation. Vomitus, blood, or other foreign matter is likely to accumulate in the mouth or the throat. Owing to depressed pharyngeal and laryngeal reflexes, aspiration of this material is likely to occur. Such aspiration gives rise to pulmonary complications.

As the heat regulating center of the unconscious patient is out of order, drafts as well as extremities of heat and cold should be avoided. Constant

* 1943 figures not available.

temperature must be artificially maintained throughout the interval of transference to the ambulance, and during transit.

Various degrees of asphyxiation result from respiratory obstruction and a depressed respiratory center. The anoxemia which accompanies acute pulmonary or cardiac disease is to be avoided if transportation is to be for any great distance.

The care of the unconscious, necessitating as it does close attention and mechanical manipulation on the part of the ambulance surgeon, calls for adequate illumination, properly insulated. Mechanical restraint for the arms and legs and ready access to the head are essential for maniacal or narcotized patients. Hypodermic circulatory stimulation is commonly provided, but intravenous fluids are conspicuous by their absence.

The unconscious patient is a desperately ill patient. His transportation is fraught with danger. He may die on the way to the hospital. There is no good reason why routine ambulance service should not provide the protection which the unconscious patient deserves. Safety and efficiency in the transportation of the unconscious has been made secondary to comfort. The present ideal appears to be to provide an artistic, portable, hotel bathroom instead of a reasonably well-equipped, portable, emergency-treatment room. Furthermore, it appears that no item connected with the safety of the patient is important, if it interferes with the aesthetic lines of the car body. Any violations of the necessary racy sport appearance will, according to the manufacturers, add serious sales resistance. Appearance in ambulance construction takes definite precedence over medical efficiency.

However, in spite of cramped head room, the necessary protection for the unconscious can readily be added to an ambulance already in commission. It may be more economically incorporated in the chassis construction in such a manner as to interfere little, if at all, with the existing requirement of appearance. The incorporation of these features would become a practice as common as the use of brakes or windshields if the needs of the physician and the patient could be made plain to the builder of ambulances.

With a view to demonstrating in a practical manner the application of fundamental principles in the care of the unconscious patient in transit, the writer secured the cooperation of a typical suburban private hospital of 100 beds. An ordinary commercial ambulance was placed at his disposal to equip as he saw fit. Adequate care for the asphyxiated patient was the objective. Patients unconscious from any cause are adequately protected. The body construction changes and the added equipment provide the following:

Adequate suction	Equipment for oxygen therapy
Air conditioning	Adequate illumination

Ready control and access to the patient in transit
Incidental facilities for circulatory stimulation

Suction provides protection against the aspiration of vomitus by the unconscious patient, whose reflexes are obtunded. Air-conditioning provides safety from drafts and from excessive heat and cold. It provides also means for heating blankets for transference to the ambulance as well as ventilation without exposure to drafts. Equipment for oxygen therapy provides facilities for the treatment of anoxemia, not only by mask, nasal tube, oral tube, and intratracheal insufflation, but also by supplying an oxygen atmosphere within the ambulance similar to the conventional oxygen tent. Adequate illumination, properly insulated, provides a means of caring for the patient second only to that of the operating room. Restraint is supplied for the patient who is to be transported from an insane asylum or under an anesthetic. Facilities for the administration of intravenous solutions heated by air-conditioning equipment makes it possible to treat cases of hemorrhage or shock without the usual delay.

Details of Equipment

Suction: Suction is obtainable by tapping the intake manifold of the ambulance engine. The suction so produced, even with the engine idling, is sufficient to empty a quart bottle of fluid in three seconds (see Fig. 21).

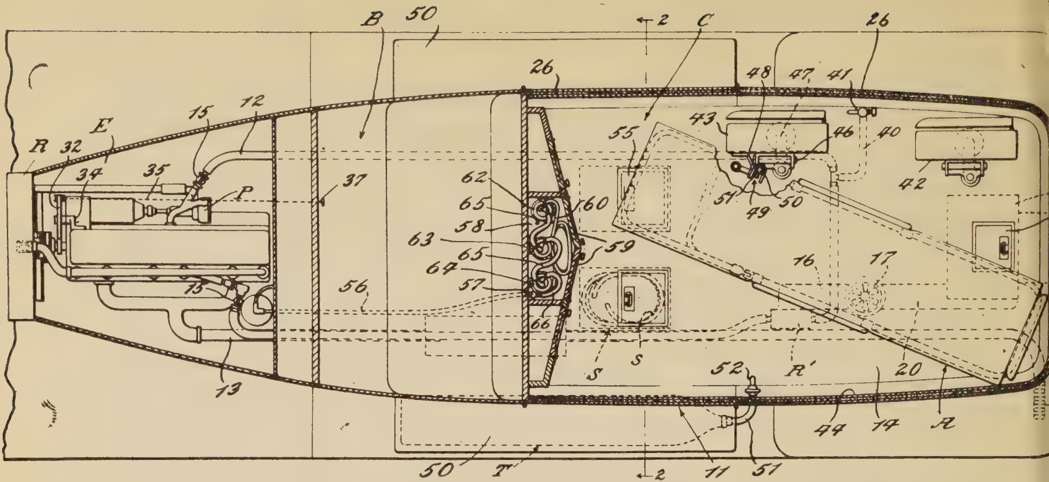
In order that this suction may become available in a patient's room, a rubber hose 100 feet in length is attached to the car suction. On his way to the patient, the ambulance surgeon carries a suction bottle and the hose. One end of the hose is dropped outside of the window adjacent to the curb where it is picked up by the ambulance driver.

Air-Conditioning: The passenger compartment of the modern ambulance may be readily and thoroughly air-conditioned. For ordinary use, exhaust of the chamber air is accomplished by a ceiling ventilator equipped by a suction fan (Fig. 84).

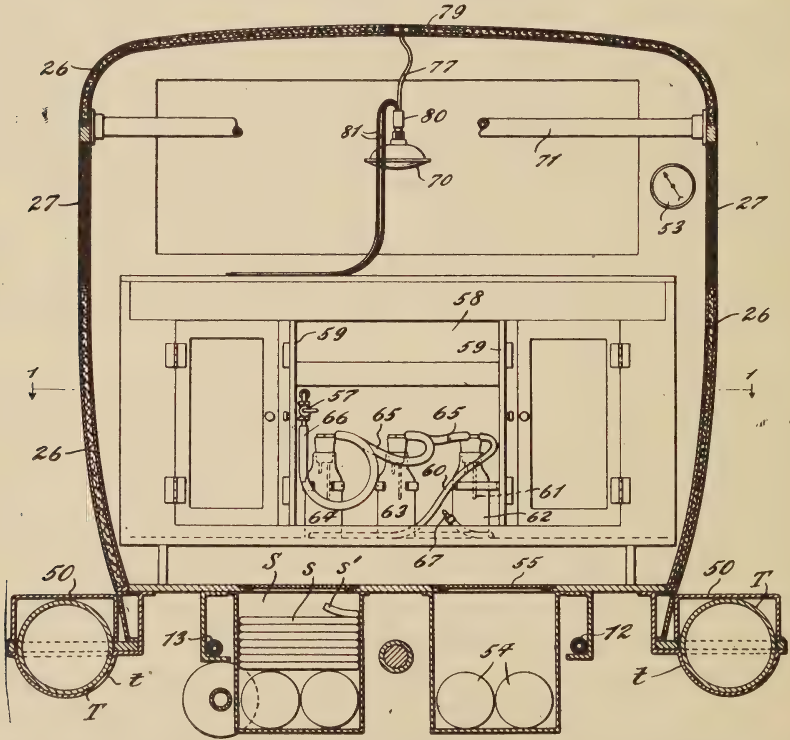
Disposal of the exhaust gases of the ambulance's own engine is secured by delivery of the gases to the side of the car, instead of to the rear or beneath, where it may be sucked into the passenger compartment by the following tail draught produced by the progress of the car.

Heat is secured by hot water circulating from the radiator, the temperature of which is raised by a radiator heating device controlled from the driver's seat (Fig. 84). Heat furnished by the automobile engine to a large floor radiator is distributed by a positive-pressure fan of conventional type. In summer a reduced temperature is secured by the substitution of ice in the heating plant. The rate of conduction of heat and cold through the plate-glass windows is reduced by a double glass with intervening air space (Fig. 83).

If it is desirable to provide an oxygen atmosphere similar to that usual in an oxygen tent, the oxygen is supplied by standard 6200-liter tanks sunk and concealed in each running board (Fig. 83). When this method is employed, the ventilator on the roof of the car is closed and the car atmosphere becomes self-contained. Humidity is controlled by ventilation over the

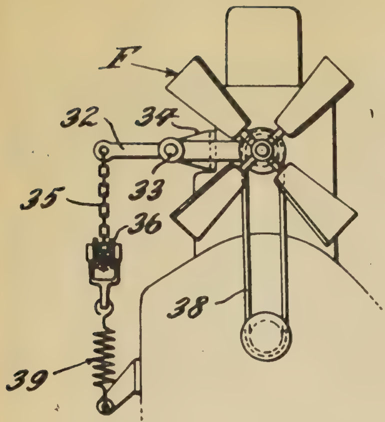


Floor plan of ambulance. Fan control to dash, 37. Suction line from intake manifold, 56, to suction bottles, 62, 63, 65. O, Tank 50T, 100 feet extra suction tubing. S.S., Floor heater. 17, Container for resuscitation apparatus. 55, Clamp for stretcher leg. 49, 50, 51, Tap for water from radiator for hot water bags. 41, Diagonal stretcher. Auto exhaust to side of car, 31.



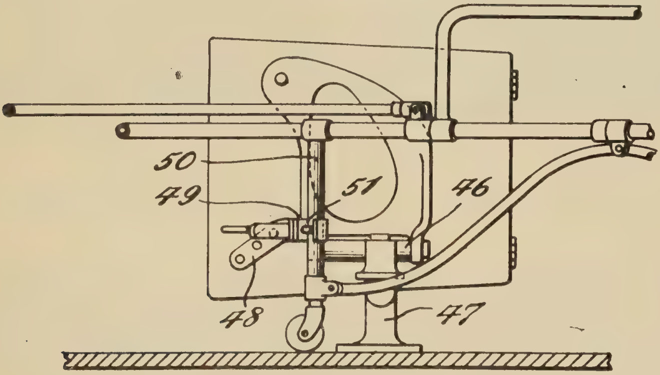
Cross-section of ambulance. 26, Insulated walls. 27, Windows, double glass. 50T, Oxygen tank under running board. SSS, container for 100 feet of suction tubing. 54, portable oxygen tanks. 55, container under floor for resuscitation apparatus. 57, suction from intake manifold with shut off. 63, 64, suction bottles to remain in car. 65, 60, suction bottle to be taken to patient with tubing SSS. 70, 80, 81, spot light. 53, thermometer.

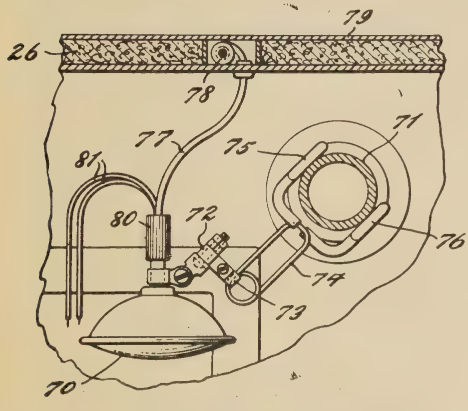
Figure 83



Fan-belt tension control to do away with radiator cover. Chain 35, attached to fan 32, pivoted at 34, passes to the dash-fan-belt, 38, slips driver's seat over pulley, 36. When chain, 35, relaxed fan-belt, 38, slips and motor heats water. When 35 is under spring tension, 39, fan rotates and water cools.

Gadget to hold stretcher in diagonal position (see floor plan). Clamp, 48, 49, 51, holds stretcher leg, 50.





Ceiling spot-light. 72, 73, 74, 75, Adjustable bracket on ceiling rod 71 running across car. 81, 77, Electrical connections. 26, 79, Insulated car walls.

21, 22, 23, Ceiling ventilator, equipped with exhaust fan, U F M, on swivel joint, 24.

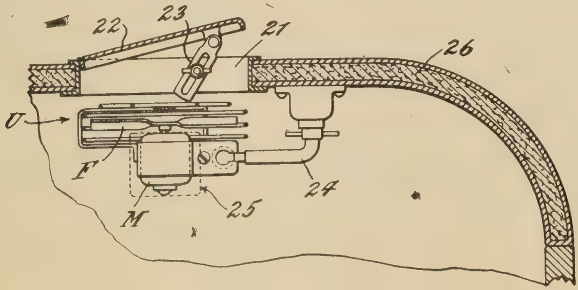


Figure 84

ice container. Oxygen concentration is secured and maintained by repeated checks made with the usual oxygen concentration analyzer.

Note: The principles involved in the air-conditioning of the automobile may be applied to conveyances used on water-ways and planes.

Equipment for Oxygen Therapy. Suitable apparatus for the administration of oxygen, by inhaler, oral tube, nasal tube, or intratracheal tube is available and should be present in every ambulance at all times. Such apparatus is now in common use. Pure oxygen, oxygen with carbon dioxide, and pure carbon dioxide in small cylinders are carried as a routine. The ambulance surgeon should be familiar with and skilled in the use of this equipment.

Illumination. Movable spotlight illumination is important. All electrical connections should be made with due regard for the fire danger present in an oxygen or an anaesthetic atmosphere. All switches should be outside the compartment. Fortunately, the oxygen fire hazard under the conditions described are minimal, as the watchful attendant, in the compartment with the patient, may immediately blanket such an ignition by opening the door of the compartment, thus destroying the concentration of the oxygen atmosphere.

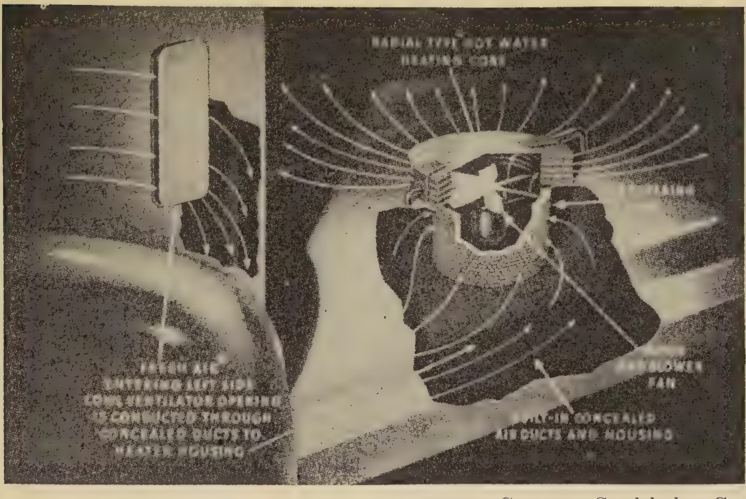
Access to the Patient. Those familiar with the care of the unconscious patient recognize the importance of ready access to and control of the head. To secure this control, it must be possible to place oneself at the patient's head. This arrangement has been made possible by placing and locking the stretcher in a diagonal position (Figs. 83 and 84).

The triangular space at the head of the patient will accommodate the seated ambulance surgeon. Suitable knee and wrist restraint increases the safety of the patient and the surgeon's control.

Conveniences for Intravenous Administration. Sterile intravenous saline solution in the usual vacuum container with needle, is readily carried in a floor compartment; but a convenient means of heating this fluid is necessary. This is provided by a tap connected with the hot-water system. Ordinary hot-water bags filled at this tap may be used to warm the patient. When the ambulance is called, blankets stored under the cot are heated by the hot-water radiator system and are ready for the patient when the ambulance arrives at its destination.

Summary. A serious gap exists in routine medical practice. Facilities for the transportation of the unconscious patient have not kept pace with modern medical progress. The desperately ill, bed-ridden patient requiring hospitalization, and those unconscious as the result of accidents find their narrow margin of safety further reduced by the lack of ordinary protection during transportation.

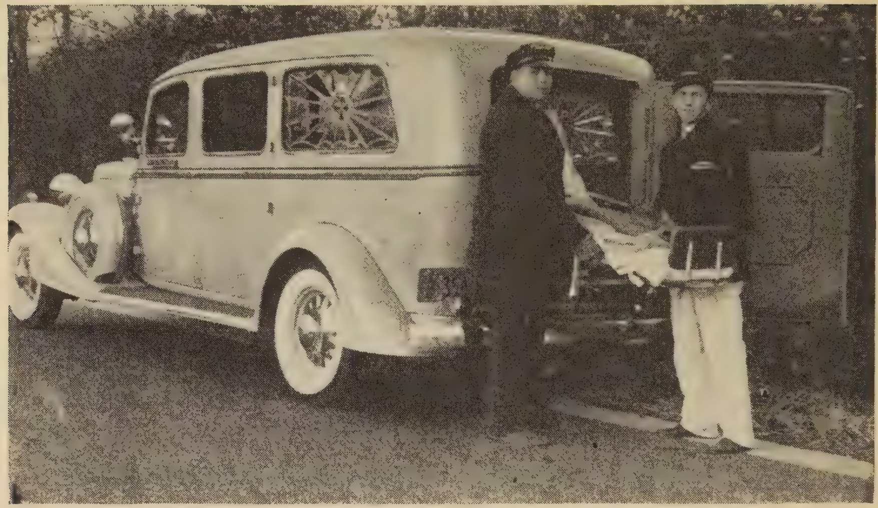
A substantial percentage of patients admitted to the general hospital service are unconscious. There is no good mechanical or economic reason why the unconscious patient should not receive the benefit of air-conditioning, suction, and oxygen therapy applied with adequate illumination,

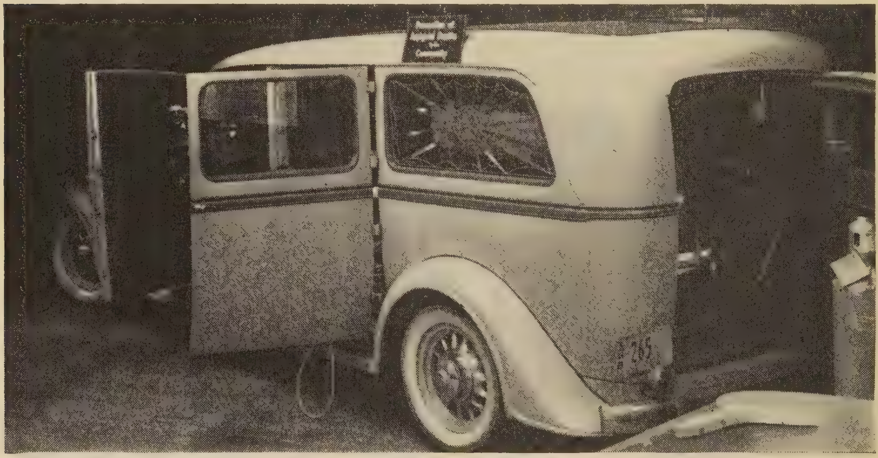


Courtesy Studebaker Co.

Fig. 85. The climatizer in action. Air enters left side cowl ventilator and passes through concealed ducts to Climatizer housing under floor. Air is filtered and then forced up through the fins of the high capacity radial-type heating core. Air emerging from core under front seat is distributed evenly to all parts of the car interior.

Fig. 86. Fire department resuscitation equipment at Omaha.





Courtesy Yonkers Professional Hospital

Figs. 87 and 88. Resuscitation ambulance exhibited at Atlantic City. See Figs. 83 and 84.

restraint, and whatever circulatory stimulation may be required. Industry may supply an articulate demand for this service.

A typical commercial ambulance correctly equipped was on exhibit at the Atlantic City Scientific Exhibit of the American Medical Association from June 10–14, 1935 (Fig. 88). Details embodying slight changes in this car with additional features not yet included have been described.

Part IV

Asphyxia as a Specific Problem

Chapter 8

Asphyxia Neonatorum

Anatomical Considerations

The head of a new-born baby of an average weight of 6 to 8 pounds (2700 to 3600 grams) is about the size of a small grape-fruit and weighs approximately as much. It is attached to the body by a relatively long, relatively thin neck which in the state of complete relaxation gives no support to the head. The normal baby's mouth will readily admit one finger, occasionally two. The pharynx in relaxation will admit the tip of the finger up to the attachment of the epiglottis. The distance from the gums to the glottis is about 2 inches, a relatively short distance. The distance between the epiglottis and the glottis is often so short that when the lip of the laryngoscope is placed against the epiglottis, it impinges against the glottis itself and obscures it. In complete relaxation, the soft parts of the airway are collapsed and as soft and adherent as the finger of a rubber glove or a collapsed toy balloon. While insufflation tends to open these passages, suction immediately causes them to collapse still further, simulating the phenomenon seen when one attempts to use a soft rubber tube on a suction instrument. The trachea is about 2 inches, from the glottis to the bifurcation. The tracheal rings are soft and easily compressible, but they maintain the lumen of the trachea under ordinary conditions. The chest wall with its intercostal muscles, accessory muscles, and the diaphragm, offer little resistance to interpulmonary gas pressure when the lungs are inflatable.

Asphyxia Neonatorum is of the greatest possible interest to the pneumatologist, for it offers an ever-present working model by which all other types of asphyxia may be approached.

Although the anatomical structures of the new-born are delicate and easily traumatized, this tissue fragility is more than counterbalanced by a resilience and a tolerance to asphyxia far beyond that which the child or the adult can endure and survive.

Furthermore, the location and the conditions under which asphyxia of the new-born occurs are under better control than is any other type of asphyxial accident. Not only are the conditions under which asphyxia neonatorum occur subject to anticipation and control, but the predisposing pathology presented by the mother issues a clear warning of the asphyxial accident about to occur.

Correct and non-traumatic treatment of the asphyxiated baby will prepare the physician to treat any other type of asphyxia with understanding and success. A swimmer pulled out of the surf in a condition of complete asphyxial flaccidity presents a condition practically identical with that of the new-born baby drowned in amniotic fluid. The attempted suicide who has taken an overdose of sodium amytal is closely akin to the asphyxiated baby whose mother has received excessive premedication. The Caesarian about to perish from an overdose of anaesthetic resembles the asphyxiated baby just delivered and about to die from the same cause.

For these reasons, State Health Commissioners will do well to encourage the use of material available for the study of asphyxia of the new-born (new-born cadaver) by the pediatricians and the general practitioners of the territory, in order that these may acquire the valuable and basic material which will permit them to take their rightful place as directors of all First-aid emergency resuscitation groups.

Asphyxia of the new-born, the most frequent cause of asphyxial death, complicates approximately one-half of the 4–5 per cent of new-born deaths.

The intensive development of the specialty of obstetrics, pediatrics and internal medicine has divided the human species into infant, child and adult groups which are so sharply circumscribed that we are inclined to forget that the infant is nothing more nor less than a very small man, that his anatomical structures are and should be in a general proportion to his size as compared to that of his mother. For example, given a woman's height and a new-born baby's length, we should by this ratio be able to form a general estimate of the size of his anatomical structures, the variations due to excessive function which occur in early life being admitted as exceptions. For example, the heart of the infant is 0.76 per cent of the body weight; that of the adult is 0.46 per cent of the body weight.

Structures having no excessive physiological activity follow a remarkably constant proportion. For example, the average length of the white baby is 50 cm (20 in); the average height of the white woman is 63 inches. The ratio of 1:3 applies to anatomical structures as generally unfamiliar as the subglottic tracheal diameter. Chevalier Jackson gives the average adult tracheal diameter as 18 mm. Gabriel Tucker states that the average full-term child presents a subglottic tracheal diameter of 6 mm, or 1:3. Jackson gives the distance from the teeth to the glottis as 15 cm or six inches, the distance from the glottis to the bifurcation of the trachea as 12 cm. One-third of these distances, namely five and four cm, would constitute a fair measurement for the average baby.

Jackson bronchoscopic tube, infant size: $3\frac{1}{2}$ mm = 6 by $4\frac{1}{2}$ mm, o.d.
4 " = 9 " 5 " "

Velvet end bronchoscopic suction tube, 3 mm.
Infant subglottic diameter, 6 mm.
Flagg infant insufflator tube, 4 mm.
Suction tube used for insufflation in prematures, 3 mm.

The ordinary respiratory rate of the sleeping new-born baby varies from 36 to 60, average 43 (Douglas P. Murphy and Edward S. Thorp). The tidal capacity varies from 17 cc at birth to 36 cc six hours after birth (Von Reus). The anterior border and apices of the lungs are the first portions to become aereated. The right ventricle of the heart is unusually large and strong. The pulse rate varies from 130 to 150. The blood pressure varies from 40–60 systolic and from 20–40 diastolic.

Pathological Physiology

We may consider the fetus as a fish, suspended in fluid, exerting an equal pressure over the surface of its body and within its open body cavities. Respiratory movement takes place, causing a gentle circulation of fluid from the amniotic bath in which the child is suspended, through the mouth and into the trachea. These movements, the precursors of those to occur in extra-uterine life, tend to maintain the patency of the airway to fluid as well as to synchronize and develop the respiratory musculature. Adequate oxygenation is provided by the placental circulation. Reifferscheidt is of the opinion that respiratory movements take place with the glottis closed, and the power of suction is so slight that the liquor amnii reaches at the utmost the nasal cavity or the entrance of the larynx. Under normal conditions the respiratory tract is always free of liquor amnii. At birth, by vertex delivery, intrauterine pressure compresses the chest and upper airway, squeezing much of the fluid present out of the mouth and nostrils. This preparation for an atmospheric environment is accompanied by varying degrees of respiratory obstruction. If the baby is vigorous and awake, as it were, that is, if he is not under the influence of an anaesthetic which has been administered to produce general anaesthesia, basal anaesthesia or asphyxiation (profound anesthesia), muscle tone and active reflexes help to keep open and clear the airway for the extrauterine respiratory effort. This effort, the accumulative effect of many repressed efforts, finally breaks through the existing obstruction and initiates subsequent rhythm. If the baby presents profound relaxation, obstruction and respiratory depression, these complications must be relieved or he will die.

Johnson and Meyers* reported a series of 500 autopsies of which 19.4 per cent showed evidence of pneumonia, 13.6 per cent of which were infected before birth. The great majority showed aspiration of amniotic material. These investigators state that autopsies should include examination of the placenta, cord and membranes. They urge that prophylaxis against infection by amniotic fluid be practiced by studying the condition of the cervix and vagina before labor. While amniotic fluid is normally present in the nose, mouth and trachea, it is not usually found in the lung. The presence of epithelial cells in the lungs indicates that aspiration has taken place as the result of intrauterine respiratory movements. Aspiration of uncontaminated amniotic fluid is not expected to produce inflammatory effects.

* *Am. J. Obst. Gyn.*, **9**, #2, Feb. 1925.

A new-born baby presents one of two extremes of reflex activity and muscle tone. It is extremely important to recognize these variations, as they form the criteria of treatment. There is no excuse to attempt to intubate a new-born baby whose head is moving about from the action of the muscles of the neck, or whose gums close upon the gloved finger of the operator. Asphyxia produces progressive relaxation and loss of the reflexes of the airway. In extreme asphyxia the gums separate without resistance, the tongue is completely relaxed, the soft palate and the pillars of the fauces are perfectly flaccid, the epiglottis drops into view, the glottis appears beneath the lip of the laryngoscope, with cords which are silent and which are separated or in contact with one another. The mucous membranes of the field are cyanotic and injected to a degree which varies directly with the vigor of the baby's circulation. A vigorous circulation gives rise to the well known asphyxia livida; a depressed circulation, on the other hand, results in the so-called asphyxia pallida.

Between the two extremes of active reflexes and muscle tone on the one hand, and complete disappearance of the reflexes and complete relaxation on the other, appears every variation. These variations depend upon the viability of the baby.

It is, therefore, practical and reasonable to make an immediate prognosis of the baby's condition by the state of the reflexes and muscle tone found upon the initial examination. If a baby permits the introduction of the finger into the mouth without resistance, exposure of the pharynx by the laryngoscope is indicated, which will be entirely non-traumatic. If upon laryngoscopy, active swallowing reflexes and spasm of the glottis occur, it will be unnecessary to intubate, for the viability present indicates that central activity will promptly result in a respiratory effort. The act of opening the mouth, lifting the tongue out of the field, and removing detritus in the airway provides adequate immediate treatment for the baby.

On the other hand, if laryngoscopy reveals the pharyngeal reflex to be in abeyance and the cords inactive, the indications are to introduce a suction tube between them, to practice endotracheal suction, and to follow this by re-intubation and insufflation of oxygen and carbon dioxide. In the presence of an active circulation, cyanosis, which may be present upon intubation and which is seen as lividity in the mucous membranes, ecchymosis of the skin or cyanosis of the extremities will promptly disappear upon insufflation of oxygen. It will be replaced by a pink color, and the reflexes which were in abeyance will reappear.

In asphyxia due to obstetrical manipulation without cerebral hemorrhage, or in respiratory obstruction from fluid in the airway, the reflexes return very promptly. However, if the asphyxia is the result of prolonged anaesthesia or of medication to the mother, a longer period will elapse before the reflexes return. A vigorous circulation will promptly pick up oxygen available in the trachea and in the bronchi. A depressed circulation will react more slowly. Care must be exercised, therefore, to provide surface heat as an initial circulatory stimulation.

The rhythm of respiration is the result of a highly complex biochemical reaction which is complicated by asphyxial factors. It will vary in accordance with the freedom with which each individual respiratory effort takes place. If the airway is absolutely unobstructed, as is the case when the endotracheal insufflation tube is in place, returning respiratory effort will be noted as an extremely shallow, but regular, effort occurring as rapidly as, or more rapidly than the pulse rate. As the vigor of the respiratory effort increases, the depth of each respiration will increase and the rate will be diminished; if the respiration is obstructed, these initial efforts will be obscured until the summation results in a forceful gasp, a final effort to overcome existing obstruction. When it is realized that adequate oxygenation of the circulation may readily be carried on for hours by means of endotracheal oxygen without any respiratory efforts whatsoever, the effort to establish an artificial respiratory rate and rhythm will be of secondary importance. The objective in every case is to support the returning voluntary respiration without in any way interfering with it by attempting to supplant an artificial rate and rhythm. The operator attempts, as it were, to feed the returning respiration with the oxygen which is required to resuscitate the respiratory center.

If, however, there is no respiratory effort whatever, one is justified in attempting to initiate such effort by stimulating the Hering-Breuer reflex by the use of maximum pressure (25 mm Hg). Following this initial stimulation, the technique described may be followed to advantage.

The return of normal vigorous respiration will be accompanied by muscular movement of the extremities and movement of the muscles of the face. The baby's head will begin to move from side to side. After normal respiration through the tracheal tube is permitted for a short period, the tube may be removed, whereupon the baby will begin to cry.

Prolonged postoperative treatment of the new-born due to drug depression or other causes when the respiration has been demonstrated to be free, is best accomplished by means of an oxygen chamber suited to the baby's size and equipped with the necessary heating and ventilating devices.

Prognostic Signs of Impending Asphyxia during Delivery

The heart is slowed; bradycardia occurs between pains during labor. There is irregularity of the heart sounds, loud umbilical souffle, and excessive movements of the baby in utero. Mecomium is seen as a result of increased peristalsis due to asphyxia. There is twitching of the visible and palpable scalp covering the skull (spastic contractions).

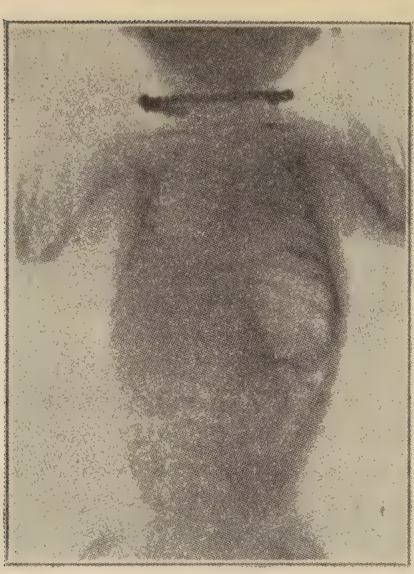


Fig. 89. Complete atelectasis of left lung in infant 61 lbs. 15 oz.

Fig. 90. Diaphragmatic hernia, left. Intestines in left chest. Infant, 6 lbs. 4 oz.

Causes of Asphyxia of the New-Born

Anoxic:
- Aspiration of amniotic fluid and debris
- Atelectasis
- Enlarged thymus
- Tracheo-bronchial fistula
- Pathologic abnormalities of glottis, web, exaggerated infantile type
- Pneumonia (Fig. 93)
- Diaphragmatic hernia (Figs. 90 and 91)

Stagnant Anoxia:
- Cerebral hemorrhage
- Cardio-vascular congenital lesions (Fig. 92)
- From analgesics and anaesthetics
- Intrauterine pressure interfering with placental circulation
- Premature rupture of membranes (causing a disturbance in the ordinary intrauterine hydrostatic pressure resulting in a disproportionate pressure on the presenting vertex)
- Premature and excessive use of pituitrin
- Post-delivery exposure

Anemic Anoxia:
- Maternal hemorrhage
- Cord pressure
- Premature separation of placenta

* Illustrations from Dennen, E. H., "Cyanosis of the new-born," *Am. J. Abs. Gyn.*, **30**, No. 1, p. 147 (1933).

Fig. 91. Diaphragmatic hernia after operation. Patient recovered.

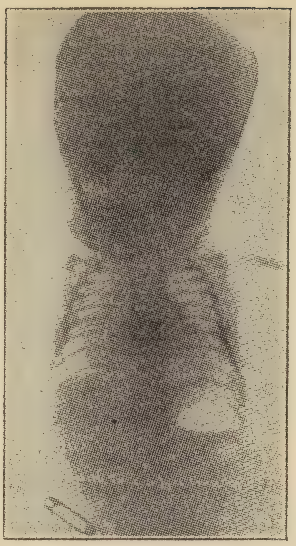


Fig. 92. Congenital cardiac lesion in 6-lb. infant. Post mortem showed three chambers in right heart, rudimentary pulmonary artery, patient foramen ovale.

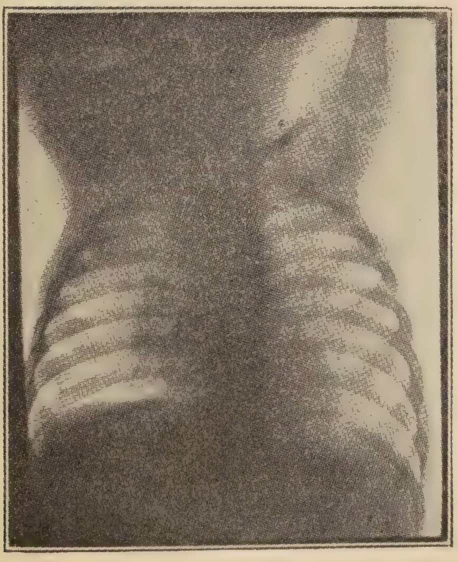


Fig. 93. Pneumonia in 8-lb. infant. Consolidation in upper right lobe; patchy consolidation in right root.

Differential Diagnosis

A definite diagnosis of congenital cardiac lesions or gross lesions of the circulatory system may be made by the infant's reaction to the endotracheal insufflation of oxygen. The cyanotic baby is insufflated and intubated in the usual manner. If the color remains constantly dusky, or fails to clear completely in the presence of endotracheal oxygen upon pressure, a portion of the circulation leaving the right heart is finding its way directly into the arterial system. We see this in a patent foramen ovale and in a congenital short-circuting of the large blood vessels. The reason for this phenomenon is that a definite portion of the circulating blood has failed to pass through the lungs and is contaminating the oxygenated stream. Neonatal atelectasis, cerebral trauma, as well as persistent thymus and even the rare, tracheo-esophageal fistula, fail to give this unique reaction. In each case, adequate oxygen insufflation temporarily but completely clears the color of the circulating blood. A tracheo-esophageal fistula may be suspected when there is a persistent discharge of fluid or froth through the glottis following satisfactory initial intubation, suction and an apparently clear respiratory tree.

Fluid within the pulmonary airway may be expected in Caesarean sections and in breech deliveries as the compression action of the uterus upon the flexed chest has not functioned to squeeze out fluid contained therein. It is therefore desirable to practice immediate oral, pharyngeal, and, if relaxation permits, endotracheal suction of babies delivered by these two procedures.

A diagnosis of cerebral hemorrhage is based upon persistent respiratory depression, regional flaccidity, or blood in the spiral fluid.

Enlarged thymus will give symptoms of tracheal obstruction when the head is extended. In this position the trachea is compressed between the thymus and the sternum. Diagnosis is confirmed by x-ray. Atelectasis may be suspected when there is depression of the lower costal margins during inspiration, intermittent spells of cyanosis, especially in the premature, which clear up with spells of crying, but reappear upon quiet breathing. Diagnosis may be confirmed by x-ray. This is caused by cohesion of soft moist surfaces by obstruction higher up, the left lung being most frequently involved. Post mortem distinction of true atelectasis involves the presence of cylindrical epithelium lining the alveolar spaces. When air has entered these spaces the epithelium becomes flattened. This is characteristic of the premature lung which is atelectatic.* Glottic obstruction is characterized by dysponea, accompanied by a sucking in of the supra-sternal and supra-clavicular regions. Diagnosis is confirmed by endoscopy and intubation.

The foregoing anatomical considerations and pathologic physiology are common knowledge included in the curriculum of the medical schools of this country and abroad. It is therefore of the greatest importance and

* Forbes and Wilson, *Am. J. Diseases Children*, **46**, 590 (1933).

interest to try to determine common practices flowing from this common knowledge. Just how are the medical schools dealing with the problem of asphyxia neonatorum?

To this end, in accordance with the recommendation of the Committee on Asphyxia of the American Medical Association, a survey was begun in 1938 to determine the policy and the practice which the obstetrical teaching centers of the United States and Canada employ in the treatment of asphyxia neonatorum. Contact was made, carried on and completed as follows:

A copy of the last annual report was secured from every medical school.

A personal letter was written to the Professor of Obstetrics of each school requesting the following information:

(a) A review of methods now in use in the treatment of asphyxia neonatorum.

(b) A statement relative to research under way.

(c) A reaction to a suggested outline proposing a new classification of the stages of asphyxia and the treatment indicated (pp. 124, 125) was sent to the Professor of Obstetrics of the following medical schools. On page 122 an outline is suggested for those who wish to assemble accurate information relative to resuscitation.

Albany Medical College
Univ. of Alberta Faculty of Medicine
Baylor Univ. College of Medicine, Texas
Boston Lying-In Hospital
Boston Univ.
Univ. of Buffalo, School of Medicine
Univ. of Cincinnati College of Medicine
Univ. of Colorado School of Medicine
Dalhousie Univ., Faculty of Medicine, Halifax, N. S.
Duke Univ. School of Medicine
Emory Univ. School of Medicine
College of Medical Evangelists
George Washington Univ. School of Medicine
Georgetown Univ. School of Medicine
Univ. of Georgia School of Medicine
Univ. of Illinois College of Medicine
Indiana Univ. School of Medicine
State Univ. of Iowa College of Medicine
John Hopkins Univ. Medical School
Univ. of Kansas
Long Island College of Medicine
Louisiana State Univ. Medical Center
Marquette Univ., School of Medicine, Indiana
Univ. of Maryland, School of Medicine
Meharry Medical College, Tennessee
Univ. of Michigan, Medical School
Univ. of Minnesota, Medical School
Univ. of Mississippi, School of Medicine
Univ. of Montreal Faculty of Medicine
Univ. of Nebraska College of Medicine
New York Hospital
New York Univ., College of Medicine
Univ. of North Dakota School of Medicine
Northwestern Univ. Medical School, Illinois
Ohio State Univ., College of Medicine
Univ. of Oklahoma School
Univ. of Pennsylvania, School of Medicine
Univ. of Pittsburgh (E. S. Magee Hospital)
Jefferson Medical College of Phila. (Penn. Hospital)
Queens Univ., Faculty of Medicine
Univ. of Rochester, School of Medicine
Univ. of Southern California, School of Medicine
Medical College of the State of South Carolina
Univ. of South Dakota, School of Medicine
Stanford Univ., School of Medicine
St. Louis Univ., School of Medicine
Syracuse Univ., College of Medicine

Temple Univ. School of Medicine, Pa.
Univ. of Toronto Faculty of Medicine
Tufts College Medical School, Mass.
Tulane Univ. of Louisiana School of Medicine
Univ. of Utah School of Medicine
Vanderbilt Univ. School of Medicine, Tenn.
Univ. of Vermont College of Medicine
Univ. of Virginia, Dep't of Medicine
Medical College of Virginia
Wake Forest, College School of Medical Sciences
Wayne Univ., College of Medicine
Univ. of Western Ontario, Medical School
Univ. of Manitoba Faculty of Medicine (Winnipeg General Hospital)
Univ. of Wisconsin Medical School
Woman's Medical College of Pennsylvania
Yale Univ. School of Medicine

Resuscitation Study Report

Note: A routine toilet of mechanical suction should be established. Suction should be sterile and should be available for use upon delivery of the head. A velvet-eyed suction tube should be provided for this purpose. Suction should precede attention to the cord, the eyes, etc., and should be applied as soon as the mouth is freely accessible.

Maternal Record

Date: Time of delivery: a.m. p.m. Para

Complications of delivery:	Medication:
	Delayed labor:
	Operation: Type:
Type of delivery:	Vertex:
	Breech:
	Caesarian:
	Miscellaneous:

Baby Record

General appearance at instant of delivery:	Respiration:
	Color:
	Heart action:
	Movements:
	Extremities:
	Face:

Treatment

Apparatus or manual methods used:		
Indications for treatment:	Relaxation:	1 2 3
	Reflexes:	1 2 3
	Apnoea:	1 2 3
	Cyanosis:	1 2 3
	Circulation failing:	1 2 3
Physical signs before treatment:	Color:	
	Lividity:	1 2 3
	Pallor:	1 2 3
Respiration: Respiratory effort:	Present	
	Frequency	
	Absent	
Is respiratory act effective:	Yes	No

Reflexes: Is jaw relaxed: Yes No
Are facial muscles relaxed: Yes No

Heart: Apex beat: Present Absent
Cord pulsation: Present Absent
Rate:

Exposure (peroral endoscopy and laryngoscopy):
Color of lips:
Spasm of masseters:
Movement of facial muscles:
Fluid in hypo-pharynx:
Appearance of glottis:
open:
closed:
Vocal cords: Not working:
Working:

Suction (amount and character of fluid in):
Hypo-pharynx
Trachea
Source of suction: Operator's mouth—hand—electric—water—steam

Intubation (instrument used): Suction tube
Insufflation tube
Active
Cord reflex Sluggish
Absent
Do cords close about tube: Yes No
Does glottis remain open on withdrawl of tube: Yes No
Insufflation: Yes No
Pressure employed:
mm Hg
Inches of water
Duration of pressure:
Seconds
Tube removed: Tube reintubated
Yes
No
How long
Total time of treatment
Minutes

Results of treatment: Sequence of method or treatment used:
Respiration established:
Time in minutes:
Character:
Color:
Muscle tone:
Reflexes:
Crying:

Remarks

Notes: The interne should differentiate between:

1. Asphyxia from central depression with its tendency to pallor in which the circulation is failing and in which the respiratory act, while complete, is ineffective because of its infrequency and small total volume;
2. Asphyxia from obstruction in which the circulation is well maintained, but owing to fluid in the pharynx and trachea or to a glottis which has failed to open, the respiratory act is spasmodic, incomplete and entirely ineffective because of the mechanical obstruction present.

Artificial respiration must be continued until circulation fails. Gradual failure of the circulation will be noted as the color begins to fade and anoxemia develops in the presence of oxygen insufflation.

The various causes of asphyxia neonatorum should be borne in mind, for example, thymus pressure, central pressure or depression, congenital abnormalities of the heart, aspiration and atelectasis.

One of the best diagnostic signs of cardiac abnormality is the continuation of anoxemia in the presence of intratracheal insufflation of oxygen, the apex beat remaining strong.

When the baby begins to breathe, try to cooperate with his respiratory effort and to fall in with his rhythm. He can breathe very freely through the intratracheal tube and, as he does so, he will inhale oxygen and CO_2 even though the pressure vent is wide open.

When the glottic reflexes return, the tube may be withdrawn. The glottic reflexes usually presuppose and are usually accompanied by a functioning respiratory center.

Your report at this time is important since the information which you will gather is pioneering in a new field and will be scrutinized with interest and appreciation.

Physical Signs Accompanying Stages of Asphyxia

The following stages of asphyxia are recognized as applying to asphyxia of the new-born:

(1) Stage of Depression.
(2) Stage of Spasticity.
(3) Stage of Flaccidity.

Depressed:

Baby does not breathe well.

Tendency to duskiness or recurring cyanosis.

Respiration free, but slow and irregular.

Spastic:

Irregular, gasping or shallow respiration occurring at long intervals.

Marked cyanosis of mucous membranes, with blotching of skin or general pallor.

The baby's gums close on the gloved finger tip.

Reflex reaction to suction of the pharynx, such as movement of facial muscles or extremities.

If pharynx is exposed, pharyngeal reflex is sluggish or active and the glottic reflex is active.

Froth or fluid is present in the mouth and pharynx.

Flaccid:

Respiration occurs at long intervals, or cannot be demonstrated.

Cyanosis or pallor.

Complete flaccidity of the musculature; all muscle tone gone.

Jaw completely relaxed.

No resistance to suction or exposure of the pharynx.

Fluid is found in the hypo-pharynx.

Apex beat may or may not be demonstrable.

Indications for Relief

On delivery, routine toilet should be practiced, including immediate suction, correct posture, and heat to the body.

Depression: Administration of oxygen and CO_2 pending confirmation of diagnosis of the cause of depression. Supply heat to the body and maintain correct posture.

Spasticity: Relieve obstruction to free respiration by suction and otherwise. Provide oxygen and CO_2 so that it may reach the glottis. Apply artificial heat and postural treatment.

Note: Artificial respiratory obstruction may be easily induced by the slightest pressure on the baby's face which tends to depress the lower jaw.

Flaccidity: Eliminate complete respiratory obstruction which has occurred as the result of flaccidity of airway by lifting the tongue from contact with the soft palate and pharynx. Examine the pharynx, and remove fluid present by suction. Examine the glottis. If the vocal cords are relaxed and silent, introduce suction tube. Follow tracheal suction by insufflation of oxygen and CO_2 under controlled pressure for measured periods. On the first appearance of spontaneous respiration, follow this newly established respiratory rhythm with stimulating doses of oxygen and CO_2 through a tracheal tube. When reflexes return, discontinue insufflation and provide oxygen and CO_2 by inhalation, employing correct posture and heat. Insufflation may be accompanied by hypodermic or intravenous medication, directed to stimulation of the respiratory center.

The details of this report which will be of particular interest to obstetricians and pediatricians will be found in appendix II.

Comments

The material in Appendix II is most significant because of the extraordinary educational influence which it represents. The directors of the leading obstetrical services in the United States and Canada have, by their thoughtful response to the queries addressed to them, indicated a nationwide interest in the problem of asphyxia neonatorum. The author, in assembling and presenting the material, is merely correlating the position of leading teaching centers on the subject. He has in this capacity attempted to report the statements of record without unduly emphasizing those factors which might be of particular interest to him.

In commenting upon the conclusions to be drawn, he has attempted to bring to focus points of acceptance and difference which are sharply marked. He has taken the liberty of adding notes based on his clinical experience in this field.

Returning to the general outline as a focal point about which conclusions may conveniently be assembled, the following facts may be noted:

The outline presenting the stages of asphyxiation, including the physical signs characteristic of each stage, has been found acceptable by more than

80 per cent of the universities contacted. In view of this approval and acceptance by the leading medical centers of the country, is it too much to hope that the medical literature dealing with this subject may adopt this classification for common usage?

Turning to the indications for and the specific method employed for the treatment of each stage, we find that the common practice exhibits differences of opinion. The first and the most important point of discussion is in connection with the third stage of asphyxia, flaccidity.

The point is taken by some that the stage of flaccidity formerly referred to as asphyxia pallida is primarily shock, which may have no relation to asphyxiation, but is closely associated with cerebral hemorrhage. Perhaps this point may be illuminated by proposing the question "What physical signs follow the stage of spasticity?" In asphyxia from other etiological causes, the picture presented by the stage described as flaccidity supervenes. We find this in asphyxia from submersion, from anaesthetic accidents, etc. The test of this condition lies in treatment. The writer has personally treated the flaccid new-born infant with nothing but artificial respiration by endotracheal insufflation, relieving the condition and rescuing the child.

Closely allied to this problem is that of cerebral hemorrhage in the newborn. Many schools favor the theory that cerebral hemorrhage is caused by instrumental delivery and antedates asphyxia. Faced with intracranial hemorrhage in the baby delivered by Caesarian section, this theory offers no explanation.

While the clotting time of the blood is noted and checked by lumbar puncture in some clinics, no reference has been submitted concerning a prothrombin deficiency and the use of vitamin K before delivery.*

From the mechanical point of view, the theory that intracranial hemorrhage is merely an expression of increased venous pressure due to asphyxiation provides a reasonable explanation for the intracranial hemorrhage which occurs without trauma, i.e., Caesarian delivery. The frequency of intracranial hemorrhage in the premature, in which the thin-walled blood vessels are more liable to rupture, may also be explained from this point of view. The clinical picture of asphyxia neonatorum in the stage of spasticity requires little imagination to extend the ecchymosis mottling of the skin to a cerebral site where it cannot be seen, but where its effects may prove fatal or appear in later life as mental derangements. The work of Schriber which has been referred to in this connection is of much interest.

Impinging upon the clinical picture of intracranial hemorrhage is that demonstrated by ante- or neonatal atelectasis. This field invites extensive research. Whether the situation is a developmental deficiency or a mechanical failure to distend the air sacks is an open question, particularly in the premature. An infant lung which it was not possible to insufflate endotracheally by a pressure of 25 mm Hg for a period of five seconds *in situ*,

* An inquiry in the fall of 1942 indicated that the respondents desired no change in the views they had expressed. Vitamin K is, however, generally employed.

and which was born atelectatic, was easily insufflated under water at post mortem. The writer has repeatedly emphasized the need of research directed to a series of new-born infants in which endotracheal insufflation could be practiced under fluoroscopic examination, an x-ray record of the progress made kept for record, and a vital capacity tidal volume of air mensuration used as a check. There is an urgent need for an air-conditioned cabinet providing known mixtures of oxygen and CO_2 under constant qualitative check and providing adequate heat, humidity and visibility.*

There is a general agreement regarding the necessity of early and thorough aspiration of the infant airway. This seems to be particularly desirable in the breech, podalic, or Caesarian delivery. In these cases the compression effect on the chest occurring in vertex birth, which expresses much of the pulmonary secretion, has failed to operate.

Snyder's demonstration of intra-uterine respiratory movements suggests that amniotic fluid may be a normal condition within the respiratory tree, and that the respiratory efforts may propel a fluid wave instead of the tidal volume of air occurring after birth.

The relief of fluid by suction is carried out by techniques which vary with the individual clinic. Common practice encourages the use of a rubber catheter operated by the surgeon's mouth. This catheter introduced into the mouth and pharynx is useful in removing fluid with which it comes in contact. The method of de Lee, in which the catheter is passed into the glottis by blind intubation, is a source of much satisfaction to many surgeons. Familiarity with the size and the position of the glottis and the strength of the glottic reflex, except in flaccidity, suggests that much of the so-called blind endotracheal suction is in reality esophagal suction.

The glottis of the new-born baby of seven or eight pounds whose laryngeal reflex is still active presents resistance to a 3-mm smooth metal tube. An attempt to intubate a flexible rubber catheter demonstrates the ease with which the tip of the catheter may be deflected into the esophagus. The finger of the average operator, furthermore, presents a diameter which is larger than the infant laryngoscope. The trauma of manipulation to accomplish blind intubation may very well injure the fauces and the soft palate. During this manipulation the baby's respiration is, of course, completely obstructed by the foreign body presented by the finger and the rubber catheter, which completely fills the glottis. Operators familiar with the use of direct exposure by an illuminated laryngoscope, presenting as it does facilities for the examination of the glottic aperture, amniotic shreds, etc., are inclined to compare the technique of blind intubation with the routine technique advocated by James O'Dwyer fifty years ago for the treatment of diphtheria.

Oral endoscopy in the flaccid new-born can be taught to a student as readily as can blind intubation. The results are those of precision instrumentation as opposed to surgery limited to tactile manipulation.

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* The author is now constructing a transparent positive pressure oxygen tent for infants.

Use of Heat in Treatment of the New-born

The use of heat in the form of a bath is urged. Avoidance of drafts, avoidance of the heat of an intense light upon the new-born skin, and of the pressure of blankets for protection are to be noted. Blankets heated for the use of the baby should not be put in a warmer which renders them absolutely dry, as the static spark and fire hazard is a common danger where inflammable anaesthetic gases are in use. An understanding of the gesture of skin stimulation as compared with other means of stimulation to provoke respiration would do much to eliminate the hot and cold tubbing, spanking, rubbing of the back, slapping of the soles of the feet, etc.

A profoundly asphyxiated infant can be expected to respond to such form of irritation in just about the same manner as one might expect a patient anaesthetized for a laparotomy to respond. The anaesthesia produced by the asphyxia is certainly as profound. The indications are, as has been noted, to stimulate the respiratory center by carbon dioxide, simultaneously supporting it by oxygen. The vitalization of the respiratory center is immediately followed in orderly sequence by the return of the more superficial reflexes, and recovery ensues.

It is quite immaterial by what apparatus or by what means one introduces oxygen and CO_2 into the respiratory tree, provided this actually takes place. Blowing oxygen into the baby's mouth or covering the mouth and nose with an oxygen mask is nothing but an idle gesture if the gas delivered does not actually find its way into the trachea and bronchii. Babies which survive with this treatment do so in spite of it, and because of their inherent vitality, rather than as a result of the treatment. The school which employs direct laryngoscopy and intubation for suction is convinced that the only way to be sure that oxygen is in contact with the respiratory alveolae is to place it past all obstructions directly in the trachea. The immediate results of this treatment have repeatedly confirmed these views.

An important point is brought out in the necessity for close post-operative observation of the baby who has been asphyxiated. He cannot be casually abandoned to nursing care. He should not be placed in an incubator with solid walls where it is difficult for the nurse to see him as she goes about her duties. Adequate illumination of a correct temperature curve (full spectrum)* should be allowed to fall upon the baby. He should not be put in a nursery where daylight is reflected from green walls to his skin. Such illumination destroys the color value and the variations which should be noted.

The position of the baby should be such as to encourage the best possible respiratory ventilation. He should never be placed on his face. If atelectasis is suspected, placing the baby on his right or left side will result in less expansion than if placed flat on his back. The side position is useful, however, in preventing aspiration in very ill babies who regurgitate fluid.

* See page 388.

Respiratory expansion should not be embarrassed by the use of tight umbilical binders, bands or other clothing.

In the absence of mechanical means of artificial respiration, mouth-to-mouth insufflation is commonly practiced and offers relief. The difficulty at once apparent to the operator familiar with the glottis of the newborn infant is the resistance offered by this tiny aperture to the entrance of insufflated air in comparison to the absence of resistance of the wide-open esophagus. There is no doubt but that the greater part of the insufflated breath of the operator distends the baby's stomach instead of the lung. To this, of course, is added the danger of the insufflation of fluid from the baby's pharynx into his airway and contamination from the operator's mouth. However, in the absence of all other means of adequate insufflation, there is no doubt that this method should be practiced.

A number of universities have noted experience with intravenous and other types of medication. There is marked difference of opinion in the case of alpha-lobelin. It may be concluded that the drug selected may be used as an adjutant, certainly never as the sole means of reestablishing the intrauterine respiration of the baby who has just been delivered. The implication that a hypodermic will produce resuscitation *per se* is dangerous, for it may frequently delay the use of methods better calculated to bring about the desired result.

Intracardiac injections are used as a last resort. In this connection it is of interest to refer to the electric Pacemaker of Hyman, by means of which an auricular impulse is stimulated by a bipolar needle activated by an intermittent current which is injected into the right auricle.*

A number of universities urge the popularization of the matter which has been submitted with emphasis upon the prevention rather than the treatment of asphyxia. There is a strong feeling expressed by an important group among our correspondents that sedatives to the mother are responsible for much of the asphyxia neonatorum which occurs, and that the elimination or the reduction of this routine sedation will do much to prevent it. Since this impression has become generally recognized through many press releases it need not be stressed.

In view of the great interest in the subject of asphyxia neonatorum the need for research can scarcely be overemphasized. Research is urgently needed at this time to throw light upon the question of atelectasis, to produce suitable equipment for asphyxia neonatorum in the premature, to throw light upon the problem of intracranial hemorrhage, and to reduce the use of maternal sedation which acts to depress the baby.

Summary

It is found that carbon dioxide with oxygen is in general use; that aspiration is regarded as essential to the treatment of the baby; that the use

* See "Resuscitation of the Stopped Heart by Intracardial Therapy," by Albert S. Hyman, U. S. Naval Medical Bulletin, Vol. **33**, No. 2.

of heat is frequently overlooked but should always be applied to protect the baby from exposure.

Sharp differences of opinion exist regarding the use of alpha-lobelin and other drugs as respiratory stimulants.

Where intracranial hemorrhage is suspected, lumbar puncture is advocated and the use of whole blood injected into the buttocks of the baby is recommended.

Mouth-to-mouth insufflation is commonly employed as a method of artificial respiration where mechanical facilities are not available. The position of the baby after delivery is considered important. Intracardiac injections are used as a last resort, but are not popular. The use of blind intubation in accordance with the technique of de Lee is common practice in many clinics. The technique of direct laryngoscopy, intubation, suction and insufflation is not generally understood or applied where indicated. Prochownick's method of artificial respiration is referred to. Confusion exists as to the sequence in which intracranial hemorrhage occurs; it is claimed as both a cause of and a result of asphyxia. It is recommended that the baby who has shown signs of asphyxia be carefully observed after return to the nursery.

The important question of atelectasis is raised and the need of research indicated. There is a strong sentiment in favor of popularizing certain general information relative to asphyxia neonatorum to the profession and to the public, with special reference to the practice of frequent sedation administered to the mother. The danger of respiratory obstruction by the pressure of an inhalator mask on a baby's face is noted.

Experience with the use of mechanical methods, including the Drinker, E & J, Kreiselman and Flagg equipment, are referred to.

The solitary reference to euthenasia in obstetrics is noted for the purpose of condemning it.

Research on the problem of asphyxia neonatorum in the various universities in this country and in Canada is noted and found to be entirely inadequate for the problem confronted.

The work of Schulte and Davis in which pure CO_2 was used in cases of extreme asphyxia neonatorum as a means of successful resuscitation would seem to bear out Yandell Henderson's hypothesis of the existence of a non-acidotic acarbia, as opposed to a true acidotic acarbia in asphyxia of the new-born. It would seem that such treatment should certainly have resulted in the death of these babies had the use of CO_2 *per se* been as hazardous as proclaimed by those opposed to its use. Henderson's chapter, "The Fallacy of Asphyxial Acidosis" has been greeted by silence. Let us hope that this is the silence of assent. It is unfortunate that those who object to the use of CO_2 under certain specific conditions are being regarded as condemning its use in a wider field than they intend to condemn. Aspiration is gaining in popularity. The use of heat continues to be overlooked. Intravenous drugs are losing their popularity.* Mouth-to-mouth insufflation continues to be recommended as an emergency measure; applied through an intubation tube it becomes effective and more useful.

As pointed out by Dr. Curtis J. Lund,† preoperative maternal oxygen therapy may serve to reduce asphyxia neonatorum at its source.‡ Lund emphasizes that "Just as the mother is evaluated as an obstetric risk, so should the fetus be considered from the standpoint of ASPHYXIAL RISK . . . . The diagnosis of fetal anoxia in utero by fetal heart arythmya and treatment by maternal oxygen administration should be kept in mind.§ Reference to Lund's original work will repay the reader.

Crotty||, pleading for a wider adoption of intratrachael insufflation concludes that the keynote in the prevention of asphyxia neonatorum is greater caution in the use of drugs for the production of forgetfulness and the relief of pain.

The record form suggested on page 122, if put into practice in institutions interested in the prevention of asphyxial death, may be expected to yield statistics of real value to the art of resuscitation.

* "The treatment of experimental anoxia with certain respiratory and cardiac stimulants," N. J. Eastman and J. Kreiselman, *Surg., Gynec. and Obstet.*, **41,** 260 (Feb. 1941).

† *Am. J. Obstet. and Gynec.*, **43,** 365 (March, 1942).

‡ *Intra-uterine Asphyxia.* Oct. 12, 1943. Para I. Premedication. Nembutal gr. 6. Scopolamine gr. 1/150, between 7 and 9 A.M. Patient first seen by author under anaesthesia gas (oxygen, ether). Slightly anoxic. Moderate respiratory obstruction. Fetal heart 60. Fetal movements active. Free respiratory ventilation was instituted in the mother's anaesthesia. Pure oxygen with rebreathing was practiced, ether being continued. Fetal heart promptly increased in rate. In five minutes it was 100. Baby was delivered by forceps. Cord about neck, slightly cyanosed, mecomium escaping. Within one minute it began to cry lustily.

Comment. Free respiratory ventilation, plus oxygen and CO_2, plus reduced intra-uterine pressure from ether relaxation promply relieved a critical intrauterine asphyxia.

§ Ibid., **41,** No. 6 (June 1941).

|| *Cincinnati J. Med.*, **23,** No. 388 (1942).

NOTE: Live birth following asphyxial death of mother. (Post-mortem Caesarian section.) Patient admitted to St. Vincent's Hospital Jan. 3, 1944, 2 A.M. Diag. Bronchopneumonia: laryngeal stridor, edema of glottis and aryepiglottic folds; cyanosis. Tracheotomy 6.00 P.M. Oxygen administration; condition deteriorated. Expired at 10.14 P.M. Caesarian section done, 35 weeks old, 5 lb. 10 oz. male baby delivered alive. Discharged in good condition 27 days later. (From service of Dr. John Francis McGrath, reported by V. W. Badia.)

Chapter 9

Asphyxia from High Altitudes

The writer's activities in connection with high-altitude problems of asphyxia began curiously enough in a motion picture which he saw in 1934. The presentation described the fire hazard to flammable material in under-water caisson work. A carelessly discarded match was shown to cause an uncontrollable conflagration of wooden structures, the fire resembling that which occurs in the ordinary oxygen tent. If such concentrations of oxygen could be secured by compressing sea-level atmosphere, why would it not be possible to compress the atmosphere of high altitudes to sea-level pressure of 760 mm, obtaining thereby ordinary sea-level atmosphere with its 20.96 per cent oxygen concentration? If this were feasible the problem of the air-conditioning of the high-altitude plane would be solved. A continuous supply of fresh compressed air would do away with the necessity of supplying oxygen to the passengers.

It was fully realized that the practical application of this theory turned upon the constant relation of the partial pressures of the gases in the high altitudes as compared with those at sea level. If oxygen comprised approximately 20 per cent of the stratosphere atmosphere its compression would create sea-level atmosphere; but if this relation did not exist compression would not yield this result. The author discussed his views with Col. Lindberg and later with Dr. R. R. Sayer, Director, Bureau of Mines, whom he met at Atlantic city in June 1935; both were in agreement that such an approach was feasible. The author was unaware of the statement made by Cruchet and Moulinier some 30 years ago: "As a matter of fact the (oxygen) problem will never be satisfactorily solved until crew and passenger sitting in an airtight cabin shall breathe at all altitudes air atmosphere practically identical with that at sea level."

A letter to the U. S. Weather Bureau brought the following reply under date of April 23, 1935.

"Your letter of April 3rd, 1935, relative to the composition of the air of the stratosphere has been received. It is practically certain from a few direct observations and from theory, that up to at least 60,000 ft. above sea-level the composition of the air is substantially constant, except as to water vapor. Owing to low temperature the absolute humidity is very small in all portions of the stratosphere that have been explored."

It was not until Nov. 11, 1935, however, that Capt. Albert W. Stevens and Capt. Orvil Anderson made their memorable balloon ascension under the auspices of the National Geographic Society and the U. S. Army. The following is taken from the *National Geographic Magazine* for May, 1936 (page 693):

"Science has long wanted samples of air from the upper regions of the atmosphere to determine whether it differs in makeup and in the proportions of its ingredients from the air near the earth's surface.

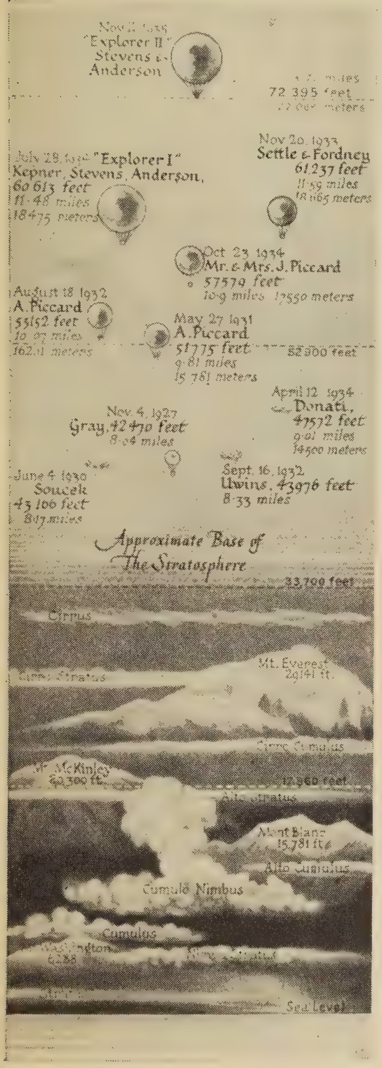


Fig. 94. The diagram shows how cloud forms mark altitudes in the lower atmosphere, and the heights reached by the most important balloon and airplane flights. Shading is used arbitrarily to indicate density of the atmosphere, greatest near sea level and growing rapidly less upward.

Courtesy National Geographic Magazine

"It has been supposed that far up in the stratosphere, since there is no turbulence due to vertical currents, the air gases tend to separate out. This action should show itself first as an increased proportion of nitrogen which, being lighter than oxygen, should tend to concentrate upward.

"During our flight we obtained two samples of stratosphere air each of nearly six gallons. When six gallons of rare air under only one twenty-fifth of an atmosphere of pressure is brought down to earth, however, it amounts to less than one quart under normal pressure. The two quart samples represent the only sizable samples that have ever been captured at such a height above sea level and their analysis is being undertaken at the National Bureau of Standards by G. M. Shepherd."

On May 20th, 1936, the following communication was received from the National Bureau of Standards: "We have completed the examination of the first two samples and found 20.89% oxygen; the percentage of oxygen normally occurring in the air is about 20.94 to 20.95, although there have been recorded what we consider to be authentic analyses of air sampled during high barometric pressure and low temperature in which the oxygen found was as low as 20.44%. The difference we have found in the stratosphere samples is very slight and would correspond to a height of approximately 21 kilometers as the base of the stratosphere at which separation of the components of the air had begun to occur." (Kilometer: 0.62 mile, 21 kilometers: 13.02 miles.)

Confirmed in the practical value of his earlier theoretical approach to the problem, the author contacted Jack Frye, President of Transcontinental and Western Airways, and discussed the problem with his representative in New York City on Sept. 30th, 1936. The end result of this conference was contained in a letter received from President Frye, Oct. 26, 1939. "Your discussion with Mr. Tomilson in Sept. 1936 did have a definite bearing on our decision to enter into the stratoliner development, and I hope when we have one of the ships in the East that you will make a flight in it and inspect the actual equipment that is being used."

The foregoing experience is cited as evidence that the integration of the field of gas therapy is entirely natural, that contributions of value to it may be expected from those who are familiar with the behavior of gases and their biochemical effects, but who may be quite unfamiliar with other major aspects of the problems under consideration.

The barometric pressure of 760 mm Hg existing at sea level is rapidly reduced as one moves skyward. At the 6,000-ft level of Colorado Springs the pressure is 620 mm; at the 14,000-ft level of Pikes Peak it is reduced to 420 mm; at the 21,000-ft level of the North Col of Mt. Everest, where one may learn to live in comfort when acclimatized, the barometric pressure is reduced to 335 mm, or approximately one-half an atmosphere.

It will be observed from the foregoing that it is not necessary to leave the surface of the earth in order to experience the effects of high altitude. Furthermore, as Yandell Henderson has shown in his "Adventures in Respiration", these effects may be studied en route, as it were, to the high destination. Mountain climbing, therefore, provides a control for the findings of aeronautical science, which may be extended over days or weeks at a given level. Here compensatory mechanisms may be studied at leisure.

Courtesy National Geographic Magazine

Fig. 95. The first photograph ever made showing division between the troposphere and the stratosphere and the actual curvature of the earth (black line). Elevation 72,395 feet. The line of sight of the camera is wholly through the stratosphere.

More recently large negative pressure cabinets in which the operator and assistants may be wholly contained to carry on experiments under conditions simulating high altitudes and temperatures have been made available. By means of these facilities, not only are investigators able to pursue their research without leaving the surface of the earth but they may be observed by others outside the pressure chamber.

Pathological Physiology

Having briefly referred to the external physical environment of high altitudes, we may now consider the problem of gas tension in its relation to the internal environment.

The normal pulmonary gas tensions at sea level reported by Yandell Henderson in his fascinating book, "Adventures in Respiration", in which he describes high-altitude studies based on mountain climbing, are as follows: In the first place, they are partial pressures of the 760-mm sea level pressure. The relations are constant: oxygen 100 mm, CO_2 40 mm, water vapor 47 mm. "As the oxygen falls, the result of increasing altitude, the CO_2 and the blood alkali are lowered proportionately. There is no such thing as a general acclimatization; there are as many acclimatizations as there are altitudes at which a man can live; each is an adjustment of the carbon dioxide and alkali to correspond to a certain tension of oxygen, and each state requires time to develop. Acclimatization fails to develop only when the altitude is so great and the tension of oxygen so low that adjustment becomes physically impossible." The following are examples of the establishment of levels in pulmonary gas tensions at various altitudes.

	Barometric pressure (mm Hg)	Water vapor (mm Hg)	Oxygen (mm Hg)	Carbon dioxide (mm Hg)
Sea level	760	47	100	40
Colorado Springs (6,000′)	620	47	79	36
Pikes Peak (14,000′)	460	47	53	28
North Col Mt. Everest (21,000′)	335	47	39	19

A worthy disciple of Paul Bert, the father of Aviation, Armstrong gives us the following figures on gas pressure at sea level:

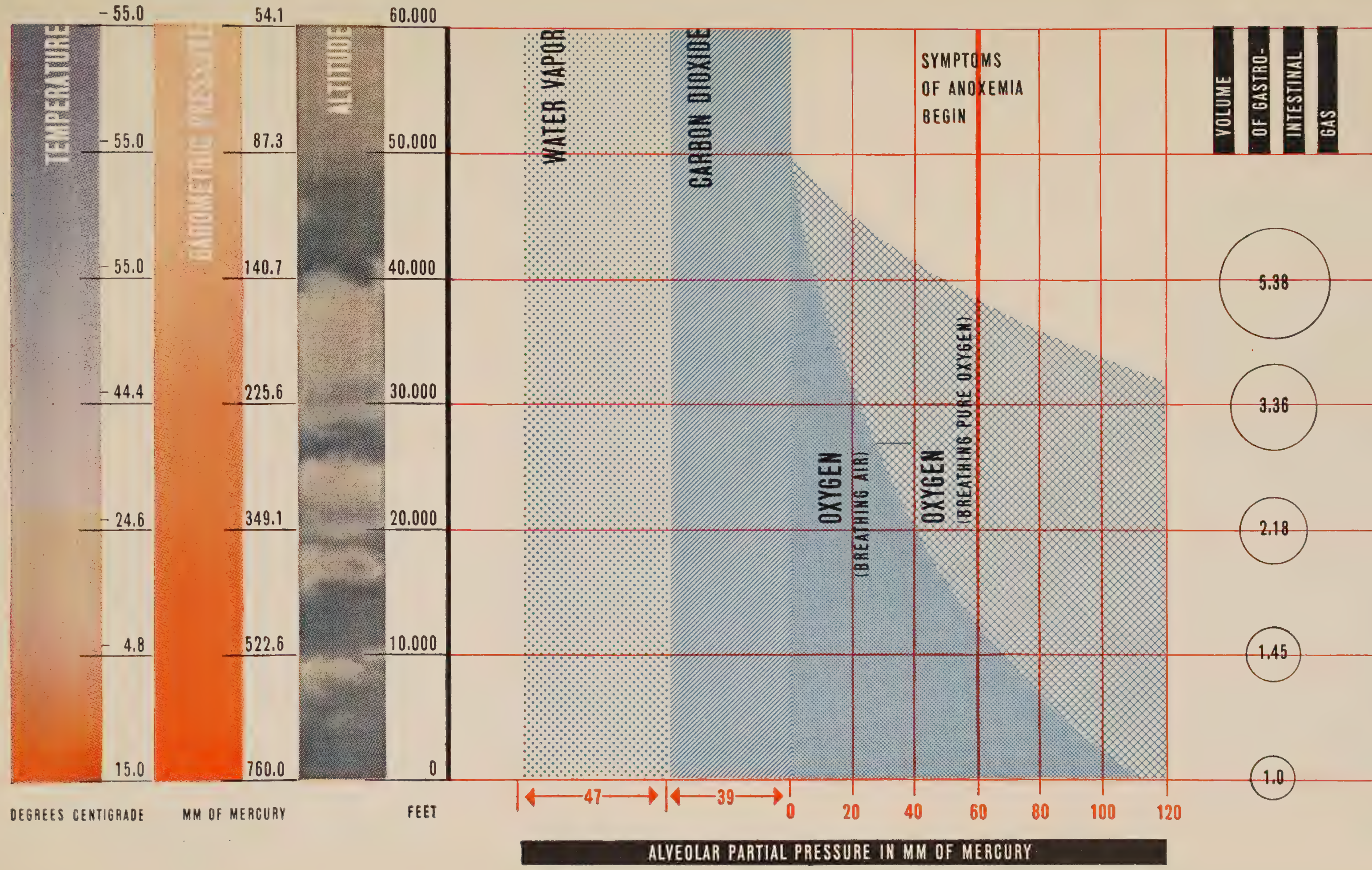
The atmosphere is made up of: Oxygen, 20.96%; nitrogen, 79%; CO_2, .04%; water vapor variable. (Oxygen 159 mm)

Pulmonary air is made up of: Oxygen, 14.5%; nitrogen, 80%; CO_2, 5.5%; water vapor, 47%.

or in terms of mm vapor tension partial pressure: Oxygen, 103 mm; CO_2, 39 mm; water vapor, 47 mm.

(760 − 47% w.v. = 713; 713 × 0.145 = 103 mm) (Page 247. 1940)

Blood 96 per cent saturated at sea level by oxygen carries about 18.5 cc of combined oxygen and 0.24 cc of oxygen in solution. Armstrong states, "This oxygen in simple solution is extremely important inasmuch as it is the immediate source of oxygen supplied to the tissues." The reader is



Courtesy The Upjohn Co.

reminded in this connection of the point (page 142) that oxygen therapy may provide support pending transfusion for shock.

The altitude pressure curve is not a straight line; 0–1000 ft, 27 mm; 29,000–30,000 ft, only 11 mm.

ALTITUDE	PRESSURE	ALTITUDE	PRESSURE	ALTITUDE	PRESSURE	ALTITUDE	PRESSURE
feet	*mm.* Hg	*feet*	*mm.* Hg	*feet*	*mm.* Hg	*feet*	*mm.* Hg
0	760	21,000	334.8	41,000	134.2	61,000	51.6
1,000	733	22,000	320.8	42,000	127.9	62,000	49.2
2,000	706.6	23,000	307.4	43,000	122	63,000	46.9
3,000	681	24,000	294.4	44,000	116.3	64,000	44.7
4,000	656.4	25,000	282	45,000	110.9	65,000	42.6
5,000	632.4	26,000	269.8	46,000	105.7	66,000	40.7
6,000	609	27,000	258.2	47,000	100.8	67,000	39.8
7,000	586.4	28,000	246.8	48,000	96	68,000	37
8,000	564.4	29,000	236	49,000	91.6	69,000	35.2
9,000	543.2	30,000	225.6	50,000	87.3	70,000	33.6
10,000	522.6	31,000	215.4	51,000	83.2	71,000	32
11,000	502.6	32,000	205.8	52,000	79.3	72,000	30.5
12,000	483.2	33,000	196.4	53,000	75.6	73,000	29.1
13,000	464.6	34,000	187.4	54,000	72.1	74,000	27.7
14,000	446.4	35,000	178.7	55,000	68.8	75,000	26.5
15,000	428.8	36,000	170.4	56,000	65.5	76,000	25.2
16,000	411.8	37,000	162.4	57,000	62.5	77,000	24
17,000	395.4	38,000	154.9	58,000	59.6	78,000	22.9
18,000	379.4	39,000	147.6	59,000	56.8	79,000	21.9
19,000	364	40,000	140.7	60,000	54.1	80,000	20.8
20,000	349.2						

From "Principles and Practice of Aviation Medicine," by H. G. Armstrong

Fig. 96. Altitude-pressure table.

Haldane and Douglass devised a simple technique for securing samples of lung atmosphere. The operator makes a sudden deep expiration through a wide-bore tube, and a sample of the air is immediately taken from this tube close to the lips. This air represents the alveolar air.

As the oxygen in the external air is reduced by altitude the oxygen in the alveolar spaces is likewise diminished. The proportion oxygen 100, CO_2 40 is disturbed; carbon dioxide is then blown off through increased respiratory effort. This effort brings more oxygen (even though of low tension) into the alveolar spaces, to supply the metabolic needs, which remain constant regardless of altitude.

In addition to the constancy of metabolic need there is another factor, emphasized by Henderson, which also remains unchanged in high altitude, *i.e.*, the vapor tension of water vapor in the lungs. This tension of 47 mm is unimportant at sea level, as there is plenty of room for it in the 760 mm total pressure which exists; but as the pressure is reduced to 460 mm at Pikes Peak, for example, the water vapor occupies almost double its former space; and at the level achieved by Captain Stevens, 73,000 ft, representing $\frac{4}{100}$ or $\frac{1}{25}$ of 760, a barometric pressure of 30, there would be nothing but boiling water vapor in the lungs of a man so exposed.

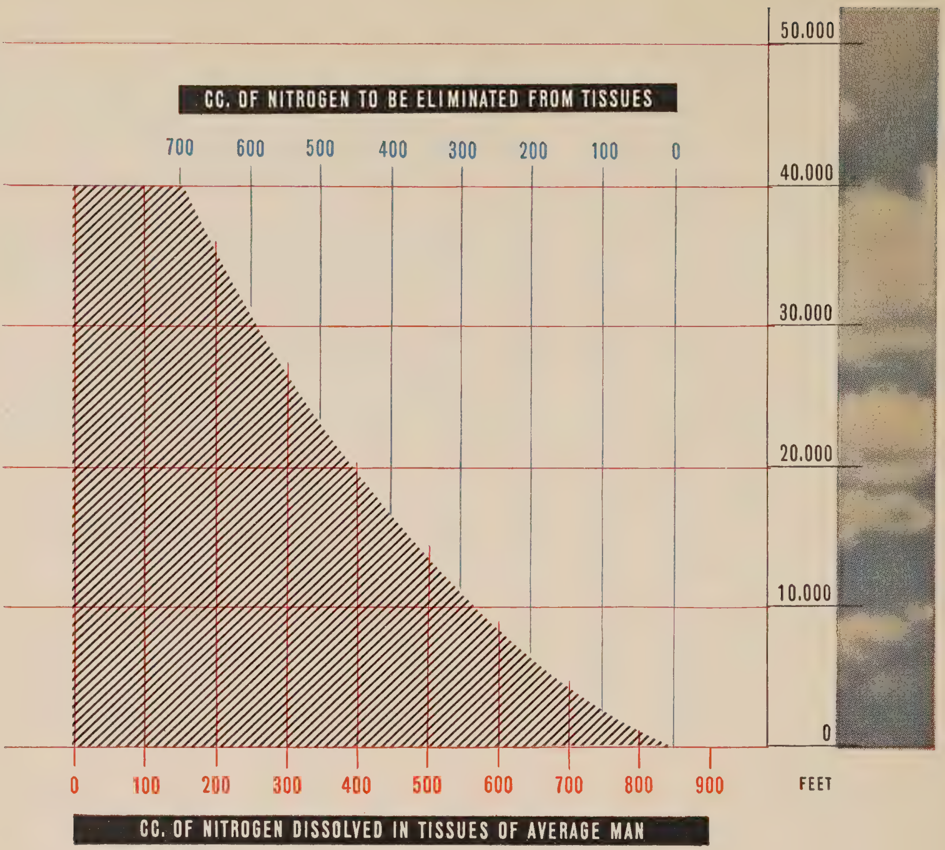
Approaching the problem from the viewpoint of the aviator, however, Armstrong states that the level at which no oxygen will appear in the alveolar air is not 62,965 ft, representing a vapor tension of 47 mm but 50,313 ft, representing a tension of 86 mm (39+47). In other words the zero altitude at which an aviator would receive no oxygen from stratosphere atmosphere would be 13,000 ft lower than is supposed (Fig. 96). He reasons that under the conditions of relative tranquillity accompanying flying, CO_2 vapor tension like water vapor is constant.

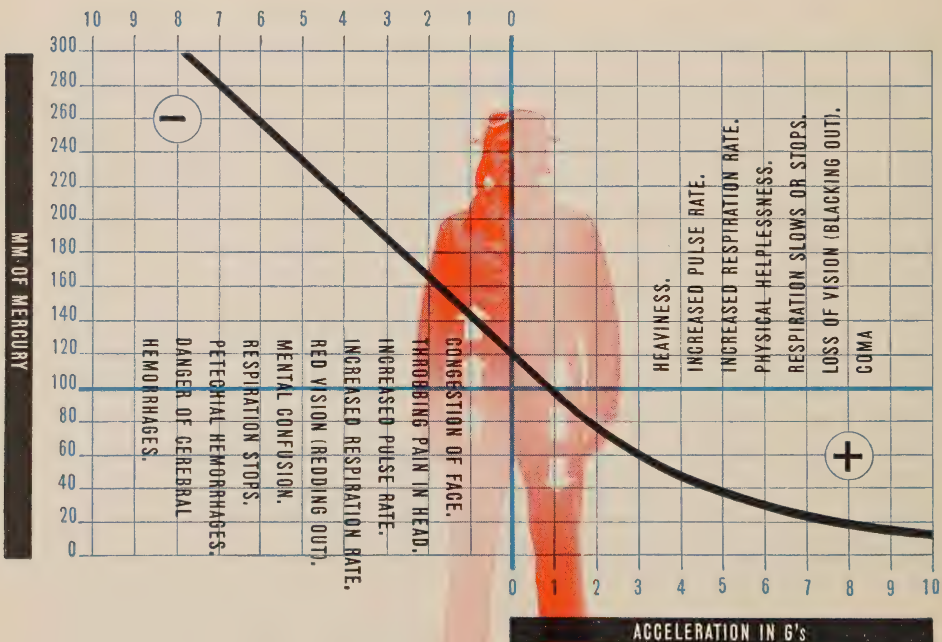
The discrepancy between these two views may be explained by the approach. Henderson presupposes physiological adaptation; Armstrong expects no adaptation, but conceives of his subject as being projected directly into the high altitude without CO_2 acclimatization.

Experiments conducted by Henderson in normal men indicate that the minimum tolerance—the point at which consciousness is lost—is 35 mm of oxygen, represented by an altitude occurring between that of the North Col of Mt. Everest (21,000 ft) and its summit (29,000 ft). He points out that, although an aviator with his normal CO_2 tension of 40 mm could ascend the summit of Mt. Everest, if he landed at the North Col equipped with a CO_2 absorption apparatus, and were deprived of such assistance he would find himself caught in a barometric pressure of 288 (335−47) with a CO_2 tension of 40 and an oxygen tension of 17, which would asphyxiate him.

On the other hand, an acclimated mountaineer who had learned to live at 21,000 ft, having developed an oxygen tension of 39 and a CO_2 tension of 19, would not be helped by oxygen. The reason is that having become acclimated to a CO_2 tension of about 20, his respiratory center would be hypersensitive. Since a man in climbing produces six or eight times as much CO_2 as at rest, and since the actual amount of CO_2 produced by the mountaineer would equal that of the aviator, he would be required to breathe not 36 or 64 liters per minute but 72 to 126 liters per minute (ordinary respiration 8 to 12 liters per minute). He could not endure such respiration for long without exhaustion.

In exposure to an environment of high-altitude atmosphere, therefore, dyspnoea and anoxia become apparent at elevations of 8,000 to 14,000 ft. Exertion by increasing the CO_2 tension in the lungs crowds out oxygen and increases this dyspnoea and anoxia. A man with a low ceiling (low resistance to lack of oxygen) may be comfortably seated in a plane at 10,000 feet, but if he attempts to walk its length and back, he may develop marked dyspnoea and anoxia. Henderson has developed a device to measure the ceiling of aviators. Healthy young men fail when the oxygen tension in their lungs falls below 35 mm, or about 5 per cent of an atmosphere. It appears from the work of Christiansen and Koegh that the critical factor is not the tension of the air inhaled but that of the air maintained in the





Courtesy The Upjohn Co.

lungs. Those most resistant to oxygen depression are those whose respiratory centers are sensitive to CO_2 at sea level.

The practical conclusions to be drawn from these physical facts are that man can acclimate himself to an altitude which he cannot accept in a few minutes; that the aviator is not protected by the margin of safety developed by the mountain climber; that while oxygen is essential in high altitudes, the role of carbon dioxide and water vapor increase in importance with the altitude, as is emphasized by Armstrong in his chapter on aero-embolism. Lastly, the nitrogen saturating the tissues and the blood must be decompressed through the circulation and the respiration, or there will follow a series of subjective and objective symptoms to which we shall now briefly refer.

Oxygen, carbon dioxide and water vapor comprise about 187 mm of the total vapor tension of alveolar air at sea level, and nitrogen makes up the balance. Nitrogen is the vehicle of the internal gaseous environment. As external pressure is reduced, all the gases present in the blood and tissues must escape or expand in their container. Were a man to be projected suddenly 15 miles into space he would literally explode from the gases within his tissues. Stevens saw a mist and clusters of "diamonds" enshrouding the ropes of his gondola—the water vapor exploding from within the texture of the ropes. A man so exposed would rapidly become completely desiccated. The effects produced by such internal combustion may readily be anticipated. They constitute the objective and subjective signs described by Armstrong as acute altitude sickness.

Considering the nose and throat, obstructed frontal, antral or ethmoidal sinuses become painful because of the increased gas pressure within them. Inflamed eustacian tubes obstructing ventilation of the middle ear may cause severe earache or result in rupture of the drum. Implanted glass bells supporting artificial eyes, if containing air, may burst. Myalgic pains occur as a result of aero-embolism.

Nitrogen within the tissues is absorbed into the circulation at high altitudes, forming air emboli. Decompression of nitrogen from the tissues will take place if the ascent is not too rapid (500 ft per minute). A technique has been developed to decompress atmospheric nitrogen on the ground by rebreating pure oxygen for a period while exercising, the oxygen atmosphere washing the nitrogen out of the tissues.

Neurological effects are closely associated with early anoxia; depression and euphoria are common. The man suffering from incipient anoxia is certainly not in a position to pass well-considered judgement on behalf of himself or others. As may be expected, abdominal distension and flatulence with increased intestinal mobility are frequent.

The subjective symptoms of acute altitude sickness are reported by Armstrong (Fig. 97). He sharply differentiates the acute altitude sickness just referred to from what he describes as air sickness or air sea-sickness. Air sickness is described as, "a condition occurring primarily as the result of acceleration in aircraft flight (chiefly vertical). It is marked by nausea, vomiting, fear and prostration". It is caused by over-stimulation of the labyrinth and visual visceral disturbances, psychic states, by toxemias and by mechanical, electrical and thermal disturbances, anemia and congestion of vestibular apparatus.

Therefore the traveller on an airplane is subject, not only to seasickness but to the additional complications of anoxic anoxia from deficient oxygen at low atmospheric pressures. Armstrong is also of the opinion that there may develop a histotoxic anoxia as the result of cell injury from protracted anoxic anoxia.

While the level at which middle ear, visual, visceral and psychic disturbances become complicated by progressive anoxia is difficult to determine, it seems fair to assume that early and adequate provision to meet the latter would greatly assist the former. To this field of therapy our attention is devoted.

12,000 FEET ALTITUDE	14,000 FEET ALTITUDE	16,000 FEET ALTITUDE
Sleepiness	Headache	Headache
Headache	Altered respiration	Altered respiration
Altered respiration	Sleepiness	Psychologic impairment
Lassitude	Psychologic impairment	Euphoria
Fatigue	Lassitude	Sleepiness
Psychologic impairment	Fatigue	Lassitude
Euphoria	Euphoria	Fatigue

Courtesy Williams & Wilkins Co.

Fig. 97.—Effect of high altitude on the human body.

In a very brief inspection of the literature relative to high-altitude flying, the author has noted with surprise that the cooperation of the physician anaesthetist has not been enlisted; and yet the unconsciousness treated by the research worker in high-altitude flying as the end of his "margin of safety" represents merely the introduction to a state which the anaesthetist deliberately induces daily and controls successfully. Why has not this viewpoint been enlisted? Why are these fields of gas therapy segregated in their tight little compartments, sealed by the complacency which may temporarily attach to each particular field? The research worker in aeronautics at this writing hardly expects assistance from the obstetrician or pediatrician, and yet a man from either of these fields, familiar with the flaccidity of terminal asphyxia in asphyxia neonatorum, might very well take over the practical relief of a patient dying at high altitude.

Equipment to Prevent Asphyxial Death at High Altitudes

Oxygen is made by liquefying atmospheric air at a temperature of −182.5°C, after which nitrogen is distilled off. While oxygen is available in four forms, as a solid, as a peroxide, as a liquid and as a compressed gas, only the liquid and gaseous form will here be considered. Liquid oxygen, which is bluish in color, was standardized for use by the U. S. Army in 1923, but in 1936 the compressed gas returned. The reason for this change was that the small demand did not make it generally available; furthermore, there was a necessary loss by evaporation of about 12 per cent in 24 hours, and for this reason it could not be stored in quantity. It was also difficult and dangerous to handle, causing skin injury. The rate of flow could not readily be estimated and the amount left over in the container was not easily determined.

The use of compressed gas in light alloy cylinders has become common practice; it is particularly important that gas so stored should be perfectly dry, for there must be no danger of the valves freezing shut, when operating at high altitudes.

Methods of Administration. The requirements for equipment with which to administer oxygen at high altitudes, especially for military purposes in open cockpits, are rigorous. Armstrong states these requirements as follows:

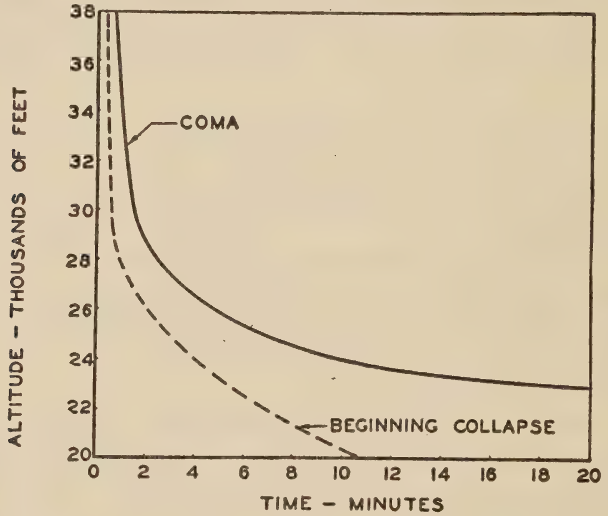
(1) The device must be perfectly comfortable.
(2) It should cover the nose and mouth.
(3) The sound of the voice should not be obstructed, for radio communication.
(4) The device must be light and compact so that it will not obstruct vision or offer wind resistance, which may cause it to be blown off.
(5) It must be free of valves and small moving parts.
(6) It should warm and humidify the oxygen.
(7) It should be 100 per cent efficient.

To date, these requirements have not been met. The human body will tolerate continuous administration of pure oxygen for five days. This tolerance, which is greater than in animals, is thought to be due to differences in the alveolar epithelium.

The following methods are in use: the simple mask, the pipe-stem tube, various closed methods, the B-L-B adaptation of Haldane's Mask and the closed-cabin plane. The simple mask, in which oxygen is led to a face piece and exhalations occur through an open vent, is said to utilize only one-third of the stream of oxygen supplied. The method is inefficient and wasteful. The pipe-stem method consists of a rubber tube held in the corner of the mouth. Inhalation is regulated by the immediate need of the flyer. The tube is difficult to retain; the dry gas irritates the oral pharynx, and yet because of its simplicity the method has been widely used. The closed method, employing a mask with a circuit (soda lime) CO_2 absorber, has not met with favor because of the tight application of the face-piece required and the possibility that decompressed nitrogen will escape from the lungs and build up pressure in the bag, thus excluding oxygen and causing dangerous asphyxia.

It is Armstrong's opinion that "The pressure cabin aeroplane is the ultimate solution, and the unreserved prediction is made that within the next five or ten years all airlines in high-altitude passenger flights (15–20,000 ft) in this country will be operating pressure cabin planes."

The question may be raised, What happens when a mask must be removed temporarily in moving about the plane at high altitudes? Fig. 98 indicates the margin of safety in minutes of time at various altitudes when, following over-ventilation, the mask may be removed. Armstrong's conclusions are that many fatal and near-fatal accidents due to anoxia have occurred in flights of between 12 and 15,000 ft. He states that under present conditions it is not safe to fly above 20,000 ft when the safety of the flight depends upon the inhalation of oxygen. Flights of 25,000 ft are considered hazardous, and 30,000 ft should be the absolute allowable limit except in unusual circumstances. No one should exceed 38,000 ft.



Courtesy Williams & Wilkins Co.

Fig. 98.—The oxygen reserve of normal healthy adults at various high altitudes after pure oxygen is suddenly replaced by atmospheric air. (Armstrong, p. 318)

Comments. "In experimental animals it has been found that death from altitude sickness is always due to a sudden failure of the respiratory center and that the heart continues to beat for 5 to 10 minutes after breathing has ceased. Of particular interest in this regard is the fact that once breathing is stopped it is seldom possible to reestablish it, even though relief measures are promptly instituted. Artificial respiration for periods of 30 minutes or more, the administration of oxygen, and other similar measures are usually fruitless even though the heart continues to function. This has led to the belief that serious damage to the respiratory center occurs. There have been many deaths reported as a direct result of altitude sickness and it is thought that, in healthy individuals, this is always due to respiratory paralysis." (Armstrong)

These questions immediately present themselves: Exactly what was done for these patients who died? Were modern medical methods to treat the spastic and the flaccid patient employed; if not, why not? Are trip graphs of oxygen concentration, barometric pressure, and the temperature of pressure cabins automatically recorded? Such records would be valuable.

Commercial resistance exists against well-equipped pneumatological facilities at air fields since it does not appear wise to emphasize acute high-altitude sickness to the traveling public. Such facilities and a trained personnel, it would seem, could be set up where traffic is heavy, without undue publicity and the asphyxial emergency given the benefit of the best treatment, instead of what it is now customary to improvise in even our largest hospital and medical centers. Until relief methods become something more than mere commercial oxygen therapy, or the unimportant sideline of some surgical, medical or anaesthetic service, the best results cannot be hoped for.

If it is worth trouble and expense to prevent high-altitude asphyxia it does seem as though it would be worth while to help save the lives of those patients who are about to die from asphyxia. The present air activity of the armed forces offers an opportunity to set up such protection with a minimum of resistance.

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Chapter 10

Asphyxia from Carbon Monoxide Poisoning

In this section an attempt is made to segregate from the vast literature covering the problem of carbon monoxide poisoning material which will assist the reader to treat asphyxia arising from this important cause.

Our consideration will be confined to pathological physiology and the various methods commonly employed for the prevention and the treatment of this type of poisoning. Reference will be made to organized efforts to acquaint the public of the hazard of carbon monoxide and the effects which an intensive drive to train lay rescue squads has had on the cooperation of physicians.

Carbon monoxide is a hazard in the following occupations:*

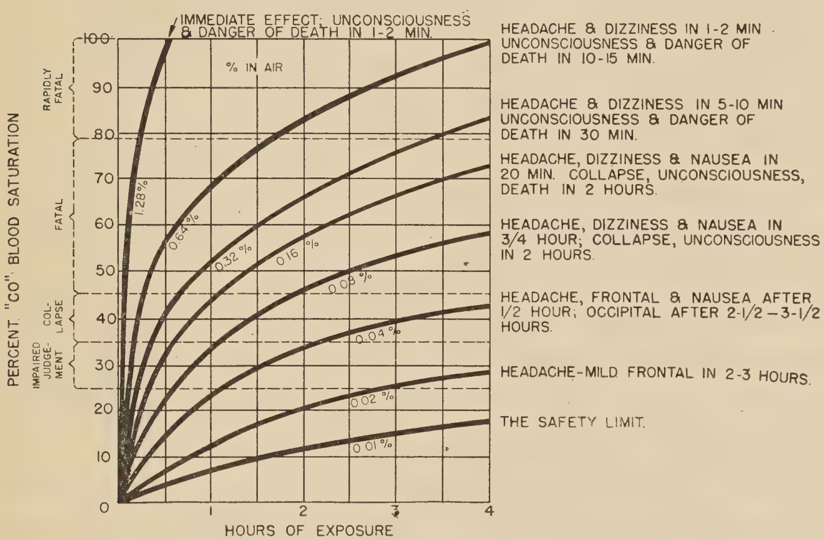
Acetylene workers
Ammonia workers
Bakers
Balloon inflaters
Bisque-kiln workers
Blacksmiths
Blasterers
Blast furnace workers
Blockers (felt hats)
Boiler cleaners
Boiler room workers
Brass foundries
Brick burners
Cable splicers
Calico printers
Carbide makers
Charcoal burners
Chargers (foundries)
Chargers (zinc smelting)
Chauffeurs
Chimney masons
Chimney sweepers
Cleaners (foundries)
Cloth singers
Coaltar workers
Coke oven workers
Cooks
Copper smelters
Core makers
Cupilo men (foundries)
Drier workers
Drying room workers
Enamelers
Enamel makers
Engineers, stationary
Filament makers
Filament finishers
Firemen
Flangers (felt hats)
Flue cleaners
Foundry workers
Fumigators
Furnace workers
Garage workers
Gas (illumin. workers)
Gassers (textile)
Glost-kiln workers
Incandescent lamp makers
Ink (printers) makers
Ironers
Kiln tenders
Laboratory workers
Laundry workers
Lead smelters
Lime burners
Lime kiln chargers
Linotypers
Mechanics (gas engines)
Mercury smelters
Methane makers (synthetic)
Methyl alcohol makers (synthetic)
Miners
Mold breakers (pottery)
Monotypers
Motion film workers
Neon light letter makers
Patent leather makers
Phosgene makers
Plumbers
Pottery kiln workers
Pressers
Puddlers (foundries)
Pyroxolin plastic workers
Refiners (metallic)
Repairers (foundries)
Sealers (incand. lamps)
Sewer workers
Silver melters
Singers (cloth)
Soda makers (le Blanc)
Solderers
Steeplejacks
Stokers
Teasers (glass)
Telephone linemen (trench work)
Temperers
Top fillers (foundries)
Tubulators (lamps)
Welders
Wood alcohol distillers
Wood charcoal workers
Zinc smelters

*Bul. U. S. Bureau of Labor Statistics #582. U. S. Gov. Prin.O ffice. Rev. 306.

The Nature of Carbon Monoxide Poisoning

The pathological physiology of carbon monoxide poisoning is that of acute anemic anoxia. It is identical with that produced by acute oxygen deprivation or by hemolysis. It differs from the anemic anoxia caused by hemorrhage in that the blood volume remains intact. There is usually an increase rather than a lowering of blood pressure. The late effects of the poisoning are the direct result of acute anoxia upon the various body tissues in the order of the abundance of their blood supply, *i.e.*, the brain and the heart suffering first and most gravely.

While the iron-containing catalyst, cytochrome, in the tissues, has an affinity for CO, as it has for oxygen, this affinity is weak and of little consequence, because such a high degree of CO is necessary to produce an effect upon it that the hemoglobin saturation has passed the fatal point long



Courtesy of F. M. Van Deventer

Fig. 99.—Carbon monoxide absorption by human blood.

before this effect takes place. The cyanogen and the H_2S radicals, on the other hand, have a strong affinity for this catalyst. Their prompt combination with it results in characteristic and profound histotoxic anoxia. Given an anoxia of equal intensity and duration, therefore, the lesions resulting from CO poisoning may be said to resemble closely those occurring in asphyxia neonatorum, submersion or electric stroke.

The Blood

Carbon monoxide has a density of 0.967 as compared with air. It burns with a blue flame. Two volumes of CO unite with one of oxygen to form CO_2. It has an affinity for hemoglobin which is 300 times that of oxygen.

The biophysical combination of CO and hemoglobin is known as HbCO. Carbon monoxide does not form a stable chemical combination with the hemoglobin, as was once supposed. It is readily replaced by oxygen when the respired atmosphere contains a high tension of this gas. In a circulation in which CO_2 has been depleted, both CO and oxygen are retained by the hemoglobin with great tenacity. The presence of CO_2 is therefore as important as that of oxygen in the liberation of CO from the blood and of oxygen to the tissues.

Three considerations influence the intensity of the effect of carbon monoxide poisoning; concentration, exposure, and respiratory exchange.

Concentration

The concentration of CO in a given atmosphere is referred to in terms of parts per 10,000, as for example, .01% is equal to one part or 0.5% is equal to 50 parts per 10,000. Because of the affinity for hemoglobin, a given concentration of CO breathed is cumulative, four parts per 10,000 saturating the blood to 44 per cent HbCO over a long period. A part of CO is thus one hundredth of 1 per cent, or 1/10,000.

According to Henderson, while a blood saturation of 25 per cent is not likely to produce symptoms in a patient at rest, 28 per cent may be expected to produce headache, 44 per cent analgesia and an unsteady gait, 66 per cent unconsciousness and spastic asphyxia and 75 per cent may result in death.

Harrison Martland states that the blood must be 20 per cent saturated to produce symptoms. Between 20 and 30 per cent saturation, vertigo, faintness, nausea and muscular weakness develop; between 30 and 35 per cent symptoms are urgent: incoördination, muscular weakness preventing movement, and death in the absence of rescue; between 50 and 70 per cent complete paralysis and coma; over 80 per cent, rapid death.

In Gettler's opinion the figures stated above are high. In the experience of the Medical Examiner's Office the majority of cases who have died from CO poisoning show a hemoglobin saturation of 45 to 65 per cent. In a few cases the saturation has been as low as 28 to 30 per cent. He believes that symptoms begin between 18 and 20 per cent. A concentration of 30 per cent is regarded as dangerous. He has found it practically impossible to locate individuals whose blood is entirely free from CO, since cigarettes, pipes and automotive vehicles provide a continual source of contamination, producing a concentration in normal individuals of about .01. Blood concentration in street cleaners is about .03, in taxi drivers .05 to .08. In cadavers the blood diffuses CO from the superficial layers of the skin. There is no diffusion from the heart blood, however which is employed as a basis of the medical examiner's findings.

The following normal CO contents of the blood and its fatal CO concentration indicate that 25 per cent of the deaths from CO had less than 60 per cent.*

* A. Gettler, *Am. J. Clinical Pathology*, **13**, No. 4 (Apr., 1943).

Normal CO Content of Blood

	Hemoglobin concentration (%)
Average person in New York City	1.0–1.5
New York City Street Cleaners	2.0–3.0
New York City Traffic Police	2.0–8.0
Persons in Rural Institutions	Less than 1.0
Heavy smokers	2.0–2.4

Fatal CO Concentration

Case	Per cent	% of 68 fatalities	Concentration (%)	History
1	31.3	4.5	30–40	Each one of our series of 68 cases was found dead in a gas-filled room or in a garage containing a high concentration of exhaust gases.
68	33.6	4.5	40–50	
19	42.2	14.5	50–60	
17	43.6	28.0	60–70	
50	49.3	48.5	70–88	
4	50.4			
37	52.9			
5	53.6			
2	58.9			
52	65.1			
67	81.1			

The Time Factor in Exposure

The affinity of CO for hemoglobin and its consequent cumulative effects render the *time* of exposure of vital importance. Henderson and Haggard have suggested a simple formula to determine the probable effect of a given concentration acting over a stated period. If the parts per ten thousand multiplied by the number of hours of exposure equals 3, the effects are negligible (1.5 parts times 2 hours). If the parts per ten thousand multiplied by the hours of exposure equals 6, effects are noticed (3 parts times 2 hours). If the parts per thousand multiplied by the hours of exposure equals 9, definite symptoms become apparent, *e.g.*, headache, nausea, etc. (4.5 parts times 2 hours). When these factors equal 15, life is endangered (7.5 parts times 2 hours). Fifty parts per 10,000 may be fatal in 30 minutes* (Fig. 99).

Respiratory Exchange

The rate at which the carbon monoxide is brought into contact with the capillary circulation directly influences the speed of blood saturation. CO poisoning at rest from a given concentration is one thing; poisoning while exercising is something quite different.

During ordinary respiration a man breaths 8–10 liters of air a minute. About six liters of this comes into contact with his capillary circulation. When exercising, a vigorous man may breath 36 to 64 liters a minute, or six to eight times the normal rate. Under general anaesthesia with a closed

* Henderson and Haggard, "Noxious Gases," 2nd Ed., p. 167, 168, Reinhold Publishing Corp., 1943 (where parts per million are given, multiply parts per 10,000 by 100).

method, without CO_2 absorption, a respiratory exchange of 35 to 45 liters is commonplace. The human organism seems well designed to accommodate itself to increased respiratory ventilation. It does not tolerate reduced respiration, especially when unusual demands are made upon tissue metabolism.

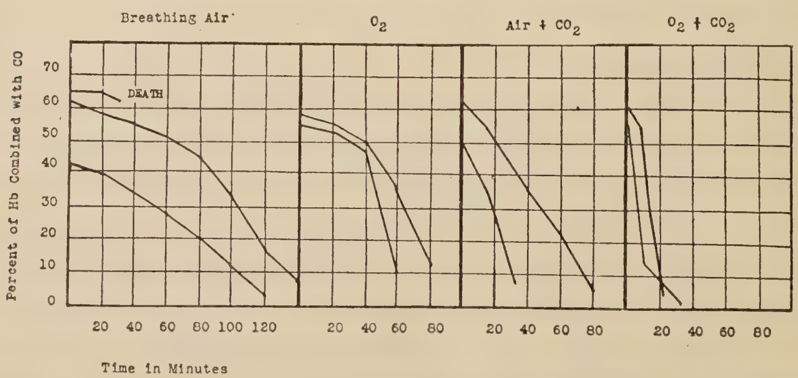
It can be readily understood, therefore, why little children whose respiratory exchange is greater than adults may be overcome by CO before their parents, asleep in the same bed-chamber, and why vigorous attempts at escape or rescue in an environment containing a high percentage of CO may produce rapid unconsciousness and death. The fireman or the civilian, without an oxygen mask, who rushes up the stairs of a burning house or attempts to carry a victim out of a burning building may easily perish. The only possible protection under such circumstances is to overbreathe pure air, to hold the breath while attempting rescue, or to use a closed circuit mask with oxygen.

Concentration, exposure and ventilation determine the intensity of CO poisoning. These three factors are constantly in operation and vary in every case. The total of these factors determines the immediate and the late lesions produced.

Immediate Effects

The immediate effects are those of a smoothly acting anaesthetic. Vertigo, headache, and impaired orientation are followed by muscular incoördination, weakness, spasticity, unconsciousness, muscular relaxation, and reduction in the activity of the reflexes.

If the time of exposure is brief, and if the anoxia has not proceeded to an engorgement of the vital organs, with ensuing petechial or gross hemorrhage with thrombosis or embolism, uneventful recovery follows a spontaneous return of consciousness or suitable treatment with oxygen and carbon dioxide. The period of recovery and the prevention of late effects turn upon how rapidly the CO is removed from the hemoglobin (Fig. 100).



Courtesy Williams & Wilkins Co.

Fig. 100.—Rate of elimination of carbon monoxide from the blood when breathing air, oxygen, carbon dioxide and air, and carbon dioxide and oxygen.

Carbon Dioxide Loss

A factor frequently overlooked, but repeatedly stressed by Henderson and Haggard, is progressive loss of CO_2 in CO poisoning. This may result in dangerous or fatal apnoea.

As a result of the anoxia produced by the oxyhemoglobin reduction, overbreathing takes place for the purpose of balancing the oxygen loss by a greater volume of respired air. Overbreathing by decreasing the CO_2 in the blood further reduces the oxygen available to the tissues (Bohr phenomenon). The use of pure oxygen under these conditions may therefore be dangerous. Carbon dioxide should invariably be administered with it.

The autopsy findings following the immediate effects of carbon monoxide are noted by Harrison Martland as follows. The most characteristic appearance is a bright pink color of the skin and post-mortem lividity. (This color is distinctive, and is not seen to such a degree in any other form of poisoning. However, a similar color, although not so pronounced, is sometimes seen in bodies which have been frozen or exposed to extreme cold, and sometimes in cyanide poisoning.) The color is most prominent on the most dependent part (in contact with the surface on which it is lying). Turning of the body will cause the color to shift if decomposition has not set in. There appears to be a great deal of cherry-red blood in the blood-containing organs. This is characteristic of the liver, brain, and stomach. Pulmonary edema is seen with bright pink froth in the airways. Patechial hemorrhages may be found in the cortical matter. Bodies exhumed retain carbon monoxide hemoglobin for months or years.

Late Effects

McNally states that central nervous involvement, severe anemias with consequent lowered resistance to infection, and cardio-vascular involvement with thrombosis and coronary disease not infrequently occur.

The syndrome of angina pectoris is attributed to anoxemia of the myocardium by Elliott and Cabot. Kiefer and Resnick note a striking resemblance between the effect of hemorrhagic bleeding and the breathing of low oxygen concentration and CO poisoning.

The classic anemic lesions in the heart of the victim of carbon monoxide poisoning are hemorrhage, necrosis and the process of granulation and regeneration (Kroetz).

Martland finds that, in cases which have been resuscitated and the respiration restored, the patient may remain in coma or become fully conscious. On examination the blood will be found free from CO. Such patients may die in coma in two or three days or develop pathological lesions due to hemorrhage into the heart or brain. The autopsy in cases which escape the immediate acute effects of CO poisoning but subsequently die reveals the following conditions. Bilateral degeneration of the globus pallidus of the lenticular nucleae, and of the pia arachnoid and parenchyma.

Other portions of the brain are affected histologically. The bilateral lesions are apparent to the naked eye; they vary from one to two centimeters in diameter, and are thought to be caused by the anoxia-producing thrombosis of the nutrient arteries. Lesions which produce late effects following apparent recovery begin with a selective anemia necrosis of the anterior portion of the pallidum, which soon drops down and becomes infiltrated with blood and leucocytes. The blood vessels become necrotic and autolysis follows. Patients with these progressive lesions recover but suffer from psychoses, disturbances of personality, or even imbecility. P. 228, Fig. 125.

Because of the ease with which lesions having no causal relation with carbon monoxide poisoning may be advanced in compensation claims, and because of the difficulty of bridging what appears to be a wide gap between cause and effect under these conditions, justice in medico-legal claims is likely to be thwarted until such time as the generic pathology of asphyxia is more generally understood and accepted. Popularization of the material offered in this book may contribute to this end.

Detection of Carbon Monoxide

While spectroscopic analysis of blood containing CO is useful, it is not sufficiently sensitive, since satisfactory readings require at least 8 per cent concentration. The Van Slyke method of gasometric analysis is preferred.

The following tests are suggested to determine the gross presence of carbon monoxide in the blood:

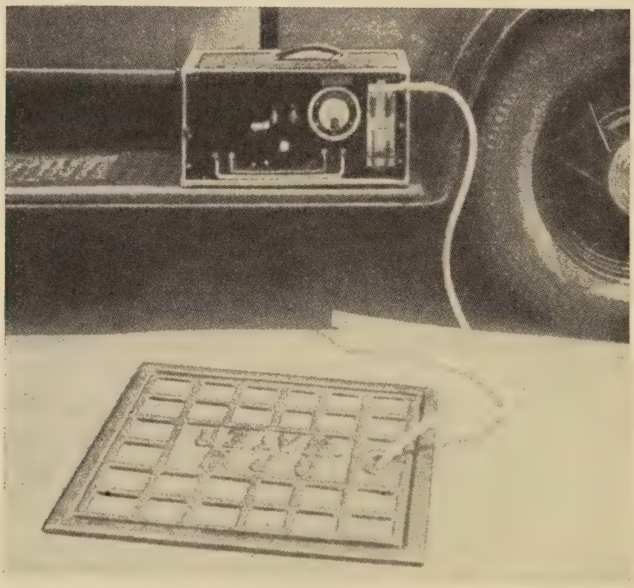


Fig. 101.—Carbon Monoxide indicator, testing sample of air from manhole. (M. S. A.)

(1) Boil blood: if normal it will be brown-black; if it contains CO it will be brick red.
(2) To 10 cc of a 2 per cent solution of blood add 2 cc of yellow ammonium sulfate and 2 cc of 30 per cent acetic acid; if normal the color will be green; if it contains CO it will be bright red.
(3) To a solution of dilute hydrogen sulfide water add blood drop by drop. If the blood is normal the color will be bright green; if CO is present the color will be bright red.
(4) To 0.1 cc of blood add 2 cc of water and 0.4 g equal parts of tannic and pyrogallic acids; if the blood is normal the color will be gray; if CO is present the color will be carmine red.

Prevention of Carbon Monoxide Poisoning

Insurance carriers are keenly interested in the prevention of asphyxial death from carbon monoxide poisoning. The Travelers Insurance Co. (Booklet, Vol. 16, No. 7) suggests a practical plan for safe-guarding workers in garages in which motors are operated indoors and indicates a ventilating system for a service station. The exhaust gases are sucked from the cars while engines are operating and discharged to the outer air from a high point in the garage by a suction fan. Multiple-intake plugs are provided at a convenient distance from the muffler pipes of automobiles undergoing repairs. The exhaust is connected to suction inlets by flexible tubing attached to a floor receptacle resembling ordinary vacuum cleaner inlets. The suction fan should be wired with the electric light circuit so that the switch will operate both simultaneously. This arrangement will prevent unwise economy in the use of the fan as well as failure to operate when the garage is in use.

The pipe line is ordinary stove-pipe buried in concrete at such a depth that the overlying concrete will not be crushed. The ducts should be laid on a slight but uniform grade, to avoid the accumulation of oil or gasoline which might ignite and explode.*

One of the best programs of prevention carried out recently has been that pursued by the Cities Service Oil Companies in an effort to reduce driving accidents due to CO gas leaking into closed cars and poisoning drivers. Reporting upon this work L. T. White states, "The smallest car will make 200 feet of exhaust gas per hour, the largest 1000. The CO content of this gas varies with the motor efficiency. Sensible differences in efficiency are not always clear to the driver. At 50 per cent efficiency the CO may be 13 per cent, at 80 or 90 per cent it may drop to 4 per cent. In two years the efficiency of 250,000 motors was analyzed. The vast major-

* The investigations of Harvey G. Beck in the field of CO are well known. See the following references:
West Virginia Univ. Bull. Series 43, No. 2-I (August, 1942).
Beck, H. G., and Suter, G. M., *J. A. M. A.*, *110*, 1982 (June, 1938).
Review Gastroenterology, **6**, No. 3 (May, June, 1939).
Southern Med. J., **30**, No. 8 (August, 1937).

ity were inefficient." It is believed that of one million non-fatal accidents occurring annually many may be ascribed to the effects of CO in affecting the driver's judgment, 85 per cent being unexplainable. Insurance groups lost $14,000,000 annually, in 1932 and 1933 (Aetna). It is stated that as a result of this situation the cost of insurance to the car owner often exceeds the cost of gas and oil.*

The contamination is due to incomplete combustion complicated by broken gaskets between the manifold and the exhaust, open seams in the muffler, muffler pipe terminating ahead of the rear wheels, and heaters drawing warm air from the front of the engine. The results of these investigations have been more tightly sealed car bodies and much desirable publicity regarding the nature of the hazard in terms easily understood by the public.†

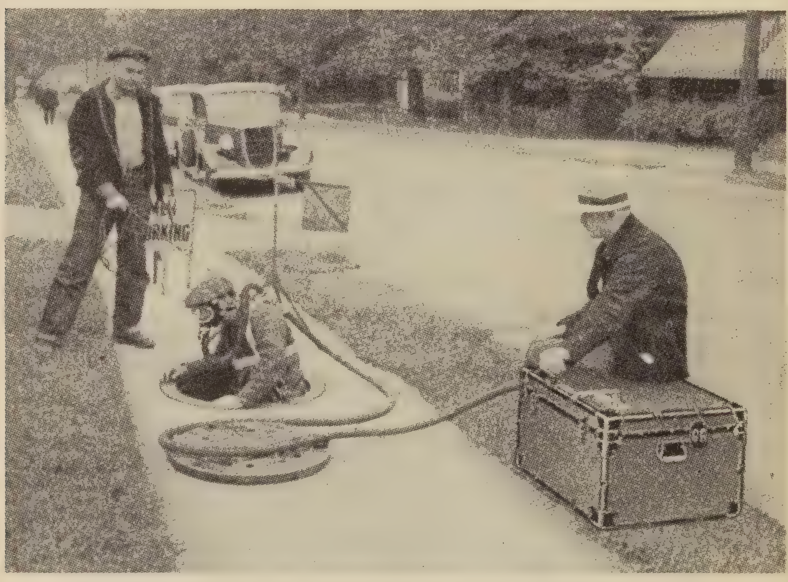


Fig. 102.—Testing sewer for carbon monoxide. Note blower, base, and mask. (M. S. A.)

* A survey was carried out by F. M. Van Deventer, mechanical engineer of the Cities Service Oil Companies, over a period of three years in cooperation with Governor's Committees, the National Safety Council, Motor Vehicle Departments and Highway Safety Groups. Two thousand cars in fourteen states were selected at random, and the composition of the exhaust gases and the car atmosphere was examined. Six per cent of these cars were found to contain enough CO to create a hazard to the driver's efficiency. It is estimated that a Derby hat full of exhaust gas in a sedan interior will produce symptoms in half an hour's drive. As Van Deventer puts it rather neatly "Imagine a million accidents on rubber rolling along the highway."

† These hazards will all increase with the use of old cars. The reduction in gas consumption will not counterbalance them.

Organization of Lay Rescue Squads

In 1921 at the request of the American Gas Association a Commission was formed, headed by Dr. Cecile Drinker, to consider the most efficient methods for artificial respiration in cases of carbon monoxide poisoning. The Commission reviewed the field and recommended the prone-pressure method of artificial respiration with oxygen and carbon dioxide as routine treatment by lay groups. In 1925 the New York Consolidated Gas Company became interested in the problem and in 1926 equipped its trucks with inhalators and trained crews. Dr. Daniel Donovan of the New York Police Department and Dr. Harry Archer of the New York Fire Department extended their cooperation and assisted in setting up a uniform technique for the use of their respective groups in accordance with the pattern suggested by the American Gas Commission. Literature was later distributed to the lay groups under instruction and to hospital administrators for distribution to their Ambulance Surgeons. This literature stressed the value of prone-pressure technique and the use of oxygen and CO_2.

Acting as a subcommittee of the American Gas Commission, Henderson and Haggard released a preliminary report in September, 1922. The point of this report was to prove that CO poisoning resulted in apnoea from overventilation of CO_2, and to point out the urgent necessity of combatting this apnoea and the mechanical means by which this could be accomplished (H. & H. Inhalator). The curve of CO dissociation from hemoglobin is shown in Fig. 100. The investigation and the report of this committee was based upon experience with 12 cases in various degrees of asphyxia.

While the investigation and the recommendations of the American Gas Commission were of the greatest possible value in establishing and coordinating a suitable routine for lay use, its effects upon the medical profession were most unfortunate. It reduced all degrees of asphyxia to a common level of importance; it implied that the treatment supplied by the mechanical performance of the lay squad aided by the inhalator left nothing more to be desired. Instead of stimulating the ambulance surgeon, as a trained medical man, to complement the first-aid work of the rescue squads, it produced not only a lack of professional interest in the problem but competition in rescue efforts, resulting in fatalities that might have been averted on various occasions by the employment of modern medical methods of treating advanced, flaccid asphyxia.

The consequence of thus freezing professional activity in the field of asphyxia has long been apparent. Calls to the rescue squads for assistance in asphyxia in the operating room have occurred. Resuscitation calls for asphyxia neonatorum have been a common occurrence, as reference to press releases will indicate. The effect of such a practice upon medical student and interne may easily be imagined; the dying asphyxiated patient was not his concern because the squad could provide a more perfect prone pressure technique than he could.

Nothing in medicine could be more absurd or cruelly fatalistic. The well-intentioned, highly informed sponsors of lay instruction certainly never anticipated that such a secondary effect to their efforts could develop, and yet it most certainly did. The immediate result of this unilateral emphasis has been that not one physician in a thousand can pass judgement upon resuscitation equipment, for the simple reason that he has never considered it worth his while to consider the problem of resuscitation seriously.

When the average physician looking upon impending asphyxial death is shocked into a realization that here and now is the supreme test of his life-saving skill as a physician, resuscitation will cease to be relegated to the care of those lay groups whose services have been and continue to be a bridge to span the period between complete helplessness and adequate medical care.

While the organized rescue efforts of the trained squads cannot be replaced, and should continue and be expanded, let us look to the medical student and the interne of today to direct and to perform the complete resuscitation procedures of tomorrow.

Chapter 11

Asphyxia from Submersion

Drowning occurs whenever sufficient fluid finds its way into the respiratory tract to produce asphyxiation. According to Gonzales, approximately 90 per cent of the submersion deaths which take place are due to obstruction from the fluid which blocks the airway; about 2 per cent are due to laryngospasm and circulatory failure. The remaining 8 per cent are caused by a combination of these. Therefore, in the language of asphyxia, anoxic anoxia accounts for more than 90 per cent of deaths by drowning, the balance being due to stagnant anoxia.

As might be expected, any accident which results in complete submersion automatically provides all the conditions necessary for 100 per cent anoxic anoxia. Fatal asphyxia can occur in water so shallow that only the nose and the mouth are immersed. Vomitus, blood, or abscess contents, if unrelieved by active coughing, swallowing reflexes, or by mechanical means, will drown the patient as effectively as sea water, 100 feet deep. The hazard of drowning on dry land is one which constantly threatens the unconscious patient.*

Because of the prompt and severe anoxemia which usually results from submersion in deep water and because of the inaccessibility of such accidents, skilled medical care is less likely to be available than first-aid treatment. Too much emphasis, therefore, cannot be placed upon immediate, skilled first-aid care, *i.e.*, posture, heat to the body, and prone-pressure artificial respiration with or without the use of an inhalator.

On the other hand, while a clear demonstration of the phenomena of asphyxia is presented by the submerged laboratory animal, it must be remembered that the victim of drowning appears for immediate attention without record of any kind. We must take him as we find him, and diagnose the degree of his asphyxiation from his physical signs (p. 167).

Furthermore, the degree or stages of recognized asphyxia are constantly changing. Asphyxia is not static. The state which we note at any given instant is but a milestone along the road from life to death, or in the event of correct treatment, from death to life. Our job is to control, if possible, the direction of this movement. It is our duty to provide the treatment which is acceptable to the patient. If accepted without resistance, it will be non-traumatic. Should mild asphyxiation with semi-consciousness ob-

* "Drowning in the Operating Room," *N. Y. Med. Week*, Feb. 3, 1940.

tain, for example, all the patient needs is to be kept warm and to be pushed gently along the road to recovery, by inhalations of oxygen and carbon dioxide. If he is spastic, more active measures are indicated; if flaccid, it is absurd to waste time by half measures. Give him all you have. There is everything to gain and nothing to lose when a man is poised on the brink of the grave.

So that the condition of the drowned may be correctly diagnosed and treated, calls for help should include medical aid. Physicians summoned to such accidents will in due time become familiar with the newer techniques. Attendance upon one submerged victim, as he passes from spasticity to flaccidity, under unassisted first-aid treatment, is all that is necessary to induce the physician to jump into the breech, relieve obstruction, supply adequate ventilation and reverse the direction of the movement from death to life.

The subject of submersion will now be considered in some detail. The viewpoint of the medical examiner, the laboratory research worker, the clinician and organized group efforts will be briefly considered. Dr. Gonzales has dedicated his popular text-books "Legal Medicine" and "Toxicology" to Dr. Charles Norris. Dr. Howard Neail, speaking of the late Dr. Norris, refers to him as the leading authority on asphyxia in the Western hemisphere. The author is fortunate in the possession of an unpublished paper given by Dr. Norris before the Biltmore Conference of the S. P. A. D. The following excerpts are therefore of special interest.

The Medical Examiner's Viewpoint (Dr. Charles Norris)

"Drowning may be classified as follows: accidental, suicidal, homicidal, and deaths from natural causes in water."

"It will be recalled that in ancient days in Europe, people were drowned as a punishment. This occurred in Scotland and England, and all over the Continent. The last cases of official drowning that I have knowledge of were in Iceland in 1776, and in Austria in 1777. Usually women were chosen because it was considered that this was a less tragic way of disposing of the life of a person. And in some parts of Germany, to show how much they thought of their women, they were drowned in mud!"

"Very few homicides are committed by drowning. In a visit to the Villa Teste at Tivoli, however, I learned that one of the favorite walks on the veranda had a trap door. When they wanted to dispose of their guest they opened the trap door and the body fell through into a stream about fifty feet below."

"It is, however, quite natural to believe that a few people may be disposed of by being thrown into the water from a boat and without witnesses; of course, these cases escape the attention of the authorities. However, there are certainly a large number of bodies which have been assaulted, and also hit-and-run cases where it is easy to dispose of the body on account of the proximity of the river or ocean. This is the reason for the most careful examination of a body if there is any suspicion of foul play. A person thrown into a river after death will not show signs of drowning."

"In regard to suicides, undoubtedly, before the advent of the general use of gas, drowning was a much more frequent way of disposing of one's life than it is now. For instance, in the depression statistics 40 per cent of the suicides in women were due to drowning, compared with only 14 per cent of men. The introduction of illuminating gas into homes made this a favorite method of self-destruction."

"Death in water from natural causes is at times difficult to determine if the pathological lesions, namely, a fibrous myocarditis with coronary sclerosis or cerebral hemorrhage, is present; and if there are none of the classic signs of drowning and the chemical examination of the heart's blood shows that the sodium chloride content is normal, the determination is not easy. I admit that if death is due to a laryngeal inhibition similar to the sudden deaths due to the choking of the larynx with a piece of meat or other foreign obstacle, that the determination is extremely difficult and may be impossible, unless the surrounding circumstances are ascertainable through witnesses. In all cases of drowning in which a history of diving is presented, a careful examination of the neck must always be made for a fracture of the cervical spinal column. Such casualties usually float on top of the water face downward after crushing the cord; naturally they die from drowning, and have the typical appearance of drowning. The death certificate, however, should include the real cause of the drowning which is, of course, a fracture of the neck with compression of the cervical spinal cord."

"We can all realize the tremendous number of tragedies which drowning has brought upon civilization. Drowning is an asphyxial death. It is essential only that sufficient water be present to cover the nostrils and mouth. Cases of drowning in shallow pools with only a few inches of water have frequently occurred, the victims being children, epileptics, or persons comatose from alcohol."

"Let me call attention to the classic description by Sidney Smith.* He states that a person falling into the water sinks immediately after the fall, and with the force of gravity breathing stops immediately. Deaths occur from shock, injury, or heart failure due to heart lesions or inhibitions. Usually the person rises to the surface because of the natural buoyancy of the body and tries to save himself. Violent attempts are made at reaching the surface; if the head is under water the victim inhales a certain amount of water and thus increases the dyspnoea. When a person strikes cold water he swallows a certain amount of it, and some of it reaches the laryngeal mucous membrane. This is really the first stage of drowning. It is known as "respiration de surprise". Inhibition of respiration ensues. This stage is of very considerable importance. In other words, a person may die in water and if respiration does not start again he will not inhale water. Thus, although he dies from asphyxia, his body will not present

* Sidney Smith, "Forensic," p. 266, London, J. & A. Churchill, 1931. Martland "The Medico-legal Necropsy," Williams & Wilkins, Baltimore, 1934.

the picture of a typical drowning case; in other words, water will not be present in the lungs, which will not be ballooned. Chemical examination of the blood of both sides of the heart will be negative. As is now well known, when a person drowns in salt water and inhales a considerable amount of it, the salt content of the blood of the left heart will be increased on account of the absorption through the air vesicles, for the reason that salt water contains more sodium chloride than does the blood."

"Drowning in fresh water is directly the opposite in this respect: there will be less sodium chloride in the left heart than in the right. The chemist who is not informed by the pathologist will report that the death is not due to drowning, because there is no essential difference in chloride content of the left and right sides of the heart. This, is, of course, a grievous error. We must also recollect that, although the chemist may find a very considerable excess of sodium chloride in the left heart, an amount which clearly indicates drowning, this finding means only that the man has died in water, that his terminal agony has taken place in it. It would be a mistake to entertain the idea that the chemist can determine the cause of death accurately, because he is not aware of the circumstances attending his presence in the water. Also he is not acquainted with the pathological lesions that are found in the body. For instance, if a man is shot on a lake—in the head, let us say—he does not die immediately from hemorrhage, but falls into the water and his suffering takes place in water. The terminal stage, of course, is drowning; but the reason he drowned was because he fell into the water after being shot. At times the determination of the actual cause of death is extremely difficult. For instance, in the Dorothy Faithful case we have a woman who had a three plus alcohol, *i.e.*, was intoxicated; she had a very large amount of luminal, and she died in the water, as indicated by the examination of the blood of the right and left heart."

"There are also a number of instances in the literature in fairly well authenticated cases in which perforated ear drums have caused syncope and consequent drowning. The post-mortem signs in the recently drowned are pronounced and they offer no difficulty in determination. The convulsive movements which occur in the terminal stage of asphyxia, in which automatic respiratory efforts cause water to be drawn into the bronchi and air into the air vesicles, produce the characteristic ballooning of the lungs. According to Smith—and the author believes that all authorities agree with him—the extent of ballooning of the lungs depends upon the duration of the terminal stage and the extent of post-mortem signs, which will vary with the struggle."

"As in almost all asphyxial deaths, the heart does not stop beating immediately, especially the right auricle, which may continue to beat after respiration has ceased. This is known as the "ultimum moriens Halleri" The beating of the heart is favorable to attempts at resuscitation; and if this is not delayed too long it will be successful."

"Authorities agree that one cannot determine that a body has died from drowning on external inspection alone. The characteristic foam at the nose, so typical of drowning when bodies are brought up shortly after death, is not typical of drowning, as it is present in all cases suffering from pulmonary edema. In ponds and other shallow waters it has been observed that the hands were grasping weeds and such material."

"Another important sign of drowning is the presence of water in the stomach. It is characteristic of drowning, but it may be concealed when a person has been drinking before the accident, or if the deceased has had a very large meal. In many cases the water extends into the duodenum, or the upper portion of the small gut. I have usually found an absence of fluid in the duodenum or small gut. I do not know the reason for this; but it is generally known that when water is swallowed in fairly large amounts it passes through the stomach very quickly along the so-called *wasser strasse*. It has been shown by x-ray plates of the stomach that water passes readily into the duodenum through the pyloris. But some fluids, such as buttermilk, will not pass through into the duodenum. It is quite possible that in drowning in fresh water the water will pass into the duodenum and the upper small gut more readily than it does in salt-water cases."

Resuscitation Experiences at Coney Island

It was the author's privelege to be associated with Dr. Pol Coryllos in an investigation of the practical aspects of submersion as these occurred on the Coney Island beach during the summer of 1930. Coryllos, former Surgeon General of the Greek Army and private physician to the King of Greece, possessed extraordinary originality and an encyclopedic memory. He was simple, charming and completely devoid of affectation; nothing mattered except the task in hand. A sufferer from angina pectoris, he paid no attention to his malady, of which he ultimately died (August 1938).

Our practice was to assemble early on Sundays and holidays at the Coney Island Hospital. Through the courtesy of the Superintendent, Dr. Scherf, we were assigned a resident, Dr. Plain, and an ambulance. We tapped the intake manifold of the ambulance engine and secured perfect suction.* This was connected to a brace of glass bottles. We were equipped with portable tanks of Oxygen 90, CO_2 10, reducing valves, mercury manometer, nasal tubes, portable suction, laryngoscopes and endotracheal tubes, and an H. and H. Inhalator.

Upon arrival at the hospital, we contacted the Police Department and awaited calls which were sent us through the courtesy of the Chief of Police. On Sundays and holidays visitors on the beach numbered upward of a million persons.

The difficulty of locating the victim is emphasized by a call which we answered on one hot Sunday morning. Weaving noisily through the Sun-

* Page 60. Fig. 21.

day traffic we made our way to a blind street ending at the board walk. Each of us carried equipment: Coryllos had a stretcher; the driver, the house man and I carried gas tanks and instruments. Over the board walk we went, dropping into a sea of legs, arms, lunch baskets, children and adults who paid not the slightest attention to us. In a few seconds, we were separated and effectively lost. Unable to locate my associates or the patient, I went back to the ambulance and stored my equipment. Then I set out to look for Coryllos, who had with him nothing but a stretcher. He was in a shack with the semi-conscious patient, doing prone-pressure artificial respiration.

Experience resulted in future close team work. We found that our patients were in extremes of asphyxiation. A woman just pulled out of a pool was in a dazed, semi-conscious state. She needed treatment for exposure; any thing more would have been meddlesome. Again, we were called to a man on the water's edge, lying flat on his stomach. His face was purple; his mouth and nose were filled with vomitus and blood. The mask of an inhalator was being pressed against this mess while he was getting prone-pressure treatment. We took off the inhaler, put a nasal tube down one nostril and blew in oxygen and CO_2. His color promptly cleared. We rolled him over on his back and with the help of bystanders lugged him back to the ambulance. Oxygen meanwhile was blowing through the nasal catheter into his pharynx. In the ambulance we laid him on his back and extended his head. We sucked out his throat with the ambulance engine suction and continued to give him intra-nasal oxygen and CO_2. His color improved. His respiratory rate and volume increased. Arrived at the hospital, we put him on a ward treatment table and observed his subsequent recovery. Coryllos in the meantime demonstrated the effects of CO_2 on his respiratory rate and volume.

Another call was at some distance. A man was pulled out of the bay. He was absolutely limp, completely flaccid. His pharynx and glottis were exposed without the slightest difficulty. His airway was filled with froth which we removed as it oozed out of his glottis. The vocal cords were absolutely silent. In death, which had arrived well before we did, this man exhibited the most complete and absolute flaccidity which we had ever encountered. Such flaccidity is absolute. It is more marked and presents a better field of exposure than a cadaver in rigor or in the refrigeration of the morgue. Flaccidity immediately follows loss of muscle tone and disappearance of the reflexes.

This was demonstrated upon another occasion. While the author was anaesthetizing a patient one day in a New York Sanatorium, the surgeon became ill, laid his head on the patient and fell to the floor. In less than a minute the author laryngoscoped and intubated him on the tile floor of the operating room. The glottic reflex had disappeared. There was not the slightest resistance to instrumentation. Mouth-to-tube insufflation was performed without result. He was dead from circulatory failure.

This case is cited to illustrate the writer's conviction that the patient will indicate the treatment which he needs. He will resist meddlesome interference by reflex and by muscle tone. In the absence of resistance he is entitled to all the operator has to give him.

Laboratory Research

With characteristic scientific curiosity and drive, Coryllos promptly proceeded to put our clinical experience into reverse. He went from patient back to laboratory animals to try to learn what order, if any, could be arrived at from our scattered observations. He drowned approximately 55 dogs under controlled conditions at the Loomis Laboratory.* Four phases of asphyxia became clear.

"The first phase is characterized by apnea and rise of blood pressure. This apnea is due to the reflex closure of the glottis. I shall call it the phase of initial apnea. Rise of blood pressure at the beginning of asphyxia is a constant phenomenon in all forms of asphyxia. The duration of this phase is about 1 minute.

"The second phase is characterized by labored respiratory movements and continuation of the rise of blood pressure. The animal fights for breath; the non-anaesthetized animal is perfectly conscious until the end of this phase and fights vigorously to liberate itself of the hood. At the end of this phase, which also lasts 1 minute, consciousness disappears and tonic and clonic convulsions appear. This phase can be called the 'phase of dyspnea.'

"The third phase is characterized by arrest of respiration, drop of the blood pressure, and disappearance of reflexes and muscular tonus. The duration of this phase is also 1 minute. The animal, which presented clonic and tonic convulsions at the end of the second phase, gradually relaxes and becomes limp; its jaw drops, the sphincters relax and the corneal and glottis reflexes disappear. I shall designate this as the phase of 'terminal apnea'.

"The fourth phase is characterized by rapidly progressing weakness of cardiac contraction and arrest of the heart. I shall indicate this as the phase of arrest of the heart." *

It is to be emphasized that these experiments were all conducted with an endotracheal tube in place. Such an arrangement completely eliminates the chief difficulty in artificial respiration, namely, respiratory obstruction in the upper airway. For this reason, the mechanics of the technique of artificial respiration employed cannot, for obvious reasons, be translated to the asphyxiated patient, whose airway is embarrassed by muscle relaxation, blood, vomitus, sea water or other material. To attempt to secure the results reported by Coryllos by the mere application of a mechanical device providing transpharyngeal insufflation (page 77) is not only to court failure but to preclude a more rational approach.

* *Surg., Gynec. and Obstet.*, **66**, 698 (1935).

Therefore, in order to simulate the successful resuscitations which were reported by Coryllos, it is absolutely essential that the original conditions of the experiments be duplicated in the patient, *i.e.*, he must be laryngoscoped and intubated before the application of mechanical resuscitation. Once the endotracheal tube is placed and insufflation of O_2 and CO_2 carried out intermittently under measured pressure, the results achieved by Coryllos at the termination of the stage of terminal dyspnoea may be expected to take place in the asphyxiated human victim. We know this to be true in the new-born baby; the submerged patient is entitled to as much. The mechanical method employed by Coryllos included the use of suction (mechanical expiration). It is unfortunate that the reasons for this feature were not stressed. We look in vain in the pathological physiology of asphyxia for indications for using suction (page 24).

When intermittent O_2 and CO_2 insufflation is made available through an endotracheal tube, the author heartily subscribes to the following statement: "This experimental evidence shows clearly, I believe, the importance of dividing asphyxia by submersion into four phases. Moreover, it shows the futility of artificial respiration and carbon dioxide in oxygen inhalation beyond the third phase and the necessity of avoiding any loss of time by the immediate application of procedures which insure insufflation of oxygen into the lung. The fact that resuscitation is still easy at the beginning of the third phase, while it is very difficult and often impossible at the end of the same phase, and that the duration of this phase is of 1 minute only, shows clearly the paramount importance of time. Seconds count in asphyxia and often determine the life or death of the patient." This experimental evidence illustrates the statement previously made that unless the phase of asphyxia is accurately indicated in the results obtained with different procedures of resuscitation, no reliance can be placed upon the results reported.

Comments on Diagnosis

Prevention. Because of inaccessibility, and the severity of asphyxia from submersion, preventive measures are to be emphasized.

The most frequent cause of death in the United States Navy is submersion. This was recently displaced by automobile accidents but owing to gasoline curtailment and present greater sea exposure, submersion once more has taken the lead.

The most recent life-saving apparatus which has appeared consists of inflatable life preservers, rafts and even life boats. Cartridges of carbon dioxide, not unlike the old-fashioned sparklets, provide the inflating gas. This container, part of the standard equipment, releases its contents spontaneously on striking the water, or is released by hand. The sustaining power of these rafts is such that a recent rescue, that of Dixon, Aldrich and Pastula, took place 34 days after they were cast adrift. A rubber doughnut inflated by CO_2 is also said to have saved the life of the only survivor of the United States Torpedo Squadron Eight.*

The "Military Medical Manual" for 1940 lists ten "don'ts" for the prevention of drowning:

"(1) Don't swim immediately after eating; wait for two hours.

"(2) Don't swim alone.

"(3) Don't swim if overheated.

"(4) Don't swim if you know that you have heart trouble.

"(5) Don't continue to swim when exhausted.

"(6) Don't wade into water with the hands above the head; you may step into a hole. Be ready to take a stroke.

"(7) Don't struggle if caught in a swift current or undertow; the force of the current will bring you to the surface.

"(8) Don't fight or struggle if you swallow water; clear your windpipe of water first.

"(9) Don't cry for help in fun; you may really need it some time.

"(10) Don't dive in strange waters."

The "Handbook of the Hospital Corps United States Navy" gives the following excellent advice:

"To treat a case of unconsciousness is one of the most difficult things that may fall to the lot of the First-aid man. In all cases of unconsciousness strenuous efforts should be made to bring the patient under the care of the Medical Officer as soon as possible. No reliance should be placed upon any special mechanical apparatus as it is frequently out of order and often is not available when needed".

Both Army and Navy recommend that the patient be lifted by the middle (jacknifed) to help drain fluid from the airways. It is also recommended that pressure be applied to the back and loins for the purpose of expressing water from the stomach as well as the lungs. Both services recommend folded blankets under the chest of the patient in the prone-pressure method.

Civilian. The drowned are usually strong swimmers 25–35 years of age. Neglected little children and babies drown in lily pools, excavations, and bath-tubs. One should never swim alone. Use the buddy system (two at a time) or be accompanied by a boat with two occupants. If overtaken by cramps in the arms or legs, submerge and pinch the cramp hard. In a heavy sea float, do not attempt to swim. To avoid cramps, keep out of water for two hours after meals. If you do not know how to swim, it is never too late to learn, until suddenly it will be too late.

Rescuer. Emphasis should be placed upon mechanical means of rescue, says Martland, instead of on swimming. Spot a board, oar, pole, or anything that will float and get it out to the victim. A line or an inflated tube may be near by. When on ice, lie flat upon it. Tie a line to the shore and

* *Science News Letter*, Jan. 9, 1943; abstracted in *Reader's Digest*, p. 83, Feb. 1943.

spread your weight on the surface. Clothes contain air and will help you to float for a time; shoes will not. If the victim grabs you, duck him until he lets go; then tow him to shore.

It is possible that the muscular cramps sometimes experienced during swimming are due to hyperventilation. This is caused by a combination of voluntary deep breathing and reflex deep breathing from exposure to cold water; sometimes too the element of fear is introduced. We suggest that professional lifeguards observe the respiratory behavior of such persone in distress because, to our knowledge, this possible explanation of the phenomenon has not been suggested previously.*

Diagnosis. The drowned sink and remain under water until gases due to decomposition form in the body and raise it to the surface. It is then known as a "floater." Exceptions occur in the case of fat persons or small children, who remain on the surface. When the body is washed ashore, the skin is found to be pallid and covered with *cutis anserina*, due to the rigor mortis of the muscles in the hair follicles. The hands resemble washerwomen's. The face is bloated and cyanosed. The eyes protrude and are injected. A thin foamy exudate is present in the mouth and nose. The hands clutch sand, sea-weed or whatever is within reach. Examination should be conducted for injuries sustained before the victim struck the water, *i.e.*, gun-shot or other wounds, a broken neck due to diving, cardiac lesions, acute alcoholism, syncope or CO poisoning.

The diagnostic sign of death by drowning is the heart-blood sodium chloride test of Gettler. The laboratory test for Gettler's sign is described by Gonzales. Gettler states that this sign has been found conclusive in 99 per cent of the cases examined. The principle involved is as follows. Water, upon entering the lungs, is absorbed by the alveolar capillaries and enters the pulmonary veins (carrying oxygenated blood to the left auricle); consequently the blood in the left auricle will have a higher percentage of salt, if sea water has been inhaled or less salt if fresh water has been inhaled, than will the right auricle. The normal sodium chloride content of blood is 5 mg to 100 cc. In a taxi driver who drove his car off a wharf into the river, Neail found the left auricle to contain 849.6 mg of salt per 100 cc and the right 447.2 mg per 100 cc, a difference of 402 mg. The longer the heart beats, the higher will be the salt concentration in the general circulation, but the auricle near the source of the absorption, the left, will be the higher. Gettler reports a case of a child drowned in an open cellar filled with rain water. Examination of the blood of the left auricle showed a concentration of 548.5 mg of salt and the right 509.6 mg. Since the site of the accident was far from salt water, the finding proved puzzling until it appeared that a grocer had emptied a barrel of salt near the excavation. Gettler therefore suggests that a sample of the water in which the victim drowned be submitted for examination. Fresh-water pools are often clorinated, and bathing salts are found in bath-tubs.

* *Proc. Staff Meeting, Mayo Clinic,* **16,** 209 (April 2, 1941).

Treatment of the Drowned *

Laboratory animals artificially drowned under controlled conditions clearly indicate four stages:

Initial apnoea (1 minute) Laryngeal irritation

Dyspnoea (1 minute) CO_2 Stimulation plus Hering-Bruer reflex; partial anaesthesia of reflexes

Fig. 103. Asphyxial death from submersion, showing foam in mouth. Drowning occurred in bathtub. (Courtesy Dr. Harrison Martland.)

Terminal apnoea (1 minute) Loss of reflexes and muscle tone

Apparent death (1 minute) Respiration completely stopped, circulation failing

Total time in complete submersion, 4–5 minutes. Spontaneous respiration may be expected through "dyspnoea" and the beginning of "terminal apnoea".

* "Functional Swimming and Water Safety Training Course", Instructor's Manual, American Red Cross, Washington, D.C. (Special War Information.)

Endotracheal insufflation of oxygen is effective through "terminal apnoea" until the blood pressure has dropped to 20, when it will probably prove ineffective. Since accidental submersion is not under control and its degree cannot be known except through a diagnosis of the signs of asphyxia, one must assume that in cases of doubt it is never too late to apply treatment.

If the rescued is semi-conscious but can be roused (depression), his submersion probably did not extend beyond "dyspnoea". He should be treated by posture, heat and oxygen and CO_2 inhalation only. If he is

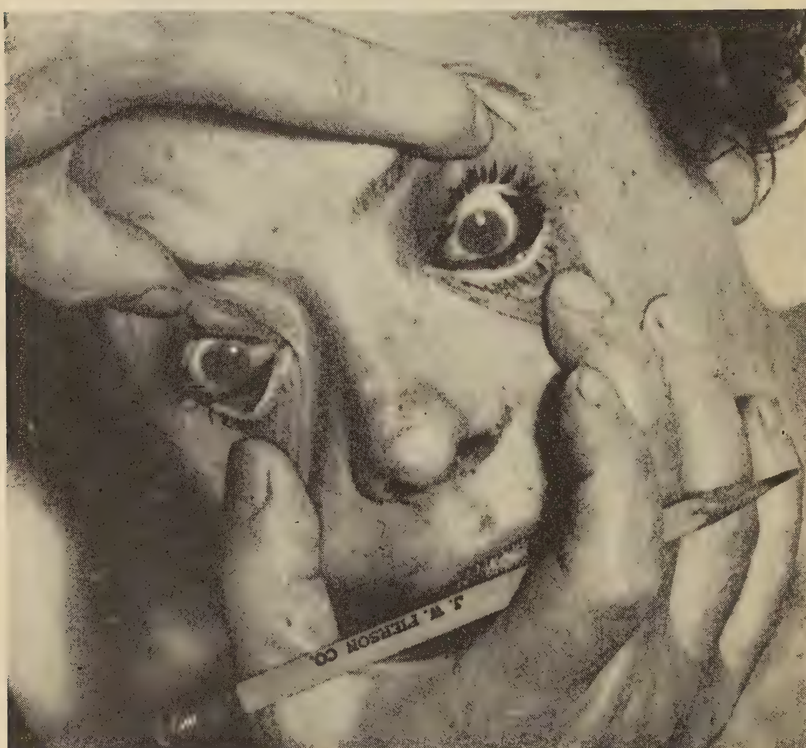


Fig. 104. Asphyxial death from submersion, showing conjunctival hemorrhage.
Courtesy Dr. Harrison Martland

unconscious and cannot be roused (spasticity), his submersion may have extended into and through "dyspnoea". He should be sucked out, if obstructed, and receive nasopharyngeal oxygen by inhaler or catheter with prone-pressure artificial respiration. Voluntary respiratory efforts will pick up pharyngeal oxygen-CO_2 if permitted to do so, and will be more effective than manual methods.

If the patient has ceased to breath and is completely relaxed without a palpable pulse (flaccidity) he was probably submerged through "terminal apnoea" into the stage of "apparent death".

If prone-pressure is used in asphyxia of this degree it cannot be expected to be of real assistance, since the loss of muscle tone of the respiratory cage renders the elastic recoil of the chest ineffective (page 57). Laryngoscopy, intubation and endotracheal insufflation are mandatory, if life is to be saved.

The indications for the treatment of asphyxia apply in submersion as well as in every other type of asphyxia. Any apparatus which is capable of meeting these indications is recommended (page 61).

The rescue squads of the fire, the police the industries and the volunteer life-saving groups are rendering excellent first-aid treatment. The use of suction, and intra-nasal insufflation might well be added.

The missing link is skilled medical diagnosis and treatment. It is to be hoped that this link will soon be strongly forged.

Physical Signs of Asphyxia in Submersion

Depression. Patient semi-conscious, can move about, swallow, vomit, and speak incoherently, opens and closes eyes.

Unconscious: At intervals but may be roused

Respiration: Normal or interrupted by vomiting or retching

Color: Normal

Muscles: Pseudo-relaxation

Reflexes: Active

Heart: Normal, pulse accelerated

Special Senses: Eyes moving; lid reflex active, pupils dilated; nose and throat reflexes active.

Spasticity. Spasm or incipient relaxation. Vital functions beginning to show embarrassment from progressive anoxic anoxia.

Unconscious: Cannot be roused

Respiration: Rate rythm and volume depend upon the degree of respiratory obstruction from spasm of masseters, sea water, vomitus or blood

Color: Moderately or deeply cyanosed

Muscles: Rigid or beginning relaxation

Reflexes: Coughing and swallowing reflex reduced in activity; laryngeal reflex active; sphincter reflexes beginning to relax

Heart: Sounds strong; apex beat visible; pulse of anoxia full and rapid or slowing

Special Senses: Eyes moving or fixed; lid reflex absent or sluggish; corneal reflex active.

Flaccidity. Muscle tone gone, patient apparently about to die.

Unconsciousness: Complete

Respiration: Gasping and irregular

Color: Pallor; heart beginning to fail

Muscles: Completely flaccid, resembling the relaxation of spinal anaesthesia

Reflexes: Sphincters relaxed, pharyngeal reflex gone; glottic reflex sluggish or absent; the tone of the vocal cords on endotracheal suction is a measure of the residual vitality

Heart: Sounds faint or not audible; apex beat feeble or not palpable; a three-inch needle injected into the heart will move if there is any heart action

Special Senses: Pupils dilated; corneal reflex sluggish or gone; eyeballs fixed.

It is to be recalled that these stages merge: there are no clear-cut demarcations; furthermore, the patient moves from one stage to another. The spastic patient may recover to depression or deteriorate into flaccidity.

Volunteer Life-saving Corps of New York

This Corps, organized in 1870, received a charter from the State of New York in 1890. Its purpose is to establish and maintain life-saving stations at various points along the waterfront of Greater New York City. It has more than 60 stations in the five boroughs. Active membership is about 5000 members, all of whom are volunteers. The Corps' duties include beach patrol and water rescue.

Submersion Studies and Rhode Island Inspector's Report

Life-saving in water accidents has reached a high point of perfection in Rhode Island under the able leadership of Captain Roger Wheeler. The Inspector's report, which appears below, is an example of the care and thoroughness which has characterized the rescue work done in that state.

Suggestions for County and State Submersion Studies

Purpose: To reduce mortality.

Method: (a) Study of physical conditions of locality in which accidents have occurred.

(b) Study of post mortem findings.

(a) **Physical Conditions**

Official vital statistic records to be scanned for location of accident.

Repeated accidents at the same spot to designate a "sore spot."

"Sore spots" to be indicated on a map.

These "sore spots" to be segregated in the order of accident incidence.

Important: "Sore spots" to be thoroughly checked up by at least two investigators.

The investigation to cover the following questionnaire, to be illustrated by sketches and by photographs of the location, and to be completed by recommendation for safety.

A full and detailed report of the "sore spots" to be assembled, and their relation to the nearest hospital facilities to be noted. Hospital authorities to be notified of these findings.

Inspector's Report

Village — Inspector
City or Town — Date

(1) Name of swimming or skating site
(2) Approximately, how many persons frequent this area?
  (a) Swimming season (yearly) — Daily — Week Ends
  (b) Ice Skating (yearly) — Daily — Week Ends
(3) Have any drownings occurred here — How many?
(4) Name of seasons: Skating — Swimming
(5) Is this site dangerous at any time?
  (a) Summer
  (b) Winter
(6) Who controls this property?
(7) What is the area of this site?
(8) How deep is the water at most dangerous area?
(9) Is night skating or swimming allowed?
(10) Are any life guards present?
  (a) How many life guards present?
  (b) How many life guards on duty?
  (c) Are these guards members of any life saving unit? If so, what unit?
  (d) What hours do the guards patrol?
  (e) In cases of ocean bathing are there life boats present?
(11) Is there any equipment in use here? (Go into details)
  (a) Is the equipment in good condition?
  (b) Is equipment needed?
  (c) Just what equipment is needed?
  (d) What is age of equipment other than boats?
(12) Are guards required to use life boats?
  (a) In cases of ocean bathing, are guards given any examination as to seamanship ability?
  (b) Who gives this examination?
  (c) What equipment is included in boat gear?
  (d) Is life boat used only as such?
  (e) What is length of boat?
  (f) How many persons does it hold?
  (g) Is it capable of the same?
  (h) What is the age of the boat?
(13) Is there a life guard tower here?
  (a) How high is tower?
  (b) If no tower at site, do guards use other elevated means for observations?
  (c) How high is this platform?
  (d) Is tower in good condition?
  (e) Is there any immediate need for additional towers?
  (f) How many?
(14) Is there any First-aid room close at hand?
  (a) How far from edge of water?
  (b) Is First-aid kit or cabinet in room?
  (c) Is present kit or cabinet suitable to present demands?
  (d) Are there any blankets in room? — How many?
  (e) Is room in sanitary condition?
  (f) Is there a First-aid man close at hand?
  (g) Is he qualified to administer First-aid treatment?
  (h) Is there any bed, cot, bench, etc in room?
(15) Are signs placed about site warning persons of dangerous areas?
  (a) Are those signs suitable?
  (b) Are more signs needed?
  (c) Are red flags used to mark dangerous conditions?

(d) In ocean bathing area, what are means of notifying people of undertow, etc?

(16) When life guard needs assistance for resuscitation who is called upon?
(a) How far is this help from site?
(b) Through what medium are they called?
(c) Is response good?
(d) Go into detail if guard has no available assistance
(e) How long does it take aid to arrive here?

(17) If fire and police departments responds to calls of assistance from guards, what equipment do they carry?
(a) Fire Department
(1) Is equipment in good condition?
(2) Is equipment sufficient?
(b) Police Department
(1) Is equipment in good condition?
(2) Is equipment sufficient?

(18) Are there organized First-aid crews in these departments?
(a) What is the equipment?
(b) Are members of the police and fire department First-aid crews qualified for such work?
(c) How often are drills held?
(d) Would these departments welcome any further instructions?

(19) Do the residents of this area realize any existing danger?
(a) Were steps ever taken for the improvement of the above?
(b) If nothing has been done for the correction of any dangerous conditions, who are the responsible persons?

(20) Give the names of the following:
(a) Chief of Police Tel. No.
(b) Chief of Fire Dept. Tel. No.

I have this day interviewed the official listed below and information here upon received are true statements:

Witness:
Address:
Phone Number:
Inspector:

Note: The above Inspector's report was supplied through Captain Roger Wheeler of the State of Rhode Island, Department of Health. The experience of Rhode Island indicates:

50% of all submersions occur in June, July and August.

32% in children of school age (Board of Education approach).

68% adults, suggesting the need of general publicity.

It was found that where equipment and shelter were provided, volunteer personnel was not difficult to secure.

Fresh-water resorts, lakes, rivers, and pools deserve close attention.

Suggestions re Post Mortem Findings

These records may be secured through the files of the Registrar's offices, coroner's records, or hospital records. They are to include the available history and findings of each case and should be translated into graphs indicating the incidence of floaters and victims who might have been saved.

Chapter 12

Asphyxia in Anaesthesia

It would be difficult to find a field more replete with opportunities to study anemic, anoxic and histotoxic anoxia than that of general anaesthesia. Indeed it is strange that these opportunities have not been utilized by those engaged in other activities and the material employed to mutual advantage. Such investigation need not be traumatic or offer additional hazard to the unconscious patient. The phenomena of mechanical obstruction, the reaction to variations in quality and pressure of gases administered, the effect of blood loss, the result of surgical trauma and the relation of reflex activity to muscle tone are constantly available for study. Why do not internal medicine, neurology, and Departments of Physics and Chemistry utilize this priceless human material? Is it perhaps because the anaesthetist, serving always as assistant to the surgeon, is deprived of initiative as a physician and may not presume to express an active interest and participation in other medical fields. A few brilliant exceptions seem to suggest this rule. While anaesthesia is by its terminology limited to the control of pain, declarations to the contrary notwithstanding, the physician familiar with deliberately induced unconsciousness is in an extraordinarily advantageous position to offer a very special service when faced with accidental unconsciousness. The recent organization and standardization of the field of anaesthesia increases the value of the operating room as a laboratory of clinical research. The anaesthetist carries on his activity throughout the entire field of unconsciousness. Other groups seemingly cease to function, except in a remote and detached manner, when the patient has lost consciousness.

Without doubt the author's insistence upon the need of a new terminology for the purpose of integrating the dissociated fields of gas therapy, has been interpreted by anaesthetists as an attempt to reduce the importance of anaesthesia by making it part of a larger specialty, *i.e.*, pneumatology. This objection, it is felt, will largely disappear if the difference between the field and the worker in the field is more clearly understood.

In medical terminology anaesthesia is a categorical imperative. Its meaning and the logical limitation of this meaning, do not seem to leave much difference for opinion. The meaning attached to anaesthesia, the control of pain, is not questioned by the author.

On the other hand, the man administering an anaesthetic is a physician first, an anaesthetist afterward. If this were not true he would be merely a technician. As a physician he has a duty to devote his special experience in the care of the unconscious to include the saving of life as well as the control of pain. No one else in the medical field is as well prepared to offer this service. Acting in this broad capacity he is obviously more than an anaesthetist. His widened activity requires a designation which is both scientific and inclusive. How, it may be asked, can such a normal evolution of the physician administering anaesthetics be resented by the true physician?

The needs of resuscitation and oxygen therapy require that the physician utilize his special skill in the wide field that he is qualified to treat.

In 1936 the Society for the Prevention of Asphyxial Death sent a request to all physicians (335) listed as anaesthetists in the "American Medical Directory" requesting each man to serve on a Committee on Anaesthesia to study asphyxia from the point of view of the anaesthetist. A questionnaire was designed, under the Chairmanship of Dr. Lincoln Sise of Boston, to study the clinical material which is continually accumulating concerning deaths under anaesthesia. This questionnaire, it will be observed, deals with the broad questions of how, when, and where, rather than upon the scientific details of the history record, whose correct assembly implies an experience and knowledge capable of arriving at a correct interpretation of the cause of death. A glance through the deaths reported will indicate how easy it is to overlook the obvious and how necessary it is to reiterate the existence of the ever-present hazard. Comments received from more than seventy men indicate so much understanding and common sense that it is a pity that space prevents their inclusion.

Causes of Death Due to Anaesthesia

Direction of Service. (1) Lack of efficient planning of anesthesia service. (Intended as a broad question to cover such matters as, choice, training and assignment of personnel, length of their service, coordination with surgeons and other departments, etc.)

(2) Faulty judgment in choice of anaesthetic agent and method.

The results of the questionnaire indicated that points should appear in the following order:

(a) The emphasis on comfort, instead of safety and efficiency.
(b) An inelastic routine.
(c) Lack of familiarity with a broad list of anaesthetic agents and methods.
(d) Use of advertising material of manufacturer as guide.
(e) Experimental use of new agents.

Personnel.

The results of the questionnaire indicated that points should appear in the following order:

(1) Lack of training in observation of signs of depth of anaesthesia, of obstruction, and of asphyxia.

(2) Lack of appreciation of the importance of these signs.

(3) Ignorance of the anatomy and physiology of the "Death Zone" (epiglottis to vocal cords), combined with a technical inability to care for this region by inspection, suction and intubation.

(4) Nurse technician.

(5) Dependence upon mechanical measuration, rather than upon physiological signs.

(6) Lack of attention and interest.

(7) Ignorance of how to force oxygen into the lungs with an anaesthetic gas machine or other apparatus.

(8) Inability adequately to care for obstruction in other portions of the breathing tract.

(9) Interne anaesthetist.

(10) Lack of surgical cooperation. (Speed in induction, insistence on relaxation in unsuitable cases, small allowance for lack of control with lighter anaesthetic agents, etc.)

(11) Constitutional inability to acquire adequate technical skill.

Equipment.

The results of the questionnaire indicated that points should appear in the following order:

(1) Poor preparation (such as tanks on wrong yokes, empty oxygen cylinders, etc.).

(2) Lack of equipment

(a) In general

(b) Equipment for administering oxygen not immediately at hand during anaesthesias (such as spinal, local, intravenous) when a gas machine is not necessarily used.

(3) Faulty equipment

(4) The complexity of modern equipment.

Methods.

The results of the questionnaire indicated that points should appear in the following order:

(1) Failure to give artificial respiration with oxygen in the presence of inadequate or arrested respiration.

(2) Administration of a general anaesthetic in the presence of vomiting or when the stomach is not empty.

(3) Failure to employ emergency tracheotomy early enough.

(4) Administration of gases without oxygen.

(5) Intentional disregard of blood pressure under spinal anaesthesia.

(6) Intentional disregard of cyanosis under gases.

(7) "Saturation" and "secondary saturation" with nitrous oxide.

Post-Operative.

The results of the questionnaire indicated that points should appear in the following order:

(1) Lack of trained post-operative supervision.

(2) Delay in the use of oxygen therapy and its relegation to untrained oxygen therapy technicians.

(3) Unfamiliarity with the ordinary principles of commercial oxygen therapy equipment.

Principles to be Advocated.

The results of the questionnaire indicated that points should appear in the following order:

(1) Simplicity and reliability of equipment. Physiological rather than mechanical indications in variations of dosage.

(2) Organized anaesthesia service with active director and trained personnel.

(3) Selection of anaesthetic agent invariably based upon safety.

(4) Intimate knowledge of appearance of and ability to care for the "Death Zone."

(5) Acquaintance with basic principles of oxygen therapy.

(6) Support and wide use of publications specializing in articles on anaesthesia.

(7) Standardization of training and qualifications of anaesthetists by a duly established National Board.

Causes of Death during and following Anaesthesia Induction. From sudden vomiting, aspiration, respiratory obstruction, and respiratory failure.

From respiratory obstruction, and relaxation and obtunded reflexes giving rise to a vicious circle of: obstruction, asphyxia, depression, further asphyxia, obstruction, depression, etc.

From idiosyncrasy to ordinary pre-medication (allergy).

From sudden complete respiratory obstruction, precipitated by pre-medication or general anaesthesia in acute inflammations about the airway.

From ventricular fibrillation, during a chloroform anaesthesia, complicated by autogenous production of adrenalin from fear and excitement.

From sudden circulatory failure, anemia of the respiratory center followed by general anoxemia and death, in cases exhibiting idiosyncrasy to local and spinal anaesthesia.

From anaesthetic overdosage.

From an agent whose use was not intended.

From explosion followed by edema of the airway.

From external pressure upon the trachea by thymus or substernal thyroid.

From liquid ether finding its way into the airway.

From biochemical causes, as: alkalinization, excessive soda-lime absorption, over-ventilation.

Maintenance. From progressive respiratory failure from basal, plus general anaesthesia.

From ventricular fibrillation in chloroform anaesthesia, where adrenalin is injected into the circulation by nasal packs or by topical application.

From progressive respiratory failure due to partial respiratory obstruction, giving rise to a low-grade continuous anoxemia.

From excessive packing of the abdomen in the obese, particularly when in the Trendelenburg position.

From gastric contents expressed by high abdominal packing finding its way into trachea and drowning patient.

From shock due to traction on abdominal viscera.

From the rupture of an abscess into the healthy airway, as retropharyngeal abscess, quinsy, etc.

From intracranial pressure.

From collapse of the lung or the aspiration of a lung abscess into a healthy bronchus.

From protracted spasm of the glottis during gas oxygen anaesthesia.

From the sudden evacuation of a large abdominal tumor.

From a deep level of anaesthesia immediately following delivery (the buffer consisting of the baby, and the placenta having been removed).

From overdose in attempting to get relaxation by paralysis of the musculature without due regard for the patient's position and the tidal volume of respiration.

From biochemical causes affecting the normal CO_2 content of the blood.

From pulmonary thrombosis in pelvic and femoral vein operations.

From coronary thrombosis in chronic heart disease.

From hemorrhage.

Recovery. From neglect of the unconscious patient on the table, in transfer, or when returned to bed.

From aspiration of vomitus.

From aspiration of blood clots and foreign bodies.

From the late effect of pre-operative medication combined with the obstructive effects of the operation due to tissue engorgement, packs, etc.

From the pressure of dressings on wounds of the neck and about the mouth.

From hemorrhage, anemia, and anoxemia.

From accidents due to a maniacal reaction.

From prolonged and slowly progressive anoxemia due to unrecognized slight obstruction, in recovery from long-acting general anaesthetics.

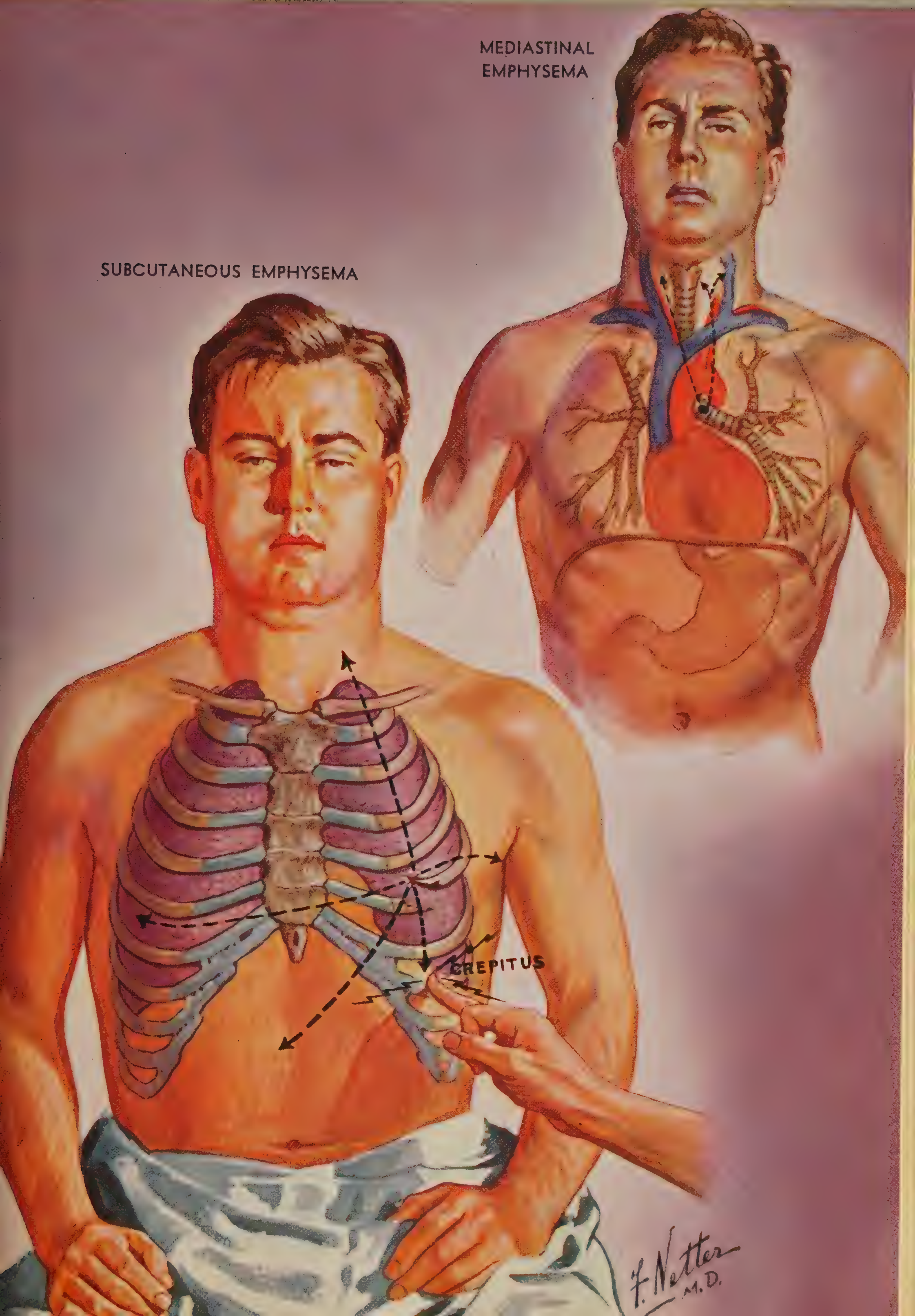
From failure to employ tracheotomy early enough.

Among more than seventy replies to the foregoing questionnaire there appeared the following deaths and comments.

It is suggested that the reader draw his own conclusions from the report in each case and compare this with the author's reaction which appears as a foot-note at the bottom of each page on which a report appears. The running comments regarding reactions to the questionnaire which appear with the reports are noted by number.

EMPHYSEMA

Courtesy Ciba Pharmaceutical Products, In



Fatality #1

Pre-operative findings: Man of 55 years with scar on tongue from previous radium insertion.
Pre-medication: H.M.C. #1 (H) 20 minutes before anaesthesia began.
Anaesthetic agents: Ethyl chloride followed by ether, 8 oz., followed Evipal, 7 cc. of 10% solution.
Method of administration: Open drop for ethyl chloride and ether.
Intravenous for Evipal.
Operation: Insertion of radium needles into tongue.
Signs preceding and accompanying fatal issue: Vomited large amount of fluid and food early. Very difficult induction of anaesthesia in alcoholic patient who was not asleep after 30 minutes of ether. Evipal then given after which: Respiration jerky. Vomiting. Cyanosis. Pupils dilated.
Method of resuscitation: Artificial respiration with O_2 and CO_2.
Post-mortem findings: Carcinoma of tongue with metastases to cervical glands. Tracheo-bronchitis (chemical).
Comment: Death during induction from a multiplicity of anaesthetic agents, in a patient difficult to anaesthetize by reason of previous alcoholism. If faced with the situation again, I should not use a less controllable agent following failure of a more controllable one, such as Evipal following ether.

No. 1 Comment:

I find myself in accord with most of the questionnaire. Would also list as possible causes of death:

(a) Continuous pressure on the eyeballs by face mask with its resulting brachycardia (Williams).

(b) Pressure on the carotid sinuses with retractors during adenectomies.

We hear of quite a few deaths where N_2O and oxygen or ethylene is given for induction in cases of Ludwig's angina, peritonsillar abscess and thymus, or any other condition preventing the free opening of the mouth. After a nasal or oral airway is established, however, this menace is erased, but I feel a general warning of this should be broadcast.

P.S.: Anaesthesia resuscitation drills should be conducted at regular intervals to familiarize operating-room staffs of their duties.

No. 2. Comment:

I believe more in the *practical observation of the reactions of the patient* than in the observations of technical appliances. That does not mean that I am not in favor of watching and keeping records or in having modern appliances. I am in favor of reducing cost of anaesthetic equipment.

No. 4 Comment:

At the present time the deaths due to anaesthesia have already reached, according to some reports, alarming proportions and seem to be continu-

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Mechanical problem (not biochemical); anoxic anoxia; patient drowned in his own secretions owing to the fact that his pharyngeal (not laryngeal) reflexes were thrown out by the Evipal. Immediate laryngoscopy and intubation plus suction plus oxygen insufflation might have saved his life.

Fatality #2

Pre-operative findings: Man of 30 with abscess of right lung. Rib resection and extensive drainage operation had been performed on right lung two months previously. B. P. 100/70. Patient a poor anaesthetic risk.
Pre-medication: Nembutal 1½ gr. at 5 a.m. Nembutal 3 gr. at 7 a.m. Morph. sulphate ¼ gr. and Hyoscine sulphate 1/150 gr. at 9:58 a.m.
Anaesthetic agent: Nitrous oxide-oxygen with ½ oz. ether, from 10:38 to 11:25 a.m.
Method of administration: CO_2 absorption technique with Heidbrink.
Operation: Drainage of right lung abscess with cauterization of lung.
Signs preceding and accompanying fatal issue: Respiration became jerky after patient coughed up a large amount of blood and pus during cauterization of lung. B. P. 60/40. Color became cyanotic in spite of oxygen administered under positive pressure. Pulse dropped to 0. Pharyngeal and laryngeal reflexes evidently active.
Methods of resuscitation: O_2 and CO_2 forced. Caffeine sodio-benzoate 7½ gr. I. V. Intratracheal catheter inserted. Adrenalin m.5 (H). Unsuccessful attempt made to give glucose solution I.V. Adrenalin 1 cc. intracardiac.
Post-mortem examination not made. No consent.
Comment: Death during maintenance due to hemorrhage from lung plus respiratory obstruction from blood clot (?). If faced with the situation again, no change indicated.

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ously rising. The efforts of the surgeon and anaesthetist, though, at the present time is not directed toward correcting this tendency, but rather toward inventing alibis for it, the most popular one being to place the blame on the patient himself—his heart, his thymus or the shape of his nose.

I've already been through this before, years ago, when I tried to convince surgeons and hospitals that they should eliminate chloroform from the armamentarium of their operating rooms, and the arguments used then were exactly the arguments in use now, to convince me that I am wrong.

Comment No. 6:

Your questions are all important. So much so that I stopped marking them after one page.

Comment No. 7:

It has been my observation that one of the greatest danger periods occurs as the patient's reflexes are returning during recovery from general anaesthesia.. It is difficult to impress upon the attending nurse the value of supporting the lower jaw at this time.

No. 8 Comment:

There is no doubt in my mind that the greatest need in anesthesia at the moment is more adequate training, particularly in respiratory physiology

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Premedication excessive. Endotracheal anaesthesia and suction facilities positively indicated. Intubation at instant of accident will not always suffice in massive suction accompanying lung pathology. Such operations are best done in the afternoon, not in the morning, since overnight secretions are then disposed of.

Fatality #3

Pre-operative findings: Carcinoma of tongue, bleeding. Pulse weak. Blood pressure not obtainable. Rales throughout lungs.
Pre-medication: Morphine sulphate $\frac{1}{4}$ gr., Atropine sulphate $\frac{1}{150}$ gr. (H) 35 minutes before anaesthesia.
Anaesthetic agent: Ethyl chloride induction. Ether.
Method of administration: Open drop (25 minutes)
Operation proposed: Ligation of external carotid to stop bleeding.
Signs preceding and accompanying fatal issue: Respiration, difficult. (Stopped gradually. No struggle for air. Color, not cyanotic.
Method of resuscitation: Tracheotomy.
Post-mortem findings: Carcinoma of tongue with metastases to cervical glands. Edema of larynx.
Comment: Death during maintenance and due to (1) shock from hemorrhage, (2) progressive respiratory failure resulting from partial respiratory obstruction (edema of larynx) and giving rise to a low-grade, continuous anoxemia. If faced with the same situation I should do no differently.

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as related to anaesthesia. Surgeons need to know more about the relative strengths of various anaesthetics, so that too much is not demanded of any one. A good example is nitrous oxide.

As soon as anaesthetists learn to maintain a wide-open airway and to keep the patient asleep without any cyanosis, anaesthetic deaths will become rarer. Adequate suction and the use of intratracheal tubes should be more generally used.

The question of nurse-technician and nurse-physician anaesthetists is constantly in the foreground. Of course physicians can and should make much better anaesthetists, but I would rather have a good technician than a poor physician administer an anaesthetic to myself. I feel that the major number of anaesthesias can and will be done by nurses under adequate instruction and supervision of a physician. Hospitals with 15 or more anaesthetists cannot fill all places with physicians. Small hospitals do not have enough work to justify a physician giving full time to it, and an "occasional anaesthetist" is, in my opinion, a dangerous one.

My feeling is that the anaesthetist has been unduly blamed for many deaths occurring under anaesthesia and immediately thereafter. I think some provision in reporting anaesthetic deaths should be made for including not only the name of the operation but also an estimate of the amount of surgical trauma and hemorrhage during it.

Here we have a meeting every week of the surgeons and anaesthetist at which we take up the deaths during the week. The causes of death are listed as follows:

1. An error in technique.
2. An error in treatment.

Morphine $\frac{1}{4}$ gr., plus anaemic anoxia (bleeding), plus overventilation (by open drop) may produce pale asphyxia. Indications: no pre-medication; transfusion; endotracheal anaesthesia.

Fatality #4

Pre-operative findings: A woman of 51 years with carcinoma of cervix Group 1.
Pre-medication: H. M. C. #1 an hour before anaesthesia.
Anaesthetic agent: Ether.
Method of administration: Open drop.
Operation: Cautery of cervix and hysterectomy performed with cautery.
Signs preceding and accompanying fatal issue: Respiration 30, but became gasping just before death. B. P. dropped suddenly after 1 hr. and 40 min. of ether. Pulse rose from 90 to 140 and became weak. Dropped to 120 before death. Pupils dilated before death.
Treatment of shock: 5% glucose solution I. V. Saline solution by hypodermoclysis. Caffeine sodio-benzoate 7½ gr. added to glucose sol. 1 hr. after drop in blood pressure. Reappeared 20 min. later.
Methods of resuscitation: Carbogen inhalation. Lobelin, 1 amp. Transfusion of blood attempted.
Post-mortem findings: Carcinoma of cervix. Surgical shock.
Comment: Considerable blood was lost, about 1½ pints. Carcinoma involved much of pelvic tissue. Death during maintenance of anaesthesia due partly to excessive packing of abdomen (a 15-yard pack was used) and partly to hemorrhage. If faced with the situation again I should follow the same procedure.

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3. An error in surgical technique.
4. Anaesthesia.
5. An unclassified group where at the time of operation and at post mortem no adequate cause for death can be found.

These cases are discussed in a very critical fashion and no one's feelings are spared. In fact, I think we are leaning over backwards in an attempt to blame ourselves. I feel that without thorough knowledge of the patient's diagnosis and the amount of surgical trauma at the operation, one must go very slowly in blaming the anaesthetist. This of course is obvious, but I should like to put in a word for the anaesthetists.

Dr. Sise:

The trouble which you mention of difficulty in determining what are anaesthetic deaths and what are not is fundamental and has disturbed us all. If anyone can offer a clean-cut, satisfactory answer, he will certainly confer a boon.

The inclusion of a question on surgical trauma and hemorrhage along with many other questions was considered and abandoned. We came to the conclusion that we could keep adding questions which would be of some value almost indefinitely, but that we had to draw the line somewhere. It was difficult to tell just where, but we wanted to keep the questionnaire as short as possible.

Overventilation from open drop ether, plus H. M. C., plus Trendelenberg position with excessive packing. Shock from loss of CO_2 plus Bohr phenomena. Indications: allow rebreathing, permit pain reflexes to return to stimulate respiration.

Fatality #5

Pre-operative findings: Man of 33 with extensive depressed fracture of frontal bone. Temp. 101.2; Pulse 84; Respiration 30; B. P. 120/80.
Pre-medication: Amytal 3 gr. given 3 hrs. before anaesthetic. Atropine 20 minutes before.
Anaesthetic: Ether.
Method of administration: Open drop.
Operation: Elevation and replacement of fragment of frontal bone after debridement of wound.
Signs preceding and accompanying fatal issue: Respiration, pulse and blood pressure rose at first, reaching a peak about 30 minutes after beginning of anaesthesia. From this time on a gradual but persistent drop came, despite stimulants used.
Methods of resuscitation: Intravenous fluids, *i.e.*, 800 cc. of saline and 800 cc. of 10% glucose solution. Caffeine sodio-benzoate 15 gr. I.M. and 7½ gr. I. V. Coramine 1 cc. I. V. Adrenalin 1 cc. intracardiac.
Post-mortem findings: Crushing injury of head.
Comment: Death during maintenance due to traumatic shock with superimposed surgical shock, plus severe intracranial contusion. If faced with the same situation, there is no reason for change in procedure.

The custom which you mention of discussing deaths critically and classifying the causes under certain headings is most helpful.

No. 12 Comment:

National Board of certification should be put through as soon as possible. Trained anaesthetists should have entire control over pre-medication, choice of anaesthetic and post-operative complications.

Dr. Sise:

All that you say is important, but the question is, how is it to be accomplished? Fortunately certification of anaesthetists seems to be well on the way, but the other matters are certainly not so fortunately placed.

No. 13 Comment:

The best way to prevent complications in anaesthesia is to anticipate the trouble. In other words, Prevent accidents.

Always keep the patient on the safe side by close attention to signals of danger. Give the patient the benefit of any doubt by lightening or stopping anaesthetic when this becomes necessary.

I do not limit my work to anaesthesia alone.

No. 14 Comment:

Standardization as to what comprises an anaesthetic death other than those occurring on the table from inexperience, hemorrhage, shock and emboli, would give us a truer understanding than statistical records.

Sodium amytal plus open drop contraindicated (reflexes increased and blood pressure raised). Avertin and endotracheal would have widened margin of safety.

Fatality #6

Pre-operative findings: Negro man of 42 yrs. Carcinoma of lip and glands of neck. Temp. 98.8; Pulse 78; Resp. 20; B. P. 138/70; Weight 120 lbs. Wassermann two plus, Kahn four plus.

Pre-medication: H. M. C. #1 (H) at 8.05 a.m.

Anaesthetic agent: Ethyl chloride induction. Ether.

Method of administration: Open drop, from 8:05 to 9:55 a.m.

Operation: Ligation of left external carotid artery (preliminary to cautery and removal of carcinoma of tongue a week later).

Signs preceding and accompanying fatal issue: Respiration became irregular 10 minutes after ether stopped and 5 minutes after left external carotid artery was tied and stopped. Blood pressure dropped from 160/70 to 110/60 in 15 minutes. Pulse rose from 100 to 135 at same time. Pupils rapidly dilated. "While skin closure being made patient suddenly stopped breathing, followed almost instantly by cardiac failure."

Methods of resuscitation: Artificial respiration for 15 minutes. Epinephrin injected into heart.

Post-mortem findings: Cancer of tongue with metastases to adjacent tissues of throat. No evidence of carcinoma in specimen taken from neck during operation.

Comment: Death during maintenance due to surgical shock and some respiratory obstruction from induration about throat. If faced with the same situation again, no reason for change of procedure.

Personally, I believe many deaths following operations are blamed on the anaesthetist by the surgeon, which in truth are of surgical nature.

Would it not be advisable to protect our weak specialty by a more intelligent investigation at autopsies, and demand that all so-called anaesthetic deaths be supported by a coroner's findings and statement?

Dr. Sise:

The two points which you mention are certainly of the greatest value. Nothing is more difficult or more important to determine than just what constitutes an anaesthetic death.

No. 19 Comment:

"Pink patients don't die."

I have the most trouble getting the interns to watch and maintain an open airway and sufficient and even anaesthesia.

Second, the nurse does not understand the importance and the technique of keeping an open airway on the post-operative anaesthetized patient.

No. 21 Comment:

In most communities, little attention is given, and in some no attention, to the question of anaesthesia. In my opinion some action should be

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H. M. C. #1 plus 1/100 ethyl chloride, plus open drop ether equals a badly depressed respiration; plus an operation on the neck plus internal obstruction plus negro equals progressive anoxic anoxia—easily overlooked. Indications: Reduce premedication, add endotracheal insufflation suction. This is clearly a preventable asphyxial death.

Fatality #7

Pre-operative findings: Man of 42 with gas bacillus infection of right leg. Compound fracture of rt. tibia and fibula (2 days). Albuminuria and granular casts. Debridement and pin through ankle. April 24th under ethylene.
Pre-medication: Morphine sulphate $\frac{1}{4}$ gr. (H) at 10:45 p.m.
Anaesthetic agent: Novocaine 100 mgm. at 11:55 p.m.
Method of administration: Subarachnoid block.
Operation: Leg amputation.
Signs preceding and accompanying fatal issue: Respiration gasping during preoperative preparation. Ceased at 12:20. Blood pressure: 11.55 p.m. 100/50; 12:05 a.m. 100/48; 12:15 a.m. 90/50; 12:20 a.m. 30/0. Pulse rose from 125 to 150 in 20 min. and dropped to 0.
Methods of resuscitation: Caffeine sodio-benzoate $7\frac{1}{2}$ gr. (H).
Post-mortem findings: Comminuted fracture of both bones of right leg with gas bacillus infection. Body organs normal except liver has appearance of fatty infiltration.
Comment: Death during maintenance due to extreme toxicity from extensive gas bacillus infection. If faced with the situation again, my choice of anaesthetic would be the same.

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taken by the A. M. A., or other authorized agent, to bring this matter to sharp attention. The use of this point as a factor to be used in the rating of a hospital might well be worth while. Under present methods reports are either withheld or made in such a manner as to be practically valueless. This is due both to ignorance and to willful concealment of negligence.

No. 22 Comment:

Skill of the anaesthetist with the agent and the method he is using is far more important than the dangers inherent in the agent and method themselves. If the anaesthetist's skill is limited to a few agents, selection should be based on his skill rather than on inherent dangers of the drugs or method.

No. 24 Comment:

The principles as noted cover the whole ground in my estimation. In addition may I state that I feel that #6 is the most important. If the anaesthetist—and by that I mean those members of the medical profession who are administering anaesthetics—will all endeavor to increase their qualifications either by travel programs or courses in various types of anaesthesia, including oxygen therapy, they will so equip themselves that fatalities will be at a minimum. I also feel that this society (S. P. A. D.) can and will do more for organized anaesthesia than would unorganized efforts.

Accident suggests circulatory shock due to loss of sustaining muscle tone in trunk and lower limbs plus $\frac{1}{4}$ gr. morphine, which further decreased irritability. Ethylene oxygen or gas oxygen ether (regardless of albuminuria) might have provided greater safety. It is to be noted that patient had previously passed safely through an ethylene anaesthesia.

Fatality #8

Pre-operative findings: Child of 6 with tuberculosis of right knee joint. Inguinal lymph nodes swollen and incised previously.
Pre-medication: Codeine $\frac{1}{6}$ gr., Atropine sulphate $\frac{1}{300}$ gr. 1 hr. 25 min. previously.
Anaesthetic agent: Ether.
Method of administration: Open drop.
Operation: Removal of tuberculous granulation tissue in right knee joint.
Signs preceding and accompanying fatal issue: Much mucus. Respiration rate rose from 20 to 36. Sudden cessation of respiration after 1 hr. 20 min. of ether. Pulse rate did not rise.
Methods used for resuscitation: Ephedrine $\frac{1}{3}$ gr. (H).
Artificial respiration with O_2 and CO_2.
Post-mortem findings: Tuberculosis of lungs, lymph nodes and right knee joint.
Comment: Death during maintenance which might have been avoided by an anaesthetist of greater experience than that of an intern. If faced with the situation again I should have a resident anaesthetist give the anaesthetic.

No. 26 Comment:

New agents not needed or desirable until proven and personally witnessed, until one is convinced of superiority in some way. Numerous new agents come and go. I do not believe all I hear or read in the new propaganda of manufactures or on the pushing of new things by anaesthetists.

The patient is a more satisfactory indicator than any medicine or machine.

A nurse-anaesthetist, trained and experienced, is more satisfactory than an interne.

No. 28 Comment:

Only the Almighty can protect the patient from ignorance and inattention. The principles suggested to be advocated are admirable and well worth working for.

No. 32 Comment:

From my experience (9 years), I feel that most deaths in hospitals occur in the hands of interns. They are often given too much freedom in anaesthesia without sufficient training. The deaths in our hospital have been mostly in the stage of maintenance due to failure to recognize overdose. Some deaths have occurred in the stage of induction, usually because of failure to provide a free airway and to a minor extent of the persistence of a low-grade anoxia due to subsequent overdosages superimposed. This low-grade anoxia is often not recognized, especially if the surgeon is intolerant about the depth and relaxation. The anaesthetic is pushed, in the face of the anoxia contraindication, with the usual result—fatality.

Spinal anaesthesia "below the umbilus" is much the safest anaesthetic when used "in anybody's hands" and when there are no contraindications.

Patient appears to have died from anoxic anoxia, drowned in his own secretions. Endotracheal suction indicated during operation.

Fatality #9

Pre-operative findings: Obese Mexican woman of 30 with ruptured ectopic pregnancy. Wassermann 3 plus, Kahn 1 plus. B. P. 140/80, Temp. 99.6, Pulse 120.
Pre-medication: Morphine sulphate ¼ gr. Atropine sulphate $\frac{1}{150}$ gr. given 21 min. before anaesthesia. Ephedrine 1 gr. preceding spinal.
Anaesthetic agent: Novocaine 150 mg., Nupercaine 7 mg.
Method of administration: Subarachnoid block. Agents mixed with 8 cc. of pt.'s spinal fluid and injected slowly in 4th lumbar space. No difficulties in administration.
Operation: Salpingectomy. Ruptured left tubal pregnancy found.
Signs preceding and accompanying fatal issue: Note by resident anaesthetist: "Operation started after patient, who was very obese, was put into Trendelenberg position. Patient could not talk English, but seemed uncomfortable and complained a little. Also was nauseated but did not vomit. Prepared to give O_2 and CO_2 while intern tried to get B. P., but B. P. was not obtainable 3 min. after operation started. Noticed some cyanosis and patient was evidently in collapse. Pulse was not obtainable."
Methods of resuscitation: Artificial respiration for 40 min. Ephedrin 1 cc. intracardiac. Ephinephine 1 cc. intracardiac.
Post-mortem findings: Post-spinal anaesthetic shock. Tubal pregnancy with hemorrhage. Obesity. Cholelithiasis.
Comment: Death during induction due probably to sensitivity to either Novocaine or Nupercaine. If faced with the situation again, barbiturates would be given before anaesthetic was administered.

I certainly agree with Dr. Henderson when he states that complications arise from loss of tone and too long a depressed state; this holds especially for pulmonary and cardio-circulatory complications.

No. 34 Comment:

I think all these questions are important to a more or less degree; however, I believe the entire series points to one question, the training and skill of the anaesthetist.

While surgical cooperation is important, any anaestheist who has his patient's welfare at heart is not going to sacrifice his patient to curry the favor of the surgeon. Moreover, good surgeons are not unreasonable.

Why not report all deaths, whether during anaesthesia or after, and then decide which may be attributed to the anaesthetic?

How many individuals are willing to report deaths as caused by the anaesthetic? There seems to be a tendency to avoid facing the facts. Last year out of 388 cases seven of my patients died. None of these deaths happened during or immediately after anaesthesia. They were not looked upon as anaesthetic deaths; however, a group of more experienced anaesthetists going over all the data on such a series might or might not find that anaesthesia was a contributing cause of death.

Anemic anoxia and central depression by morphine ¼ (some patients, especially women, will not tolerate ¼ but they will accept ⅙). Diaphragmatic embarrassment from packing, plus obesity; shock from loss of muscle tone due to spinal. Indications: reduce pre-medication; preserve reflexes and muscle tone. Hemorrhage contraindicates spinal anesthesia.

Fatality #10

Pre-operative findings: Cleft hard and soft palate in baby of 1 yr. X-ray of March 3, 1935 showed no enlargement of the thymus.
Pre-medication: None. Atropine $\frac{1}{250}$ gr. given 15 min. after anaesthesia begun because of mucus.
Anaesthetic agent: Ether.
Method of administration: Open drop followed by vaporizer, 25 min. in all.
Operation: Repair of cleft palate.
Signs preceding and accompanying fatal issue: Respiration: Irregular and of very small volume, then stopped entirely. 2 or 3 gasps obtained after artificial respiration. Color yellow. Never cyanotic. Pulse not obtainable just before respiration ceased. Pupils half dilated when respiration stopped.
Methods of resuscitation: Artificial respiration with O_2 and CO_2, continued for 20 min. Coramine, $\frac{1}{2}$ cc. into right jugular vein. Adrenalin, $\frac{1}{2}$ cc. into heart muscle. Heart massaged through abdominal wall.
Post-mortem findings. Enlarged thymus, 25 gms.
Comment: Patient had two previous operations for cleft palate. Feb. 15, 1934, chloroform for 45 min. Apr. 5, 1934, chloroform and ether for 60 min. If faced with this situation again, I do not think I should do otherwise. A more experienced anaesthetist would be advisable.

No. 36 Comment:

We need better anaesthetists here, but are faced with a reciprocity problem among all physicians whereby anaesthetics are used to pay off favors. Thus even those of us who have endeavored to be better prepared are not given the opportunity to show how good we are or to have sufficient experience to be called specialists.

No. 37 Comment:

Most of our accidents during anaesthesia here in the past two years have been due to the fact that inexperienced anaesthetists (interns) have not recognized obstruction and oxygen want soon enough.

No. 40 Comment:

The outline seems so complete that it is difficult to find much to add. However, the following suggestions are offered:

1. Emergency tracheotomy outfit, sterilized and ready for instant use when needed.
2. Drill of operating team of assistants in getting out apparatus for intravenous saline and glucose solutions in emergency.
3. Instruction in intratracheal intubation in emergencies (see pages 80, 85).

No. 42 Comment:

To my mind too many accidents follow the use by physicians of agents recommended only by advertising literature or the exaggerated claims of detail men. This, plus the desire of some to be the first in the community

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This report suggests overdosage of ether. It also suggests pale anoxia. Both conditions may result where the respirations are exceptionally free. Little children are easily overdosed; they may easily lose their carbon dioxide by overventilation during anaesthesia (operation of Bohr Phenomenon).

Fatality #11

Pre-operative findings: Emaciated man of 50 with ulcerative carcinoma of neck following carcinoma of tongue operated upon 3 mos. previously. Wassermann four plus. Pulse 130, B. P. 110/70. Poor risk.

Anaesthetic agent: Ethyl chloride for induction, followed by open drop ether.

Operation: Tracheotomy. No operation performed on carcinoma of neck.

Signs preceding and accompanying fatal issue: Respiration was unsatisfactory almost from the beginning of administration of ether, so towel was removed from around mask and an airway was inserted, but this did not seem to relieve the apparent obstruction to breathing. Color increasingly cyanotic beginning soon after ether administration began. Relaxation barely sufficient to permit airway to be inserted. Eyelids winking. Pharyngeal and laryngeal reflexes present. Patient coughed and cheesy, purulent material was extruded from opening of the airway. This was thought to be causing the respiratory obstruction and tracheotomy was performed.

Methods of resuscitation: Artificial respiration with carbogen, intracardiac injection of adrenalin ($1\frac{1}{2}$ cc.).

Post-mortem findings: 1. Carcinoma of tongue (operated). Some softening of the tissues at the base of the tongue which communicates with the ulcerated area beneath the mandible on the left. Marked induration of cervical tissues. Lumen of trachea empty. False vocal cords moderately edematous. 2. Carcinoma, metastatic, of cervical glands with necrosis and ulceration. 3. Tracheotomy wound, recent. 4. Atelectasis, pulmonary, partial. 5. Cerebral softening, minor.

Comment: Cause of death: Carcinoma of tongue. Acute medullary failure. If faced with the situation again, I should give ether with oxygen instead of by the open drop method as soon as cyanosis began, but I doubt if the results would differ at all. There was insufficient relaxation to permit the insertion of an endotracheal catheter, even had there been no ulceration at the base of the tongue or laryngeal edema.

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to use or advocate a new agent, is followed by disatrous results. Detail men and manufacturing concerns encourage men to use agents which they have no right to use, *i.e.*, the encouragement by certain men to dentists and general practitioners to use Evipal, etc. This is but a single instance of a practice employed by many manufacturing concerns.

My feeling is that as anaesthetists, we should not only be entrusted with the patients that come under our immediate care but should have some restraining influence on the advertising claims and statements made by manufacturers. A committee should be named by a national society outstanding either in clinical anaesthesia or laboratory research. To it should be entrusted new agents or apparatus for laboratory or clinical tests. It should send these to some qualified person for approval. All reports should come back to this central committee. These reports should be published with the comments and suggestions of the members. The manufacturer should limit himself, in his claims, to the findings of this committee. Example: Drug A, a new drug for spinal anaesthesia, recommended to the committee by a manufacturer. Committee forwards drug to Dr. X, a physicist, for physical properties, to Dr. Y, a physiologist and

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Supra-glottic respiratory obstruction. Ether increases spasm. Chloroform with facilities for laryngoscopy intubation or finally tracheotomy might have widened this margin of safety. The most difficult and dangerous type of case for general anaesthesia.

Fatality #12

Pre-operative findings: Gall-bladder.
Pre-medication: Morphine ¼ gr.
Anaesthetic agent: Nitrous oxide plus oxygen.
Method of administration: Gas machine with rebreathing through soda-lime.
Operation: Gall-bladder.
Signs preceding and accompanying fatal issue: Respiration normal. Blood pressure 140–80. Color: blue. Pulse: rapid. Relaxation: fair. Pharyngeal and laryngeal reflexes: jactitation.
Method of Resuscitation: oxygen.
Post-mortem findings: Edema of brain.
Comment: Apparently carbon-dioxide asphyxiation. If faced with the situation again I should give air and stop re-breathing.

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pharmologist, for its toxicity as related to other agents, and to Dr. Z for clinical use. Upon their findings the committee bases its report.

I feel the work you are doing is of great value and wish you every success.

No. 44 Comment:

In California, we do not train nurses for anaesthetists, as we do not believe they have sufficient medical background to enable them to make medical decisions.

Interns coming from the central and eastern states make very poor anaesthetists at first, as they are accustomed to have nurses giving anaesthetics and seem to feel that anaesthesia is beneath them.

One of our most serious shortcomings is allowing the interns on the surgical services to order the premedication.

All medical schools should have a course in anaesthesia and make it a required subject.

No. 46 Comment:

My personal feelings are that many of the anaesthesia deaths are due to inadequate training on the part of the anaesthetist and to the relative lack of interest in anaesthesia. There are too many men who give anaesthetics by reflexes and not by skill.

I think one of the most important things in the way of prevention of asphyxial death will be to establish anaesthesia as a department in the medical schools and teach it as a separate branch as they do surgery and medicine. It is generally considered in this and the surrounding community that anaesthesia is just a minor part of the surgical team and too many routine proceedings are preferred because our great-grandfathers did it that way.

No. 47 Comment:

I feel that a competent experienced anaesthetist should select the anaesthesia or anaesthetics and method of administration to the existing con-

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Anoxic anoxia, due to oxygen deficiency in respired mixture, not carbon dioxide poisoning.

Fatality #13

Pre-operative findings: Pharyngeal and peritonsillar abscess with edema of vocal cords and epiglottis; very cyanotic.
Pre-medication: Morphine sulphate $\frac{1}{4}$ gr., atropine $\frac{1}{150}$ gr.
Anaesthetic agent: Ethyl chloride 2 min., ether 10 min. Open drop.
Operation: Tracheotomy.
Signs preceding and accompanying fatal issue: Respiration: 20. Blood pressure: 130/90. Pulse: 100. Pharyngeal and laryngeal reflexes: complete obstruction developed after 10 min. of anaesthesia with cessation of breathing.
Methods of Resuscitation: Tracheotomy, caffein 3 gr., alpha lobelin 1 amp. intracardiac, coramine 1 amp., adrenalin into heart; artificial respiration as soon as tracheotomy tube was in position.
Comment: This patient should have a tracheotomy done under local anaesthesia before attempting anything to relieve abscess.

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dition and not the surgeon. Again, the surgeon often selects spinal anaesthesia for his own comfort, when this is not the safest for the patient. I feel that perfect teamwork between surgeon and anaesthetist is essential at all times.

No. 55 Comment:

Your questionnaire is extremely valuable as it reminds me that I am frequently careless on numerous counts.

No. 56 Comment:

Choice of anaesthetic agent and method is a most vital factor. Regardless of training, etc. some students will never master the art and science of anaesthesia. Such should be encouraged to try some other specialty.

Teaching of interns is most vital. The average medical student considers anaesthesia of no particular importance—a minor detail relegated to nurses or anyone who happens to be available.

No. 58 Comment:

Unfortunately the great majority of surgeons feel themselves gifted with infallibility, and any suggestion from outside their closed circle is considered an infringment upon their celestial domain of knowledge.

No. 60 Comment:

It is my opinion that as soon as the A. M. A. recognizes that the anaesthetist is a specialist, more physicians will devote themselves to serious study of the subject and operating rooms will have better reports to give.

No. 61 Comment:

Equipment: It is my opinion that the simpler the equipment is, the better.

Ethyl chloride and ether contraindicated owing to tendency to engorgement and muscle spasm. Chloroform by open drop or with helium oxygen sufficient to permit laryngoscopy and intubation with facilities for tracheotomy is indicated. Dissection under local down to trachea without opening until acute emergency develops may be practiced. If intubation is accomplished the skin and muscle may be closed without ill effects or delayed convalescence.

Fatality #14

Anaesthetic agent: Novocaine 150 mg.
Method of administration: Spinal
Operation: Elective, right inguinal hernia
Signs preceding and accompanying fatal issue: Respiration 60. Color cyanotic. Pulse 128. Pupils pin point.
Post-operative medication and support of respiration: This patient was returned to ward in good condition at 9:50 a.m. At 10:15 a.m. he received $\frac{1}{6}$ gr. morphine sulphate. At 2:25 p.m. he was given another $\frac{1}{6}$ of M. S., after which he became cyanotic almost immediately. He was given carbogen inhalations at five-minute intervals until his death at 2:20 p.m. on 29th. He also had caffeine sodio-benzoate and adrenalin. Caf. sod. ben. gr. $7\frac{1}{2}$. Carbogen thru face cone. Caff. sod. given 13 different times and adrenalin 5 gr. twice. Glucose 2000 cc., 10% given intravenously.
Post-mortem findings: No post-mortem made.
Comment: I think this case was due to morphine overdosage and then overdosing with caffeine. Should have used mechanical respirator.

No. 64 Comment:

Trained medical anaesthetists.

Careful examination as to physical condition and emotional stability of patients, before deciding type of anaesthetic.

Absolutely unobstructed respiration during general anaesthesia.

Let the patient, not a machine, be the barometer as to amount of various agents to be administered.

No. 67 Comment:

This is a very thorough and complete questionnaire. Allow me to commend highly the articles in the heading "Principles to be advocated". Allow me to suggest one more principle. Namely, "Closer relation between surgeons and anaesthetists for the good of both".

Under methods, #4 depends entirely upon your training as to whether cyanosis is a relative thing or not. Persistent, deep cyanosis is very materially different from moderate cyanosis for short periods of time.

No. 68 Comment:

The questionnaire herewith presented is both full and comprehensive and embraces practically all causes of anaesthetic deaths during anaesthesia. I most heartily commend the work of those who were responsible for its preparation.

No. 69 Comment:

It is interesting to note how quickly the academic candor and detachment of the Latin mind has seized upon the proposal to coordinate the field of gas therapy. There is here no hint of expediency, of profit or loss to the individual involved. The question is viewed, as it should be, from the

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Reliance placed upon circulatory stimulation instead of upon counteracting the effects of the respiratory depression. O and CO_2 endotracheally might have carried this patient to a return of normal respiratory function.

Fatality #15

Pre-operative findings: Patient of 20, with pulsating swelling of left occiput, nystagmus, choked discs and ataxia.
Pre-medication: Sodium amytal 6 gr. seventy minutes before anaesthesia.
Anaesthetic agent: Ether.
Method of administration: Open-drop induction and vaporizer with nasal catheter for maintenance.
Operation: Exploratory craniotomy for cerebellar tumor.
Signs preceding and accompanying fatal issue: Sudden cessation of respiration just before opening dura. Blood pressure and pulse constant until just before respiratory failure.
Methods used for resuscitation: Artificial respiration with O_2 and CO_2. Caffeine sodio-benzoate 7½ gr. (H). Coramine, 1 amp.
Post-mortem findings: Brain tumor 4th ventricle, malignant.
Comment: Death during maintenance from respiratory failure possibly due to pressure from 4th ventricle tumor. If faced with the situation again, I see no reason for change in procedure.

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point of view of whether or not coordination under a new terminology is logically indicated. The author believes this to be the case. The following would appear to confirm this view.*

"In the last chapter of the sixth edition of "Art of Anaesthesia" by Dr. Paluel J. Flagg . . . Dr. Flagg inserts a full article previously published in the *Journal of the Maine Medical Association*, in which he advocates the use of the term *pneumatology* to include in its wide scope, as we will see later, three scientific disciplines apparently scattered and unrelated at the present time: anaesthesia, technique for the resurrection of accidents and intoxication, and oxygen therapy.

"These three disciplines, the first fully established today, and the other two in the process of evolution, have a common denominator; the use of gases. Dr. Flagg states that no one but the anaesthetist is fully acquainted with the physical properties, therapeutic indications and use of gases. The specializing physician is the one trained to treat the unconscious patient, and it is interesting to note that this unconsciousness, whether produced by an anaesthesia or by any other of the actions of asphyxia, has a common anatomic and physiologic nature.

"Dr. Flagg, with very sound reasoning, proposes the acceptance of the term *pneumatology*, which among others has the advantage of being a time-honored word, as is proved in the following citations:

" 'Pneumatology: The science dealing with air and gases, their physical and chemical properties, and of therapeutic applications.'
(Stedman, 12th Ed., p. 853, 1943.)'

" 'Pneumatology: The science of gas and their therapeutic applications. (Lippincott's "New Medical Dictionary," 3rd Ed., p. 751).'

* Urrutia Leo, Migue, "Pneumatology," *Terepeutica al Dia*, **2**, 32 (Jan.) 1942.

Avertin with endotracheal provides reduced cerebral pressure and eliminates respiratory failure from all but aggravated and continuous cerebral pressure.

"'Pneumatology: The sum of what is known regarding air and gases and their therapeutic applications. ("American Illustrated Medical Dictionary," p. 898, 1925).'

"The advantages of the adoption of this term for society and for those specializing in anaesthesia are as follows:

"The community calling the pneumatologist has the most competent expert to solve its problems; and the general public will become accustomed to resort instinctively to him. Hence the term must be popularized and its meaning well explained.

"Another advantage for those specializing in anaesthesia is that it widens their professional scope, inasmuch as they can provide for the nation, state, city and private enterprise technical personnel trained to direct catastrophe brigades and oxygen-therapy outfits.

"It would be useful also in North America to clear up the confusion in the public mind concerning the physician-anesthetist and the nurse-technician, because it would increase the personality of the anaesthetist and correct the inferiority complex that sometimes develops with the idea that because of association, the anaesthetist is one more assistant to the surgeon.

"Considering that in the United States 1000 people die weekly from asphyxia, the necessity of specialization of technical personnel as proposed by Doctor Flagg is quite apparent.

"We venture to forecast for pneumatology as brilliant a development as that of orthopedics, which, according to Dr. J. Andrieu de Labenne, from the Science of Malformations and Deformities, has become traumatology, the branch of medicine controlling extra-visceral surgery."*

Technique for the Prevention of Asphyxia in General Anaesthesia for Surgery of the Head and Neck

While competent physicians are well aware of the usual methods employed for the maintenance of general anaesthesia about the head and neck, namely: (1) by ether vapor delivered into the mouth by the ether vapor hook or into the posterior pharynx through an intranasal catheter; and (2) by methods more recently advocated such as the use of Avertin, Evipal, or other intravenous drugs, supplemented by the vapor of ether or chloroform, experience indicates that the most satisfactory routine method is endotracheal anaesthesia administered by a thoroughly qualified anaesthetist.† "Endotracheal" and "intratracheal" are used interchangeably. Since the Greek derivative tracheal requires the prefix "endo," instead of the Latin "intra," the term endotracheal deserves popularization.

There are two generally recognized and widely used endotracheal techniques. The first, now rarely used, popularized by Meltzer and Auer and

* This comment was not the result of the original questionnaire, it is added, however, as the opinion of an experienced anaesthetist.

† For a full treatment of this topic, see "Endotracheal Anaesthesia," Gillespie, University of Wisconsin Press.

used by Elsberg, employs the insuffiation or blowing of vapor under pressure through an endotracheal catheter of relatively small diameter. The volume of the insufflated vapor must be sufficient to supply the respiratory requirements of the patient, 500 to 1000 cc. per respiration, or a continuous delivery of ten to twelve liters of vapor per minute. The tube must be small in order to allow the escape around it of the insufflated vapor from the glottis. As may be imagined, this technique implies the proper functioning of an electrically driven motor capable of supplying an adequate volume of vapor under measured pressure.

The second method depends entirely upon the patient's respiratory efforts. Vapor supplied is at ordinary atmospheric pressure. The patient's own efforts provide both inspiration and expiration. In this technique the endotracheal tube must be large enough to permit free to-and-fro respiration. The anaesthetic vapor is respired through the catheter to a connecting tubing from a bag or other source. The anaesthetic gas may be ether, chloroform, nitrous oxide, oxygen, cyclopropane, or ethylene, with or without a carbon dioxide absorption technique.

For purposes of endotracheal anaesthesia the patient is intubated by one of two methods: (1) by blind introduction of the tracheal tube through one of the nostrils deflected by the posterior wall of the pharynx into the glottis, according to the method of Magill; (2) by direct or laryngoscopic exposure of the larynx and intubation under direct vision, according to the method of Chevalier Jackson, as practiced by the author.

Since this matter is not intended as a discussion of the technical details of varying methods of endotracheal anaesthesia, the author will confine himself to a detailed consideration of the method which he has found not only satisfactory but ideal for the purpose.

The reason for selecting this technique is that when properly carried out it is surgically clean, non-traumatic, providing with the simplest equipment perfect ventilation as well as facilities for continuous or intermittent tracheal or bronchial suction, and the most modern type of artificial respiration in the event of respiratory failure from any cause.

When endotracheal anaesthesia is considered, the usual resistence offered by peroral endoscopic exposure of the larynx immediately comes to mind. The nose and throat surgeon and the bronchoscopist are familiar with these difficulties, and may question the wisdom of assuming what may seem to be unnecessary technical difficulties when the method of blind intubation appears to make this unnecessary.

It is surprising in practice to find that, where anaesthesia is satisfactorily induced, the technical difficulties of ordinary laryngoscopy are practically eliminated. In other words, laryngoscopy in the relaxed, unconscious patient whose reflexes are in abeyance ordinarily presents no difficulty whatever. Inspection under these conditions is reduced to the simplicity of observing any closed cavity with an illuminated instrument.

Since this common objection to the technique of laryngoscopy is being considered, it may be well to refer briefly to other common objections which are frequently raised against it.*

Method is old, tried and abandoned: In this objection it is confused with the insufflation technique of Meltzer and Auer.

Trauma to pharynx, vocal cords and trachea: Trauma to these structures is entirely unnecessary if the correct technique is carried out. The endotracheal tube should be lubricated, intubation in the presence of spasm should always be avoided, pharyngeal packs should be moistened with alboline or saline.

Injury to teeth: The protection to the teeth where artificial dentures, loose teeth, porcelain inlays, fillings, etc. are present implies complete relaxation and more than usual care in exposure. This is a difficulty which involves the skill of the operator.

Pulmonary complications: Endotracheal anaesthesia reduces the incidence of pulmonary complications. The presence of the endotracheal tube causes no postoperative embarrassment.

Needless instrumentation: A knowledge of the advantages to be secured compensates for the technique of intubation.

Difficulty of ordinary use: Skill in technique can be acquired only by practice, through which all art is developed.*

Requirements of unnecessarily deep anaesthesia: Deep anaesthesia is for a short period only. After intubation the patient may be carried with less anaesthesia than by any other technique.

Failure to prevent aspiration: Adequate suction before intubation during the maintenance of anaesthesia and after extubation completely eliminates the danger of aspiration.

Soft tube, such as rubber, less irritating than metal: It is common surgical experience that a smooth metal surface with velvet edges are non-irritating.

Production of edema of the glottis: Not only is there an absence of any irritation resulting in edema from the presence of the tube, but on the contrary an indwelling tube frequently assists in the elimination of glottic edema resulting from other causes, such as an infection of the neck.

Obstruction to the operative field: When used for tonsilectomies and intraoral operations, the presence of an endotracheal tube passing through the mouth is sometimes found objectionable until the operator is accustomed to its presence and learns to displace it as required.

Failure of glottis to close after extubation: When the anaesthetic is carried on with the ordinary level of anaesthesia, the glottis usually falls into spasm upon extubation. This objection arose from the use of oversized endotracheal tubes forced into a glottis which had been cocainized and extubated from a patient who was still under deep anaesthesia.

Having discussed in some detail the objections frequently raised against endotracheal anaesthesia, let us now consider our reasons for selecting this as a routine method. From the point of view of the surgeon, satisfactory,

* "Intratracheal Inhalation Anaesthesia," *Arch. Otolaryngology*, **25**, 405–429 (1937).

smooth, general anaesthesia is as a rule difficult to secure. The surgeon who first makes use of the technique described for endotracheal anaesthesia will be impressed by the following:

The patient's respirations are without sound. This, at first, is disturbing. The only evidence that the operator has that the patient is breathing is by observing movements of the chest.

The operative field, be it nasal or oral, may be rendered aseptic and kept so. The posterior pharynx is packed with lubricated gauze, placed about the endotracheal tube, preventing the passage of blood into the trachea or esophagus from the site of the operative wound, and on the other hand preventing saliva, mucus or gastric contents from emerging from the nose or mouth to the sterile field of operation.

Because of complete ventilation venous congestion is eliminated. There is no venous ooze from the wound; bleeding is found to be arterial in character and the blood bright in color. Owing to the free, unobstructed respiration reducing respiratory effort, perspiration (loss of fluid) is reduced.

Obstruction to the respiration having been completely eliminated and replaced by an open tube, absolute control of the anaesthetic level may be exercised. The patient who becomes *light* may be immediately controlled, and the patient who suddenly passes into deep anaesthesia can be allowed to recover without disturbing the operative field. As a matter of fact, once a tube has been placed, the patient can recover to a cough or vomiting reflex, without the complications and dangers usually attendant upon such recovery.

In the event of vomiting during anaesthesia, vomitus remains in the esophagus, held in place by the pharyngeal pack. Solid material which may be vomited and accumulate about the glottic opening is prevented from entering the glottis by the presence of the endotracheal tube, securely clasped in the spasm of the glottis.

Post-operative illness because of the complete ventilation is reduced to a minimum; patients recover from prolonged ether anaesthesia in a few hours without ill effects.

In spite of the apparent simplicity of the apparatus employed, the author prefers this to more complicated equipment incorporating the resistance of soda lime absorbers and other technical details which, in certain cases, detract from continuous observation of the patient, thereby increasing the hazard.

Summary

In conclusion, the author is convinced that routine endotracheal anaesthesia induced with oxygen and ether and maintained with ether is the method of choice for operations about the head and neck. If the details of the technique as advocated (by him) are consistently carried out, the ordinary objections raised to endotracheal anaesthesia are overcome. The

instrumentation required by this technique is simple and rests upon the broad field of bronchoscopic experience. Once the patient has been intubated, the hazard ordinarily faced with anaesthesia for surgery of the head and neck is completely eliminated. The surgeon may operate without haste in a field ideal for the purpose.*

It will be observed that the preceding matter is viewed from the standpoint of anaesthesia as an art. It emphasizes the clinical aspect of asphyxia as this occurs in the practice of anaesthesia. As such, the matter is of the first importance. Where it is desired to carry on research investigation into the biochemical, physical or other causes of anaesthetic deaths, or the science of anaesthesia as practised in strictly supervised university clinics, other questionnaires more closely paralleling the usual laboratory routine should be employed. (See form prepared by American Society of Anaesthesia, below.)

These two approaches to the same problem emphasize once again the difference between the art and the science of medicine, that is, the field in its broad implications, as opposed to ultra-scientific laboratory research. While scientific research is priceless in revealing avenues of progress, it is merely complementary to the field which it serves. As a specialized field of medicine, anaesthesia shows no disposition to be anything more than a technical activity in which research of questionable value has become the chief issue. A solution of this difficulty is suggested in Chapter 28.

Form Prepared by American Society of Anaesthesia

Outline to Be Followed in Submitting Case Reports to the Anaesthesia Study Committee of the American Society of Anaesthetists, Inc.

Hosp. No.—— Age—— Sex—— Race———— Wt.—— Ht.—— T.—— P.—— R.—— Phys.———————— State————————.

1. Essential details of pre-operative clinical condition of patient.
   (a) Respiratory findings: rales, dyspnea, empyema, cyanosis, bronchiectasis, obstruction, etc.
   (b) Gastro-intestinal pathology: distention, obstruction, peritonitis, hemorrhage, etc.
   (c) Genito-urinary pathology: distention, pyelitis, nephritis, hematuria uremia, etc.
   (d) Circulatory conditions: B. P., pulse, shock, hemorrhage, decompensation, hypertension, myocardial degeneration, rheumatic, thyrotoxic, luetic, coronary disease, arrhythmia, functional capacity of heart, amount of fluid, blood or stimulants given.
   (e) Central nervous system: paralysis, neuritis, neurosis, irrational, stupor, coma, brain lesion.
   (f) Miscellaneous: toxic appearance, cachexia, anemia, hemorrhage, malignancy, pregnancy, obesity, dehydration, diabetic, hyper-thyroid, jaundice, etc.
   (g) Laboratory findings: Hb., RBC., WBC., Polys., CO_2 combining power, blood sugar, urea, creatinine, chlorides, proteins; urine: alb., sugar acetone, etc.
   (h) History of previous anesthetics; general health and habits.
   (i) Clinical surgical diagnosis, including pertinent facts of surgical lesion and contemplated operative procedure.
   (j) Date admitted to hospital; date and time scheduled for operation, and time operated.

* For details refer to "Art of Anaesthesia," Flagg, 7th Ed., Lippincott, 1944.

2. Pre-medication: include doses and time of all drugs given 24 hours preoperative; reason for giving and result: satisfactory, too depressed, work off, insufficient medication, insufficient time, apprehension, etc.

3. Induction: time anaesthesia started, agent, method, machine used, position of patient, excitement, retching, emesis, suction, laryngo-spasm, cyanosis, obstruction, mucus, respiratory volume, rate, etc.
   (a) If regional, spinal, block or intravenous was given, state agent, strength, amount, location, method, and result.

4. Condition during course of anaesthesia: respiratory volume, obstruction, laryngo-spasm, passive respiration, passage of airway, passage of intratracheal tube, depth of anaesthesia, cyanosis, diaphoresis, arrhythmia, drugs given, hemorrhage, shock, operation done.
   (a) If an anesthetic fire or explosion occurred, describe the physical hazards present in the operating room just prior to the occurrence: humidity, type of flooring, use of actual cautery, electrical equipment in use at the time, method of grounding operating team and patient at the time, if any such method was employed, types of drapes covering patient, etc.

5. Post-operative treatment and condition of the patient: follow outline under (1) above, giving details of treatment instituted and time.

6. Conditions of patient on discharge from hospital. Give in detail any residual symptoms present.

7. If patient died, give physical signs immediately prior to death, *i.e.*, B. P., respiratory and pulse rate and rhythm, color, condition of airway, presence of reflexes, muscular relaxation.

8. Details of methods of attempted resuscitation, including drugs, dosage, time, and method of administration.

9. Clinical diagnosis of death, including autopsy findings and pathological report, especially of brain, lungs, liver, heart, and kidneys.

10. Surgeon's impression of the case.

11. Anaesthetist's impression of the case. Would you handle a similar case in the same manner?

12. Anaesthesia administered by ....
    Attended during anesthesia by physician anaesthetists..........................
    Surgeon..
    Resident anaesthetist..
    Resident ....
    Intern ....
    Nurse.anaesthetist ....

Attach copy of anaesthesia record. If hospital has no regular anaesthesia record, so state.

All cases submitted to the Anaesthesia Study Committee should be sent to the chairman. A report of the findings of the Committee will be sent to the one submitting the case, who shall retain the privilege of publication. The report submitted to the Committee for study, however, will not be returned, so before submitting the case, be sure to retain a copy for your own records. The original case report along with a copy of the findings of the Study Committee will be retained as the property of the Society to be used for study and reference in compiling reports.

George Percival Mills, F.R.C.N., *British Medical Journal* for January 26th, 1935, reports, "Recovery after complete stoppage of the heart for five minutes." The author would appreciate case reports of death, with the psychic and physical phenomena of record.

Chapter 13

Asphyxia in Poliomyelitis

Anterior poliomyelitis may be regarded as a specific cause of asphyxia because of the neuro-muscular disease and incoordination which it precipitates. The progressive nature of the pathological lesions produced may be viewed as both an advantage and a hazard. The fact that asphyxia is not instantaneous, as in the anoxic anoxia of foreign-body obstruction, is an advantage, for to be forewarned offers suitable preparation to meet the emergency. On the other hand, since the pathological lesions precede the objective signs of asphyxia, to be unfamiliar with prodromal signs, or to postpone treatment until asphyxia occurs, is to invite a fatal issue.

The early signs of impending asphyxia are noted by Lewin as follows: (1) The patient becomes irritable, restless and unable to sleep. (2) The respiration becomes more rapid and shallow. (3) The patient has difficulty in saying long sentences and appears to be breathless. (4) Coughing is mechanically weakened.

George Draper quotes the late Prof. F. W. Peabody of Harvard as follows: "The typical picture is that of one with a clear, alert sensorium, fighting for every breath until he is literally suffocated. . . . Often the respiration is more rapid and a trifle more difficult than the degree of paralysis warrants. Then paralysis begins to increase. A laryngeal disturbance with hoarseness and aphonia or difficulty in swallowing may be the first evidence of the spreading lesion. If the intercostals are still active, the movements of the chest becomes less marked. If the diaphragm has hitherto been intact its movement, as represented by the abdominal wall, becomes weaker; or there may be asymmetric movement suggesting a paralysis of one side of the diaphragm. The alae nasi dilate with inspiration and the accessory muscles of the neck come into play. As the diaphragm weakens, the neck muscles become more and more prominent until it seems as though the whole work of respiration depended upon them. The head is thrown back and with every breath the lower jaw is pushed downward and forward in an attempt to get air. . . . The mouth becomes filled with frothy saliva which the child is unable to swallow so he collects it between his lips and waits for the nurse to wipe it away. The pallor is distinctive; the lips are blue, cyanosis is absent, and sweating is profuse. The mind becomes dull, unconsciousness follows and—an hour or more later—respiration ceases."

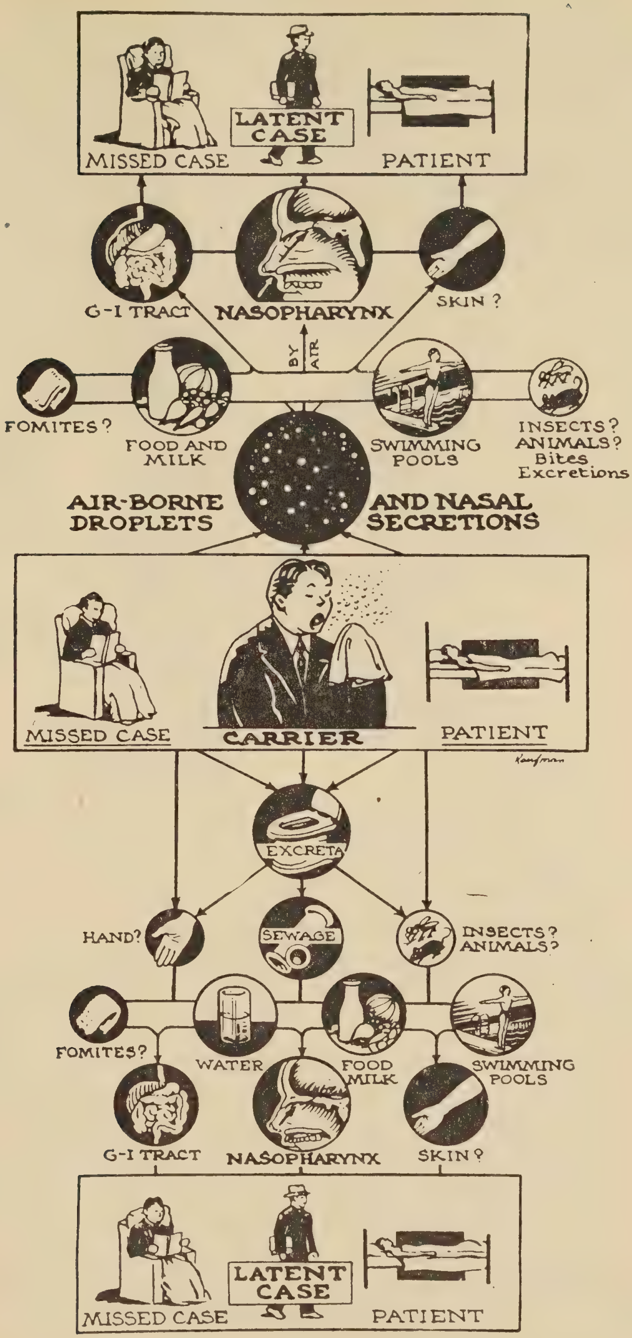
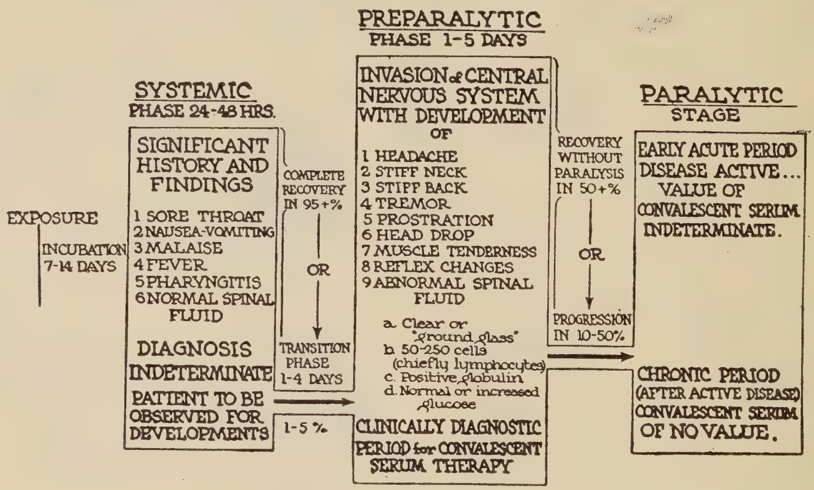


Fig. 105. Hypothetical means of dissemination of the virus of poliomyelitis.

Courtesy W. B. Saunders Co.

Since, however, a mere recitation of symptoms and signs, no matter how appealing, will make little impression on the mind unless these are supported by present knowledge of the pathological physiology of the nerve and muscle tissue involvement, the reader is invited to direct his undivided attention for a few pages to the refresher reference which follows. This will indicate a framework to which reason and memory may adhere. As the present consideration may be said to deal exclusively with problems of pneumatology (lungs and gases) our attention will be directed to only two of the major systems—the nerves and the muscles.



Courtesy W. B. Saunders Co

Fig. 106. Chart showing clinical course of poliomyelitis (Dr. S. O. Levinson).

The Nervous System in Polio

The etiology, symptomatology and clinical course of the infection are graphically represented in Figures 105 and 106.

Illustrations by R. G. Gordon and Forester Brown are reproduced in Figures 107, 109, 110–113 incl. These diagrams will serve as a condensed refresher reference of the anatomy and the histology of the nervous system. The acute inflammation of polio (as well as other infections) occurring at various points in this system result in conditions which are now well recognized, *i.e.*, anterior poliomyelitis, Landry's paralysis, etc.

Figure 108 shows a typical neurone. Figure 109 indicates what may be expected when inflammation attacks masses of such cells in the anterior horn of the spinal cord. Figure 107 represents the spinal cord and its connections and a sketch of the cerebellum. Figure 113 shows the pyramidal system, both spinal and cortical.

Having suggested the ignition system, as it were, we may now turn to the motor, which is represented by the muscle fibers.

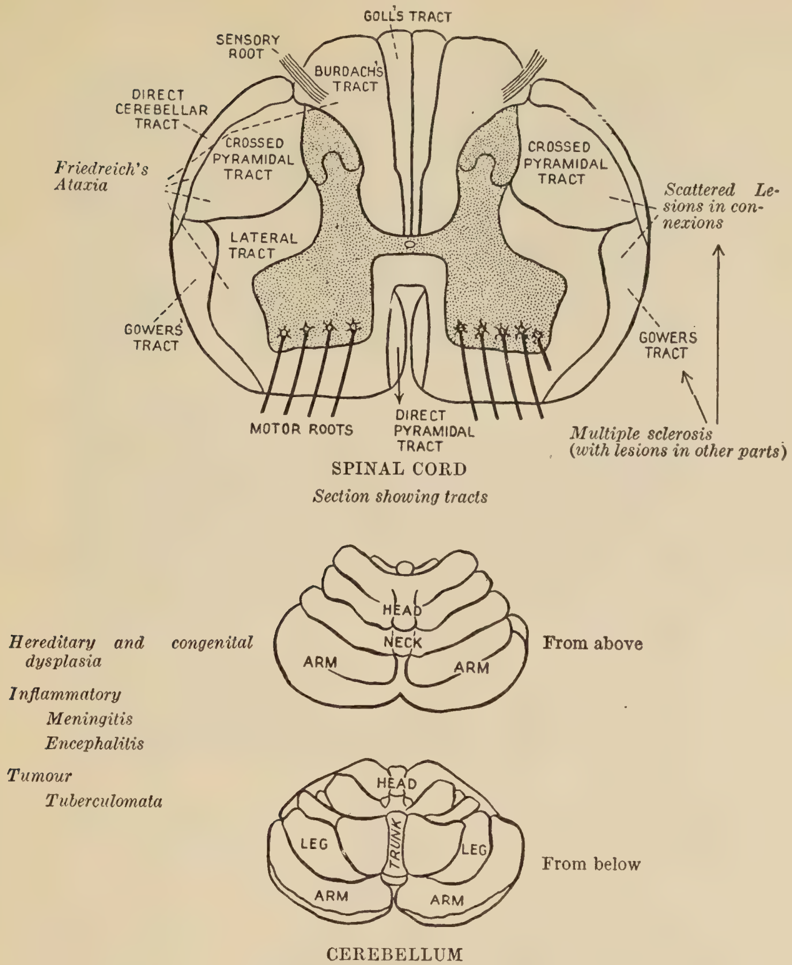
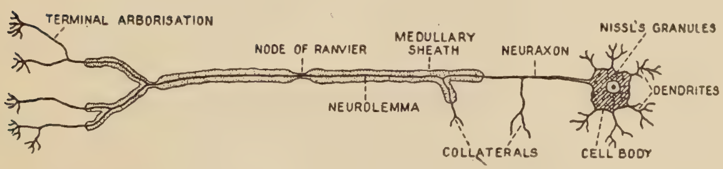
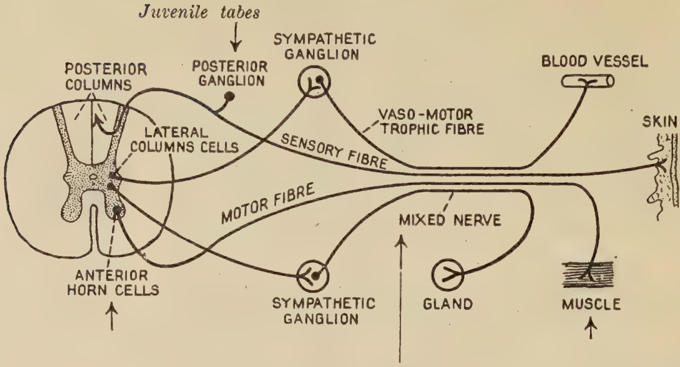


Fig. 107. The Cerebellum and its connections.



Courtesy Oxford University Press

Fig. 108. Diagram of a neurone (after Lewis and Stohr).



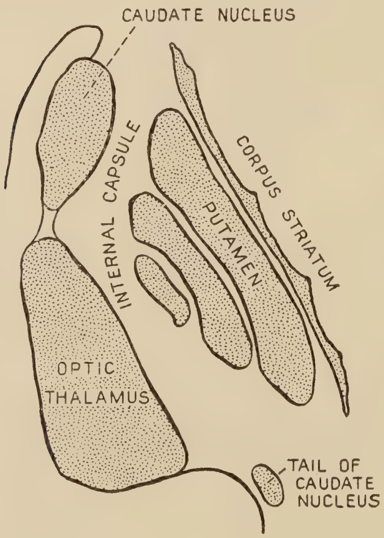
Courtesy Oxford University Press

Anterior poliomyelitis
Some cases of Landry's paralysis
Progressive muscular paralysis of infants
Motor system disease (with lesions in other parts)
Tubercular disease

The muscular dystrophies
Myatonia congenita
Inflammatory neuritis
Traumatic injuries and Birth palsies
Facial paralysis
Developmental Charcot-Marie tooth dystrophy

Fig. 109. Peripheral Arc Lesions.

Double athetosis



Parkinsonianism
Wilson's disease
Torsion spasm
Striate hemiplegia
Frog baby

Courtesy Oxford University Press

Fig. 110. Basal Ganglia.

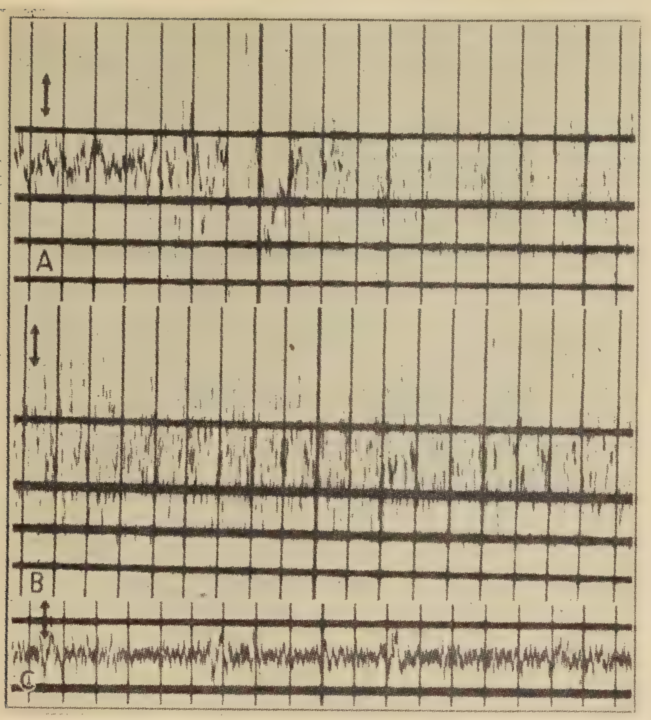


Fig. 111. Records from neck muscles of patient suffering with infantile paralysis: (A) slow passive movement of head. (B) head supported, in resting position. (C) head supported in resting position for minimum of action currents. Length of calibration arrow is potential of 10 micro-volts. Time unit is one-tenth second [Schwartz R. P., and Bouman, H. D., *J. Am. Med. Assn.*, **119**, 925 (1942)].

The Muscular System in Polio

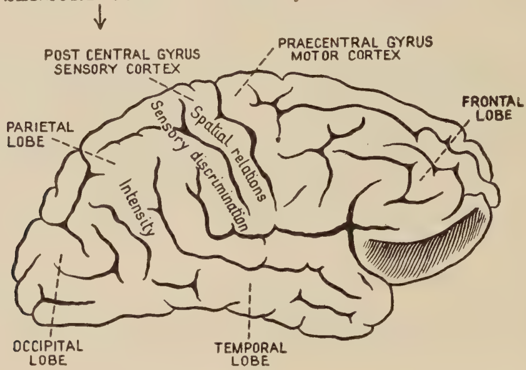
The former conception of the muscles as fibers bound together in striated or smooth bundles, reacting to impulses resembling electrical stimulae transmitted through their respective nerves, has been greatly amplified. The muscle "ensemble" is now regarded as a vital tissue which is constantly alive. Functioning normally, the tissue respiration in this internal environment is under the immediate influence of its capillary circulation, which in turn responds to neuro-vascular control.

Carey* presents evidence "which casts serious doubt on the prevalent concept that muscle fibers are permanently fixed into two types, *i.e.*, coarsely and finely striated. The manifold forms of the motor-nerve plates

* Carey, p. 237, 239.

and structural variations of the related muscle cross striations defy any simplification by a static morphologic classification. An understanding of the dynamics of functional ameboidism of the motor nerve ends and the influence of the changeable capillary chemistry in producing the different periodic structural changes in striped muscle may help to clarify this complex problem." He furthermore states that protoplasmic movement, or ameboidism, occurs during the initial growth and during the regeneration of nerves; "and finally, and most elusive of all, there remains the question of the mechanism which transforms the chemical energy derived from oxidative or anaerobic reactions, into the mechanical work which is its function in the body."

SENSORY CORTICAL LESIONS SYNDROMES



Courtesy Oxford University Press

Lesions of fibers impinging on the praecentral gyrus from other parts of the cortex. (Choreas, Athetosis, Myotonia Congenita.)

Fig. 112. Cerebral Cortex.

Spasticity

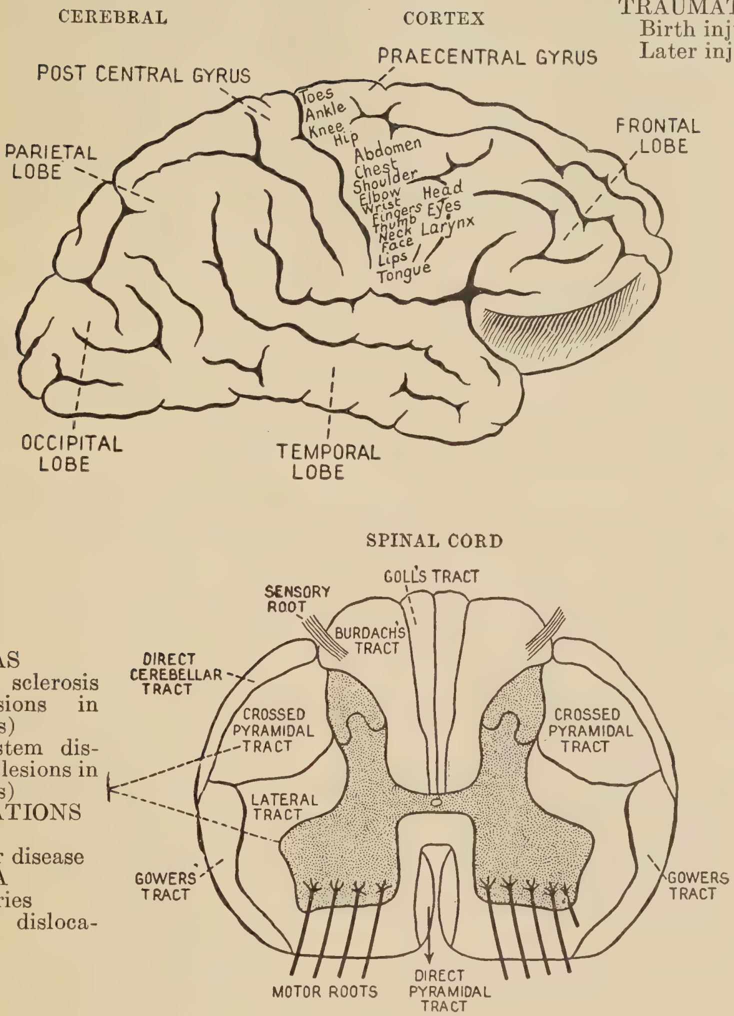
The complex muscle with its ever-present "muscle tone" responds to electrical stimulation by a so-called action current. One of the early effects of the systemic infection of polio is progressive disturbance of this electrical reaction by reason of the onset of spasticity.

This spasticity may be so slight as to be determined only by laboratory methods, or it may be clearly apparent in the grossly spastic muscle commonly seen. It may be precipitated by passive motion of the affected muscle. There is usually a "blind spot" in this range of passive motion at which the muscle will be at rest and in which it will present reduced spasticity or none at all. Spasticity is habitually present in the antagonist of the muscle affected by polio; it may be demonstrated in muscles in which the antagonist is not weakened by paralysis.

Schwartz concludes,* "In infantile paralysis spasticity of the muscles exists not only in the antagonist of the weakened muscle but also in the

* Schwartz, R. P., and Bouman, H. D., J. Am. Med. Assn., July 18, 1942.

DYSPLASIAS
Diplegia
Hereditary spastic paralysis
INFLAMMATIONS
Encephalitis
TRAUMATA
Birth injury
Later injury



DYSPLASIAS
Multiple sclerosis (with lesions in other parts)
Motor system disease (with lesions in other parts)
INFLAMMATIONS
Myelitis
Tubercular disease
TRAUMATA
Birth injuries
Fracture dislocations

Courtesy Oxford University Press

Fig. 113. The Pyramidal System, Cortical and Spinal.

weakened muscle itself and in muscles in parts of the body in which clinical symptoms of the disease are not evident.

"The spasticity is of a reflex nature and is not present in the completely paralyzed muscle.

"The spasticity can be stronger than the voluntary contraction that the muscle is able to perform, as adjudged by action currents. When the strength of the voluntary contraction increases through treatment, the spasticity decreases."

The prevalance of spasticity indicates the scientific rationale of the Kenny treatment, which assumes its widespread presence, its control by moist heat, passive motion so applied as to yield minimum stimulation to the muscle and its antagonist, and the discovery and utilization of the rest position for the given muscle or group of muscles.

Combined Neuro-Muscular Pathology in Polio

Respiratory embarrassment with its ensuing anoxia arises from both the spinal and the bulbar type of involvement. The spinal type, as might be expected, results in sensory and motor disturbances. The reflex irritability of the pharynx and the supraglottic region is reduced; swallowing and coughing are gradually weakened or abolished. Spasticity of the auxiliary muscles of the respiration, the sterno-clido mastoid, platysma mayoides, etc., as well as of the intercostal muscles and the diaphragm may result; flaccidity follows if the infection progresses. As a result of these lesions, stagnant and anoxic anoxia quickly make their appearance. The terminal reaction reported by Draper, *i.e.*, cyanosis of the lips, pallor of the skin, sweating, etc., is the typical picture of stagnant anoxia and shock encountered in dehydration and loss of muscle tone from spinal anaesthesia, shell shock, loss of CO_2, etc. Fluids, plasma, and oxygen therapy would seem to be indicated quite as much as use of a respirator. Once again the use of elaborate mechanical equipment appears to have obscured obvious indications for treatment. Secretions accumulating in the upper airway and permitted to remain there will sooner or later be aspirated, resulting in atelectasis, pneumonia, or abscess of the lung.

Muscle spasticity is likely to result in a disturbance in the rate, the rhythm and depth of the respiration, with its vicious circle of altered oxygen-carbon dioxide tissue tension. Flaccidity of the intercostal muscles with or without the diaphragm will promptly result in an interruption of the normal rhythmical variation of the intra-pulmonary gas pressures (page 39) essential to pulmonary ventilation. In other words, the patient's mechanical efforts to breath will be ineffective, anoxic anoxia will occur, and death will ensue. Lastly, in bulbar lesions the fourth ventricle—the respiratory center—may become affected. Terminal anoxia will then promptly supervene.*

* Dr. T. C. Galloway (J. A. M. A. 123: 17, Dec. 25, 1943) suggests that anoxia may play the leading role in the high mortality rate noted in cases of bulbar paralysis.

Attention in the past has been focused upon the factor of muscle flaccidity which was assumed to follow all ascending infections. The apparent simplicity of this conception gave rise to the popular demand for the use of respirators. While the result of this treatment was highly successful in those cases requiring pulmonary ventilation, its failure in those types in which muscle spasm and loss of sensation predominates was not fully understood. Strangely enough, by alleviating muscle spasm and permitting minimum but regular respiratory efforts, the Kenny Treatment now takes precedence over the respirator in selected cases. Since the respirator by accentuating muscle spasm through muscle motion and predisposing to aspiration may constitute a hazard, its use should follow and not precede more simple methods, designed to meet the pathological indications. These indications may be summarized as follows: (1) Loss of pharyngeal reflexes and consequent respiratory obstruction form accumulating secretions. (2) Muscle incoordination by spasm. (3) Anoxia by incoordination of respiratory muscles. (4) Anoxia by terminal flaccidity of spastic muscles due to progressive involvment of anterior horn cells. (5) Anoxia from central (bulbar) involvment impinging upon the respiratory center.

Treatment

The foregoing pathological indications suggest the following:

(1) The discovery of a specific cure which will kill the organism or neutralize the virus responsible for the poisoning and the leucocytic strangulation of the nerve cells in the anterior horn, bulbar region, and elsewhere. (The question has been raised as to whether such mechanical strangulation ever takes place.) All other means are merely supportive. They are designed to keep the patient from deteriorating through the period of infection and recovery.

(2) Loss of irritability of the upper airway suggests the necessity of keeping this region free of all foreign matter at all times by postural drainage or by mechanical suction, *e.g.*, a No. 10 indwelling nasal catheter, multiple-holed, terminating at the end of the soft palate or base of the tongue, connected with water or some other form of mechanical suction may be used (p. 60).

(3) Control or elimination of muscle spasm by hot wet packs and sedation.

(4) Oxygen therapy to meet the anoxia and the respiratory depression accompanying the sedation. (Certain clinics have been unable to meet all indications.) Endotracheal tubes and laryngoscopes are necessary emer-

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The post mortem findings suggest that he is correct. Tracheatomy in his hands has shortcircuted antemortem aspiration of pharyngeal secretions which the patient was unable to swallow. It is suggested that endotracheal intubation and an indwelling endotracheal tube be first employed. Patients tolerate indwelling tubes for 48 hours without ill effects. Tracheotomy may be done subsequently if it is found necessary. Those who care for Polio should become asphyxia conscious.

gency equipment to bridge the gap between a progressive respiratory failure which cannot be controlled by the foregoing treatment and removal to a respirator.

(5) Transmission to a respirator under endotracheal insufflation of oxygen.

(6) Continuation of artificial respiration in respirator with endotracheal tube in place.

(7) Establishment of postural drainage and suction in respirator.

(8) Endotracheal tube replaced by pharyngeal tube. Pharyngeal tube to be removed if the return of the reflexes indicate that it is causing embarrassment.

(9) Constant trained nursing attendance to insure freedom of the airway.

Cases which have developed true and extensive muscle flaccidity or which have experienced a bulbar involvment with central pressure present a practically hopeless prognosis. They are entitled to respirator treatment, however, in order that whatever muscle integrity remains may be given full opportunity to recover.

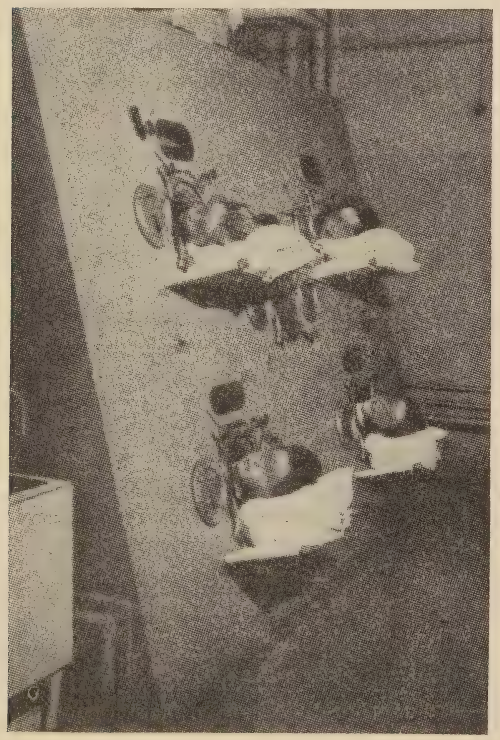


Fig. 114. Multiple respirator room for four children (Warren E. Collins).
Courtesy W. B. Saunders Co.

Use of the Respirator

The following summary is taken from an editorial which appeared in the *Journal of the American Medical Association* for July 26, 1941 (a report of J. L. Wilson). "At least 680 respirators were in use in 1940 in the U. S. A.

Replies covering 420 respirators owned by 335 hospitals and others reported 331 polio cases treated. Of the 331, 60 per cent or 204 were of the bulbar type, of which 127 died (62 per cent); 127 were of the spinal type, of which 23 died (18 per cent). The age groups were: 29 per cent under 10 years; 41 per cent from 10 to 20 years; 30 per cent over 20. As to time of treatment, 52 per cent were placed in the respirator before the 4th day, and 25 per cent after the 7th day.

"Difficulty was experienced in distinguishing between the bulbar and the spinal type, since these constantly overlapped. Three cases of diaphragmatic involvment were reported. The high mortality of the bulbar group was to be expected. Lending of machines was common. This practice, however, implied delay and inexpert service. Diagnosis and judgement of the respirator should be much improved."

Delay in the use of the respirator should be curtailed. Experience with the respirator as reflected in this report, "does not approach the high expectations of saving lives which was originally anticipated."

Comments

Clinical comparison of the neuro-pathology of polio with that of other forms of nerve-cell poisoning suggest that the phenomena of spasticity and the disturbance of the spinal reflex arc is to be expected. It would be of the greatest possible interest to parallel these findings with others which might be made to determine the electrical reactions of muscles under general, spinal or regional block anaesthesia. Abundant material is constantly available. The results would be of interest to numerous fields. Nerve trunk tension increases irritability. Is it possible that the "blind spot," or rest of the spastic muscle, is the position at which the least tension is brought to bear upon the nerve trunks supplying the part?

The picture of terminal asphyxia presented by Draper, *i.e.*, blue lips, pallor without cyanosis, and profuse sweating, is that of circulatory shock. Whether this arises from the anoxic anoxia of respiratory muscle failure or from the stagnant anoxia of a large bed of paralyzed muscles into which the circulation has bled seems secondary to the apparently clear-cut indications for treatment—intravenous plasma and oxygen therapy. Furthermore, it is stated that after an hour or more of unconsciousness respiration ceases.

To the pneumatologist the impotence of treatment in this last hour is shocking. Why is it that these patients, in the absence of a respirator, are permitted to die, when all that is necessary to bridge the gap is to expose the larynx, to intubate, and to insufflate oxygen pending their transportation to, or the arrival of, the respirator? The author has personally carried on artificial respiration for more than 10 hours in at least three cases, one of which is reported in the *Journal of the American Medical Association* for Feb. 17, 1940.

Lastly, it is suggested that the instructions in resuscitation proposed by the Special Committee on Infant Mortality, of the Medical Society of the County of New York (page 278), to be extended to pediatricians, feature as one of its important objectives the treatment of asphyxia in poliomyelitis. The physician familiar with the treatment of asphyxial spasticity and flaccidity in the new-born will find little difficulty in treating similar degrees of asphyxia in polio. By making such treatment available he will supply the missing link between death and safety in the respirator.

Chapter 14

Electrocution

Case 1. Judicial Electrocution

The famous report made by Donald Francis Mason "Sighted sub, sank same," has been matched if not improved upon, by the report of a judicial electrocution handed to the author by the Chief Prison Physician as we stood one April midnight viewing a post mortem outside the death chamber of a state prison. The report was this: "In 11:01, out 11:05."

A revolting human tragedy, deliberately planned and consummated without haste in less than five minutes.

At 11:00 p.m. the witnesses and representatives of the press were seated on their benches. The prison physicians and officials stood silently along the wall. The door to the death house opened. Guards, the prisoner and a tall priest entered and made their way directly to the chair. The prisoner made a brief ante-mortem statement. He was seated on the chair. He fixed his dark eyes on the crucifix which the priest held before him as numerous broad, soft leather straps were passed about his body, his arms, and his legs. A leather-covered, wet, metal helmet, sponge lined, wet with salt solution through which the current was to enter his body, was placed upon his head. The edge extended to above his eyebrows. The right leg, the trouser having been cut, was encircled at the calf by a wet leather-covered metal sponge lined cuff some three inches wide through which the current was to escape after having passed through him. Finally, his face was covered tightly by a black leather mask extending to the chin with openings for air.

Adjustments complete, all stepped aside. Now the man was alone. The executioner was at the switch. Then . . . .

A shock of 2000 volts at high amperage was thrown. The man rose in the chair against his straps. The chest was contracted and the head in extreme extension against the solid chair back. The hands clenched and the arms rotated outward on the chair arms. With the current still flowing, but with amperage reduced, spasm relaxed somewhat, to be sharply resumed as the amperage was again raised. The muscular contraction was accompanied by a sizzling sound as a visible white eschar appeared and spread above the cuff encircling the calf. At the end of two minutes the executioner signaled the attendants. They stepped to the man, stripped open his shirt, wiped the perspiration from his chest with a towel. Two physicians applied their stethescopes and listened. The chief physician stepped back and announced, "The man is dead." The time was 11:05 p.m.

Attendants removed the helmet and straps. The head, completely flaccid, dropped back. An attendant on each side lifted the corpse out of the chair, placed it on a stretcher and wheeled it out of the death chamber into the adjourning morgue.

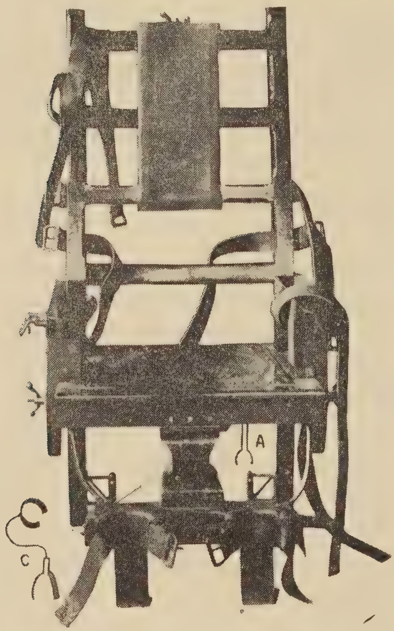


Fig. 115.
The electric chair at Sing Sing Prison. A—Anode, C—Cathode.

Courtesy N. Y. Daily News

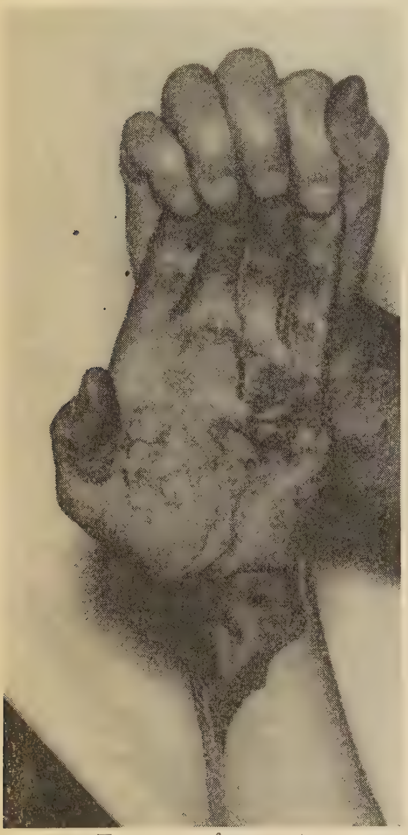
The skull was hot to touch, fifteen minutes after the passage of the current. A second-degree burn marked the edge of the helmet. This burn appeared as a yellowish-white eschar. There were no evidence of heat within the skull when the cranium was opened. The meninges appeared normal. The point of exit at the right calf was marked by exfoliation of the skin and extensive blistering. While the muscles of the airway were completely relaxed, the muscles of the back of the neck and right leg were fixed and rigid. Upon microscopic examination of the cerebral tissue, heart muscle, interventricular septum and cornea, the only changes noted were those of early cellular degeneration. "From a study of electrocution in man, it had been demonstrated that after contact with electric circuits at high voltage changes occur in a majority of the cells of those medullary centers concerned with respiration. These pathologic changes appear as dislocations of the nucleoli, with deep staining of nuclear materials and cytoplasmic loss of granules. When nuclear injury is evident the cells probably no longer have the potentiality of recovery. The suggestion is made that if the injury of these cells is slight, only a temporary respiratory block occurs; if greater, breathing will not be resumed."

"Nerve cell injuries in cases of human electrocution," O. R. Langworthy and W. B. Kowenhoven, *J. Ind. Hyg.*, **12**, No. 2 (Feb., 1930).

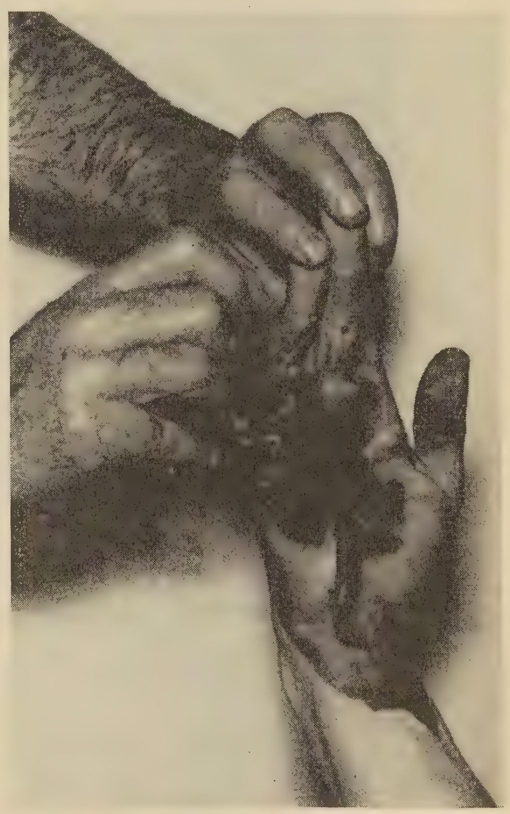
"Injuries produced by contact with electric circuits," O. R. Langworthy, *J. Exp. Med.*, **51**, No. 6 (June, 1930). Contains interesting matter re ventricular fibrillation.

Case 2. Shock by High-tension Circuit (21313)

The victim was an electrician's helper. He was working in the transformer room of an industrial plant. Electricity had been shut off in the



Entrance of current



Exit of current

Fig. 116. Fatal electric shock from high-tension current. (Courtesy Dr. Harrison Martland).

equipment undergoing repair. About nine feet overhead, however, were high-tension wires of the Public Service. Near these wires was a ladder. The deceased was found on the floor. Both hands were badly burned and his skull was fractured. The accident may be reconstructed as follows.

The man ascended the ladder to complete his work. The ladder having become unsteady, he may have reached for support, catching the wires in his hands. The muscle spasm from the high-tension current was so great that he was thrown free of the wires, fracturing his skull as he fell.

Fig. 117. Fatality by lightning. Singed hair and rupture of ear-drum at point of entrance. (Courtesy Dr. Harrison Martland).

Case 3. Lightning (21807)

The deceased and her daughter were attending a baseball game in a public park in New Jersey. A thunderstorm came up and they sought shelter under a tree. A bolt of lightning struck the tree, rebounded to woman, and entered the right side of her head, singeing her hair and rupturing her ear-drum (Fig. 117). It then passed down her body, tore off her stocking and the enamel from her shoes, stockings and shoes being rain-soaked (Fig. 118). The daughter was struck simultaneously at the tip of the nose

Fig. 118. Shoes of mother and daughter of Figures 117 and 119. Note split shoe and enamel torn off heel. (Courtesy Dr. Harrison Martland).

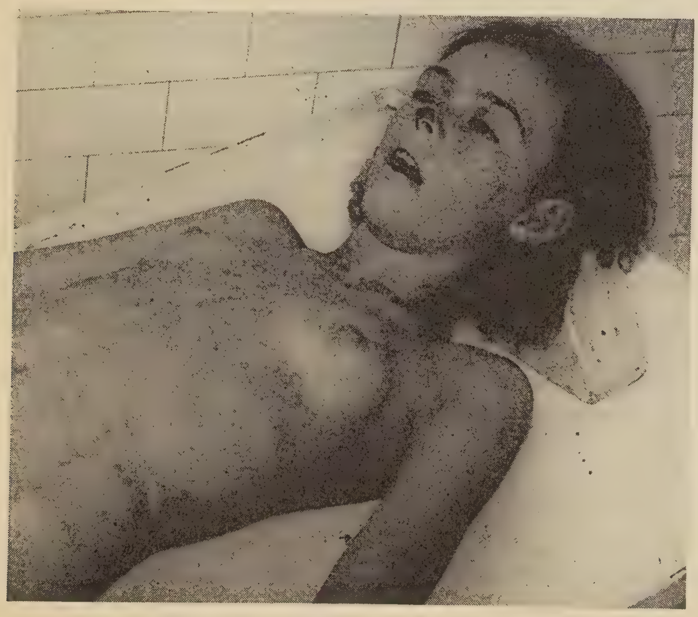


Fig. 119. Fatality by lightning. Entry at tip of nose; exit shows typical arborescent scar on abdomen. (Courtesy Dr. Harrison Martland).

(wet) the bolt leaving her body at the point of the aborescent scar shown in Fig. 119.

The crowd which witnessed this accident, fearful of touching the bodies of the stricken, made no effort to apply first-aid treatment. The deceased was unattended until the arrival of the ambulance half an hour later. Autopsy revealed a general asphyxia. Fig. 120 shows the effect of a lightning bolt striking and killing a man in an open boat.

Note. *The electric shock produced by lightning is instantaneous. The body struck contains no electric potential and may be touched at once.*

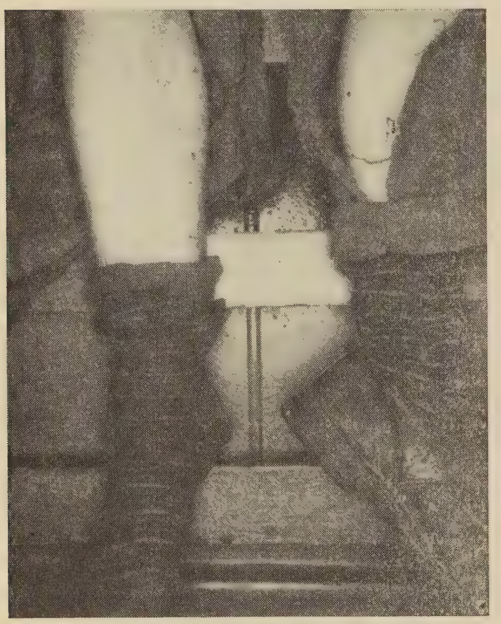


Fig. 120.
Man struck by lightning in an open boat. Trousers ripped down the legs. (Courtesy Dr. Harrison Martland).

Electricity and its Effects on the Body

These five deaths were due to electricity. Were they similar, or essentially different? Were they deaths from asphyxiation or not? A brief consideration of what is generally known about electricity will help make sense out of these reports.

Electricity is electronic energy of varying potential seeking a level. It may be natural or artificial. It occurs in nature as static electricity in potentials varying from the crackling of a cat's fur when it is stroked, to a gigantic innocent bolt of lightning, of a million or more volts, flashing across the darkened summer sky.

Artificial electricity, generated for the use of mankind, is met in the pocket dry battery and is noted in the massive cross-country cables carrying high-tension wires with up to 100,000 or more volts. For power purposes electricity is transmitted as a direct or alternating current. Direct current is difficult to transmit long distances because of the resistance which develops in the conducting circuits. Alternating current of high voltage (pressure) and amperage (volume) alternating at the rate of 120 times a second (60 cycles) is readily transmitted for long distances. If the alternations are extremely rapid, the current is referred to as of high frequency. The voltage in high frequency is also high, 20,000 to 40,000 volts or more. This type of current in very low amperage is used for medical purposes.

Electricity produces two effects: heat and stimulation.

Heat. When electricity finds resistance in the path of its movement, (though practically instantaneous, it is still much slower than the movement of light) it creates heat in the body offering resistance. If the current is high and the resistance great, the conductor is destroyed. Fuses, electric toasters, etc. operate on this principle. The body tissues offer varying resistance to the passage of current. The resistance of dry skin is high, 50,000 ohms; if it is wet, the resistance is only 1,200 ohms. The moist subcutaneous tissues and the vital organs offer little resistance.

The greater the surface of contact and the longer its duration the more pronounced will be the effects of the current passing through the conducting body. The effects of heat are important when burns occur, as the penetrating effects destroy the blood vessels nourishing the affected part.

We find the effects of heat, as we would expect, in the tissues offering the greatest resistance to the passage of the current, *i.e.*, at the point of entrance into the skin and at the point of exit from it. The dryer the skin the greater the burn. If the skin is wet, there may be no marks in a fatal electrocution, *i.e.*, contacting a live high-tension wire under water.

The effects of a low-tension current (110 volts at 1/10 ampere) are frequently fatal because of their selective effect upon the neuro-mechanism of the heart. The rhythmical, forceful contraction of the ventricle, supporting and distributing the blood stream against an average pressure of a column of water five feet high, is suddenly replaced by a feeble fluttering which brings the movement of the blood stream to a standstill. Anoxia promptly supervenes. Artificial respiration by helping to aspirate some of the blood in the right heart into the capillary bed of the pulmonary alveolae is of some help. The slight massage of the heart by the pressure of the chest wall is also helpful and may bridge the gap to recovery of the heart, if the fibrillation is not too long continued. As a rule, if true and marked fibrillation has begun the patient does not recover.

Ventricular fibrillation is often complicated by paralysis of the respiratory center. Fibrillation may be induced in small animals by a voltage as low as 20 volts. Fibrillation has a tendency to wear off. If therefore life can be maintained through the period of imperfect circulation, the patient may be saved.

Now let us go back to our cases and attempt to apply the preceding facts.

Judicial electrocution.

The current used in this case was a high-tension circuit of 1700 volts and the amperage was 9 amps. The duration of the passage of the current was two minutes (a long period); the contact area was large and the skin was moist at the point of contact. The immediate effect of the passage of the current was stimulation, evidenced by general muscle spasm. It may be assumed that ventricular fibrillation was immediately induced or that heart action was instantly stopped. The respiration ceased. These effects were followed by the heat effects of the current, protoplasmic destruction occurring. Nerve cells and heart-muscle fibers were irreparably destroyed. In spite of the broad wet contacts, second-degree burns occurred.

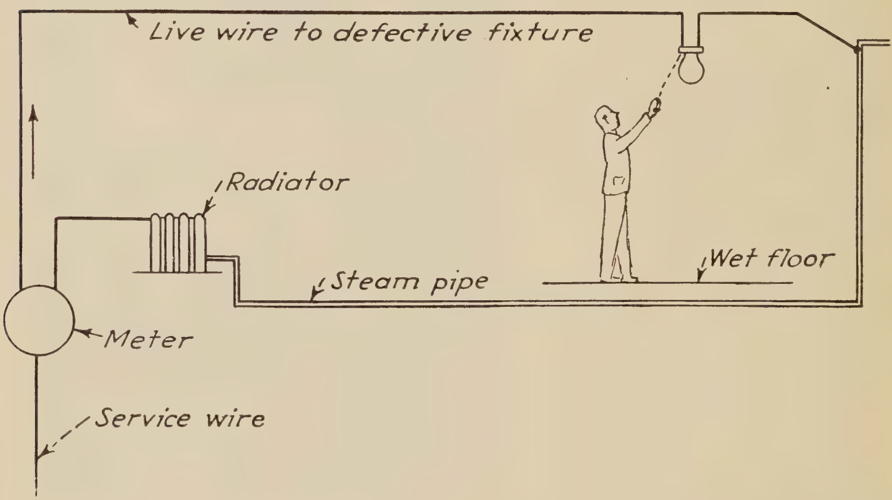


Fig. 121. Course of current in low-tension circuit shock, resulting in ventricular fibrillation. Wet floor touches steam pipe.

Electrocution by low tension and high tension circuits.

As we have noted, electricity always takes a short cut if possible; it will not detour if it can jump a gap. The human body makes a perfectly good bridge for such a short circuit. Let us elaborate this simple short circuit by a diagram of the principles underlying the wiring of a house circuit.

Fig. 121 illustrates the fact that one of the wires coming from the electric service passes through the meter and to the various light fixtures and base plugs. The other wire, however, does not follow the first, but stops after passing through the meter, and is fastened to a water pipe. In other words, the water pipes, the plumbing, the heating fixtures, etc.—everything in contact with the ground—acts as the second wire. The wire going to the fixtures is called the hot wire the other is the neutral wire.

Examining the light fixture, we find two wires attached to separate insulated, screws. One of these wires is the hot wire, coming straight from the electric light company. The other is the neutral connected with the ground, in common contact with plumbing, radiators, etc. Suppose the neutral wire gets loose and touches the metal frame of the fixture. The whole assembly, including the pull chain, will then have the same potential as the radiator or water pipe. If, while holding the chain in one hand, you stood on the wet basement floor or leaned against the faucet, nothing would happen. But on the other hand, if the other wire was loose and touched the fixture when you touched the pull chain your body would act as a bridge between the two potentials, 110 volts and 15 amperes on one side and zero volts and zero amperes on the other. If only $\frac{1}{150}$ of the total amperage passed through you, it would be sufficient to induce ventricular fibrillation and death. The death induced would be asphyxial; as such, every effort should be made to prevent it.

The author has seen Dr. Horatio Williams induce ventricular fibrillation in sheep by electric shock and stop the fibrillation by counter-shock. After the counter-shock, the heart assumed its normal rhythm. Difficulty would of course be experienced in the practical application of such treatment. Sudden death from ventricular fibrillation in chloroform and cyclopropane anaesthesia, especially where adrenalin has been employed, is not uncommon.

The prevention of household electrocutions deserves the utmost publicity. As Martland says, "It is better to curl your hair in the bedroom than to curl up on the floor of the bathroom."

Case 2 represents what is obviously an irreversible process. Owing to the high-tension circuit, *i.e.*, 10,000 to 130,000 volts with an amperage of ——a temperature of 2500°C. was produced in the body which closed the circuit. As a result, the tissue was melted, vaporized and incinerated, by the reason of the resistance which his body offered to the enormous current which passed through it.

Fortunately, contact with high-tension circuits is sometimes imperfect. Strong currents, if they are survived, do not necessarily result in fibrillation.

Caution: A stream of water played upon a third rail on one occasion served as a conductor. The current traveled back on the water stream and killed the victim.

Electrocution by lightning.

Death by lightning illustrates a perennial hazard. As is well recognized, there is nothing stereotyped about the behavior of lightning. It runs the entire gamut of electrical effects. The enormous potential which accumulates in the clouds can only be leveled off by contact with opposing charges in the heavens (90 per cent of lightning never comes to earth) or by coming to the ground as a bolt. Water reduces the resistance of the air gap, so that with the downpour comes the crash of the charge. Trifles on the earth's surface, such as trees or even a man in an open field, especially

Fig. 12[illegible]. Safe positions in an electrical storm are shown by asterisks. All others dangerous.

when wet by the rain, provide an infinitesimally shorter path to the earth. Lightning is quick to utilize this path. A wet tree is a favorite conductor: the lightning follows the wet bark, ripping it off. A man standing nearby is a little better conductor than the wood of the tree. The lightning leaves the tree and strikes the man about at the shoulders, at the level of his wet coat. It may rip these wet clothes off, as it does the bark of the tree. If it travels down a man's legs it may split his shoes or show the point of exit by small punctiform hemorrhages at the soles of his feet. Occasionally it leaves one victim in a group to pass to another. In this case the point of exit is marked by a first-degree burn shaped like the branches of a tree. Since the duration of the shock is extremely brief and the amperage is low, severe burns rarely occur. Post-mortem examinations of fatal accidents show distinctive red mottling of the heart muscle, suggestive of ventricular fibrillation.

Lightning stroke should be looked upon, as is accidental electrocution by domestic appliances, as asphyxial death. It is to be prevented whenever possible.

Martland makes the point that in lightning accidents the rescuer cannot be shocked. Artificial respiration should be practised immediately. Martland reports a case in which by-standers, for fear of shock, refused to touch the victim for 30 minutes.

Each thunderstorm brings the question, Where is the safest spot? Figure 122 illustrates the answer to this question.

It is dangerous to remain in an open boat, in an open field, on a beach, in bathing, or near a tree.

The safest place is in a house, in an automobile, under a steel bridge, or if exposed, lying flat on the ground or sandy beach.

Treatment of Electric Shock

High-tension Currents. Prevention: Drive or walk with care in heavy storms, which may dislocate high-tension wires.

Keep children at a respectful distance from third rails, especially in outlying districts. Never play a stream of water on a third rail or on high-tension wires.

If caught in a storm in an open boat make for shore if possible. Seek shelter or lie flat on the sand. If in an automobile, remain there. You are safe under a steel bridge. Keep dry.

Low-tension Currents. Use common sense in caring for electrical fixtures at home.

Don't monkey with the lights when part of your body is in contact with the plumbing or heating system. If you must fix a short, throw the main house switch. Don't rely on the fuse distributing current to a local group of lights. Don't use lamp sockets to supply current for electrical appliances. Lamps should not flutter and plugs should not hiss. All pullcords should be of string or silk, not metal chain. Use no metal sockets near water.* Neurotics, cardiacs and persons with status lymphaticus are more susceptible to ventricular fibrillation than normal persons.

Treatment. If called to a victim who has touched a high- or low-tension wire, do two things: (1) Send a telephone message to have the power shut off. (2) Break the contact of the wire with the body by means of a long dry pole, stick, or board. If possible, stand on a dry coat, or on a rubber or glass surface when making the attempt. When contact is broken or if it is impossible to move the wire, grasp the victim's leg or arm by means of a dry coat (with no metal in the pockets) and attempt to pull him out of danger. If a dry rope can be secured he may be lassoed and pulled away. Avoid touching the skin of the victim. Remember that as long as the victim maintains contact with the circuit his body is the equivalent of the wire, as a potential source of shock.

Unless the victim is obviously dead, (1) Call for medical help. (2) Apply prone-pressure artificial respiration. Do not mistake muscle rigidity for rigor mortis.

Strong currents do not necessarily induce ventricular fibrillation. All observers are agreed that the victim of electric shock should be regarded as an asphyxial accident, suffering from ventricular fibrillation or paralysis of the respiratory center. As a consequence, artificial respiration should be practiced for long periods.

The reader who has familiarized himself with the principles of resuscitation will understand that the prognosis is not as good as in other forms of asphyxia. The reason is that here we face the highly problematical question of spontaneous recovery of the heart from what is usually regarded as a fatal neuro-cardiac lesion. Unlike many other types of asphyxia in which heart action is intact and in which the reestablishment of the respiration is the end in view, in electric shock artificial respiration is carried on in the hope that its successful performance will induce the heart to recover. In other words, we must resuscitate both respiration and heart action. Emphasis upon the need of cardiac resuscitation in electric shock has led to the impression that the prone-pressure method provides efficient cardiac massage. The literature on this technique makes little mention of this advantage. Perhaps the wish has become father to the thought. While prone pressure is decidedly better than nothing, the physician desiring to massage a heart will usually do so by approaching it anteriorly, making intermittent pressure through the abdominal wall and diaphragm. It is

* "At the Children's Home in the Hawaiian Islands we had an "electric stick" which resembles the steel that comes with a carving set. This was used to heat the babies' bottles at night. One day I used it to heat water for a bath by placing it in the bath-tub. After a little while I stepped into the tub without removing the electric appliance. Much to my surprise jolts were running through my body and I was unable to release my hand from either side of the tub. My screams, however, finally brought help, the current was turned off and all was well." (Private communication)

recommended that resuscitation be maintained for hours, as recoveries are reported to have taken place at the end of long periods. All such reports should be studied in the light of Resuscitation Records, p. 122.*

Summary

Case reports of electrocution are cited. The behavior of electricity and its effect upon the body is noted. An attempt is made to reconcile these effects with the case reports cited. The prevention and treatment of electrocution is rehearsed.

Death from electrocution is asphyxial. The asphyxia induced by the shock is due to an instantaneous failure of the heart from fibrillation of its ventricles. It is believed that ventricular fibrillation has a tendency to wear off if oxygenation of the heart muscle can be maintained during the period of failure. Ventricular fibrillation is induced by three types of current—

a. Low tension currents—Voltage 110, amp. 0.1 to 15. Household equipment. High frequency medical equipment if accidently grounded.
b. Lightning.
c. High tension service currents. Not so likely to induce fibrillation is low tension.

In electrocution by lightning and by high tension currents the resistance offered by the body to the passage of the current may give rise to fatally destructive heat effects paralyzing the respiration and incinerating the tissues (judicial electrocution).

Every flaccid recently electrocuted victim is entitled to endotracheal insufflation of oxygen and massage of the heart.

* Martland, H., and Blumer, G., "The Therapeutics of Internal Diseases," Vol. 3, p. 256, 1941.
Gonzales, Vance and Halpern, "Legal Medicine and Toxicology," D. Appleton, 1937.

Chapter 15

Asphyxia from Gases Encountered in Fire-Fighting

Asphyxial accidents, occurring as they do under dramatic circumstances which call for immediate action for the preservation of a multiplicity of interests, may often be overlooked. Fire or conflagration has become No. 1 offender in this respect. Sudden and devastating properly loss, the immediate danger to life from exposure to flames, the compelling urge to effect rescue—all serve to overshadow and frequently to conceal the all-pervading and deadly presence of toxic gases of combustion which strike unseen and without warning.

It is not within the scope of the present discussion to deal with the science of fire-fighting; but we believe it is extremely important that the asphyxial hazards accompanying fires be more generally recognized. Unless the physician has become acquainted with these hazards by reason of special inquiry, he is not at all likely to realize the very real dangers to which both the victim and the rescuer are exposed.

A single astonishing reference will make clear the need of better coordination between the physician and the fire-fighter. In the vast literature which has accumulated respecting one of the most dangerous fire hazards, carbon monoxide, the experimental work reported has to do almost exclusively with concentrations of less than one per cent. And yet the average CO concentration which may be expected in conflagration is 6 per cent or more. We have been warned by scores of investigators of the danger of 50 parts per 10,000, or 0.5 per cent, but nothing has been said of the danger of 15 per cent! It would appear that the chemical engineer and the experimental physiologist have yet to meet.

In the following pages we shall attempt to bring to a focus a few points of practical interest to the physician and the technician who may be called upon to effect a rescue or to treat the asphyxiated rescued.

Let us first consider briefly the identity and the concentration of the gases evolved by the combustion of common substances. Particular attention will be directed to the CO present, to the reduction of atmospheric oxygen common to fires, to the pathological lesions resulting from the chemical gases produced, and to the indications for treatment and the methods available to meet them.

Asphyxia from exposure to fires and from fire-fighting results not only from the direct effects of heat and flame but more often than is generally realized, from the immediate and remote effects of inhaling the products of combustion under the following conditions: (1) burning in an excess of air; (2) burning in an atmosphere deficient in oxygen, as in closed rooms (laboratory experiments carried on in an atmosphere of nitrogen); (3) in the presence of heat; (4) in a cool atmosphere; (5) at varying levels from the floor.

The effects of actual flame and heat upon the upper respiratory airways are seldom seen, because smoke and gases resulting from the fire produce their effects first. By the time actual flame touches the victim, he is unconscious. Unable to escape, he is charred or consumed. Exceptions may occur from explosions of volatile gases or liquids inhaled by the individuals exposed.

Of common occurrence is the asphyxia which results from consumed cr partially consumed materials whose compositions are known, as for example wood, paper, wool, rubber, silk, and gasoline, as well as various combinations of these substances. Let us consider each in the light of analyses provided by J. C. Olsen.

Wood. Wood or other material consisting chiefly of cellulose is made up largely of carbon, oxygen and hydrogen. Decomposition by heat, as might be expected, yields varying amounts of CO_2, CO, and hydrocarbons. In an excess of air (short period of decomposition), there will be relatively little CO—1.9 per cent as against 7.6 per cent of CO_2. If air is limited, the CO rises to 2.7 per cent and the CO_2 drops to 5.7 per cent. Oxidation changes CO to CO_2. Low temperatures favor the formation of CO_2; high temperatures, that of CO.

Newspapers. Newspapers while burning give rise to gases which are much more dangerous than wood. In an atmosphere of free ventilation they yield 6.6 per cent CO_2 and 6.4 per cent CO. In an atmosphere where oxygen is limited or consumed, so that only smoldering occurs, the CO_2 may approach 43 per cent and the CO 41 per cent, both of which concentrations constitute enormous hazards.

Rubber. Rubber insulation is an omnipresent and well recognized fire hazard. While the quantities of CO_2 and CO produced (CO_2 20.1 per cent; CO 10.1 per cent) are greater than that from burning wood, there exists the complicating factors of sulfur gases and the anaesthetic effect of the unsaturated hydrocarbons.

Oils and Gasoline. Oils and gasoline, strangely enough, give a CO concentration of only 0.6 per cent. This is because they are only partly consumed, a large residue remaining as soot.

Nitrocellulose and Cellulose Acetate Films. An investigation of the potential danger of x-ray film combustion made by Olsen, as a result of the Cleveland Hospital fire in which 125 persons lost their lives, brought to light the following facts relative to the gases which may be anticipated in such fires. The experiments simulated conditions obtaining in closed rooms in which oxygen is present in reasonable quantity.

The combustion of cellulose films produces three toxic gases, carbon monoxide, nitrous fumes and hydrocyanic acid. Acetic acid fumes are also evolved. These fumes are irritating but not highly toxic. In the absence of oxygen, CO is given off in concentrations of 30–40 per cent. As this is approximately the same concentration as that from burning newspapers or other material containing cellulose, the CO hazard from films is no greater.*

Nitrous fumes (NO) are given off in high concentration of from 33 to 42 per cent; this is greatly reduced in an excess of air. Nitrous fumes are entirely absent in the combustion of cellulose acetate. The combustion of nitrocellulose films produces 0.1 to 3 per cent of hydrocyanic acid; none is formed from the combustion of cellulose acetate. When this is coated with gelatin, however, the hydrocyanic acid produced is high, from 0.8 to 0.9 per cent (woolen clothing 1.5 per cent). Olsen stresses the following point as to the presence of CO in an excess of air. The percentage of CO in the absence of air varies from 34 to 49; in an excess of air it drops to 5 to 8; however, the total amount of CO produced increases greatly in an excess of air, as the volume of gas produced per gram of material is much greater. The fire hazard from cellulose acetate is much less than from nitrocellulose and from newsprint paper; since the flash point is higher and the cellulose acetate does not support combustion, the gases evolved from the film explode when mixed with air.

Danger: do not store films in a confined, hot, unventilated space!

Wool. Wool occurs in blankets, drapes, carpets, clothing and upholstery. The decomposition of wool by fire constitutes a very special hazard, for in addition to producing a high concentration of CO_2 (33.1 per cent) and CO (17.7 per cent), it also gives rise to a very high percentage of hydrogen sulfide (25.8) as well as 3 per cent of ammonia and 1.8 per cent of hydrocyanic acid.

If the fire happens to be hot instead of cool, the CO_2, CO, and H_2S are lowered to 13.4, 6.8, and 9 per cent respectively; but the ammonia is raised to the utterly irrespirable concentration of 57.2 per cent.

Silk. The combustion of silk is similar to that of wool, the only difference being the absence of sulfur.

Another point of practical importance brought out by Olsen is that the gases of combustion tend to stratify in layers at various heights from the floor. If the air is hot, the gases rise; if it is cool they tend to sink to the floor. In quiet air the area between 3 and 5 feet offers the lowest percentages.

* *N. Y. Herald-Tribune*, Dec. 13, 1943: "Two trucking company employes were suffocated inside a large, closed furniture moving van when a fire started in a pile of burlap furniture coverings. The fire was discovered by a pedestrian who saw smoke coming through cracks in the body of the van. A motorist took the men to a hospital, where attempts to revive them failed.

Let us reconstruct briefly the potential hazard which may result, for example, from a fire in a well furnished bedroom. There are present woolen draperies, blankets, carpets and upholstery. There is a bedspread and wearing apparel containing silk. The floor and the trim is of wood and there is a shelf of books and wall paper. There are electrical appliances with lengths of rubber-insulated wire. The heavy sash windows can be opened only with difficulty. The air of the room is hot.

The following gases in the concentrations stated may be anticipated:

Gas	Source	Per Cent
Carbon dioxide	Paper, wood, wool, silk	20 –30
Carbon monoxide	Paper, wood, wool, silk	10 –15
Hydrogen sulfide	Wool, silk	8 – 9
Ammonia	Wool, silk	40 –50
Hydrocyanic acid	Wool, silk	1.5–2.0

According to laboratory investigations by Sayers, Yant, *et al.*, animals succumb quickly in the following gas concentrations:

Gas	Per Cent
Carbon dioxide	12–30
Carbon monoxide*	0.5
Hydrogen sulfide	0.1
Ammonia	0.5
Hydrocyanic acid	0.04

*In this concentration of 50 parts in 10,000, consciousness is lost in 10–15 minutes.

Comparison of the above figures will immediately indicate the extraordinary hazard faced by firemen and others in rescue attempts. Exertion such as climbing stairs, and the stimulating effects of high CO_2 concentration tend to overcome the warning and choking effect of ammonia, acetic acid, sulfur and other gases, and to bring about a rapid loss of consciousness from the inhalation of the extremely high concentration of the toxic gases.

In conflagration, therefore, the immediate effects of fire may be a minor danger compared with the devastating asphyxial consequences of combustion. Possibly this knowledge may provide solace, for while a man may be burned or charred alive, the chances are all in favor of his being thoroughly anaesthetized before he is aware of the pain of fire.

It is to be noted that the asphyxial hazards outlined above are occasioned by (1) the anemic anoxia of CO and H_2S, accelerated by the overventilation resulting from high percentages of CO_2; (2) anoxic anoxia precipitated by the choking effects of ammonia and acetic acid; (3) the histotoxic anoxia produced by the cyanogen radical of hydrocyanic acid, and hydrogen sulfide.

The anoxic effect of low concentrations of oxygen has not yet been alluded to. A minimum of 10 per cent oxygen is required to maintain safety in anaesthesia. In submarine air-conditioning, 17 per cent is the low limit; below this a candle flame is extinguished. Symptoms of oxygen lack become evident when the oxygen has dropped to 13 per cent (Brown). At an altitude of 15,000 feet, oxygen concentration is 12 per cent. Long before 30,000 feet (6 per cent) is reached, anoxic asphyxial effects are to be expected (see pages 137 and 138). The critical point at which all healthy men are expected to fail is 6 per cent (Henderson).

In fires, therefore, we are faced with overbreathing from both CO_2 and oxygen deficiency, and the overwhelming effects of CO, H_2S or hydrocyanic acid.

A moment may be well spent in directing our attention to the carbon monoxide hazard. The average of ten different conditions stated by Olsen (36; 6; 22; 7; 6.2; 10; 7.9; 17; 6; 12.4) is 10 per cent carbon monoxide. As has been noted, it would appear that medical literature has been largely limited to the investigation of very low concentrations of this gas. The terminology employed deals with concentrations or parts of CO per 10,000 parts of air. One part per 10,000 may be found in the blood of the ordinary city dweller. If a car is allowed to run in a closed, one-car garage for 10–15 minutes a concentration of 50 parts may be anticipated. If this is breathed for ten minutes, helplessness will ensue, followed by death in 30 or 40 minutes. The undiluted gas of the exhaust of an automobile contains from 3.5 to 6.5 per cent; illuminating gas contains from 6 to 34 per cent.

The carbon monoxide respired unites with the hemoglobin of the blood 300 times as readily as does oxygen. Its effects are purely biophysical, a percentage of the oxygen-carrying capacity of the hemoglobin being thrown out of commission. The action and the combination is reversed if pure oxygen is breathed. (CO is not in chemical solution with the hemoglobin of the blood.) The anticipated percentage reduction of the oxygen-carrying capacity of the hemoglobin thrown out of action, or the concentration of CO in the circulation which follows exposure to a given concentration of the gas, has been worked out simply by Henderson as follows:

The total available oxygen in the circulation is about 600 cc. At rest a man uses about $\frac{1}{3}$ of this, or 200 cc.; while exercising he may require $\frac{4}{5}$ (80 per cent) of it, or 480 cc. If he reduces his oxygen-carrying power by 25 per cent (150 cc.) by hemorrhage or CO and this deficit is not supplied, if exercising he will faint, for he cannot supply the necessary 480 cc. of oxygen.

The problem is to estimate how much CO will be required to eliminate 25 per cent of the carrying power of the blood, thus bringing about unconsciousness, in a stated period of exposure or in exercise. One part per 10,000, or 0.01 per cent, is a common saturation in automobile traffic. A man at rest breaths 8 to 10 liters a minute, of which 6 liters, or 6000 cc., reaches his capillary circulation. Breathing an atmosphere of 0.01 per cent concentration, he would inhale 0.6 cc. of CO in a minute, 6 cc. in ten minutes. Six cc. is $\frac{1}{100}$ of 600 cc., his total oxygen capacity; therefore his blood would be one per cent saturated in ten minutes.

Owing to the affinity of CO for hemoglobin, however, (300 times that of oxygen) there is a cumulative effect from continued inhalation, so that with 0.04 per cent blood concentration, for example, the saturation may rise to 44 per cent; 1.5 per cent may produce 75 per cent saturation and death. (Suggested formulas: 1 per cent saturation of blood for every part of CO, at rest, at the end of ten minutes; 25 per cent saturation by 0.25 per cent). If exercising, the effect is proportionate to the overbreathing which occurs.

Summary of the Pathological Lesions Produced by Gases of Combustion

Carbon monoxide acts exclusively by forming a physical combination with the hemoglobin of the red blood cells. All other immediate and remote effects are due directly or indirectly to the resulting asphyxia. Insects such as cockroaches, which have no red blood cells but which possess tissue cytochrome for oxygen utilization, are entirely unaffected when placed in an atmosphere of 20 per cent oxygen and 80 per cent CO. Haldane placed rats in such an atmosphere. When they were overcome by the anemic anoxia produced, he revived them by increasing the gas pressure in which they were placed to two atmospheres. The oxygen then in solution in the blood plasma was sufficient to meet the tissue needs, and the rats recovered (Henderson).

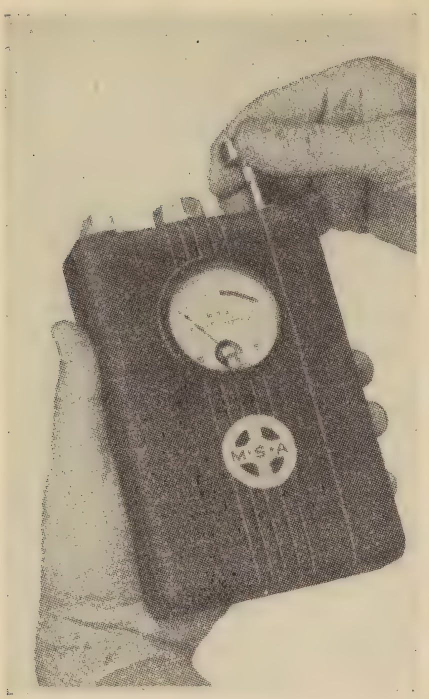
Carbon Dioxide. The pathological lesions resulting from CO_2 poisoning are produced indirectly from (a) the effects of the increased inhalation of toxic gases from the overbreathing stimulated by this gas; (b) the asphyxia which results from low concentrations of oxygen due to its physical displacement by CO_2.

Ammonia. Inhalation brings about immediate irritation of upper airway. Anoxic anoxia may develop through spasm of the respiration (not unlike the asphyxia produced by drowning). If ammonia is present in respirable concentration, laryngeal, tracheal or bronchial inflammation or pulmonary edema may follow.

Hydrogen Sulfide. More attention should be directed to the hazard of hydrogen sulfide. The familiar odor of rotten eggs may not be apparent if the gas is cold or in high concentration. It acts in concentrations of 0.5 per cent or more, as does hydrocyanic acid. The danger of its presence has been largely overlooked.

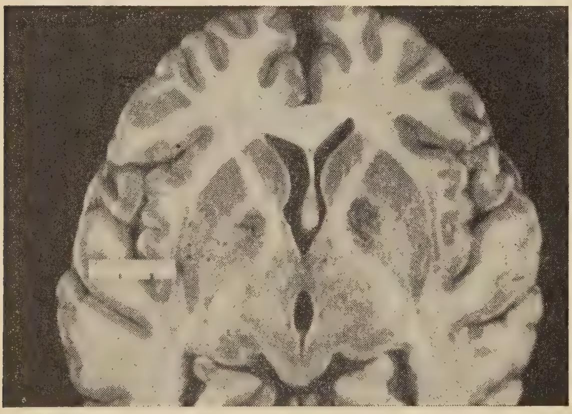
Hydrocyanic acid (**Prussic Acid**). The rapidly fatal nature of hydrocyanic acid is due to the double effect which it produces in extremely low concentrations. (a) It combines with the hemoglobin of the red blood cells in the manner typical of CO. (b) It combines with the catalyst cytochrome, an iron-containing substance in the tissues, preventing the cells from utilizing oxygen in the circulation. The affinity of cytochrome for the cyanogen radical is high as compared to its affinity for CO, which is so low as to be unimportant in lethal concentrations of the gas.

Smoke. The indications for the treatment of fire accidents suggest a brief reference to the nature of smoke.



Courtesy Mine Safety Appliances Co.
Fig. 123. Explosimeter used to determine combustible gas hazards.

Courtesy Mine Safety Appliances Co.
Fig. 124. Mask used for protection against sulphur fumes when working about electrical refrigeration.



Courtesy Dr. Harrison Martland
Fig. 125. Kolioko's lesion (carbon monoxide poisoning).

Courtesy Mine Safety Appliances Co.
Fig. 126. "All-service" mask with "all-vision" facepiece.

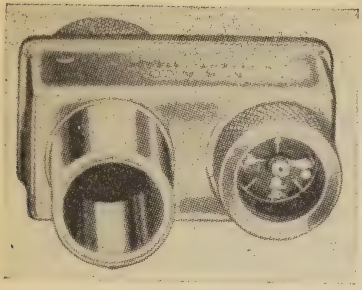
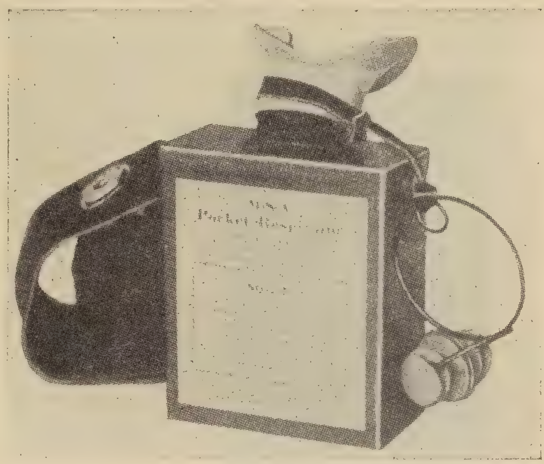
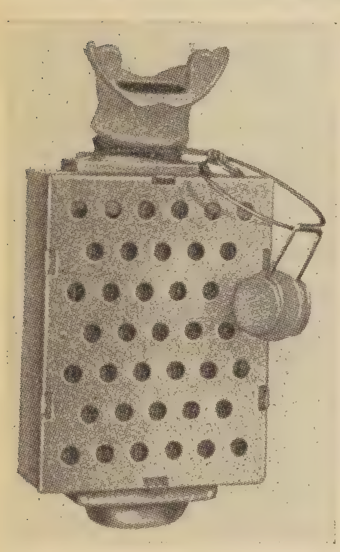


Fig. 127. Automatic timer, which indicates the service time undergone by the canister shown in Fig. 126.
Courtesy Mine Safety Appliances Co.



Courtesy Mine Safety Appliances Co.
Fig. 129. Pocket respirator.



Courtesy Mine Safety Appliances Co.
Fig. 128. "Self-rescuer" for short periods of emergency protection.

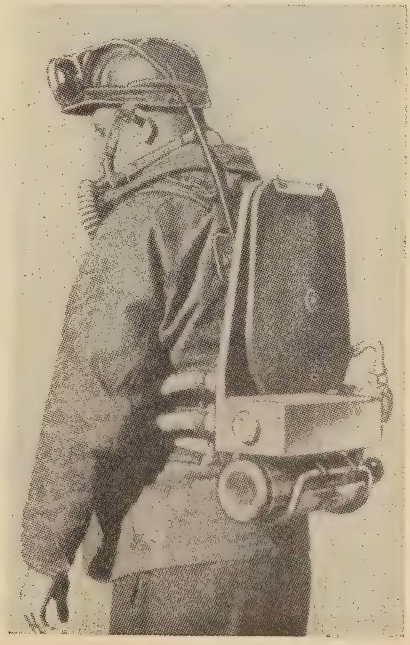


Fig. 130.
Oxygen rebreathing apparatus.
Courtesy Mine Safety Appliances Co.

Smoke is an aerial concentration of minute solid particles, accompanied by toxic gases resulting from combustion, or an aerial concentration of minute liquid particles resulting from chemical reaction not involving combustion. It does not follow laws of gaseous diffusion. If smoke is released in warm air it will rise; if in cold air it will cling to the floor. It follows the movements of air.

Precautions. The pattern indicated for the treatment of asphyxiated fire victims is clear from the pathology just referred to. Rescue precau-

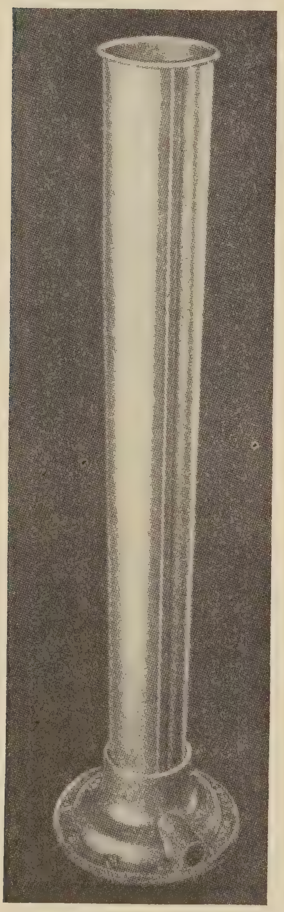


Fig. 131. "Air Mover." This tube, open at both ends, produces strong suction when compressed air is blown through it. Suction is used for ventilation. *Courtesy Mine Safety Appliances Co.*

tions include: gas-tight oxygen rebreather masks for all persons entering gas-contaminated atmospheres; immediate removal of victims from such atmospheres. For the depressed asphyxiated, oxygen and CO_2 administration by the methods and equipment previously described. (Dilute acetic acid fumes for ammonia poisoning should be added to the inhalation bag. For the spastic case, oxygen and CO_2 by the techniques and equipment described earlier. For the flaccid, completely relaxed, asphyxiated victim, endotracheal suction, intubation and oxygen-CO_2 insufflation, as an immediate life-saving measure and for safety during transportation.

The treatment for shock or for pulmonary edema is described on page 43. It includes the use of intravenous methylene blue or other suitable oxidizing agents to help neutralize the toxic effects of the cyanogen or sulfur compounds in cyanide or hydrogen sulfide poisoning.*

The Cocoanut Grove Disaster (Boston, November 28, 1942)

In this conflagration, out of an estimated attendance of 1000 persons, approximately one-half (498) lost their lives. According to Churchill and Faxon, who released a preliminary report of the participation of the Massachusetts General Hospital, approximately 20 per cent of the fatally injured were received by this institution (75 dead on arrival or died within 15 minutes). Thirty-nine additional victims entered for emergency treatment. Of this number seven subsequently died. Those who succumbed were reported to have inhaled flames and fumes. Of six patients who were tracheotomized, four died. The authors state: "Some of the dead showed no burns; they had obviously been asphyxiated. Many showed the cherry red of carbon monoxide poisoning. Others showed burns, but death from asphyxia." It is stated that most of the women were found to have been without shoes. This fact suggests that these were kicked off in the struggle to escape. Extreme physical exertion is implied—insuring rapid asphyxiation. It was noted that some of the patients were maniacal, "apparently from cerebral asphyxia."

"The 39 living patients were frightened, cold, and dripping wet. Subnormal temperatures were observed. Every patient received at once $\frac{1}{4}$ gr. of morphine sulfate hypodermically from a large common syringe, one needle only being employed. The solution was made up by adding a $\frac{1}{4}$-gr. tablet to each 1 cc. of sterile water. [This is common-sense treatment of a critical emergency.]

"Units of 250 cc. of plasma, from a frozen store, were administered to all patients with low blood pressure. Difficulties encountered in the intravenous administration of these solutions in irrational patients suggest the more general use of intrasternal injection. All temperatures taken were rectal. The late appearance of respiratory lesions (24 cases) in the apparently uninjured, demonstrates in this disaster the wisdom of hospital treatment for everyone until the pattern of trauma was fully disclosed." The late pulmonary edema following the inhalation of poison gases (page 297) may well be recalled here.

"The question of poisonous fumes was immediately raised, but definitive evidence on this point is still lacking."

The investigations of Olsen which have been quoted in this chapter would seem to suggest that there is scarcely need for a search for such definitive evidence; the fact of the fire provides it.

* A. M. Prentis, "Chemicals in War."

Summarizing the experience gained from this disaster, the authors conclude:

That the immediate demand for telephone service was enormous.

That immediate segregation of the living and the dead should be effected by two physicians.

That all women should carry personal identification, such as bracelets, rings, etc.

That intrasternal administration of plasma should be popularized.

That casualty-clearing facilities should be immediately set up (see page 45, British War Office—*author's note*).

That information as to gas hazards at the accident be made available in order that trauma may be anticipated.

That adequate morgue facilities be provided with segregation of bodies and means of identification.

That supervision be exercised by medical examiners as to bodies and personal effects.

That official count and identification of living and dead be made available to the press as early as possible.

Chapter 16

Asphyxia from Mechanical Obstruction of the Respiration

The patient is clinically **well** *before the accident*

Mechanical obstruction of the respiration is the simplest form of anoxic anoxia. The multiplicity of terms designating specific forms of obstruction, such as choking, suffocating, strangulation, etc., although descriptive of the lesion produced, diverts attention from the principal issue, which is *asphyxiation.*

It seems to the author that, if such emphasis could be placed upon what is occurring, *i.e.*, impending death, instead of upon the remote medico-legal aspects of the case, a life could now and then be saved. In other words, if the patient who is choking, suffocating or strangling were viewed as alive but about to die, instead of already dead, the internist, the surgeon or the general practioner in attendance would be more alert to his responsibilities.

Asphyxiation from mechanical causes occurs as follows:

In Infants by

Smothering: Respiration through the nose and mouth is prevented by bedclothes and pillows, or by their parents or animal pets overlying them in their sleep.*

Choking: By inhalation of material vomited, such as milk curds; by small toys put into the mouth and caught in the pharynx or trachea.

Strangulation (*Ligature*)*:* By strings tied to toys and by tight ribbons around the neck. By homicidal suffocation and strangulation.

* Mary Casey, four months old, suffocated in her carriage when she rolled face-down on the pillow. Her mother, Mrs. Gertrude Casey, discovered the tragedy a half hour after she had placed the baby in the carriage. Efforts by a doctor, an inhalator squad of the Westchester Lighting Company and the Fire Department Rescue Company, to revive the infant were unavailing. (Yonkers Herald Statesman, Dec. 8, 1943)

Four-months-old Valerie Schulze was suffocated in her bassinet in a house where she was being boarded while her father is serving in the United States Army and her mother is working in a war plant, according to police. Mrs. Gertrude Gulick, who boards children at her home, went to the room at 7:40 A.M., found the covers over the baby's head and the infant apparently a victim of suffocation. Mrs. Gulick called to her husband, who carried the baby to the first floor. They tried to revive her, pending arrival of police, and Dr. Robert Minervini, who responded with an ambulance from Yonkers General Hospital. Patrolmen of the Emergency Squad, summoned by Dr. Minervini, administered oxygen without success. Dr. Minervini pronounced the baby dead of suffocation, police said. Yonkers Herald Statesman, Dec. 9, 1943)

In Children by

Choking: By toys and odds and ends placed in the mouth and either partially swallowed or aspirated into the trachea (Fig. 132).

Strangulation (Ligature): By accidents during games with ropes, lassos, etc.

In Adults by

Smothering: By postural occlusion of mouth and nose while unconscious, occurring in alcoholism, under hypnotics, in epilepsy, etc. (Fig. 133).

By homicidal attacks, in which the mouth and the nose of the victim are closed by the assailant's hands (mugging). When the victim is attacked from the rear and thrown to the ground, with the assailant's weight on the victim's back and his hands closing the nose and mouth, the assault is referred to as Burking.

Fig. 132.
Foreign-body obstruction: peanut in trachea (17-months infant).
Courtesy Dr. Harrison Martland

Suffocation: By foreign matter which finds its way into the air passages when the head is covered by sawdust, flour, sand, ashes, or coal; in the collapse of buildings or excavations; and in landslides (Fig. 134).

I would like to describe a most unusual death. A man went into a river for a swim and was found on the bank dead. The ambulance bearer who was summoned started artificial respiration. On examination of the mouth something was seen which on further examination proved to be a fish. The fish was firmly lodged and attempts at removing it only removed the tail. A tracheotomy was performed and artificial respiration was continued without any response. At post-mortem examination I found a bream about five inches long in the pharynx with its head firmly fixed in the larynx and completely occluding it. Unfortunately, there were no witnesses to observe this extraordinary accident. Medical Journal of Australia, July, 1943)

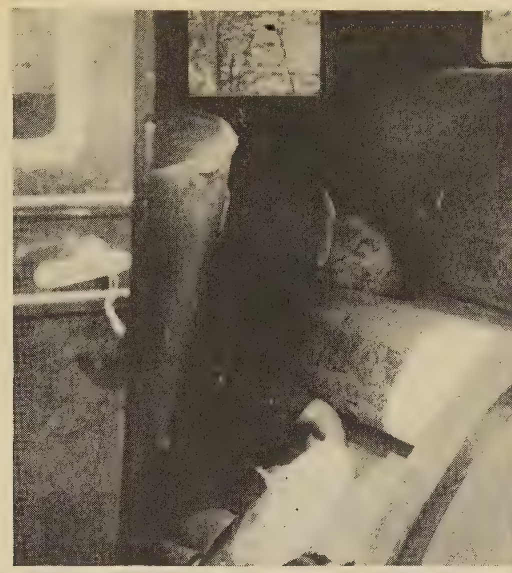


Fig. 133.
Postural smothering (alcoholism).

Fig. 134.
Soft coal in lungs as a result of suffocation in a coal bin.

Courtesy Dr. Harrison Martland.

In sudden laryngeal spasm from chemical gases in the ordinary combustion of fires, or in chemical warfare by chlorine, ammonia, hydrogen sulfide, etc.

Choking: By partial swallowing and impaction of a bolus of food between the epiglottis and the glottis (Figs. 135 and 136). By the inhalation of vomited material, especially in the unconscious, alcoholic or epileptic (Fig. 137).

Strangulation (Manual) Homicidal: By closure of the air passages from external pressure upon the neck (Fig. 138).

Strangulation (Ligature) Homicidal or suicidal. By pressure applied around the neck by a rope, wire, suspenders, straps, etc. (Figs. 139, 140, 142).

Hanging (Homicidal or suicidal): Hanging is strangulation by ligature in which compression of the neck by the rope, wire, etc. is produced by the partial or the full weight of the body drawing the ligature tight. Note: weight of the body is applied without dropping through a distance. (Figs. 143, 144).

Fig. 135. Bolus of food in glottis.

Fig. 136. Glottis after removal of meat.

Lynching or Judicial Hanging: If the body is dropped a distance before hanging or strangulation the death is not asphyxial, but is due to fracture of the cervical vertebrae. Judicial hanging is not asphyxial.

In asphyxia from smothering, the patient may present no local evidence of mechanical obstruction. Superficial cyanosis and venous engorgement of the head, neck and ears may be present, however, accompanied by pallor of the face due to the pressure which caused the smothering. Hemor-

Fig. 137.
Asphyxial death during anaesthesia due to aspiration of vomitus. Operation for lacerated wrist.
Courtesy Dr. Harrison Martland

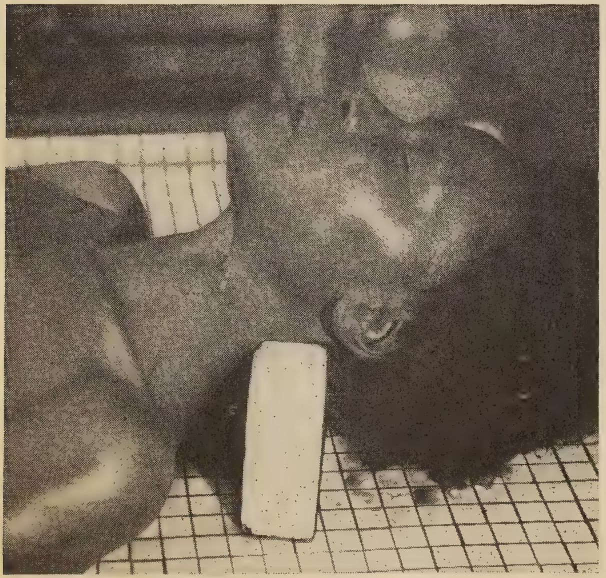


Fig. 138.
Homicidal strangulation showing marks of assailant's fingers on neck.
Courtesy Dr. Harrison Martland

rhage of the conjunctiva, bulging of the eyes, etc., will depend upon the vigor and the persistence of the circulation preceding death. An early circulatory failure may result in general pallor with gray mucous membranes.

Asphyxia from choking is likely to be accompanied by engorgement of the upper parts of the airway; this swelling produces a secondary mechanical obstruction which must be dealt with, when and if the foreign body which has induced the choking is removed. It would seem obvious that something more than prone-pressure Schaeffer is indicated in such accidents. An airway, oral, nasal or tracheal, through which air can easily pass, is mandatory.

In asphyxia due to manual strangulation, contusions of the neck are common. Fractures of the cricoid, or thyroid cartilage or the hyoid bone is of frequent occurrence. The head and face are cyanotic, the face often swollen and almost black, the tongue protruding and firmly caught between the teeth, the finger tips cyanotic; often numerous asphyxial petechial hemorrhages occur over the shoulders, back and chest.

The position of the parts in manual strangulation in those dying with the hands of the murderer still on the throat is almost identical with that found in suicidal hanging. The air passages are completely blocked by the base of the tongue being shoved backward and slightly upward, the epiglottis closing the glottis, its posterior surface in apposition to the posterior pharyngeal wall. Should these injuries involve edema of the glottis, immediate intubation or treacheatomy is indicated (p. 248).

In asphyxia due to strangulation by ligature, tightened manually or by hanging, the larynx or tracheal rings are likely to be fractured. Edema of the soft parts of the upper airway and enormous engorgement of the tongue result (Fig. 141). Gonzales states, however, "I have found that in a series of about fifty cases of asphyxia due to ligature—suicidal deaths—in most instances there is little change in the internal tissues of the neck, such as is ordinarily described as hemorrhage, tears, and so forth, of the thoracic arteries and ligaments. The striking feature of these cases is the strangulation mark on the skin and of the neck (Fig. 143). This is probably because in most suicides the body drops a short distance (Fig. 144), in contradistinction to legal executions in which the body may drop 6 or 8 feet, with trauma to the neck, even resulting sometimes in a fracture. In some strangulation cases, the marks on the neck are slight or absent, especially when soft material has been used in the strangulation." (Fig. 142.)

Observations have been made of a short stoppage of the heart due to irritation of the vagi at the moment of suspension; the inability to resuscitate persons cut down almost at once has been accounted for in this way. Others have called attention to the superficial location of the superior laryngeal nerves and their important connections with cardiac ganglions and nerve plexuses. In these hangings death usually results in from five to ten minutes after consciousness is lost, even though the heart may

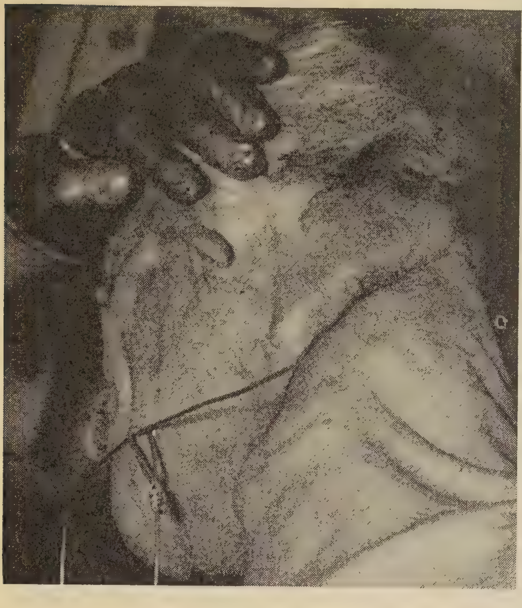


Fig. 139.
Mechanical respiratory obstruction due to hanging with a steel wire.
Courtesy Dr. Harrison Martland

(Courtesy Dr. Harrison Martland)
Fig. 140. Mechanical respiratory obstruction by ligature.

Fig. 141.
Edema of glottis (pathological respiratory obstruction)
Courtesy Dr. Harrison Martland

(Courtesy Dr. Harrison Martland)
Fig. 142. Mechanical respiratory obstruction by ligature.

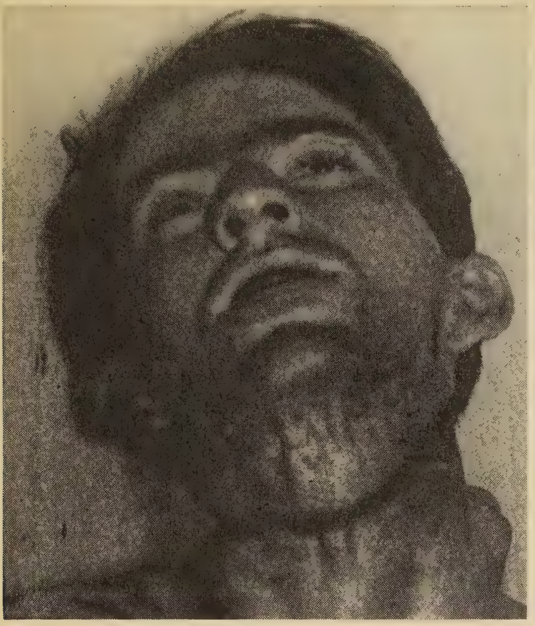
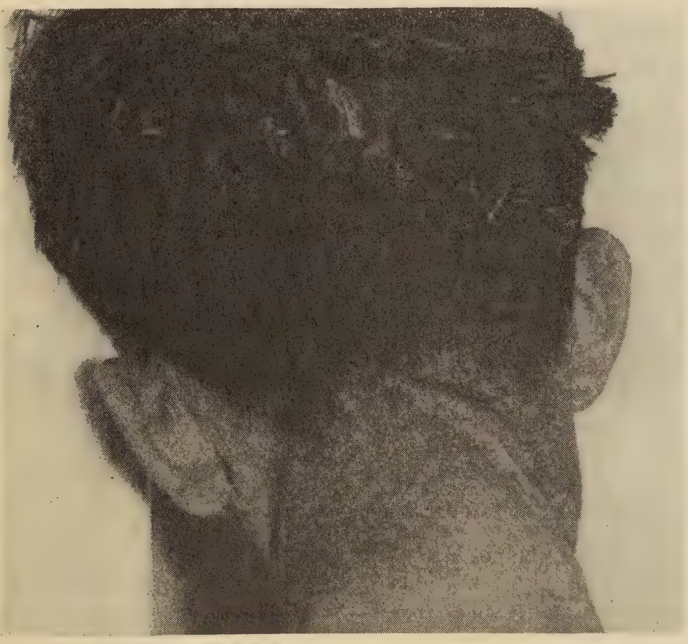


Fig. 143.
Mechanical respiratory obstruction by hanging. Note furrow made by clothesline attached to a door.
Courtesy Dr. Harrison Martland



Courtesy Dr. Harrison Martland
Fig. 144. Suicidal hanging from clothesline attached to a door Note furrow.

continue to beat for eight to ten minutes after the cessation of respiration. In view of this latter fact, resuscitation is possible, and artificial respiration should be started not later than five or six minutes after suspension.

Experience is that the main factor in these hangings is asphyxiation due to closure or shutting off of the air passages and that all other factors are usually of secondary or contributory importance. If the ligature is soft, such as a sheet or scarf, one may find only a broad, pale depression which may disappear in time and leave practically no mark. If a belt or suspenders were used, the marks of the buckles may sometimes be recognized.

The face above the furrow is often pale, however, if the strangulation has been slow and not too tight, resulting in compression of the veins before the deeper arteries. Marked cyanosis may be present above the ligature.

Martland found in most cases of hanging that the direction of the constricting force, which is almost always upward and backward, pulls the hyoid bone with the base of the tongue backward and upward. The normal space between the base of the tongue and epiglottis is obliterated, so that the latter lies in apposition with the anterior surface of the epiglottis. The epiglottis is pushed backward so that its posterior surface is in apposition with the posterior pharyngeal wall, effectively blocking the glottis. The same is true of the soft palate and uvula, so that passage of air from the nasal cavities is blocked (Figs. 145 and 146).

The position assumed by the tip of the tongue usually depends on the relation of the ligature to the hyoid bone and the direction of the traction. If the ligature comes just below the hyoid, the tongue is forced upward and slightly backward, its tip being forced forward so that it may either protrude through the lips, or may be caught between the teeth or, if the jaws are firmly closed, behind them. If, however, the ligature is very high and the main pressure is above the hyoid, the base of the tongue is forced upward and backward and the tip will be found behind the teeth and pulled well back into the mouth. This same position occurs when the ligature is very low and the traction is more circular (see Fig. 147).

Relief of these forms of mechanical obstruction is urgent and technically difficult. Exposure of the airway should be immediately attempted; this alone may permit the passage of air through the damaged and swollen tissue.

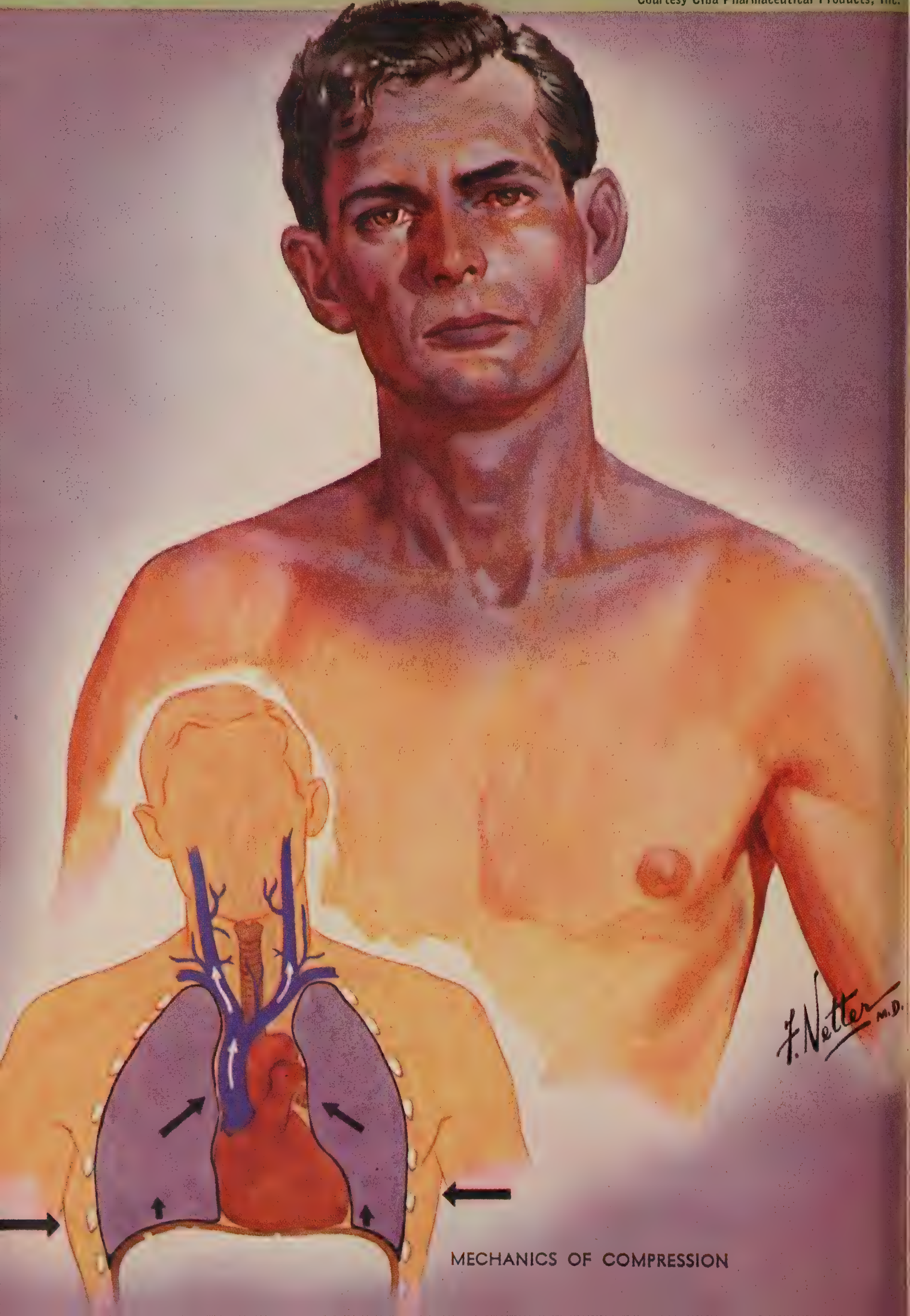
Asphyxia by Pressure upon the Chest Wall

Compression of the chest by moving objects, such as automobiles, elevators, laundry equipment, etc. is not infrequent. In the collapse of buildings timbers may fall across the chest, restricting respiratory movement.

Occasionally in such crushing accidents the sudden mechanical pressure may overdistend the capillaries of the face and neck, causing a deep purple

TRAUMATIC ASPHYXIA

Courtesy Ciba Pharmaceutical Products, Inc.

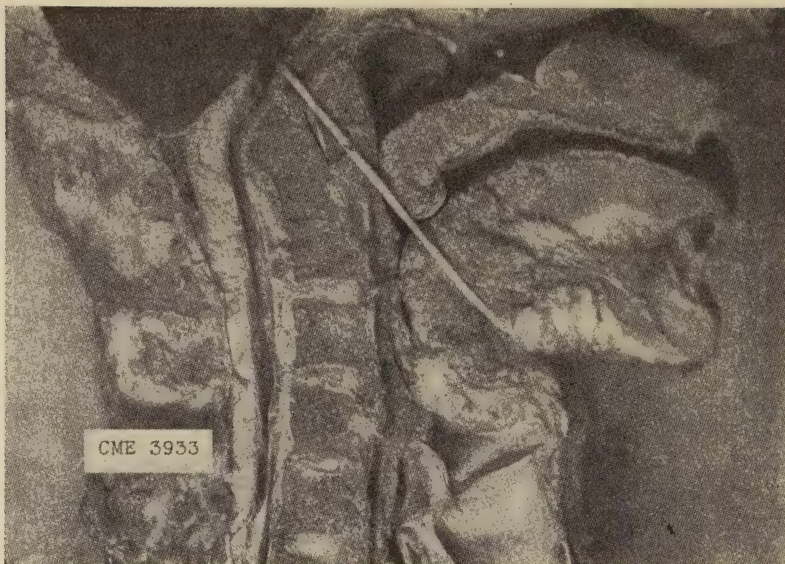


discoloration down to the clavicles, where a sharply demarcated horizontal line occurs with apparently normal skin below it. The appearance of the patient is striking and weird. The discoloration may disappear in one or two weeks without passing through the color changes of a bruise. In this connection it is well to note in passing the splinting effect of upper abdominal pain, the pressure of dressings, the use of sedatives in reducing normal excursions of the chest (raising of the ribs and lowering of the diaphragm) in post-operative surgery. Mechanical pressure effects are likely to be overlooked because of concurrent injuries which may be urgent and acute. Collapse of the lung through punctured wounds (accidental pneumothorax) of the chest wall, either by fractured ribs or otherwise, must be kept in mind.

Courtesy Williams & Wilkins Co.

Fig. 145. Median section of normal head and neck to illustrate patency of air passages.

Note the normal position of the tongue, soft palate and uvula; the normal space between base of tongue and epiglottis; the erect position of the epiglottis; the normal space between posterior surface of the epiglottis and the posterior pharyngeal wall; the distance of at least 3 cm. between the top of the epiglottis and the interarytenoid arch, allowing full view of the vocal cords from above; the space between the soft palate, uvula and the posterior pharyngeal wall allowing free access of air from nasal passages to glottis; and the wide open structures through which air has a free passage back and forth with respiration. All these spaces with their accurate measurement is of prime importance in the establishment of death due to strangulation, and not the ordinary data usually furnished in these cases, such as the color of face, presence of fluid blood, petechia, etc. Most of the real evidence has escaped notice because the usual autopsy technique almost entirely destroys it. (Office of Chief Medical Examiner, Essex County, N. J.) From "The Medicolegal Necropsy," ed. by T. B. Magath.



Courtesy Williams & Wilkins Co

Fig. 146. Median section of head and neck in suicide by hanging with clothes line.

Note position of tongue. Its base is pushed backward and upward by the traction of the rope, and because the point of traction is just below the hyoid bone, the tip of the tongue has been pushed anteriorly, projecting between anterior dental arcade (which in this case is formed by artificial plates).

Note that the base of tongue is jammed back into throat completely cutting off the entrance and exit of air in respiration. The normal space between base of tongue and epiglottis is obliterated so that the base of tongue lies in apposition with the anterior surface of the epiglottis. The epiglottis is pushed backward so that its posterior surface is in apposition with the posterior pharyngeal wall, effectively blocking the glottis.

The soft palate is pushed backward and upward, its posterior or superior surface coming in apposition with the posterior pharyngeal wall, effectively blocking the passage of air from the nasal cavities. The uvula in this case is caught and bent anteriorly against the inferior surface of the soft palate. (Office of Chief Medical Examiner, Essex County, N. J.) From "The Medicolegal Necropsy", ed. by T. B. Magath.

The Conservative Treatment of Respiratory Obstruction from Mechanical Causes

Asphyxia occurring from obstruction comes entirely without warning. Interference is most likely to be successful in infants and small children, first because of the degree of punishment that they will tolerate and survive, secondly because they offer less mechanical resistance to relief. An infant or a child exhausted by his struggle for air and partially anaesthetized by the onset of asphyxia offers little objection to instrumentation (p. 128). Vomitus, small foreign bodies, and post-operative pharyngeal or nasal packs can easily be seen and as easily removed, if above the glottis. In-

Courtesy Williams & Wilkins Co.

Fig. 147. Median section of head and neck in suicide by hanging with leather belt. Note the position of the tongue. On account of the width of the belt there has been compression and traction on tissues above and below the hyoid bone. On account of the high compression and angle of traction, the tip of tongue remains in mouth in back of anterior dental arcade. Otherwise, note the same obliteration of the normal spaces as seen in the preceding case with effectual blocking of air from nasal cavities and from mouth. Note complete blocking of glottis by epiglottis. (Office of Chief Medical Examiner, Essex County, N. J.) From "The Medicolegal Necropsy", ed. T. B. Magath.

spection of the airway should always follow ineffective slaps on the back or suspension by the heels. Vomiting induced by these First-aid methods may complicate the existing obstruction.

Adults asphyxiated to a point of relaxation by suffocation, choking or strangulation are entitled to medical care. The problem is not only immediate but involves post-operative care. The progressive edema which may result from the trauma of foreign body removal should be anticipated. Dr. Chevalier Jackson makes the following lucid and forceful comments*:

"When a patient is asphyxiated, he is as limp as a rag. With an instrument one can look directly down into the trachea without any resistance on the part of the patient, because he is completely relaxed. One thing about asphyxia is complete and absolute relaxation after respiration has stopped. We are careful to lift the patient's upper lip to put it out of the way. The directoscope is put in, and by a lifting motion the curves (of the airway) are obliterated. In a patient asphyxiated from suicidal intent, everything is crowded back against the posterior wall and the larynx is completely obliterated.

* J. Am. Med. Assn., p. 314 (July 22, 1933) Abst. of S.P.A.D.

"Suppose that a man is cut down before his heart has stopped. We put in a laryngoscope, lift up on it, and pull away all of the tissues that have been crowded back by the rope (Figs. 146 and 147); we lift those away to clear that area. In nearly all anaesthesias there is a dropping back of the tongue and all the tissues. It is to clear this airway that Dr. Flagg devised a laryngoscope available for general use for the prevention of asphyxia. It has prevented thousands from passing away under anaesthesia by clearing the airway. This instrument is not used to pry on the upper teeth. It is a lifting motion. The whole head is lifted off the table in order to clear this area. The operator can look straight into the windpipe, put in an aspirating tube and clear out the obstructing mucus. He is then ready for the insufflation of the oxygen with its 5 to 10 per cent of carbon dioxide.

"There is no use trying to force in oxygen and carbon dioxide when the larynx is plugged up. This drives the plug in tighter. The only way one can determine whether or not there is any obstruction is to look and see. One looks into the mouth, but can't see down to the larynx unless the tongue is displaced with the direct laryngoscope.

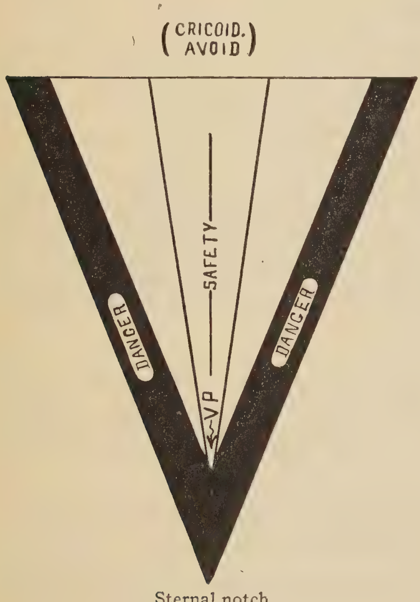
"A bronchoscopic operating room is prepared to deal with obstructions in the air and food passages and with every emergency and problem that can arise. It is adapted for the relief of asphyxia in patients *who can be brought in*, but there must be some sort of ambulance service that can get promptly to the patient and that can be equipped not only with the small instrumentarium required, but with men trained in its use. That is an absolute necessity (See pp. 82, 105).

"A patient who is asphyxiated from any obstruction above the level of the clavicles gives unmistakable signs of what is the matter with him. At every inspiration his chest sinks in. There is also a sinking in at the suprasternal notch. On expiration those depressions fill out. The patient takes another inspiration and again this sinking in occurs. The patient is becoming asphyxiated from an obstructive laryngeal dyspnea. It does not occur in asthma, in cardiac dyspnea, or in any form of dyspnea but the obstructive laryngeal type, which is the most common of all forms of surgical dyspnea. I am not speaking now of gas accidents in asphyxia or of industrial accidents.

"Here is the other symptom: The patient is restless. He is working so hard to pump in air that he doesn't want to be bothered. He doesn't want to take food. He just wants to be left alone. He is putting in all his time to pull in air, because there is a sinking of the suprasternal notch. That is a typical picture of asphyxia from an obstructive larynx dyspnea. After the breathing is stopped, one has to get it started again."

Dr. Charles J. Imperatori, agreeing with Dr. Jackson, adds the following comments:

"I agree with the method that Professor Jackson has presented. The suggestion is made that an emergency transportation service be made available in contradistinction to bronchoscopic clinics (the repair shops). With regard to trauma and other objections that have been raised to this method, I can positively say that there are no traumas that will ensue provided the individual doing the laryngoscopy and the intubation has been given sufficient instruction and has sufficient intelligence. Any method, no matter how simple, can be made, one might say, a destructive method if the individual using it does not use his intelligence. What is done by this method is, as it were, to extend the trachea and bronchi out beyond the mouth and beyond the danger zone which has been described by Dr. Jackson. Then, by insufflation, the needed oxygen is supplied to the individual. Gas therapy is not a new thing. It has been used for a long time. This method developed by Flagg has made it easy for those who are not continuously doing bronchoscopic procedures. Formerly oxygen was used simply by a mask or a tube and the patient's mouth and face were sprayed with it. But if the tongue had dropped back into the mouth and there was a great deal of secretion, even though the tongue was pulled forward, there would not be enough pressure to get the oxygen down into the trachea. These instruments that have been devised are of great importance. They may be used before tracheotomy."

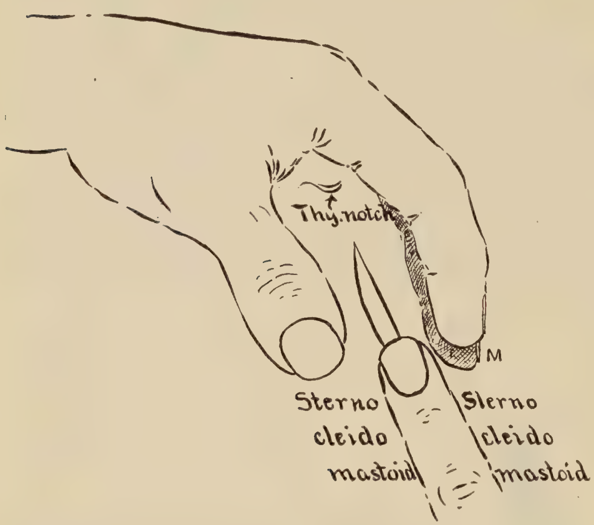


From "Bronchoscopy and Esophagoscopy" by Chevalier Jackson
Courtesy W. B. Saunders Co.

Fig. 148. Schema of practical gross anatomy to be memorized for emergency tracheotomy. The middle line is the safety line, the higher the wider. Below, the safety line narrows to the vanishing point VP. The upper limit of the safety line is the thyroid notch until the trachea is bared, when the limit falls below the first tracheal ring. In practice the two dark danger lines are pushed back with the left thumb and middle finger as shown in Fig. 149, thus throwing the safety line into prominence. This is generally known as Jackson's tracheotomic triangle.

Emergency Tracheotomy*

"Stabbing of the cricothyroid membrane, or an attempted stabbing of the trachea, so long taught as an emergency tracheotomy, is a mistake. The author's [C. Jackson's] two-stage, finger-guided method is safer, quicker, more efficient, and not likely to be followed by stenosis. To execute this promptly, the operator is required to forget his text-book anatomy and memorize the schema [Fig. 148]. The larynx and trachea are steadied by the thumb and middle finger of the left hand, which at the same time push



From "Bronchoscopy and Esophagoscopy" by Chevalier Jackson
Courtesy W. B. Saunders Co.

Fig. 149. Schema showing method of rapid tracheotomy. First stage. The hands are drawn ungloved for the sake of clearness. The upper hand is the left, of which the middle finger (M) and the thumb are used to repress the sternocleido-mastoid muscles, the finger and thumb being close to the trachea in order to press backward out of the way the carotid arteries and the jugular vein. This throws the trachea forward into prominence, and one deep slashing cut will incise all of the soft tissues down to the trachea.

back the important nerves and vessels which parallel the trachea, and render the central safety line more prominent [Fig. 149]. A long incision is now made from the thyroid notch almost to the suprasternal notch, deep enough to reach the trachea. This completes the first stage.

"Second stage: The entire wound is full of blood and the trachea cannot be seen, but its corrugations can be very readily felt by the tip of the free left index finger. The left index finger is now moved a little to the patient's left in order that the knife may come precisely in the midline of the trachea, and three rings of the trachea are divided from above downward [Fig. 150]. The Trousseau dilator should now be inserted, the head of the table should be lowered, and the patient should be turned on his side to allow the blood

* The operation for emergency tracheotomy described below is adapted from 'Bronchoscopy and Oesophagoscopy,'' by C. Jackson, Saunders, 1922.

From "Bronchoscopy and Esophagoscopy" by Chevalier Jackson
Courtesy W. B. Saunders Co.

Fig. 150. Illustrating method of quick tracheotomy. Second stage. The fingers are drawn ungloved for the sake of clearness. In operating, the whole wound is full of blood, and the rings of the trachea are felt with the left index which is then moved slightly to the patient's left, while the knife is slid down along the left index to exactly the middle line when the trachea is incised.

to run away from the wound. If respiration has ceased, a cannula is slipped in, and artificial respiration is begun. Oxygen insufflation will aid in the restoration of respiration, and a pearl of amylnitrite should be crushed in gauze and blown in with the oxygen. In all such cases, excessive or continuous pressure of oxygen should be avoided because of the danger of producing ischemia of the lungs. Hope of restoring respiration should not be abandoned for half an hour at least."

Chapter 17
Asphyxiation from Pathological Obstruction of the Respiration

The patient is clinically ill *before the accident.*

It would appear from the following account that George Washington died as the direct result of an acute asphyxial accident. The reader may judge for himself. It is interesting to speculate upon the result which might have followed the prevention of this asphyxial death by methods available today.

The Death of George Washington*

"On Friday† the 13th of December, 1799, while attending to some improvements upon his estate, he was exposed to a light rain, by which his neck and hair became wet. Unapprehensive of danger from this circumstance, he passed the afternoon in his usual manner; but in the night, he was seized with an inflammatory affection of the windpipe. The disease commenced with a violent ague, accompanied with some pain in the upper and fore part of the throat, a sense of stricture in the same part, a cough, and a difficult rather than a painful deglutation, which were soon succeeded by fever and a quick and laborious respiration.

"Believing bloodletting to be necessary, he procured a bleeder who took from his arm twelve or fourteen ounces of blood, but he would not permit a messenger to be dispatched for his family physician until the appearance of day. About eleven in the morning Doctor Craik arrived; and perceiving the extreme danger of the case, requested that two consulting physicians should be immediately sent for. The utmost exertions of medical skill were applied in vain. The powers of life were manifestly yielding to the force of the disorder; speaking, which was painful from the beginning, became almost impracticable; respiration became more and more contracted and imperfect, until half-past eleven on Saturday night, when retaining the full possession of his intellect, he expired without a struggle.

"Believing at the commencement of his complaint, as well as through every succeeding stage of it, that its conclusion would be mortal, he submitted to the exertions made for his recovery rather as a duty than from any expectation of their efficacy. Some hours before his death, after repeated efforts to be understood, he succeeded in expressing a desire that

* John Marshall, "The Life of George Washington," Vol. 5, Chap. 40, p. 761.

† This account is extracted in part from a certificate signed by Doctor Craik and Doctor Dick, and in part from a private letter of Doctor Craik.

he might be permitted to die without interruption. After it became impossible to get anything down his throat, he undressed himself and went to bed, there to die. To his friend and physician, Doctor Craik, who sat on his bed, and took his head in his lap, he said with difficulty, 'doctor, I am dying, and have been dying for a long time, but I am not afraid to die'."

Causes of Asphyxial Accidents

Acute asphyxial accidents due to obstruction from pathological lesions are encountered in:

(1) Malignant diseases of the upper airway.
(2) Pulmonary hemorrhage in tuberculosis.
(3) Aspiration of the contents of a retro-pharyngeal or tonsilar abscess.
(4) Rupture of an aneurism into the airway.
(5) Pressure of mediastinal growths.
(6) Acute laryngeal diphtheria.
(7) Status lymphaticus.
(8) Acute edema of the glottis from trauma.
(9) Esophago-tracheal fistula.
(10) Acute edema due to allergy or anaphalactic shock.

Malignant Disease

In considering acute respiratory obstruction due to pathological lesions we approach the line of demarcation which separates asphyxial death and terminal asphyxia, a common phenomena preceding death. As Martland states, "The argument may be advanced that we all die of asphyxia, as in many deaths from natural causes asphyxia often terminates life. The test of true asphyxial death, however, readily disposes of this contention." This test is: Will the relief of the asphyxia save life, directly or by bridging

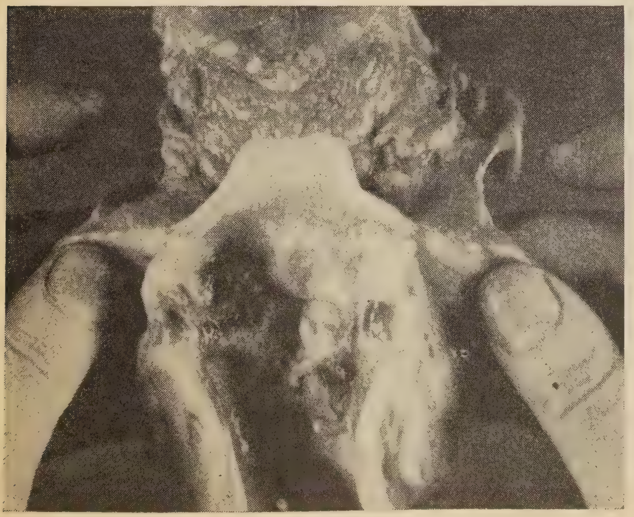


Fig. 151.
Intrinsic carcinoma of larynx.
Courtesy Dr. Harrison Martland

the gap which will allow successful treatment of the threatening pathological lesion? For example, Fig. 151 shows a malignant tumor of the larynx. By occlusion of the laryngeal airway it caused asphyxial death. Intubation past this tumor or tracheotomy would have saved this life by bridging the gap required for removal of the growth or of the larynx. This was a reversible death (Fig. 152).

On the other hand in an inoperable mediastinal tumor producing asphyxia, the relief of the anoxia cannot be calculated to save life; it will merely postpone inevitable death. Such a death, being irreversible by resuscitation, is therefore not an asphyxial death.

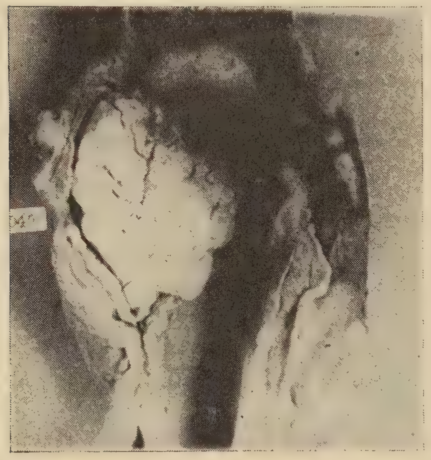


Fig. 152.
Pathological respiratory obstruction due to carcinoma of the larynx.
Courtesy Dr. Harrison Martland

Fig. 153. Asphyxiation due to Suffocation; Aspiration of Barium Sulfate into Lungs during X-ray Examination.

Sudden death following diagnostic x-ray examination for stricture of esophagus. Patient very weak when taken to x-ray department and could not cough up the aspirated barium. X-ray taken just before death showing bronchial tree filled with barium (CME 9350—Office of Chief Medical Examiner, Essex County (Newark), N. J.).

Courtesy Dr. Harrison Martland

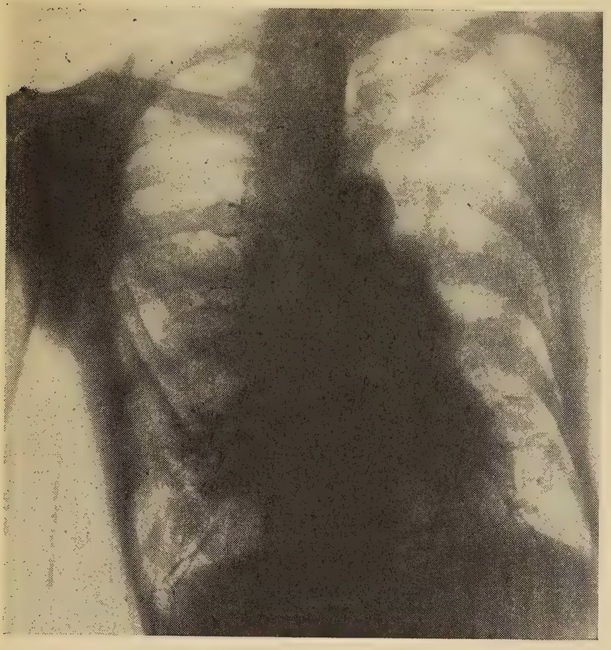


Fig. 154.
Same as Fig. 153, front view.
Courtesy Dr. Harrison Martland

Pulmonary Hemorrhage in Tuberculosis

Recent advances in thoracic surgery have reduced the incidence of massive hemorrhage into the airways. While continued bleeding from a ruptured artery in a tubercular cavity would of course result in death from anemic anoxia (direct blood loss), there are no mechanical reasons why death from anoxic anoxia cannot be prevented. Given exposure, intubation, strong endotracheal suction, a tracheal airway could be maintained. And it is also reasonable to suppose that, after the massive obstruction was relieved, positive intrapulmonary oxygen pressure could be practiced to reduce slow ooze from the bleeding field (p. 71). Almost any ordinary hazard in such critical emergencies would seem well justified. The field is a virgin one which awaits investigation.

Inhalation of Pharyngeal or Tonsilar Abscess Contents

The danger of suffocation or choking to death following the rupture of a pharyngeal or tonsilar abscess is well recognized. Two safeguards immediately suggest themselves.

(1) Maintain the activity of the swallowing and coughing reflexes.

(2) If local or general anaesthesia must be used, or if asphyxia has occurred from pressure, the larynx must be exposed and intubated, so that the abscess contents may be prevented from entering the trachea, or so that in the event of inhalation its removal may be facilitated by suction.

The method to select in approaching such hazards is as follows. The conscious patient is placed flat on the back with the head extended, and lower than the chest. A mouth gag is adjusted and the field is viewed through a laryngoscope, which may serve as a mouth gag as well as a speculum. With a small, velvet-eyed suction tube which is attached to strong suction, operating in readiness, an incision is made into the mass, large enough to allow the entrance of the suction tube which is instantly introduced. By this technique, a large abscess may be evacuated without spilling any pus into the pharynx. Gagging and vomiting from the effects of the instrumentation should be ignored; vomitus should be removed by suction as it appears. Once exposure has been begun, it must not be interrupted. To remove the laryngoscope or the mouth gag before relieving the obstruction is to court sudden, complete, and perhaps fatal asphyxia. With the speculum in place the patient may be carried through by oxygen, plus suction. If thrown back upon his own resources by removal of the gag or laryngoscope, he will be worse off than before relief was attempted. Local anaesthesia is dangerous as it will trickle into the larynx, reducing the cough reflex and permitting aspiration. General anaesthesia is contraindicated because of the danger of operative and post-operative aspiration. Analgesia, however, may be secured, after the mouth gag is placed, by N_2O, chloroform, ethylchloride, or by intravenous pentathal, evipal or intravenous ether in saline. It is to be remembered that anoxemia is a good analgesic. The only effect desired is loss of pain. No attempt should be made to control the reflexes or the movements of the patient; the hazard is out of all proportion to the advantage. The analgesic agent chosen should be one with which the anaesthetist is perfectly familiar. Dilute ether vapor, properly handled, will serve perfectly well as an analgesic, provided the airway is under control before it is administered.

Asphyxia from Acute Edema of the Glottis

Edema of the glottis and supra-glottic structures should always be suspected in acute inflammations of the nose and throat, manual strangluation, etc. Ante-operative obstructive signs are by no means uniform, for the tone of the muscles of the pharynx and larynx compensates for difficulty in inspiration. Expiration merely blows the obstruction aside. Viewed through a laryngoscope, edematous tissue appears as glistening, translucent, polypoid masses, obscuring normal structural outline and moving with the respiratory currents. The glottis may appear as a dimple in this mass, through which air may be seen to pass. Intubation through this dimple has saved life.

It is obvious that under such conditions, asphyxial death is "touch and go." Between the extremes above cited, however, and simple swelling of the pharyngeal and tracheal mucosa there is every degree of obstruction. Ante-operative mirroring of the larynx is not conclusive.

If an operation is contemplated in edema of the larynx, there is only one safe approach to the problem: deliberate anaesthesia with as little excitement as possible and exposure of the airway with facilities for tracheotomy instantly available.

The intubation tube may be left *in situ* for 24–48 hours, or until the edema has subsided. It provides facilities for endotracheal suction, through the period of recovery. As the edema subsides, the patient will breath around the tube as well as through it. When this occurs, it may be withdrawn. If intubation cannot be affected, tracheotomy should be performed at once, through the field already prepared and draped (p. 190).

By this sequence of technique the patient will have been given every opportunity to escape tracheotomy. Should it be considered desirable, following intubation, it may be performed with precision and at leisure. It is to be recalled that progressive anoxia is a first-class analgesia and a good anaesthetic for brief periods.

Asphyxia from Tracheo-Esophageal Fistula

After delivery of a baby, should frothy mucus continue to appear, a tracheo-esophageal fistula may be suspected. Intubation and aspiration of the baby's trachea will confirm the diagnosis.

Traumatic tracheo-esophageal fistulas are not uncommon. They may follow injury or growths in the esophagus. The author recalls a case in which a small boy, suffering from stricture of the esophagus and under treatment with an indwelling string attached to a dilator in the stomach, broke through his tracheal wall, aspirated coils of string and almost died from acute obstructive asphyxia. His life was saved by emergency tracheotomy. He was subsequently anaesthetized through his tracheal tube, intubated from above, and his fistula repaired. He made an uneventful recovery.

Asphyxia from Allergy

Allergic and anaphylactic shocks occur more frequently than is supposed. Vaughn reports seven sudden deaths and a number of serious reactions, in answer to an inquiry directed to 75 physicians, not professional allergists. The most frequent cause was the administration of serum.

The advent of prophylactic toxoid for diphtheria and tetanus has reduced the use of serum and the reactions to be anticipated from it. During the S. P. A. D. exhibit (Fig. 8), Vaughn interviewed fifty visitors. Twenty-five reported anaphylactic or allergic shock in their own or in an associate's experience.

Allergic shock is rapid. Anaphylactic shock lasts for some time. The basic pathology in allergic shock is a generalized interstitial edema due to sudden capillary permeability. The following statement illuminates the entire problem of allergic and anaphylactic shocks.

"Capillaries of the muscles of the average man have a total area of approximately 6,300 square meters, or 3000 times the area of the body surface. In the absence of restraining forces, the entire plasma volume could pass from the capillaries to the body tissues in 10 seconds."

Death has been reported in the British Army from repeated injections of tetanus and diphtheria antitoxin. These fatalities were due to generalized edema.

Anaphylactic shock may occur in allergy and in drug therapy in dilutions as high as 1 part in 10,000. The chief causes of serious reactions are:

(1) Failure to anticipate possible reactions.

(2) Too rapid desensitization.

(3) Short cuts in treatment due to haste.

(4) Wrong concentrations.

As a prophylactic treatment, give the patient tablet-propadrin $\frac{1}{2}$ gr., theophyllin 4 gr. after his allergy treatment, before he leaves the office.

Emergency Treatment in Office. (1) Have the patient lie down. (2) Place a tourniquet above the injection.

Inject $\frac{1}{2}$ cc. $\frac{1}{1000}$ adrenaline into the other arm and then $\frac{1}{3}$ cc. into the site of the inoculation. Improvement usually takes place in 60 seconds. If the adrenalin effects are too pronounced, release the ligature slightly. A sphygmomanometer may be used for a tourniquet; it gives good control.

Asphyxia from Status Lymphaticus. The diagnosis "*Status Lymphaticus*" has so often been used to cover up a death from obstructive asphyxia that its existence as a possible hazard as an allergic or anaphylactic factor has been underestimated.

The author is of the opinion that, unless the lymphatic patient is biochemically stimulated by a drug such as chloroform, the danger of the lesion is reduced to that of mechanical obstruction. He does not believe that ether will bring about untoward effects. In this conviction he had deliberately anaesthetized a child who suffered preoperative respiratory embarrassment from the mechanical effects of the thymus. With ether there were no ill effects. The mechanical factors involved are, however, of special interest. The thymus is located in front of the trachea. Thus, when the head is extended and the chin and the jaw raised, the usual position for relieving the respiration, the thymus is drawn upward and forward. A large thymus between it and the sternum may compress it. The more vigorous the attempts to free the patient's respiration by extending the head, the more pronounced may be the tracheal obstruction. These children are therefore intubated with a non-collapsible endotracheal tube after being anaesthetized (p. 190). The mechanical hazard of asphyxia is thus obviated.

Viewing sudden death on the operating table from its pathological aspect, Martland is impressed by its existence as a potential hazard, quite apart from the size of the thymus. He states,* "Notwithstanding the opinion of Greenwood and Woods and the report of the Status Lymphaticus Investigation Committee of the Medical Research Council and Pathological Society of Great Britain and Ireland tending to show that it is as accurate to attribute the cause of death to 'the visitation of God' as to *status lym-*

* "The Medicolegal Necropsy." Williams & Wilkins.

phaticus, and that in the future such a condition for a cause of death will probably not be accepted by reliable authorities, I still believe persons suffering from this characteristic type of constitution die suddenly under anaesthesia and during trivial surgical or other procedures."

In a leading editorial in the *Journal of the American Medical Association*,* the opinion is expressed that *Status Lymphaticus*, or *Status Thymicolymphaticus*, may be due to a dyscrasia of adrenalin cortical function.

It would appear that the symptom complex characterized by susceptibility to infection, intoxication, drugs, anaesthesia and physical trauma accompanied by general lympatic hyperplasia (especially in the small gut, as noted by Martland) and resulting in sudden death by circulatory failure cannot be easily dismissed as a pathological entity.

The correct Post Mortem diagnosis of most of these cases will rest upon a competent toxicologist.

Asphyxia from Diphtheria. Bullowa, an ardent admirer of O'Dwyer recently said to me, "Do you know that when a man attended a diphtheria case in O'Dwyer's time he automatically lost his practice, for through fear, other patients would not permit him to enter their houses. There was scarcely a city block in which there had not been a death from diphtheria. Oh, yes! we still use O'Dwyer's tubes, but for the last ten years we have looked at every affected larynx. We make our diagnosis by inspection and by clinical signs rather than by culture. Culture confirms the clinical signs. A carrier may give a positive culture; a concurrent infection may render a positive culture negative."

All children at the Willard Parker Hospital are placed in the mummy restraint before exposure of the larynx.

1. *Purpose:* To immobilize a patient so that treatment may be carried out effectively.

2. *Equipment:* (1) A small sheet. (2) 3 Safety pins.

3. *Preparation of Equipment:* (1) Fold the sheet crosswise, so that it will extend from the point midway between the elbows and shoulders to the ankles of the patient.

(2) Select pins with sharp points. If points are very blunt, run them through a cake of soap.

4. *Preparation of Patient:* (1) Remove clothing from chest. (2) Reassure the patient.

5. *Procedure:* (1) Put the sheet under the patient with the folded edge toward the shoulders.

(2) Place the patient on his back with the right arm close to his side and the right hand under the buttocks.

(3) Draw the sheet across the chest under the left arm and side, leaving the left arm on the outside.

(4) Place the left arm close to the side and the left hand under the buttocks.

* Vol. **104**, No. 21 (1910).

(5) Draw the opposite side of the sheet over the left arm and pull it snugly across the chest. Pin it about 2 inches from the right shoulder.

(6) Fasten the second safety pin at the level of the right wrist, to immobilize the arms.

(7) Tuck the lower right portion of the sheet securely under the legs.

(8) Pull it tightly around, nitre the sheet above the knees, and secure the third pin along the fold in the dart.

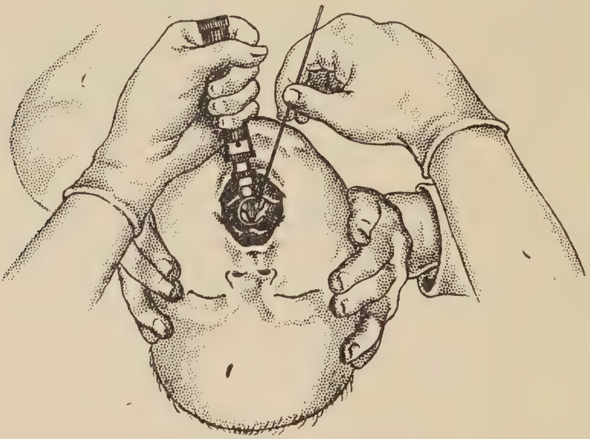


Fig. 155. Inspection of infant's airway using suction (Bullowa).

Children do not seriously object to exposure of the airway. This should be done deliberately. If the baby holds its breath it will soon relax and let go. Suction is applied directly to the secretions in the field. The condition of the cords and the larynx can easily be seen. Any serious dyspnoea or asphyxia can be immediately relieved by direct intubation under vision followed by insufflation of oxygen. Should œdema due to croup indicate tracheotomy, this may be performed at leisure following intubation. Fig. 155 demonstrates the technique of inspecting a child's airway.

The head may be placed flat on the table, but the assistant's hands are needed to keep the head from moving.

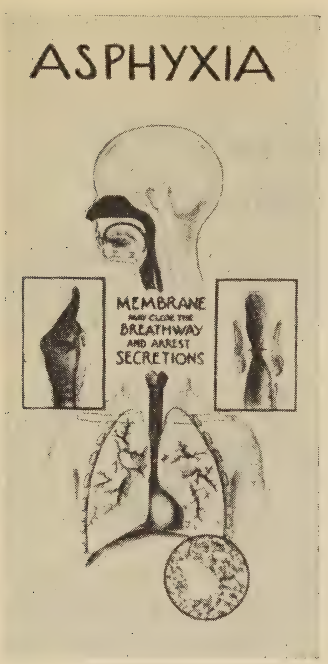


Fig. 156.
Asphyxia in diphtheria.

Site	Chief Symptoms	Treatment: General	Treatment: Local
Nose: membrane edema and mucus exudate	mouth breathing	diphtheria antitoxin	suction
Throat: membrane mucus and exudate	pain, difficulty in swallowing, rarely dyspnoea	diphtheria antitoxin	general irrigation, Geudel breathway
Larnyx: Supraglottic membrane edema	dyspnoea	diphtheria antitoxin	humid air, tracheotomy
Glottic membrane mucus exudate	dyspnoea and aphonia	diphtheria antitoxin	suction and/or intubation tube
Subglottic membrane edema	dyspnoea and aphonia	diphtheria antitoxin	intubation and/or suction tracheotomy
Trachea: membrane mucus exudate	dyspnoea and cough	diphtheria antitoxin	suction
Bronchi: membrane mucus exudate	dyspnoea and cough	diphtheria antitoxin	suction
Alveoli: asphyxial membrane actelectasis	asphyxia	diphtheria antitoxin	oxygen suction

Chapter 18
Asphyxia from Clinical Disease

The author's conception of gas therapy, or pneumatology, expresses itself in three fields of activity: gases for the control of pain (anaesthesia); gases for the saving of life (resuscitation); and gases for the treatment of clinical disease (inhalation therapy).

Inhalation therapy has the same relation to internal medicine as anaesthesia to surgery. Both are contributory. There is one important difference, however: the physician practicing inhalation therapy is a consultant first and a technician second, whereas the physician practicing anaesthesia is a technician first and a consultant second, if indeed, as is usually the case, he is recognized as such.

Inhalation therapy for clinical disease has been intensively developed over a long period in a number of university clinics, notably at the Mayo Clinic and at Columbia University. This field will enlist and undoubtedly receive the attention and the support which it deserves. It is also widely practiced outside of university clinics.

Inhalation therapy is concerned chiefly with the conscious patient who has developed a sublethal progressive anoxia, *i.e.*, cardiac decompensation, coronary artery disease, asthma, pneumonia, cerebral thrombosis, intestinal decompression in intestinal obstruction, the treatment of burns, the acceleration of reparative processes in plastic surgery, etc. Sudden asphyxial death in these conditions is obviously rare. The clinical management of these cases is outside the scope of this volume.

As pointed out by Boothby,* the transition from anoxia to asphyxia and back again through anoxia to normal oxygenation should be clearly understood.

An attempt has been made in the present volume to focus the reader's attention upon the extremes of anoxia or asphyxiation. It is these extremes which demand resuscitation by the methods described.

The value and the practical safety of 100% oxygen† is a commonplace to anaesthetists and to those familiar with the care of the unconscious patient. The more general employment of full oxygenation‡ as prescribed should be popularized.

* Boothby, Walter, "Clinical application of Oxygen Therapy," *Arch. Clin. Therapy*, **23,** 598 (Oct., 1942).

† *J. Am. Med. Assn.*, **113,** 477 (Aug. 5, 1939).

‡ "The Value of High Concentrations of Oxygen for Therapy," *Proc. Staff Meetings Mayo Clinic*, **13,** 641 (Oct., 1938).

The author views inhalation therapy as a clinician, rather than as a research worker. His suggestions are directed to the small institution and the general practitioner rather than to the university research worker, who must remain the source from which medical principles and procedures flow. It is clear that university departments of anaesthesia and inhalation therapy should be everywhere strengthened and encouraged.*

It appears equally clear that in the absence of an intelligent coordination of these departments, now the rule rather than the exception, resuscitation as an intramural and as an extramural activity continues to be neglected to a disgraceful degree. Should the reader doubt this statement, let him apply to any local medical examiner's office for factual confirmation (see p. 390).

Human nature being what it is, those who have developed and who direct departments of anaesthesia and inhalation therapy are reluctant to encourage any change which might result in what might seem to be a curtailment of independence. It may be asked, would it not be more in accord with the spirit of medical practice to pool resources for the greater good, to select from present personnel a committee or a director to care for the coordination of the pneumatological field?

Unfortunately such an arrangement turns upon the self-sacrificing action of a spirit which, it is feared, does not exist. However, should the Faculties of Medicine of the universities, viewing the needs, the present totally illogical trend, and the results to be achieved by such coordination, elect to create Chairs of Pneumatology (see p. 374) and appoint assistant professors in anaesthesia, inhalation therapy and resuscitation, models would be created which the profession at large would quickly follow. Parallel organizations already function in the major fields of medicine and surgery.

Outside university circles, the need for the integration of gas therapy in hospitals, in public health, and in general practice is even more acute. This is because the total volume of anaesthesia, resuscitation and inhalation therapy is here much reduced. Again the question of directing personnel will determine the success or the failure of meeting these needs.

The author is convinced that the specialty of anaesthesia is circumscribed by its terminology to the control of pain; that, since it is in fact contributory to surgery and cannot exist in its absence (the occasional treatment of medical pain excepted), it is neither qualified nor acceptable as a coordinator of inhalation therapy or resuscitation, outside the immediate circle of operating-room influence. The present trend toward "building up anaesthesia" by adding any other duties which may appear of interest or value is illogical, futile and actively delays the relief which is sought. It is absurd to call an anesthetist to care for carbon monoxide poisoning, terminal polio, suffocation, strangulation, or an anaphylactic shock.

* "Inhalational Therapy"; Barach, A., 1944.

That this view is shared by those interested in shaping medical policy is implied in a recent release by the Committee on Public Relations of the New York Academy of Medicine, in a special article entitled "Standards of Effective Administration of Inhalation Therapy."* This article, which stresses the various phases of inhalation therapy, makes no mention of the Department of Anaesthesia which is supposed to direct it. Furthermore, no anaesthetist appears upon the committee which drew up these standards. One looks in vain in the voluminous literature dealing with asphyxia in aviation medicine, electric shock, polio, foreign-body obstruction, asphyxia neonatorum, submersion, etc. for mention of the Department of Anaesthesia. The author believes that this reaction is correct.

On the other hand, the contributions which have been made to the fields of resuscitation and inhalation therapy by men trained in anaesthesia are absolutely priceless. The physician-anaesthetist, *unlike* the field with which he has been identified, is *not* restricted; he is a doctor of medicine, and the practice of medicine is his. He is familiar with the pathological physiology of those unconscious from both anaesthetic agents and accidental causes. He is technically trained to deal with these emergencies, either personally or by directives which he issues. He may readily be trained, through refresher courses in internal medicine, to provide the most efficient and most up-to-date inhalation therapy.† As a case in point, the best survey of the pathological physiology of the hazards of fire and the most lucid discussion of its management which has come to the attention of the author appears in an article entitled "Resuscitation and sedation of patients with burns which include the airway: Some problems of immediate therapy arising from the Coconut Grove Disaster," by Henry K. Beecher.‡

Men formerly active in anaesthesia, upon finding it impossible to secure the recognition which their work outside of the operating room deserved, have left it, to develop inhalation therapy with conspicuous success.

The author is convinced that relief will come only through the adoption and the popularization of a new comprehensive terminology which will permit the logical integration of anaesthesia, resuscitation and inhalation therapy. Such integration will lead to the open road of progress in research and achievement. It may not come swiftly; but when it does arrive it will usher in a new era of low morbidity and mortality in the vast, virgin field of pneumatology.

* *J. Am. Med. Assn.*, **121,** 755 (March 6, 1943).
† Evans, J. H., *N. Y. State J. Med.*, **39,** (1939).
‡ *Annals of Surgery*, June, 1943.

Part V

The Field of Asphyxia and Resuscitation

Chapter 19

Asphyxia as a Problem of Organized Medicine

Report of the Committee on Asphyxia of the American Medical Association*

"Asphyxia as a terminal complication precedes numerous deaths and widespread morbidity from a variety of causes. The question as to what can be done through the prevention and treatment of asphyxia has been raised and your committee has been called on to make a short report without the benefit of an exhaustive study of the question.

"It is conceded that the problem had best be attacked without haste. In time, the work may be so organized as to permit a reasonable expectancy of success. Of necessity, ommissions of more or less important aspects of the problem will occur. We propose, however, to consider these as they appear, limiting our attention at this time to those groups in which the prevention of asphyxia or its treatment offers a problem of sufficient importance to engage the attention and enlist the action of the Trustees of the American Medical Association. The fields which we believe need immediate attention are as follows:

1. Asphyxia neonatorum.
2. Asphyxia from gases, used industrially.
    - (a) Carbon monoxide from illuminating gas and from engine exhaust.
    - (b) Refrigerants such as ammonia, carbon dioxide and Dry Ice.
    - (c) Fumes in the manufacture of chemicals.
    - (d) Gases associated with the oil industry.
    - (e) Gases in the mining industry.
    - (f) Fumigation for disease; the destruction of rodents on board ship and elsewhere.
3. Asphyxia from gases in warfare.
4. Asphyxia from drugs, hypnotics, narcotics and sedatives, including acute alcoholism.
5. Asphyxia from disease, such as acute pulmonary conditions, asthma and cardiac decompensation.
6. Asphyxia from developmental and mechanical abnormalities, such as neonatal atelectasis and collapse of the lung.
7. Asphyxia from anaesthesia due to overdosage, idiosyncrasy or a failure to meet mechanical obstruction, occurring in relaxation.

* *J. Am. Med. Assn.*, **108**, No. 18 (May, 1937).

8. Asphyxia from drowning (submersion).
9. Asphyxia from flying at high altitudes.
10. Asphyxia from fire fighting (smoke, chemical poisoning).
11. Asphyxia from obstruction by foreign bodies.
    (a) Material caught in the esophagus or inhaled.
    (b) Tumors or infections within or without the airway.
12. Asphyxia from electrocution.
13. Asphyxia from strangulation.
14. Asphyxia from allergy.
15. Asphyxia from terminal poliomyelitis.

Treatment Is Similar, Irrespective of the Cause

"It will immediately be asked, Why are so many apparently unrelated entities considered under the head of asphyxia? The reason for so considering the many and various causes of asphyxiation is that, once the predisposing or immediate causes are removed, the treatment, regardless of the etiology, is always the same. For example, once the electrocuted person is detached from the current causing the accident, the submerged patient lifted out of the water or the foreign body removed, the basic problem in each case is reduced to the introduction of oxygen into the respiratory tract, the support of the centers governing respiration, and the stimulation of the circulation.

"The brevity of this report precludes the consideration of detailed, specific information for the conditions inducing asphyxiation.

Methods by Which the Problem of Asphyxiation May be Attacked

"Your committee believes that the attack on the problem of asphyxia lies in:

"Publicity, by which the attention of the medical profession and the public may be directed to the frequency of death due to asphyxiation.

"Organization to coordinate available information as well as agencies and groups whose normal activity brings them into frequent contact with asphyxial emergencies.

"Action, developed as the result of publicity and organization, to stimulate research and to develop a personnel, both lay and medical, capable of carrying out treatment appropriate for the specific case.

"The problem of asphyxiation is one that extends outside the immediate confines of the practice of medicine. It impinges directly on industry and the utilities and forms a vital problem in the defense of the nation against gas attacks by a foreign power.

"While much valuable work has been done through statistical and other studies conducted by industrial organizations attacking the problem of carbon monoxide and other specific causes of gas poisoning, and while measures to reduce asphyxiation from drowning have been intensively carried out, notably by the state of Rhode Island, and while first-aid measures made available through many police and fire departments leaves little to be desired, your committee is nevertheless impressed by the sporadic and limited nature of such efforts, the lack of publicity attending successful organization, and the entirely unwarranted publicity frequently accompanying unscientific efforts to treat asphyxiation.

"Your committee is furthermore impressed by the need of proper organization and supervision of lay groups by a trained and experienced medical personnel. Coordination between the physician and the lay group is often entirely lacking and might well be encouraged. There is not so much a lack of information on the subject of asphyxia as a lack of a general knowledge of available technique and equipment. Furthermore, while the specific treatment of any of the stated causes of asphyxiation is well known to those who have done special work on the problem, such treatment developed as a medical routine is by no means the common property of practicing physicians.

"The recognition of the problem of asphyxia, implying as it does the loss of at least 50,000 lives a year, bring with it the challenge to reduce these deaths by a carefully organized program to be carried out over a long period.

Proposed Committee Program

"*General.* Your committee believes that it may be of immediate service to the American Medical Association by acting in an advisory and coordinating capacity.

"This assistance and coordination may take the form of a continued program, including a method of attack by public education, research, and the post-graduate instruction of physicians.

"Arrangements may be made for the preparation of a series of articles in the *Journal of the American Medical Association.*

"Questions relative to resuscitation apparatus and technique referred to it from the Council on Physical Therapy may well come under its purview.

"Your committee suggests the desirability of taking over the work and carrying on the program initiated and maintained for the last four years by the Society for the Prevention of Asphyxial Death. Informative material accumulated by this organization might be filed by the committee for the use of the Association, and contacts with organizations and groups interested in the prevention of asphyxial death may be preserved and developed.

"Your committee believes that it may be helpful and awaits instruction and power to act as a coordinating agency in the assembly of information from and the dissemination of information through the various committees and bureaus of the American Medical Association.

"Realizing the magnitude of the task presented by the problem of asphyxiation, its presentation and control, your committee feels it necessary to reaffirm its conviction that progress can be made only by a well planned attack over a period of time.

"*Specific and Immediate Steps.* Of fundamental importance in the preparation of this attack is the assembly of exact information to be used in the listing of asphyxial deaths. There must be established, first of all, a clearcut and generally accepted list of causes of asphyxiation; secondly, these causes must appear in national death returns, where they may be segregated and studied. Such returns may be reported each year, and reduced mortality due to preventative measures noted. Your committee suggests that such an annual report of vital statistics relative to asphyxiation appear annually in the *Journal of the American Medical Association* at a stated date.

"Following the accumulation and analysis of asphyxia statistics, intensive efforts may be made to reduce asphyxiation in the various groups in which it is most prevalent. For example, an early and concentrated effort might well be made to reduce asphyxia in the new-born, since this type of asphyxiation constitutes more than 60 per cent of total asphyxia mortality. Simultaneously, or at a later period, asphyxiation from gases used industrially, from gases in warfare, from submersion, from foreign-body obstruction, from anaesthesia, from drugs and from disease may each receive attention in the order of importance.

"In conclusion, your committee requests the necessary financial assistance to carry out the recommendations reported.

"Respectfully submitted,

Paluel J. Flagg, *Chairman*
John S. Lundy
Thomas J. Vischer"

Chapter 20

The United States Public Health Service

Safety Work of the Bureau of Mines

History

Before the present century there existed a fairly persistent demand for the establishment of a Federal Department of Mines, but relatively little was done about it until a considerable number of very serious coal-mine disasters in 1906, 1907, 1908 and 1909 created what might almost be termed a national scandal and virtually forced the Federal Government to take action to try to solve the problem of the cause and prevention of such disastrous occurrences. Sections of the Geological Survey, which had been doing some coal-mine safety work since 1907 or 1908, were transferred into the Bureau when it began to operate in July, 1910.

Within a few months of the creation of the Bureau, an arrangement was made with the United States Public Health Service by which doctors of that organization could cooperate with engineers of the Bureau in investigations to improve the health of persons engaged in the mining and allied industries. A similar arrangement is in effect at the present time.

The mining, quarrying, metallurgical, and petroleum industries of this country give more or less direct employment to well over 2,000,000 persons; if their dependents are included, the welfare of probably 10,000,000 persons is fairly definitely affected by the Bureau's work of various kinds.

With the passage of the Coal Mine Inspection Act by Congress on May 7, 1941, the Bureau obtained for the first time the authority to enter coal mines to obtain information regarding operating methods and conditions affecting the health and safety of employees. The Bureau now has 107 coal-mine inspectors and a corps of mining-explosives and mining-electrical engineers in the field inspecting coal mines, noting both commendable and unsafe practices and making recommendations for improving undesirable conditions. Such recommendations are not mandatory upon the operators, and the Bureau continues to rely upon cooperation of those in the coal-mining industry for adoption of safe operating standards. During the 1942 fiscal year, which ended June 30, 1942, 400 coal mines were inspected in 20 states and many of the recommendations of the inspectors were adopted by the managements of those mines. While administering the Coal-Mine Inspection Act, the Health and Safety Service of the Bureau has not departed from its policy of maintaining close cooperation with workers, employers, state inspection and enforcement organizations and numerous other agencies engaged in promoting health and safety in the mineral industries.

During the 1942 fiscal year, the work of the Health Division of the Bureau expanded greatly because of the Federal Coal-mine Inspection Act. The number of employees increased from 15 to 40, and a new unit, composed of doctors, engineers and chemists, was established for the purpose of making investigations of occupational diseases. The staff of the gas and dust laboratory was increased to handle analyses of air and coal-dust samples

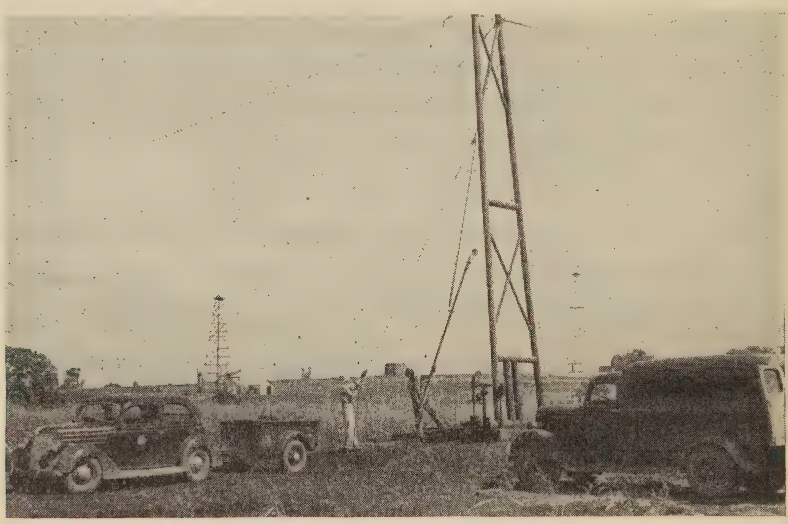


Fig. 157. Petroleum industry.

submitted by the Federal coal-mine inspectors. During the 1942 fiscal year, the Health Division analyzed 5,300 air samples and many hundreds of coal-dust samples submitted by coal-mine inspectors in the field. Meanwhile, the Health Division continued its work in air-contamination studies, tests of respiratory devices and preparation of reports designed to promote health and safety.

In fact, the Bureau acts as a sort of clearing house for the assembling and disseminating of information on health and safety in mining.

First Aid and Safety

While the Bureau of Mines was given numerous functions, unquestionably the promotion of safety in the mineral industries was and continues to be one of its main "jobs."

Before the end of the first fiscal year (June 30, 1911) 6 mine safety stations had been established in various parts of the United States, and

6 mine safety cars had been put into the field; by October 1910, First-aid-training work was being done in mining regions.

To conduct the work of the Safety Division to the best advantage, the United States and Alaska have been divided into nine districts. A field staff of 40 engineers and 30 safety instructors operates from 17 safety stations and two of the seven safety cars owned by the Bureau. Other employees such as clerks, bring the total field force to 95. The mine safety cars are railroad coaches which are useful in giving instructions to miners and mine officials in first aid, mine rescue and recovery procedure, and accident prevention. Four of these cars have been transferred to the Bureau's Manganese Beneficiation and Pilot Plant at Boulder City, Nev., for the duration of the war.

The work of the Safety Division of the Bureau falls under two general headings: educational and investigative. Under educational work, the Safety Division sponsors first-aid training, mine rescue, advanced mine rescue and recovery operations, accident prevention, and a petroleum-gas course; First-aid and mine-rescue contests; exhibits and demonstrations; Holmes safety chapters; safety motion pictures and safety meetings. The investigative work comprises investigations of mine explosions and fires, miscellaneous accidents and health hazards; safety inspections of mines and investigations and development of safety devices.

The first-aid training work of the Safety Division of the Bureau's Health and Safety Service constitutes the most intimate and in some ways the most effective method of putting before the mining industry (both employer and employee) up-to-date health and safety data; in other words, the First-aid training course, as now given, is excellent accident-prevention "propaganda" and probably its most valuable feature is that it reaches workers in large numbers, approximately 100,000 now taking the course annually. More than 1,200,000 mining people have taken this course; it is estimated that through it at least 200 lives are saved annually, and probably at least 10,000 non-fatal accidents are prevented in the mining and allied industries because of the "safety consciousness" given to those who take the course of instruction as taught by present-day Bureau of Mines methods and instructors. During the past fiscal year, the Bureau's First-aid training and mine-rescue training were given in 964 mining communities and other localities in 37 states, Alaska and the District of Columbia. During that period, 88,262 persons were trained in First Aid and 2,008 in mine rescue.

Naturally, the Bureau of Mines, immediately upon coming into existence with a mandate from Congress to try to stop mine disasters, addressed itself intensively to the matter of aiding in preventing loss of life in mine disasters, and it was found advisable in this as well as in all other phases of safety work to attack the problem from several points of view, as safety work does not thrive at all well when all the "eggs are in one basket." An experimental coal mine was established near Pittsburgh, Pa., and in a short time valuable information was obtained as to how coal-mine explosions and fires were caused, and many data were obtained as to feasible methods of preventing them, or of limiting their extent and destructiveness if they should start. Many other laboratory studies were made, and are continuing to be made, with regard to mine-disaster limitation or prevention, such as investigations about explosives, electrical and other equipment, miners' lamps, respiratory equipment, and ventilation; field studies including all of the above as well as other matters pertaining to explosions and other mine disasters and their prevention were and still are carried on in essentially every mining section of the United States.

Since 1910, the Bureau's First-aid and mine-rescue courses have been given to more than 15,000 persons in the cement industry, more than 1,095,000 in coal mining, 170,000 in metal mining, 40,000 in metallurgical plants, about 26,000 in non-metallic mining, 126,000 in the petroleum industry, about 9,000 in tunnel work, and about 57,000 in miscellaneous activities related in some way to mines or the mineral industries. This training has also been given to about 28,000 persons not connected with the mineral industries. In all, approximately 1½ millions of persons have been trained in the Bureau's First-aid methods.

To encourage persons proficient in First-aid work or in the teaching of it, the Bureau has issued more than 16,000 First-aid instructors' certificates, which are much in demand as the holders very often are given a preferential right to obtain or retain employment. These certificates have been issued to residents of 43 states, the District of Columbia, Mexico, Alaska and the Philippine Islands.

Up to June 30, 1942, 2,816 mines and plants in the mining and allied industries had been given 100 per cent training certificates indicating that every person in the mine or plant had taken the full Bureau of Mines First-aid training course. Some mining companies have had these certificates annually for 10 or more consecutive years. During the 1942 fiscal year, 100 per cent certificates were issued to 117 mines or plants, the total trained being more than 18,000. This 100 per cent First-aid training has been given in 40 states, Alaska and the District of Columbia to a total of more than 510,000 persons.

Every year the Bureau's field safety workers assist or in some manner participate in nearly 70 First-aid and mine-rescue contests or meets; these meets are now staged by organizations connected with the mineral industries to stimulate safety in general as well as First-aid and mine-rescue work in particular. In the fiscal year ended June 30, 1942, the Safety Division workers of the Bureau of Mines aided in 67 of these meets staged in 11 states; about 7,000 persons participated as contestants and more than 150,000 persons were present as spectators.

In the fiscal year ended June 30, 1942, the Safety Division employees attended 617 safety gatherings of various kinds, at which there was an estimated attendance of 94,000 persons. These meetings are held in addition to instructional classes of various types. In the past 6 years the field workers of the Safety Division have attended about 4,300 safety meetings in 42 states, the District of Columbia, Alaska, British Columbia and Ontario, Canada.

Since the Bureau was created, one of the important activities of the field safety personnel has been attendance at mine accidents and disasters; the engineers prepare reports on them after investigating to ascertain the causes and possible methods of prevention. In the past fiscal year, 24 mine explosions and 27 mine fires in 19 states were investigated by members of the Safety Division and 83 miscellaneous accidents in 24 states were reported.

Courtesy Bureau of Mines

Fig. 158. Pennsylvania State Annual First Aid Contest.

In addition to coal-mine inspections, the field workers make special underground inspections investigating equipment and health or safety conditions and later submitting written reports, giving the ascertained data with recommendations as to improvement. In the past fiscal year, 23 such reports were made in 11 states. The safety inspections included a study of practices, conditions, and equipment, and sampling and analysis of mine air and coal dust.

Another essential service that the field workers of the Safety Division give to the industry is the inspection of privately owned mine rescue stations and their equipment. Forty-three such stations in 12 states were inspected by men of the Safety Division.

In trying to give the mining industry as much as possible of the safety and health information obtained by the Safety Division, 41 manuscripts of various kinds were prepared for publication by 27 members of the division.

Results

As has been indicated, the occurrence of major disasters in coal mines of the United States has been reduced nearly 80 per cent, and the fatalities from major disasters have been reduced more than 90 per cent since the Bureau came into existence.

From 1936 to 1940, inclusive, (the last 5-year period for which complete statistics are available) there were 6,326 fatalities in our coal mines, the rate being 2.70 persons killed per million tons of coal mined. During the 5-year period 1911 to 1915, inclusive, the fatality rate was 4.76 per million tons mined. Thus, the fatality rate has been cut nearly in half.

Courtesy Bureau of Mines

Fig. 159. Rescue squad emerging from a mine.

The coal mine fatality rate has been reduced enough to indicate an average annual saving of life of more than 1,000 persons.

The annual saving of somewhat more than 1,000 lives may be considered as "worth" more than $5,000,000. The prevention of the death of 32,000 coal-mine workers in the past 31 years obviously has avoided a vast amount of suffering, pain and misery of various kinds to the families who would have been bereft of their loved ones. Moreover, the prevention of 50,000 or more nonfatal accidents annually for 31 years certainly has saved the miners, their families, and the community a large amount of pain, inconvenience, misery, and other losses.

The average age of the coal-mine worker who is killed is about 35 years, whereas under normal conditions he should have a future active working period of at least 20 years more.

The saving of the lives of about 32,000 coal-mine workers in the past 31 years through lowering the death rate in coal mining has therefore prevented a financial loss to them of at least $640,000; and this does not account for the financial losses to workers avoided through elimination of large numbers of nonfatal accidents. Hence, for the past 31 years an annual saving of approximately $21,000,000 has been made to coal-mine workers in the prevention of fatalities.

Unquestionably, much progress has been made in eliminating health and safety hazards in the mineral industries since the Bureau of Mines was established in 1910, and there is absolutely no doubt that a considerable amount of the improvement is due to the fact that the Federal Bureau of Mines, through its numerous health and safety activities, acted as what might be termed a "spark-plug" in keeping safety to the front. There is also no question that much remains to be done before our mines can be considered as being operated with anything like the degree of safety that can and should be reached. Unquestionably, mine-accident occurrence can be reduced at least 50 per cent (possibly as much as 75 per cent) below what it now is, and this can be accomplished readily if the various interested agencies—the mine operators, the workers, the State inspection departments, the safety workers of the Federal Bureau of Mines, and others engaged in mine-safety work cooperate as they can and should to make mining a relatively safe occupation.

Artificial Respiration*

Artificial Respiration is a method by which normal respiration is imitated by manual movements to restore breathing. As we have no means of influencing muscular action in an unconscious person to produce normal inspiration, we have to depend on manual means of forcing air from the lungs by compression and on natural elasticity of the walls of the chest cavity plus the drop of the arch of the diaphragm to draw fresh air into the lungs when the compression is released.

Where breathing has stopped or is very irregular and feeble artificial respiration is usually required. The most frequent conditions under which artificial respiration is required are electric shock, gas poisoning, drowning, and suffocation from various causes. It has been estimated that 12,000 deaths occur each year from the above causes but that 40,000 to 50,000 lives are saved annually by application of artificial respiration with the use of the oxygen inhaler, where available.

In many conditions where breathing has ceased or apparently ceased, the heart action continues for a limited time. If fresh air is brought into the lungs, so the blood can obtain the needed oxygen from the air, life can be sustained. That can be accomplished in a great number of cases by artificial respiration. Every moment of delay is serious. As the breathing may be so faint that it will not be detected by the layman or first-aid man, it is advisable that artificial respiration be started immediately, even if the person seems dead. Continue artificial respiration without interruption until natural breathing is restored, or until a physician declares that

* Bureau of Mines "Manual of First-aid Instruction," 1940.

the patient is dead. Persons have been resuscitated after as long as 72 hours of artificial respiration. If natural breathing stops after being restored, give artificial respiration again.

An assistant should remove from the patient's mouth all foreign bodies, such as false teeth, tobacco, gum, and any loose material, see that the tongue is forward and not over the windpipe, and loosen tight clothing from the victim's neck, chest and waist. Keep the patient warm by wrapping with blankets, clothing, or other suitable material. Hot-water bottles, hot bricks, or other heated objects, well wrapped in cloth or paper and tested, should be placed about the victim. Place aromatic spirits of ammonia near the patient's nose, determining a safe distance by first trying how near it may be held to your nose. Do not give any liquid by mouth until the patient is fully conscious, when aromatic spirits of ammonia (1 teaspoon in one-half glass of water), hot coffee, hot tea, or hot water may be given slowly. Keep the patient lying down. If after being partly resuscitated he must be moved, carry him on a stretcher.

Perform artificial respiration gently and slowly. Roughness may injure the patient. Always treat the patient for shock in addition to the artificial respiration. Do not permit bystanders to crowd around. Assistants should not do anything that will interfere with the operator.

The Schaefer (prone-pressure) method and the Silvester method of artificial respiration are both recommended. The prone-pressure method being the simpler and easier to perform, it should be used in all cases where possible. However, there may be a few instances where the Silvester method can be employed more satisfactorily.

How to Give Artificial Respiration by the Prone-Pressure Method*

This method is essentially the same, except that wording and description have been changed to make it more understandable, as that approved by the following organizations: American Telephone & Telegraph Co., American Red Cross, American Gas Association, Bethlehem Steel Co., National Electric Light Association, National Safety Council, Bureau of Medicine and Surgery (Navy Department), Office of the Surgeon General (War Department), Bureau of Mines, National Bureau of Standards, U. S. Public Health Service, and various others.

Lay the patient on his belly, one arm extended directly overhead, the other bent at the elbow, and turn the head toward the extended arm so the side of the face will rest on the hand or forearm of the bent arm, and the nose and mouth will be free for breathing (see Fig. 12, p. 55).

Kneel, straddling the patient's thighs with your knees placed such a distance from the hip bones as will allow you to place the palms of the hands on the small of the back with the fingers resting on the ribs, the little finger just touching the lowest floating rib, the thumb and fingers in a natural position, and the tips of the fingers just out of sight on the sides of

* Bureau of Mines "Manual of First-aid Instruction," 1940.

the trunk (see Fig. 13). If conditions make it impracticable for the operator to straddle both thighs, he may assume a position where but one thigh is straddled.

With arms held straight, swing forward slowly, so that the weight of your body is gradually brought to bear upon the patient. The shoulder should be directly over the heel of the hand at the end of the forward swing (see Fig. 13). Do not bend the elbows. This operation should take about 2 seconds.

Now, swing backward immediately to remove the pressure completely. In actual work the hands may or may not be entirely removed from the patient. In contests, for uniformity, the operator will entirely remove the hands and assume the position shown in the picture in figure 14 between each complete respiration and the next. After 2 seconds swing forward again. Thus, repeat deliberately 15 times a minute, the double movement of compression and release, a complete respiration in 4 seconds.*

As soon as artificial respiration has been started and while it is being continued, an assistant should loosen any tight clothing about the patient's neck, chest or waist. Keep the patient warm. Do not give any liquids whatever by mouth until the patient is fully conscious.

To avoid strain on the heart when the patient revives, he should be kept lying down and not allowed to stand or to sit up. If the doctor has not arrived by the time the patient has revived and is conscious, he should be given some stimulant, such as 1 teaspoon of aromatic spirits of ammonia in half a glass of water or a hot drink of coffee or tea. The patient should be kept warm.

Resuscitation should be carried on in a safe place nearest possible to that where the patient received his injuries. He should not be moved from this point until he is breathing normally of his own volition and then moved only in a lying position. Should it be necessary, due to extreme weather or other conditions, to move a patient before he is breathing normally, resuscitation should be continued while he is being moved.

A brief return of natural respiration is not a certain indication for stopping the resuscitation treatment. Not infrequently a patient, after a temporary recovery of respiration, stops breathing again. The patient must be watched; if natural breathing stops, artificial respiration should be resumed at once.

* Red Cross technique, p. 55.

Chapter 21

Asphyxia as a Problem of the Federal Government Children's Bureau

The Future of Asphyxia Neonatorum

If progress is to be made in the prevention of asphyxial death, it is not likely to occur through modification of the methods just considered or the addition of new ones. The author is of the opinion that intensive training of personnel responsible for the care of the asphyxiated baby offers the greatest hope of success. We have the necessary facts, we have the technical equipment, but the widespread ignorance of both results in unnecessary deaths.

In addition to training physicians and technicians, a total reorganization of the field of gas therapy is urgently indicated. The vast field of pneumatology is likely to develop rapidly under the direction of physicians familiar with the use of gases for the control of pain (anaesthesia). On the other hand, it is absurd to attempt to subordinate it to the limited intramural field of Anaesthesia.

The problem must be approached from two angles simultaneously: (1) as an emergency problem to be met now; and (2) as a long-range problem, to be integrated into medical school instruction. The immediate approach is briefly referred to below. The long-range approach is discussed in chapters 28 and 30.

On December 8, 1941, The Special Committee on Infant Mortality of the New York County Medical Society passed the following resolution:

"The recommendation of the New York County Medical Society is as follows:

"That the Special Committee on Infant Mortality be permitted to contact the heads of obstetric departments of Manhattan hospitals to inquire if these departments would be interested in contributing to the setting up of a special training service under the auspices of the Society for the Prevention of Asphyxial Death, so that they may have present in the delivery room an individual competent in the use of resuscitation apparatus."

As a result of this resolution a communication was mailed to the Directors of Obstetrics of all private Manhattan hospitals, eleven of which expressed interest in the proposal. The resolution was simultaneously forwarded to the State Director of Maternal Health and Child Welfare of every state in the union with the accompanying statement:

Feb. 1942.

"The above resolution indicates such an important trend that we think that it will be of interest to you.

"We believe that a routine based upon the pathological physiology of asphyxia should displace the present 'apparatus first' policy which commercial interests have fostered with so much vigor.

"Because of the ease with which an exact knowledge and correct treatment of the needs of a particular case may be avoided by placing all responsibility upon a mechanical robot, the prevention of asphyxial death is now and threatens to remain upon the low level of commercial competition. We regard resuscitation of the new-born as on a par with or of greater

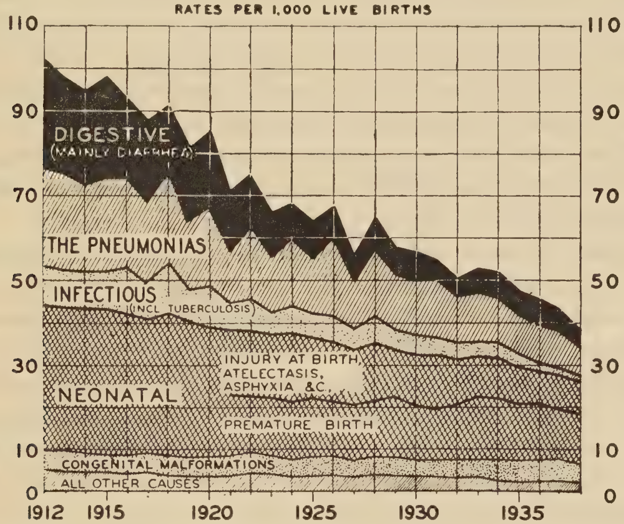


Fig. 160. Causes of infant deaths in New York City, 1912–1935.

importance than the Caesarian which may have preceded it. For of what use is a Caesarian if the baby dies? Many courses in operative obstetrics are provided. We believe that correct artificial respiration should be broadcast upon a national basis.

"The incidence of asphyxial mortality in the obstetric service is such that it may readily be overlooked or regarded as a regrettable accident. Where residents rotate on a short-term service, experience in the care of asphyxia neonatorum is limited.

"The attending obstetrician and his assistants have their hands full at the time of delivery. They cannot direct infant care at this period. If the baby survives there is no asphyxial problem. If the baby dies, it is too bad, but it is also too late to do anything about it. After the accident, plans to prevent recurrence are usually discussed, but the necessary prophylactic measures are seldom put into operation.

"While each institution has its particular environmental problems, and while secondary difficulties and objections naturally arise, the basic question, 'Would you consider it a good plan to have at all times in your delivery room an individual qualified to care for asphyxia?' deserves careful consideration. Furthermore, it is pertinent to recall that those who can treat asphyxia neonatorum can also treat asphyxia in adults regardless of its cause, be this in civil or military practice.

"The desirability of setting up a resuscitation service may be briefly considered from the following points of view (1) the value of routine; (2) the personnel; and (3) the subject matter to be taught.

"**The value of routine.** The importance of resuscitation routines has been demonstrated by the First Aid provided by the Fire, Police, Consolidated Gas and other groups, which maintain trained rescue squads. More recently, so-called "advanced First Aid," conducted by non-professional persons addressing physicians and medical students, has become popular. Students in these classes, while having a better theoretical understanding of the subject than their instructors, admit by their presence that they are deficient in the practical application of the theory which they possess. They attend for the purpose of acquiring a 'practical routine.'

"It is to be noted that medical resuscitation routines are further complicated by the fact that the asphyxial accident is relatively infrequent. In other words, the expected accident does not happen often enough to insure by its own momentum an efficient routine to meet it. It is this momentum or stimulus which the proposed service will endeavor to supply.

"**Personnel.** The instruction and the supervision of asphyxia neonatorum by the visiting staff is handicapped by the fact of a constantly divided interest—the mother and the baby. Experience has demonstrated that resuscitation technique is not passed from senior to junior house man with any greater success than is surgical technique. Furthermore, the senior cannot impart details and the experience which extended practice implies. Primarily interested in other aspects of the case, he is very likely to regard this duty as an undesirable chore. The importance attached to a field is often measured by the formality of its approach. Casual instruction by the resident does not constitute a formal approach. In the resuscitation service proposed it is suggested that the instructor in resuscitation serve in the capacity of visiting or consulting pneumatologist, in charge of resuscitation.*

"**The Subject Matter to be Taught.** Granted the desirability of a resuscitation routine directed by personnel expert in the field, the subject matter to be taught remains to be determined. The basis of this may be found in "A Clinical Study Based Upon an Expression of Opinion Received from the Professor of Obstetrics of Sixty-three American and Canadian Medical Schools". It is suggested that procedures which have been found

* *J. Med. Soc. County of N. Y.*, p. 4, Feb. 7, 1942.

acceptable to the majority of these schools be used as a ground-work upon which more recent matter may subsequently be added. For example; correct posture, heat to the body and removal of the secretions from the infant's airway are generally accepted as constituting "necessary procedures" and provide an acceptable ground-work.

"Theoretical instruction may be developed as follows:

(1) Review of the anatomy and physiology of the infant respiratory tract.

(2) The causes of asphyxia and their diagnosis.

(3) Diagnosis of the stages of asphyxia.

(4) Indications for the treatment of each stage.

(5) Facilities required for the treatment of each stage. (Apparatus available is usually adequate.)

(6) Toilet of the airway, removal of secretions, oxygen inhalations, exposure of the airway of the flaccid baby, intubation and suction of the airway of the flaccid baby, insufflation of the flaccid airway (p. 79).

(7) Post-operative care and oxygen therapy.

(8) Resuscitation records based upon findings noted above (p. 122).

"Resuscitation apparatus is regarded as a means to an end. The simpler it is the safer it is, and the more likely to function when needed. The care of the asphyxiated has not observed the classic formula, "To follow the physiological indication." The approach has been simultaneously too simple and so complex as to be dangerously meddlesome. It is too simple in that it has disregarded varying degrees of asphyxia, with their respective indications for treatment. It has been meddlesome in attempting to supplant the delicate balance of respiratory rate and rhythm, instead of encouraging and supporting the returning function."

Responses were received from all over the country: Juneau, Alaska; Santa Fé, New Mexico; Jacksonville, Florida and Providence, R. I., as well as from Nebraska, Pennsylvania, Washington, South Carolina, Oregon, Arizona, New York, Maryland, Georgia, Illinois and Texas.

From Pennsylvania: "Your letter of March 21st is of great interest to this Bureau and personally I can agree with you on many points. . . . Although the percentage of mothers delivered in a hospital is increasing rapidly, the percentage delivered at home is still high in rural communities of this State and measures to prevent asphyxia are admittedly more difficult in the home. In my opinion, careful consideration of your suggestion by the obstetrical staffs of all of our hospitals would lead to a further reduction in infant mortality."

From Washington: "During the last year we have been conducting a neonatal mortality survey. My medical advisory committee and I have become particularly interested in asphyxial deaths and the resuscitation of infants through this survey. We have discussed very seriously the matter of trying to arrange for a panel discussion on the resuscitation of infants at a joint meeting of the pediatric and obstetric sections of the State

Medical Association at its annual convention, perhaps not this year, but next."

From South Carolina: "It is true that the subject, Asphyxia of the New-born, has been given little consideration in most hospitals, and it does seem to be most important that one person be specially trained as a consultant and an instructor, and placed in charge of such a service. Your suggestions under consideration are fine, and when they are put into action we should certainly like to be notified.

From Oregon: "I have been interested in this subject for some time and I am interested, not only in the importance of infant asphyxia when it has developed, but also in preventing the asphyxia in the first place. By this I have in mind more instructions to the physicians as to the danger of heavy analgesia in the later stages of pregnancy and unwise use of nitrous oxide anesthesia during the second stage of labor."

From Arizona: "Your suggestions for having an instructor in resuscitation to serve in the capacity of visiting or consulting pneumatologist in charge of resuscitation is a very good idea."

From Maryland: "I would like to assure you of the deep interest of this Bureau in any program pertaining to this subject, and I would appreciate receiving from time to time any further suggestions or literature which you may have."

From Texas: "With regard to the subject of asphyxia of the new-born, I may say that I thoroughly agree with the thought that lies behind this study, that is, the development of preventive measures for asphyxia neonatorum which are not based upon mechanical apparatus but rather upon the anatomy, physiology and pathology involved. From our own standpoint, it would seem that there is a definite need for a scientific approach to this question through the means of well regulated and well controlled research. The practical features are, in my opinion, going to produce the greatest difficulties, and except for about fifteen of the 367 hospitals licensed by this department, there would have to be a very definite revision of the plan because there are only about fifteen to twenty hospitals in this stage having a daily patient average of one hundred or more.

"I feel that the training in the hospital should be given to both obstetrics and pediatrics residents, and the maintenance of a visiting or a consultant pneumatologist might be excellent for training purposes; but these questions remain to be solved. First, if a member of the resident staff is trained and truly interested in the program, the difficulty lies in the fact that he will probably move on after one or two years. If the individual selected to render the care is a consultant or visiting man of the hospital, many situations will arise when he will not be available. This leads to the third question. Should not the training also be directed to some of the nursing staff, possibly the supervising nurse of the operating room or the obstetrical nurse assistant? While I agree that the training of a limited number of persons in large institutions is of value, this training should not be confined to large hospitals. A definite routine which can be adjusted to variable factors should be taught in all medical schools in conjunction with the departments of obstetrics and pediatrics.

"I believe that the points outlined under your theoretical plan of instruction cover the subject well, and certainly the use of a stillborn is preferable for practice in intratracheal catheterization. However, the possibility should be kept in mind of development of a manikin for this purpose, such as that used in obstetrics. We would, of course, emphasize that the material for teaching purposes should be concise and practical enough to be used at any time and at any place."

Granted the need of intensive training in the prevention of asphyxial death, the course of instruction set must meet the needs of urban, suburban and rural practice. In this new activity the pediatrician is destined to become well acquainted with the Art of Resuscitation.

Chapter 22

Asphyxia as an Army Problem

Asphyxial Accidents in the United States Army

It has been the author's great good fortune to have enjoyed the acquaintance of Charles Ransom Reynolds, Major General, U.S.A. M.C. who was Surgeon General from 1935 to 1939. The present interest of the United States Army in the coordination of gas therapy, as indicated in Army Regulations A.M.B. #54, Oct. 1940, may readily be traced to the keen interest of this far-sighted administrator.

On the evening of Feb. 19, 1934, a blizzard swept New York City. Dr. Wendell Phillips, past President of the American Medical Association had just read a paper entitled, "Asphyxia, a Major Problem in Medical Education" to a handful of listeners in the ballroom of the Biltmore Hotel. Col. C. R. Reynolds, Commander of the Second Corps area arose and commented on Dr. Phillips' paper as follows:

"While the Medical Department of the Army is thinly spread out in stations in the United States, in Alaska, in the Philippine Islands, and Puerto Rico, it is not in those places that we can hope to make very much of an impression in the training that is necessary to give emphasis to the principle for which the Society [S.P.A.D.] stands. But it is rather at the nerve center, or the headquarters in Washington, which is represented by the Surgeon General's Office and the Army Medical School, that most can be done. It is there that the principles of training are enunciated and formulated and passed out in the service in the form of Army regulations and training regulations.

"From the standpoint of therapy we go along with the medical profession. There is nothing peculiar in the method of medical practice in military service except, perhaps, in preventive medicine. Our medical officers are graduates of civil institutions. They have studied medicine and have come to us as doctors. The training they get at the Army Medical School, however, fits them for the peculiar duties they are required to perform in military service. It is in the nature of a post-graduate course. I believe it is just there that greater emphasis can be given and is now being given in the matter of anesthesia and oxygen therapy. At the Army Medical School particularly, considerable emphasis is placed on anesthesia and also on oxygen therapy, along with the practice which is going on in civil life. I believe right there is where the greatest effect can be made in putting over the principles of this Society in military service.

"Another thought comes to my mind regarding the use of the Army in advancing this idea. Dr. Baker, who facetiously refers to himself as the greatest college professor in the world, means by that that the Army of the United States is an educational school. We in the Medical Corps are constantly giving or receiving training. He refers to himself as the president of the largest university in the world because he has the largest student body, consisting at one time of some 500,000 officers and men. I mention that today not because we have that number of officers and enlisted men, nor are we such a great school as he thinks we are, but the Army of the United States has quite a little opportunity to spread the gospel, whether it is for good or evil, in civil life, because in its military establishment it comes in contact with a large element of so-called citizen components. We are constantly training R.O.T.C. students in the R.O.T.C. units at the medical schools. Then we get them in camp for about six weeks during their college course. There they are given training in First Aid and principles which have been discussed here.

"We also train many thousands of the C.M.T.C. boys between the ages of 17 and 21. There again is a great opportunity of putting over a training program. We come in contact also with a large number of Reserve Officers. In the Medical Corps in the Army now there are something like 17,000 men of the civil profession who come to the Army from year to year to receive special training. It is among those that we try to emphasize First Aid and the care of the injured.

"Then again we come in contact with the National Guard. It is in that field I think that the Army Medical Corps can do its greatest work in emphasizing such emergency work as resuscitation. We have a large number of drowning cases, as has the Navy. One of our chief causes of death is suicide. The emergencies we get in the Army are no different from those of civil life. But we have an unusual opportunity of training some 120,000 enlisted men and, as I mentioned, a large number of citizen components that come to us for training.

"I believe a real step can be made by emphasizing the importance of the subject to the Surgeon General and to the officials of the Army Medical School, to the end that the subject should be given more importance in the curriculum at that school where we instruct the Army Medical Officers, both in anaesthesia and oxygen therapy, as well as calling more attention to resuscitation. Of course there are also the measures which go out to our forces by way of training regulations."

The Surgeon General referred to by Col. Reynolds, General Robert Urie Patterson, 1931–1935, was already a member of the Advisory Board of the S.P.A.D. His letter of acceptance, dated May 17, 1933, contains the following interesting excerpt:

"Your letter of December 7, 1932, inviting me to accept membership in the Society for the Prevention of Asphyxial Death, Inc. in an honorary advisory capacity was received the next day; however, I postponed replying until I could get information on which to base my decision on your request. You can readily understand that I do not wish the official title of the Surgeon General to be associated with any organization engaged in activities of a commercial nature or with one not entirely ethical in its relation to the American medical profession. I have, therefore, had my Executive Officer take the time and make the effort to determine the standing of your Society.

"Data regarding the Society has been secured from various sources. I am pleased to find that all information received has been favorable."

On the day following Col. Reynolds' comments, Major Leon Fox, representing General Patterson, presented a paper which considered the factor of chemical injury in asphyxia. Quotations from Major Fox's paper appear at intervals in the following pages.

In 1935, Col. Reynolds became General Reynolds, succeeding General Patterson. During the course of General Reynolds' administration the author was guest speaker at the Walter Reid Hospital, stressing the coordination of gas therapy. At the opening of the New York World's Fair, General Reynolds accepted an invitation to state the position of the U. S. Army in its relation to the problem of asphyxiation. General Renolds' letter, as of Feb. 28, 1939, is as follows:

"War Department, Office of the Surgeon General, Washington. Reference is made to your letter of January 17, 1939, relative to publicity material for the World's Fair dealing with the Army's interest in and researches on asphyxia. From the viewpoint of the United States Army, the problem of asphyxia primarily resolves itself into three divisions: (1) carbo monoxide poisoning; (2) war gas poisoning, and (3) anoxemia incident to high-altitude flying.

"Carbon monoxide poisoning is encountered in situations similar to those in civil life; also, where firearms are discharged in closed spaces and ventilation is inadequate, as a result of collections in shell holes and dugouts, and under conditions of incomplete combustion, such as fires in buildings and barracks. An all-purpose gas mask with special cannister which catalytically decomposes the carbon monoxide is prescribed for use as a protection from this gas.

"The war gases present a problem peculiar to the military for all practical purposes, and one that is of great import. The true asphyxiating gases or lung irritants of most importance are phosgene, diphosgene, and chloropicrin. Of these, phosgene is perhaps the most effective in poisoning the air. At present intensive investigation is being conducted at the Chemical Warfare School on the treatment of phosgene poisoning. The vesicants such as mustard gas, lewisite, and ethyldichlorarsine may also cause asphyxia if breathed in sufficient concentration or over a prolonged period. The military gas mask affords considerable protection against these and other warfare gases. Oxygen is essential in the treatment of asphyxia from such gases; artificial respiration is contraindicated.

"The problem of anoxemia is of utmost importance to the Army. The Physiological Research Laboratory at Dayton, Ohio, has been particularly equipped to study anoxemia. Here a huge vacuum chamber has been constructed that can simulate ascents at the rate of 10,000 feet per minute up to an altitude of 130,000 feet and a descent of 20,000 feet per minute. Temperatures as low as −65°F can be produced in this chamber. It is now well known that anoxemia causes effects similar to alcoholic intoxication. The individual may feel alert and stimulated and believe that he is performing brilliantly when in reality his judgment is seriously impaired and he has marked psychomotor retardation. It can be appreciated readily that such effects are highly dangerous to the individual, and that the potentialities for disaster are very great in mass-formation flights at such critical altitudes. It has been found in experimental tests that the average individual loses consciousness from anoxemia at an altitude of 20,000 to 25,000 feet unless oxygen is breathed. At about 35,000 to 40,000 feet unconsciousness supervenes in spite of the use of oxygen (unless administered under pressure) because the alveolar tension falls below the level necessary for the oxygen to combine with the red blood cells. For sub-stratosphere flying pressure-cabin planes are essential. Such a plane has been successfully developed in recent months.

"Parenthetically, it may be of interest to note that the body cannot accommodate itself to the terrific rate of ascent of which modern airplanes are capable. The gases released in the body tissues consequent to such rapid decompression may cause a condition similar to caisson disease. A rate of ascent greater than 3000 feet per minute is dangerous because of this factor.

"From these considerations it is apparent that the problem of asphyxia other than carbon monoxide poisoning differs markedly in the Army from that in civil life and that the solution is vital not only for the safety of the individual, but also for the security of the country."

General Reynolds' concise statement is a prelude to the brilliant results since accomplished by the Army in the prevention of asphyxial death. Outstanding in this field has been the pioneer work of Capt. H. G. Armstrong and the recent activities of the Committee on aviation of the National Research Council headed by Dr. Eugene DuBois.

As noted in the letter just refered to, asphyxia in the Army may be considered from three points of view (1) high-altitude flying, (2) chemical warfare, (3) civilian accidents (see page 290 and frontispiece).

A few comments may serve to refresh the Army officer's knowledge of civilian causes of asphyxiation and to suggest the manner in which warfare may serve to accentuate these causes. It is believed that special emphasis may well be placed upon the pathological physiology of chemical warfare and on the indications for treatment.

Accentuated Civilian Accidents

It would appear desirable that the Army medical officer be perfectly familiar with the usual causes of asphyxia, as he may at any time be called upon to face such unexpected accidents. The suggestions offered below may one day prove of service.

Submersion. A common accident in the Army, often complicated by impedimenta, cold, exposure and darkness, by coexisting wounds or shock from blood loss. The factor of submersion may be overlooked unless the existing signs are so striking as to demand immediate relief. It is quite conceivable that asphyxial death may take place while the attention of the rescuer is focused upon the treatment of a local injury. It is suggested that treatment be habitually directed to the man's *general* rather than his *local* condition (see page 155).

Carbon Monoxide Poisoning. Too much publicity cannot be directed to this form of asphyxial accident. It should be constantly borne in mind that carbon monoxide is odorless and tasteless, that its presence is often first noted from its *effects*, and that *those affected promptly lose their power of observation and judgement.* In other words, the only safe-guard against carbon monoxide poisoning is knowledge and a conviction of danger which will result from exposure. The presence of carbon monoxide may be anticipated: (1) where the exhaust of automotive vehicles is not well ventilated by draughts or open spaces (automobiles, airplanes, stationary engines, tanks). (2) *As the residual effect of high explosives* in gun turrets, tanks, new shell holes, shelter tunnels, dugouts, fox-holes, and swampy lowlands. The gas may penetrate earth, seeping through bomb craters and demolished buildings to rescue shelters and tunnels. (3) *Wherever illuminating gas exists* (Improvised fixtures, leaky connections, broken gas mains, etc.). The odor of the illuminant may be obscured by liberated chemical gases, nitric fumes, chlorine or ammonia. (4) *From incomplete combustion of fires* made for heating or cooking purposes in poorly ventilated spaces, blacked out or dimmed out. From damaged flues of otherwise first-class heating plants in well constructed buildings.

"An enormous amount of carbon monoxide accompanies the combustion of high explosives. Explosions occurring in the vicinity of badly ventilated spaces, in subterranean galleries and dugouts may produce severe and immediately fatal cases of carbon monoxide poisoning. Its extreme toxicity is due to the fact that its affinity for the hemoglobin of the blood is 210 times greater than that of oxygen. Its presence in the system diminishes the oxygen-carrying properties of the blood. During World War I it was observed in exceptional instances that dangerous concentrations of carbon monoxide accumulated in the depths of shell craters. Poisoning by carbon monoxide was not infrequently observed in areas remote from the original sites of explosion, the gases having diffused through the permeable soil."

Oxygen Deficiency. It is to be recalled that in warfare as in civilian life a reduction of oxygen in the respired air may be expected in shelters, galleries, or poorly ventilated compartments. Oxygen in such spaces may be consumed by human metabolic needs, by fires used for heating purposes, or the gas may be diluted or displaced by carbon dioxide, methane, or other gases.

The reduction of oxygen at high altitudes when an army is on a march over mountains is not to be overlooked (see page 136). "When the oxygen content of air falls below 17.5 per cent a lighted candle will be extinguished."

Asphyxia of the New-born. While the military surgeon does not expect to meet obstetrics, it would be a pity were he unable to do what was possible to relieve the asphyxia of a baby whom he had been called upon to deliver. In this connection it is well to bear in mind that a new-born baby is not a detached entity dependent solely upon the pediatrician and the obstetrician. The baby is nothing more or less than a little man (or woman) whose over-all as well as detailed measurements may be roughly estimated as approximately $\frac{1}{3}$ that of his mother (at full term). While the structures are relatively small with delicate bone and cartilage framework, the mucous membranes are not unlike that of the adult, but resilience to asphyxia is far greater. Procedures indicated for the relief of asphyxia, if carried out with reasonable care, will cause no injury. Finally, the prevention of asphyxial death is no mere academic consideration. It is the supreme medical act—the real saving of a life otherwise lost forever. Because of this fact how can half measures be deemed sufficient? The issue is "*how can this life be saved?*" not "*what may happen when the baby recovers?*". Recovery by any procedure is too often taken for granted. Death, when it occurs, is not referred to the inefficiency of the resuscitation but to the failure of the baby to fall into a preconceived reaction. The indications for the treatment of asphyxia have been given (page 46), and also the means of meeting them (chapter 6). Familiarity with these indications and these means of relief will suggest appropriate improvisation as occasion offers.

Foreign-body Obstruction. Obstruction of the airway is a common complication of wounds of the head and neck. This is especially true of the unconscious injured. It requires but little imagination to reconstruct such accidents. A penetrating wound of the jaw, hemorrhage, unconsciousness, continued hemorrhage into the mouth, broken teeth and splinters of bone, patient lying flat on his back, morphine administered—all the necessary elements for progressive anoxia, reduced pharyngeal and laryngeal reflexes, aspiration, terminal asphyxia and death. (Signed out as hemorrhage and shock!)

The possibilities of asphyxial accidents in bombing raids are pointed out in the following letter which appeared in *New York Medical Week* for

August 17, 1940: "In battle formation come the bombers, wreathed in clouds . . . Ten seconds after dust has filled the air comes the sound of the explosion, a good two miles away. A woman pinned beneath a rafter, her chest compressed, anoxic, suffocating, asphyxial death impending. Nearby, a jagged shell hole in his cheek, a man lies prone, hemorrhage filling his flaccid airway, drowning him. And in a cellar shrink two small children, dazed, drowsy, for the shadow of anoxia is upon them. Comes the first-aid stretcher, its bearers picking their way carefully through the smoke, the heaps of stone and plaster. Back to the ambulance with their burden. There is suction, let us hope, and oxygen for pharynx and trachea. And the woman with right lung penetrated, collapsed—can we hold her till the hospital is reached? For everything we need is here: sterile team at work; eyes for the freedom of the airway, the color of the blood, for the care that must follow when the work is done. Asphyxial death in September, 1940, in this world at war, in this Britain so close to us in every way."

Drugs and Allergy. The internist is accustomed to view his problem as the *director* of the treatment to be applied. He looks to the interne, the nurse, the technician, for the correct application of the physical measures which he prescribes. He is too often satisfied with his detached consideration of symptoms and signs and regards his diagnosis as the peak of achievement. He orders a blood count, an infusion, a transfusion, or artifical respiration, as the case may be. If the patient does not respond, it is most unfortunate but is in no wise a reflection on his own skill or ability.

It is high time to assail such complacency. An acute asphyxial accident is an immediate matter of life and death. The outcome is the responsibility of the physician in charge. If technicians are employed, they operate under his directions. It is the duty of the attending physician to see that this technical assistance is in every respect efficient. He can never resign in favor of the rescue squad or be satisfied to follow it. He must figuratively roll up his sleeves and get into the battle. Should he be unfamiliar with the "feel" of the unconscious airway or with simple technical methods for controlling its freedom, it is his duty to acquaint himself with these matters. He can do so by administering a few general anaesthetics.

Acute or progressive respiratory failure may readily be caused by the simple administration of drugs; *but it cannot be relieved by this method!* Too often the internist fails to realize this fact until too late. Re-orientation of men who are established practitioners is difficult. The medical education of students and internes may well be directed to this point of view.

Allergic reactions are closely allied to drug poisoning, and are likely to occur under similar conditions in the hands of men who are unfamiliar with pneumatological technique (see page 255).

Fire and Smoke. In actual warfare the medical officer is exposed to and in charge of all the hazards known to fire-fighting (page 222). To these hazards are added those of gunfire and high explosives. Crushing injuries from collapsed buildings, from compression between moving objects, etc., complicate the hazards of the wounded, and if over-looked may result in sudden death.

Electrocution. The danger of accidental electrocution is always imminent in frontline advances and in bombed areas. Its immediate causes and effects should be entirely familiar. In the smoke and the confusion of battle, electric shock may easily be overlooked and supportive treatment too long delayed. The important features of this form of asphyxial death are discussed on page 209.

Accidental garroting or hanging upon wire entanglements may occur; for the pathological physiology of this form of accident see page 238.

Asphyxia as a Problem of Chemical Warfare

The general medical aspects of chemical warfare are referred to briefly as follows: "Medical officers are primarily interested in the physiological and pathological action of chemical agents, and classify all chemical warfare materials on this basis. The physiological classification must be considered as only a general grouping, inasmuch as all the gases, if in sufficient concentration, are irritating to all tissues with which they come in contact. Under field conditions, however, where concentrations are generally weak, certain tissues and organs are more quickly or obviously injured by a given gas than others. It must be remembered that several chemicals, each affecting different parts of the body, may be used at the same time, thereby producing a variety of symptoms. Some chemical agents produce effects with great rapidity; others are slower in their action. Thus the flow of tears provoked by lachrymators, the loss of consciousness induced by a direct poison to the nervous system, and the sneezing caused by a sternutator may ensue almost immediately after exposure. In other cases symptoms of material severity may not appear until some time after exposure.

"According to their *principal action* on the body, chemical agents are classified as follows:

Action	Gas
(1) Lung irritants (suffocants)	Phosgene Chlorine Chloropicrin
(2) Sternutatory (sneezers)	Diphenylaminechlorarsine Diphenylchlorarsine
(3) Lachrymators (tear producers)	Chloracetophenone Brombenzyl cyanide
(4) Vesicants (blisterers)	Mustard Gas Lewisite
(5) Direct poisons of the nervous system	Hydrocyanic acid gas
(6) Gases interfering with the respiratory properties of the blood	Arseniureted hydrogen Carbon monoxide
(7) Incendiaries (burn producers)	Spontaneously inflammable liquids Solid oils White phosphorus Thermite

"It is important to remember that the above classification is based entirely on the primary action of the agent and that the secondary action may be a very important factor in the production of casualties. As an example, mustard, while classified as a vesicant because of its principal action, may in fact under certain conditions be the cause of numerous casualties due to its lung-irritant action."

As a problem involving asphyxial hazards the number of the chemicals referred to above may be reduced to those which produce anoxic, stagnant, anemic and histotoxic anoxia.

Anoxic Anoxia (anoxia from obstruction of the passage of air to and from the alveolar capillaries) may occur from:

(a) Spasm of the pharynx or glottis, complicated by an inflammatory process of the lining mucosa of the upper airway.

(b) Pulmonary edema, resulting from inflammations of the endothelial lining of the alveolar spaces or from the cupping effects of suction produced by excessive inspiratory efforts to overcome the inflammatory narrowing of the smaller bronchial tubes.

(c) Reduced oxygen concentration in the respired air in the presence of excessive CO_2 or reduced oxygen.

Stagnant Anoxia occurs in the pulmonary capillary bed as the direct result of serum exudate (pulmonary edema). The hemoconcentration which develops raises the pressure in the pulmonary artery, which in turn is transmitted to the right ventricle, creating back pressure on the right auricle delivering systemic venous blood (hence the blue anoxia of venous congestion). As the return flow from the lungs via the pulmonary vein (carrying oxygenated blood) is reduced by the viscosity of the dehydrated blood, arterial flow and oxygenation are reduced, resulting in the terminal grey anoxia (small volume of anoxemic blood).

Anemic Anoxia, in which red blood cells are thrown out of commission by CO poisoning or by exposure to hydrocyanic gas.

Histotoxic Anoxia, as in cyanide poisoning.

Anoxic Anoxia

From upper airway spasm and inflammation following the inhalation of:

Chlorine: odor of bleaching powder, greenish yellow color, activated by moisture, makes smoking unpalatable.

Nitric vapor: aromatic odor, reddish-brown fumes.

Ammonia: odor sharp and penetrating, colorless.

Mustard: odor of garlic or onions, brownish in color.

Lewisite: odor of geranium, brownish in color.

If the above gases are inhaled for an extended period in respirable concentrations they may act like

Chloropicrin: odor of flypaper or licorice, colorless; causes in addition to inflammation of the upper airway, inflammation of the epithelium lining of the alveolar spaces, resulting in pulmonary exudate (pulmonary edema).

Hydrogen Sulfide: odor of rotten eggs, rapidly tolerated, rendering the gas particularly dangerous.

Phosgene: odor of cut corn, musty hay, making smoking unpalatable; inhalation may be followed by a quiescent period of 12–24 hours, culminating in an overwhelming fatal pulmonary edema.

Histotoxic Anoxia

This form of anoxia is precipitated by the inhalation of hydrocyanic gas. The methemoglobin formed by the cyanide radical is as effective as hemorrhage in causing an anemic anoxia. The stability of this combination is greater than that formed by carbon monoxide. Rebreathing oxygen-CO_2 is therefore not as effective as in the case of the latter form of poisoning. It forms a bridge, however, to the transfusion of whole blood. The positive pressure suggested by Behnke [at a pressure of $2\frac{1}{2}$ atmospheres (33 lbs.), sufficient oxygen is taken up by the blood in physical solution to meet all demands of the tissues, and therefore hemoglobin is not necessary as an oxygen carrier] should be kept in mind and employed where such facilities are available and where whole blood cannot be obtained.

Anemic Anoxia

In warfare, oxygen supply is often inadequate in shelters, galleries and other poorly ventilated spaces. Oxygen is consumed by the respiration of human beings and animals, and by fires used for heating purposes; it may be diluted or displaced by an excess of CO_2, methane or other gases.

The reduced supply of oxygen at high altitudes where troops are fighting over mountain passes, if uncompensated by adequate training, may prove serious (page 136).

Recapitulation. Asphyxia in chemical warfare may be produced as follows:

Upper Respiratory Obstruction, by spasm and inflammation (chlorine, nitric vapors, ammonia, mustard, lewisite).

Lower Respiratory Obstruction, by inflammation (phosgene, chloropicrin, hydrogen sulfide).

Reduced Oxygen Content of Respired Vapor (carbon monoxide, carbon dioxide and primary oxygen deficiency).

Stagnant Anoxia (phosgene, chloropicrin, hydrogen sulfide, as factors complicating serum exudate following inflammation of the lower respiratory tract).

Anemic Anoxia (carbon monoxide and hydrocyanic acid).

Histotoxic Anoxia (hydrocyanic acid).

Pathology of War Gas Poisoning*

Chlorine. The reaction is acute, occurring within 24 hours. It consists of severe injury to the mucosa of the upper respiratory tract and leads to superficial sloughing followed by membranous tracheitis. Irregular dilation and contraction of bronchi result in alternating patches of emphysema and atelectasis. There is extreme congestion and edema of the entire respiratory tract, including the peribronchial tissue and the sheaths of large blood vessels. Acute infectious processes begin in a few hours and develop into pneumonia.

Phosgene. The upper respiratory tract is unaffected. There is intense edema and congestion of the bronchioles. This leads to plugging, with consequent emphysema and atelectasis. Fibrin is present in abundance in and on the alveolar walls. This fibrin crosses and obstructs the capillaries everywhere, offering an explanation of the obstruction to the capillary flow which dams back the circulation into the right heart.

Chloropicrin. Extreme edema and congestion of the lungs occur. There is necrosis of the bronchial epithelium and of the bronchiolar walls, as well as dilatation of the right heart.

Diphosgene. There is intense irritation of the bronchioles and pulmonary parenchyma. Tremendous congestion and edema occur quickly, with dilation and contraction of the bronchioles, as in chlorine poisoning. The epithelium of the bronchioles becomes necrotic and is bathed in inflammatory exudate.

The upper respiratory tract is unaffected.

Mustard Gas. There is marked congestion of the lungs with little edema. Patchy atelectasis occurs. There is great destruction of the epithelium of the upper air passages with false membrane formation. Necrosis of the alveolar and bronchial walls follows invasion of bacteria.

Cyanogen and its Halogen Derivatives.

Cyanogen Chloride. Acute changes are confined to pulmonary edema and congestion. There is mild inflammation of the bronchi.

Cyanogen Bromide. Intense respiratory irritant. Half an hour after exposure there is contraction and dilation of the bronchioles and the lungs are bathed in inflammatory exudate.

Arsine. Arsine is an intense hemolytic agent. After four to six hours hemoglobinuria and hemoglobinaemia occur. Hemolysis is later associated with icterus.

Arsine Compounds. Methyl and phenyldichloroarsine destroy epithelium, as does mustard gas, and produce edema, as does phosgene. Both actions are milder, however. Circulatory interference appears to be from fibrin rather than from edema.

Note. With the idea of applying direct treatment to lungs affected by

* Adapted from the work of M. C. Winternitz, Major, M.C., U.S.A., Yale University Press, 1920.

poison gases, Winternitz conducted experiments in perfusing the lungs with saline solutions. He concludes that the lung is not sensitive to the introduction of fluid—perfusion can be done throughout all the lobes. The lungs can tolerate salt solution in large quantities without damage. Quantities up to lung capacity can be insufflated without difficulty. Absorption is rapid. There is no post-operative change. Phenolpthalein is absorbed from the lungs as quickly as from the muscles. Pulmonary irrigation is possible. There need be no ill effects. Irrigation removes material from the trachea and penetrates the smaller air passages (in this connection the reader is referred to the discussion of submersion, page 320).

The above conclusions would appear to confirm the impression that the hazard in drowning is anoxic anoxia. The mere presence of fluid is of secondary importance. Is it perhaps possible that in a pressure chamber of two atmospheres (page 293) tracheal and pulmonary fluid could be charged with oxygen to a degree permitting life during lung irrigation procedures. A man about to die from pulmonary edema would certainly have nothing to lose.

Edema. Technical Manual No. 8 of the War Department suggests the following explanation for the development of edema after gas inhalation.

"In contact with the moisture of the alveolar walls, the gas hydrolyzes, producing hydrochloric acid and carbon dioxide: $COCl_2 + H_2O = 2HCl + CO_2$. The irritant effect is supposed, but not proved, to be due to the action of the hydrochloric acid thus formed. As a result of this irritation the permeability of the alveolar and capillary walls is increased, causing a pulmonary edema. Pulmonary vessel thrombosis may develop. There are relatively few changes in the upper respiratory tract."

The lung irritants react with the cells of the pulmonary blood vessels and the alveoli (air cells) injure or devitalize them to the extent that they cannot perform their normal function. In the healthy lung these cells form a vital membrane that allows the exchange of the respiratory gases without allowing the tissue fluids to pass through the membrane. When the cells are injured they no longer act as a vital membrane but take on the properties of a devitalized, semipermeable membrane; as fluids are not held back or restrained, transudates occur. The extent to which this fluid fills the lung depends on the degree of pulmonary involvement. When the area is small, local effusion occurs. With more general severe injury, profuse transudations develop, involving large areas in both lungs. With the loss of the serum of the blood there is an increase in its viscosity; it becomes dark and tarry in color, and its flow through the capillaries is greatly retarded. The pumping of this heavy fluid places an extra burden on the heart which may result in dilation and failure. Undoubtedly some of the inhaled warfare gases decompose, thereby producing a direct toxic action on other organs, particularly the kidneys and central nervous system, which may become affected secondarily.

Treatment of Asphyxia due to War Gases

The treatment noted below has special reference to the care of patients who have been exposed to gases which may induce pulmonary edema.

Immediate treatment. This consists of absolute rest, application of heat (blankets, warmth, hot drinks), inhalation of oxygen, and venesection in the "blue" cases. Even though rest appears unnecessary, it must be enforced, the patient being kept prone on a litter, the foot of which may be elevated to assist lung drainage. He should be kept well covered with blankets and given hot drinks of coffee, tea, or water; alcoholic drinks should not be used. Even if the patient is very restless and apprehensive, morphine should never be given. Contraindication of morphine is based on the uniform experience of the First World War. Restlessness and apprehension may be combated with guarded doses of barbiturates. However, the relief afforded by oxygen inhalation is the best means of quieting and reassuring the patient.

Oxygen Therapy. While oxygen therapy is strongly recommended, the following statements appear in Training Manual 285: "Mixtures of helium and oxygen offer no advantage since the difficulty is not bronchial obstruction but the filling of the alveolae with fluid."

"Oxygen should be administered as soon as pulmonary edema develops; if the military situation limits the supply of oxygen it should be remembered that oxygen therapy is most useful in cases that verge on the desperate, especially those which are still deeply cyanotic, on the second day."

Venesection. "Because it can easily be performed under field conditions, bleeding is probably the most valuable therapy for the cyanotic or blue stage of phosgene poisoning, but *it should never be employed in the gray or* collapsed stage; 400 to 600 cc of blood should be withdrawn as early as possible and another 500 cc may be removed 6 hours later and the procedure repeated if necessary. A large-bore needle should be used unless the viscosity of the blood makes direct incision of the vein necessary.

When it is necessary to employ *general anaesthesia*, chloroform or nitrous oxide should be used.

"In situations where the use of spinal anaesthesia may appear logical, it should be remembered that it may predispose to the development of cardio-vascular collapse which so often occurs following exposure to pulmonary irritants."

"If pulmonary edema has developed, general anaesthesia is absolutely contraindicated."

Comments

Beecher*, who stresses the factor of inspiration against resistance, states: "Probably every anaesthetist has observed that if he is unable to

* "Physiology of Anaesthesia."

provide a free airway during anaesthesia, he is inordinately troubled by mucus in the bronchi, which may slowly drain out as a fine froth and increase the difficulty of inducing or maintaining a smooth anaesthesia. The effects of partially obstructed breathing in causing edematous, draining lungs have been reported by a number of observers.

Graham (1921) showed in excised edematous lungs that the negative "intrapleural" pressure of artificial respiration is adequate to produce seepage of fluid into the lung parenchyma and copious weeping of the lung surface. Hoover (1922) stated that if inspiration is obstructed, great negative pressures are produced in transudation of serum into the alveoli. This process he likens to dry-cupping of the lungs."

Is it not possible that the mechanical factors referred to may help to explain at least a portion of the pulmonary edema of chemical gas poisoning and might not recommendation be made to eliminate this factor?

As to the stagnant anoxia which develops as the result of the serum exudate with its ensuing hemoconcentration, it would be well to recall the enormous capillary surface offered by the lung (equal to that of a balloon 20 feet in diameter, or an area of 1256 sq. ft.). The fact that this enormous surface is folded away in the chest like the leaves of a fan may explain why it has been overlooked. Would it not be well to consider the vast inflamed surface of the endothelial lining of the lung as an injury comparable to a superficial burn with its accompanying dehydration, serum loss, and hemoconcentration? Under these conditions, would we withold fluid or practice venesection, thereby increasing the viscosity of the blood and superimposing an artificial anemic anoxia by loss of red blood cells? Does not the location of the serum exudate confuse the usual indications for treatment? A revision of the present treatment is suggested on page 300.

Viewing the injury to the respiratory tract inflicted by the inhalation of chemical gases in the light of the general pathological physiology of asphyxia, the question may be asked, Are the indications for treatment met by the routine prescribed? In raising this question the author freely admits a lack of clinical experience in war-gas therapy. He bases his comments entirely upon seemingly parallel problems in more familiar fields.

The vital problem in war-gas poisoning is that created by the development of pulmonary edema.

(a) The cause of this edema is ascribed solely to the toxic inflammatory effects of the gases involved upon the alveolar lining membrane, resulting in serum exudate. Inflammation of the bronchioles, reducing their lumen and producing a suction (cupping action) which predisposes to serum exudate is not mentioned. The use of helium is consequently considered useless ("The difficulty is not bronchial obstruction but filling of the alveoli with fluid"). If there exists inflammation of the walls of the bronchioles which reduces their lumen, helium therapy would appear to be indicated.

(b) No mention is made of the effects of upper respiratory obstruction upon the production of pulmonary edema, in the manner pointed out by Beecher. Consequently measures to prevent or to relieve obstruction are not noted.

(c) The unfortunate universal experience with morphine, to the effect that it should not be used, is undoubtedly due to failure to observe the respiratory obstruction which it may induce, and to relieve it by increasing the activity of the reflexes. It would seem that morphine, used with due regard for respiratory embarrassment, with oxygen, would give greater satisfaction than the use of barbiturates with their frequent post-dosage excitement. In the absence of such precautions morphine undoubtedly constitutes a hazard, as does the use of all analgesics and hypnotics.

(d) Of first moment, however, is the question of pulmonary edema. Judging from the existing pathology, the transudate of serum from the pulmonary capillary surface, a relatively vast area, is not regarded in the same light as is the transudate of serum from other inflamed surfaces. Possibly the thought of adding fluid to a lung already embarrassed by exudate has served to short-circuit what would otherwise be routine treatment for hemoconcentration due to loss of blood.*

* Clinical experience reported by J. H. Evans, *N. Y. State J. Medicine*, Dec. 1, 1943. "If we are to be prepared to give adequate oxygen therapy to large numbers of persons, whether on the fighting front or on the home front, immediate steps should be taken to obtain equipment and to convince the clinician that continuous 100 per cent oxygen can be given with safety to patients suffering from anoxemia and that it should be given at least intermittently as early as possible to all gassed cases in order to prevent pulmonary complications and to reduce edema of the bronchial mucosa.

The use of positive pressure oxygen is absolutely essential in the treatment of marked pulmonary edema and very important in oxygenation of the blood when there is swelling of the mucous membranes lining the respiratory passages.

Case 1.—A woman, aged 45, had gas poisoning and pneumonia. Tracheotomy had been done the day before I saw her. Her face was covered with vaseline gauze, and her arms and hands were bandaged because of burns. She was gasping for breath. Although she was not delirious, it required three nurses to keep her in bed and under the oxygen tent. Her toenails indicated marked cyanosis. When positive pressure oxygen was given her she quieted down and whispered, "Don't leave me." After ten minutes her physician wished the 100 per cent oxygen discontinued and the patient was again placed under the tent with the oxygen kept at the maximum flow permitted by the gage. She again became restless, cyanotic, gasped for breath, and had to be forcibly kept in bed. Her physician then said, "I give up; please go back to positive pressure oxygen." As soon as this was done she settled down and breathed easily again.

Case 2.—A man, aged 50 and weighing 240 pounds, had gas poisoning, pneumonia, and burns of face, neck, arms, and hands. Toenails showed there was deep cyanosis. Tracheotomy had been done. The face and eyelids were so swollen that patient could not open his eyes. His physician plugged the tracheotomy tube and positive pressure oxygen was given by the mask method. After about thirty minutes the doctor made the observation that the edema of the face had been sufficiently absorbed to enable the patient to open his eyes, and asked if the edema of the upper respiratory mucosa would also be reduced in the same way. When I replied that it surely would he remarked, "Then all these tracheotomies might have been avoided." However, in spite of the improvement in this patient the doctor wished to have the 100 per cent oxygen discontinued because he believed that ten minutes was the maximum length of time it could be given with safety."

The viscosity of the blood is reported to be so great at times that it will not flow through a large-bore needle. This state of affairs is accepted without comment.

Hemoconcentration (serum loss elsewhere) is a critical emergency demanding immediate treatment. Blood plasma or serum must be given without delay. Fluids by mouth or glucose intravenously are not expected to support the volume of blood and its fluidity. This can be accomplished only by plasma or serum containing the necessary blood protieds. Given relief of the existing hemoconcentration by suitable intravenous therapy, resulting in reduced viscosity of the blood, would it not be reasonable to expect that the pulmonary capillary stasis producing the back pressure on the right heart would be promptly relieved? Is it not a characteristic of an active, fluid, oxygenated circulation that it absorbs tissue edema?

How, it may be asked, does the removal of from 500 to 1600 cc. of blood assist the existing dehydration and hemoconcentration? Does not this treatment, from which men undoubtedly appear to recover, serve but to add the anemic anoxia of blood loss to the stagnant and anoxic anoxia which already exists?

(e) As to oxygen therapy, it is stated, "Oxygen should not be given under too high pressure for fear of rupturing the already damaged lung tissue." Unfortunately, the fear of rupturing lung tissue creates resistance to the best possible means which we have at hand to meet the fear which exists in treating hemoconcentration, namely that of adding to the existing pulmonary edema.

As fluid may be cupped into the lungs by suction, so can edema be reduced, indeed prevented, by positive pressure, by breathing air or preferably oxygen under pressure, as pointed out by Beecher. It has even been suggested by Poultron that the respiratory phenomenon presented by the patient suffering from pulmonary edema is an automatic effort to create intrapulmonary pressure and reduce exudation, *i.e.*, a short, quick inspiration and a long expiration against a partly closed glottis. As to the danger from positive pressure, it may be pointed out that the injured portion of the lung, in which fluid already exists, will be penetrated by gas pressure only with the greatest difficulty, if at all.

(f) As to the time of oxygen administration, this would seem to depend upon whether it is to be employed as a preventive of serum exudate or for purely symptomatic treatment. It is a source of great satisfaction to turn blue blood pink, but it would seem wiser, if possible, to prevent the blood from becoming blue. A closed system for rebreathing oxygen under pressure would do much to conserve a depleted supply. Its early use might well be calculated to prevent the terminal stage for which its use is otherwise to be reserved.

(g) "When general anaesthesia is to be used, chloroform or nitrous oxide is to be preferred." Chloroform is a circulatory depressant. The blood pressure curve follows the muscle relaxation which accompanies its use. Chloroform would seem to contribute to the existing stagnant anoxia. Nitrous oxide is contraindicated wherever anomemia exists.

That spinal anaesthesia may predispose to cardio-vascular collapse in an existing pulmonary edema may be explained upon the basis of the destruction of muscle tone which spinal anesthesia produces. This loss of muscle tone causes the shock of stagnant anoxia so frequently described by Henderson.

If the rationale of intravenous plasma administration for serum loss in pulmonary edema is acceptable, and if oxygen under pressure is found desirable, the problem of general anaesthesia in pulmonary edema is simplified. Ether, cyclopropane, or intravenous pentothal sodium may be used in conjunction with oxygen in a closed pressure system. In view, therefore, of the pathological physiology of pulmonary edema resulting from war gases, it is respectfully suggested (1) that such patients receive intravenous plasma in quantity sufficient to overcome hemoconcentration and to dispose of blood viscosity; (2) that oxygen under pressure be administered for the first 24–48 hours; (3) that morphine and atropine be given, rather than the barbiturates, with due regard for respiratory obstruction and with oxygen; and (4) that venesection be discontinued. Recent Army research and practice confirm these views.

Mechanical Means for the Treatment of War-gas Asphyxia

The mechanical treatment of asphyxial accidents in warfare suggests no deviation from the indications pointed out and repeatedly emphasized in this book. These indications should be familiar to the military office. Familiarity with commercial mechanical equipment which is now available to meet pathological indications will suggest methods of improvisation under emergency conditions. If we know what an apparatus is expected to do we can usually devise something to take its place.

At this point one suggestion cannot be too greatly emphasized: become familiar with the anatomy of the upper airway by developing the technique necessary to expose it. The prevention of asphyxial death in the unconscious flaccid patient turns upon the elimination of the death zone. For means of exposing the death zone, see page 79.

Preventive Treatment

The Gas Mask. At the outset let it be said that though there is no mystery associated with the gas mask used as a protection against chemical warfare, there are numerous features which set it apart from the usual pneumatological equipment. For example: It is to be worn by the normal, acutely conscious man engaged in the most laborious and exacting of all tasks—warfare. It must function with complete efficiency in the hands of hundreds of thousands of men entirely unfamiliar with the principles of gas therapy. It must be so constructed as to respond satisfactorily under the most difficult conditions imaginable. It must be capable of repair and upkeep many miles from the original source of supply.

As stated in "Training Manual No. 3205" (Copy #1), p. 5, the mask must (1) protect against all chemical warfare agents; (2) have a low breathing resistance; (3) be light in weight; (4) be comfortable; (5) be simple in design and easy to operate and repair; (6) not interfere greatly with vision; (7) be rugged enough to withstand field conditions; (8) be reasonably easy to manufacture in quantity; (9) not deteriorate in storage for at least several years; (10) have a service life in the field of at least several months; (11) be inexpensive; (12) be made of non-strategic materials as far as possible.

Many of these requirements are mutually opposed, for example, the requirements of maximum protection and light weight, since the amount of protection varies with the weight of chemicals used and the capacity of the filter. The greater the weight of chemicals and the larger the filter, the heavier the canister. Low breathing resistance depends upon a large surface area for the filter, but this increases the size and weight. A small, light canister would have less weight of chemicals with resultant lower protection, and a smaller filter area would cause greater breathing resistance. Therefore, the military mask is a compromise among the above twelve factors, of which the first three are the most important.

The foregoing considerations suggest that the mask cannot be improvised. Unlike pneumatological equipment operating under the eye of an experienced pneumatologist, the efficiency of the mask must be self-contained. Changes and improvements are constantly being made. Consequently, older models must be used simultaneously with the latest. This involves a vast amount of appropriate classification and checking. These difficulties have been met by an ingenious system of nomenclature which also serves to test the new model.

Since the present consideration has to do with the prevention of asphyxial death, the reader is referred to the War Department ("Training Manual 3205") for details dealing with maintenance and supply. The following matter is quoted directly from this manual. It provides a well though out, concise explanation of the construction and operation of a gas mask.

Service Gas Mask

General. (a) "*Definition:* The military gas mask is an apparatus designed to purify the air which the soldier breathes. It also protects his eyes and face when he is in an atmosphere contaminated with toxic or irritating gases, vapors, or smokes.

"(b) *Components:* The complete gas mask consists of three principal parts: the face-piece assembly, the canister, and the carrier. (1) The *face-piece assembly* generally consists of the face-piece, containing eye-pieces, an outlet valve, a hose connection or angletube, and with a head

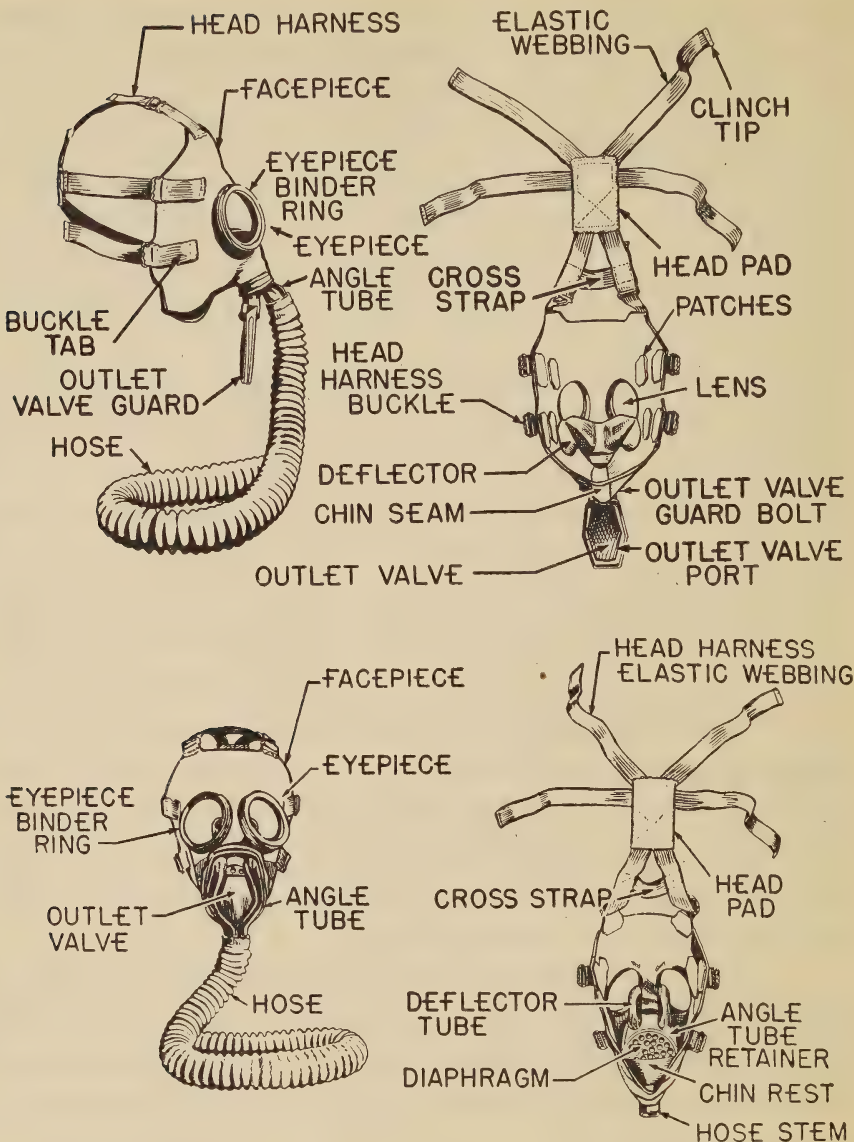


Fig. 161. Service gas mask. (From U. S. War Department's Technical Manual "The Gas Mask")

harness attached. When a connecting hose forms part of the gas mask, it is included in the face-piece assembly. (2) The *canister* has a hose connection which may or may not include an elbow nozzle, and also one or two inlet valves, depending on the type of canister. (3) The *carrier* is made of a canvas pouch or sack with various straps and buckles for slinging to the body of the wearer.

"(c) *Principles of operation:* (1) The gas mask is an air filter. The path of the air flow in the service gas mask is as shown in Figure 162. Air is drawn into the mask when the soldier inhales, and the mask is so constructed that this air must first pass through a canister containing a filtration system. This system comprises both mechanical and chemical filters, the former filtering out solid and liquid particles (smoke and dust), and the latter absorbing and neutralizing toxic and irritating gases and vapors. The air, after being purified by filtration, is drawn to the soldier's face and after being inhaled and exhaled is expelled from the mask through

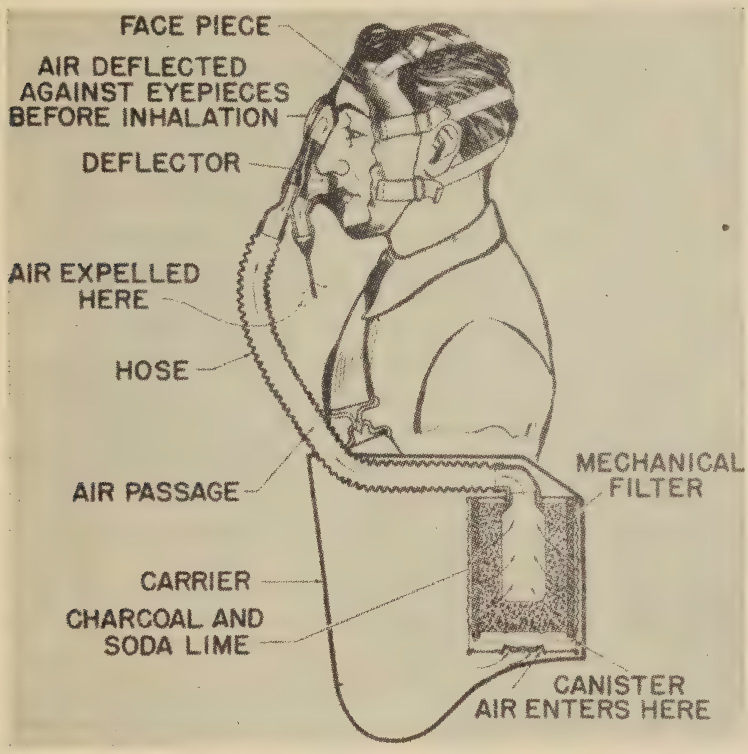


Fig. 162. Air flow in a gas mask. (From U. S. War Department's Technical Manual "The Gas Mask")

an outlet valve. (2) The face-piece fits snugly to the face so as to be gas-tight and is held in place by an elastic head harness. It is equipped with eye-pieces of safety glass or other transparent material, and a deflector which deflects incoming air against the eye-pieces so as to prevent condensation of moisture on the lenses. In addition to this method of reducing dimming of the eye-pieces, a soap-like substance, known as "anti-dim," is furnished with each mask, except the training mask, which, when spread as a thin layer over the inner surfaces of the eye-pieces, tends further to keep down eye-piece fogging. The face-piece is equipped with an outlet valve through which exhaled air is expelled and a metal tube connected to the canister by a corrugated rubber hose. (3) The canister is a metal cylinder or oblong-shaped box containing a filter and chemicals comprising the filtration system. Ordinarily, it is equipped with an inlet valve which permits the entrance of incoming air and a nozzle for the exit of purified air. This inlet valve prevents exhaled air from passing through the canister. The nozzle of the canister usually is connected to the face-piece by the corrugated rubber hose tube through which the purified air passes to the wearer's face. The face-piece, hose, and canister are contained in a canvas carrier.

"(d) *Limitations and use:* (1) The protection afforded by the gas mask is due primarily to the canister, the other components of the gas mask merely preventing air from entering the face-piece by any other route. There is at the present time no known toxic gas, vapor, or smoke against which adequate protection cannot be provided by means of a suitable canister. (2) Special canisters can be made up with filtration systems that will eliminate any one or all of the known toxic gases or dusts from the air breathed. No canister, however, affords protection indefinitely. The life of the canister is dependent upon the total quantity of gas which it is capable of filtering out of the air. Thus, for very high concentrations, the life of the canister may be short, while for low concentrations, it may be many weeks or months before the canister becomes so saturated with gas that it fails to protect. Normal field gas conditions are such that a canister does not break down suddenly. Except in case of mechanical damage, any failure of the filters occurs very gradually and can be noticed by the wearer as it happens. (3) The filtration system of the Army canister provides excellent protection against field concentraions of the chemical agents of warfare. Generally, field concentrations are relatively low. There are, however, toxic gases such as carbon monoxide and ammonia against which the Army canister will not protect, although adequate protection could be furnished if necessary. Neither will the Army canister nor any canister type of gas mask protect in an atmosphere containing an insufficient amount of oxygen to support life, nor, for a certainty, in cases where concentrations of the toxics are extremely high, such as might be encountered in inclosed spaces, in chemical manufacturing plants, storage tanks, and the like. The service canister will protect against high concentrations of gas for only a short period of time. It is therefore assumed that protection for longer periods is assured only if the concentration of gas does not exceed one per cent by volume. For such contingencies, which do not occur in the open air, hose masks or self-contained oxygen breathing apparatuses are required."

Description of Canisters and Canister Parts. (a) "*General:* The very earliest of military gas masks had no canister; instead, the air was passed through layers of treated cloth. These filters were improved by introducing a box of chemicals in place of the cloth. The safety and protection furnished by the gas mask was very greatly increased thereby because of the increase in surface and the greater quantity of the chemicals. Modern developments of the canister have been along the lines of producing greater effectiveness in particulate (or smoke) filtration, and in increasing the efficiency of the absorbents by means of various added chemicals.

"(b) *Parts:* The canister includes three main parts (Fig. 163): a container made of metal and provided with an air inlet and outlet; a filter which removes solid and liquid particles by mechanical filtration; and a chemical filling which removes gases by absorption, neutralization, or both.

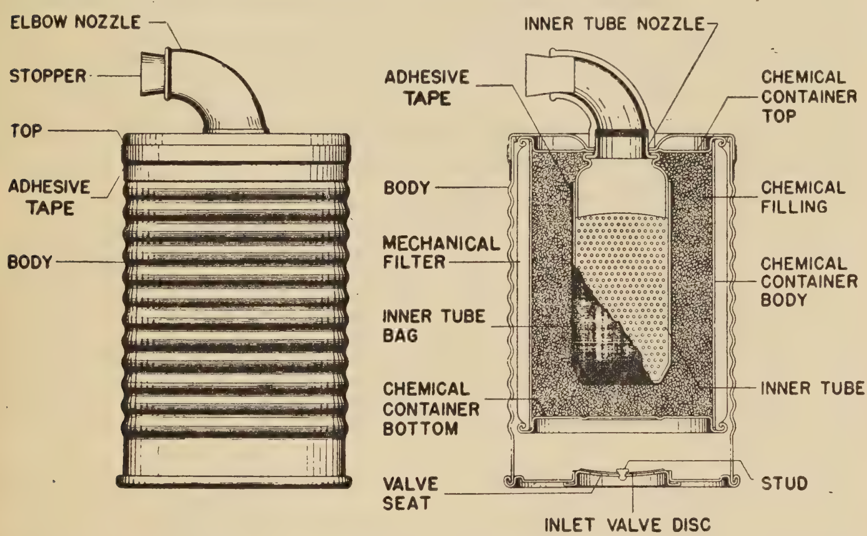


Fig. 163. Gas mask canister. (From U. S. War Department's Technical Manual "The Gas Mask")

"(1) *Container:* The container is the box in which the mechanical filter and chemicals are placed. It may be made of lithographed or enameled sheet iron, galvanized iron, aluminum, tin plate, or some other metal. Tin plate is most generally used.

"(2) *Mechanical Filter:* (a) The interstices of the mechanical filter must be fine enough to stop the extremely small solid and liquid particles in which irritant gases and smokes occur. Most of these particles cannot be seen under an ordinary high-power microscope since they are of minute (colloidal) dimensions. Even under the ultra-microscope they are visible only as points of reflected light. On the other hand, the filter must not be so dense as to cause a high resistance to the flow of air through it and thus cause increased breathing resistance. A filter which fulfills these requirements has been developed.

"(b) The materials used are fragile, and great care must be exercised in handling and repairing gas-mask canisters to the end that these filters are not damaged. For this reason the canister will not be opened for repairs in the field except by special authorization.

"(3) *Absorptive and neutralizing chemicals.*

(a) *General requirements.* The chemicals used for absorption must:

(1) Be very minutely porous so as to provide a huge surface area for adsorption in as small a space as possible.

(2) Not react with each other, nor corrode the container.

(3) Not be greatly affected by humidity.

(4) Remove the gases very rapidly since the air moves through the canister at such a velocity as to be in contact with the chemicals for only a fraction of a second.

(5) Have a high capacity, since the canister should last several months when in use.

(6) Be easily and cheaply manufactured from domestic raw materials.

(7) Not cause a high resistance to breathing.

(8) Resist crushing and abrasion.

"(b) *Activated charcoal.* (1) Of all the materials tested for the requirements listed above, the only one which approximately fulfills all the requirements is activated charcoal made from a very dense material and in the form of finely divided granules. During World War I, coconut shell was found to be the best raw material, but various nut shells, fruit seeds, etc., were also used. Since then methods of activation have been greatly improved, and now domestic raw materials can be used instead of coconut shells.

"(2) Charcoal is made from wood and wood-like materials which consist of cellulose and other components. In charcoal manufacture, the cellulose is heated and thus converted to carbon, water is given off, and other constituents are also evolved. The residue is called primary charcoal. If the primary charcoal is then subjected to treatment with heat and steam or to some superheated vapors or gases in the absence or in limited amounts of air, more of the constituents are driven off, leaving purer carbon. This activated charcoal is full of tiny spaces which were formerly occupied by some other constituents of the original material and now gives an extremely great surface to a small amount of charcoal.

"(3) Charcoal may also be processed in part by chemical action, as for example, by the peculiar charring action of zinc chloride, concentrated sulphuric acid, or phosphoric acid.

"(4) When air containing a gas or gases used in warfare is passed through activated charcoal, the molecules of the gas are held physically on the surface of the pores of the charcoal while the purified air flows through. This process is called adsorption, and may be considered comparable to the action of a magnet in attracting and holding iron filings on its surface. The heavier the gas, the more easily it is adsorbed.

"(5) One of the characteristics of chemical warfare agents is that any such substance must be capable of producing the desired effect in very small quantities or doses. Casualty-producing chemical warfare agents are therefore chosen for their high toxicity and relatively great effect in low or minute concentrations.

"(6) The chemicals used in the gas-mask canister, especially the charcoal, must therefore be able to seize and securely retain exceedingly small quantities of the chemical warfare agent. Also, these canister chemicals must be able to retain relatively large quantities of the toxic substances. Modern gas-mask charcoal is chosen for these specific qualities. However, the total weight of toxic substances adsorbed and the tenacity with which they are held will vary with the vapor density of the gas and the temperature. Thus it is easier to adsorb and retain mustard gas vapor, which has a high vapor density, than some other, lighter substance as, for example, hydrocyanic acid gas, or even phosgene. At very high temperatures, these lighter gases will be retained with difficulty, or if previously adsorbed by the charcoal, may slowly be released again.

"(c) *Removal of specific chemical agents.* The following table shows which components of the canister adsorb or neutralize the various known war gases:

Type of Gas	Neutralizing Component
Adamsite	Filter
Brombenzylcyanide	Charcoal
Chloracetophenone	Charcoal and filter
Chloracetophenone solution	Do.
Chlorine	Charcoal-soda lime
Chloropicrin	Charcoal
Cyanogen chloride	Do.
Diphosgene	Charcoal-soda lime
Diphenylchloroarsine	Filter
Ethyldichloroarsine	Charcoal-soda lime
Lewisite	Do.
Mustard gas	Charcoal
Phosgene	Charcoal-soda lime

"(d) *Neutralizing chemicals.* (1) There are several chemical substances which react with and neutralize those gases or vapors not readily adsorbed or not easily retained by the activated charcoal. These may act on the gas in any or all of several ways as, for example, by decomposition, oxidation, catalysis, or by direct chemical combination. The principal neutralizing chemical in the service, optical, and training canisters is soda lime, several different mixtures of which have been developed so that the exact composition may vary.

"(2) Those gases which are not easily or firmly held by the charcoal or are given off by it come into contact with the soda lime. This latter substance may combine directly with the gas, as in the case of phosgene, or may decompose the gas to form harmless substances, which in turn may combine with the sodium and calcium hydroxides in the mixture. The soda lime reacts more rapidly at higher temperatures. Thus there is an advantage in having a combination of charcoal and soda lime, in that while a rise in temperature decreases the adsorptive capacity of the charcoal, it increases the chemical activity of the soda lime, and the two effects tend to balance each other."

Limitations of service canister. The service canister does not protect against carbon monoxide or ammonia nor does it supply oxygen. Carbon monoxide and ammonia are excluded by the use of an all-purpose canister. Ammonia is restrained by silica gel. Carbon monoxide is restrained by the use of Hopcalite, a mixture of manganese dioxide and copper oxide.

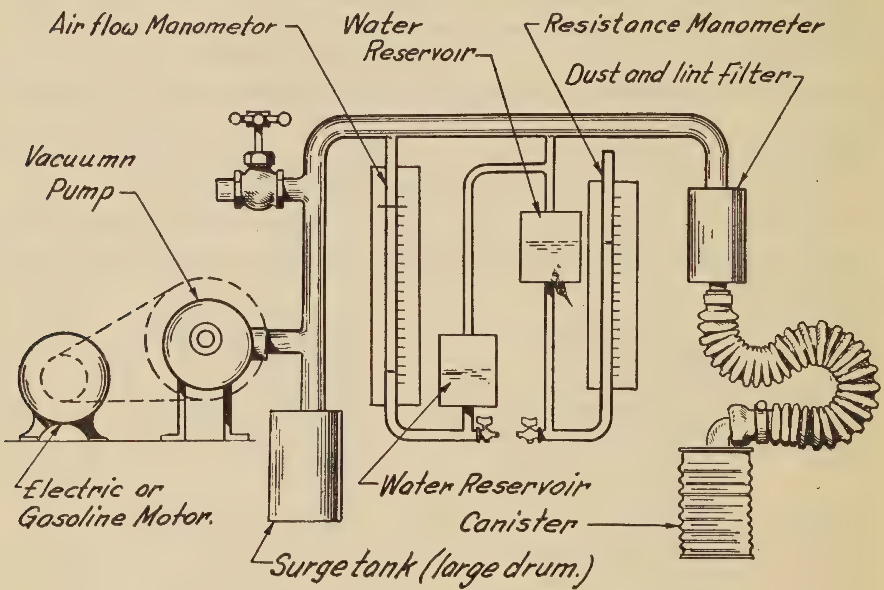


Fig. 164. Diagram of a breathing resistance indicator. (From U. S. War Department's Technical Manual "The Gas Mask")

The Hopcalite acts as a catalyst, uniting the oxygen in the air with the CO; it operates until moisture enters the mixture. A limit of two hours is placed on its use.

Of great interest to the pneumatologist is the negative and positive pressure created against the respiration as the result of breathing through the mask.

Canisters are checked for resistance by equipment shown in Fig. 164. The check is made upon the basis of the maximum air volume breathed by a man doing hard labor (32 liters per minute). According to the type of canister employed, the resistance varies between a minimum of 45 and a maximum of 92 mm of water, or a minimum of 35 and a maximum of 77 mm of water (divided by 13 for mercury).

Masks and respirators are supplied for the following purposes:

Optical gas mask: designed for use with optical instruments, range-finders, gun-sights, etc.

Aviator's gas mask: designed especially for the use of oxygen in aviation.

Dust respirators: made especially for drivers of vehicles.

Paint respirators: to stop pigment and liquid particles from being inhaled.

Hose mask: for atmospheres low in oxygen.

Non-combatant mask: for civilian employees.

For details concerning these various types of masks the reader is again referred to "Training Manual No. 3205."

The foregoing references to chemical gas poisoning have to do with its employment in military operations. It is to be noted that nothing has been said of the problem of creating the conditions for which protection is required, of preparation for the gas attack. As stated by Zanetti, there has been a great deal of irresponsible sensationalism regarding gas warfare. As a matter of fact, the requirements for a successful attack are exactingly severe. While an infection by a bacillus may radiate from a tiny focus and by growth and multiplication create enormous havoc, gas is inert, and must be delivered in full force.*

Two factors influence the effects of a gas attack: concentration and time of exposure. Chlorine, for example, is dangerous in concentrations of 1 to 10,000 parts per million over a period of two hours; phosgene in concentrations of 1–50,000 parts is equally dangerous. The velocity of the wind and its direction, as well as the temperature, influence the success of the gas attack. Extreme cold reduces the effect. Extreme heat, by causing convection currents of air from the earth, tends to dilute the gas. A gas wave (mustard) requires 65 lbs. per yard, or 60 tons for a mile. The British in 1917 released 160 tons from 5000 cylinders to gas an area of only about 230 acres.

Mustard gas stays where it is placed for a long time. Areas or important roads may be so treated and rendered useless. This is called "denying terrain." It is said that 12,000 tons of mustard gas was used by the Allies and Germany in World War I. Of the 350,000 casualities which resulted, 9,000 died. Compare this to the comment ascribed to the Duke of Wellington, who said, "On the field of battle it takes one ton of lead to kill a man."

If London or New York City were subjected to a gas attack, only small areas could be gassed successfully, and this at great hazard and expense to the enemy. Planes are required to fly low for such attacks. The World War I average of gas casualties from mustard gas precisely placed was 29 per ton. Civilian casualties might well be expected to be less. James

* Zanetti, J. E., *Independent Jour.*, Dec. 13, 1935.

Kendall, quoting Davidson Pratt's "The Truth about War Gases" makes the following statement, "Those who may feel uneasy on this score will, it is hoped, find confidence in the fact that during the great war French and Flemish peasants living on the forward area came unscathed through big gas attacks by going into their houses, closing the doors—the windows were always closed in any case—and remaining there until the action was over."*

* R. I. N. Greaves, "Production of blood derivatives to meet war requirements in Great Britain", *J. Am. Med. Assn.*, **124,** 76 (1944).

Chapter 23

Asphyxial Accidents in the United States Navy

Much interest has been exhibited by the Medical Department of the United States Navy in problems of asphyxia. The author made the acquaintance of Admiral Rossiter during a lecture on resuscitation at the Brooklyn Navy Yard before the Admiral became Surgeon-General. On Feb. 20, 1934, Captain E. W. Brown, representing General Rossiter, presented a paper before the Society for the Prevention of Asphyxial Death, entitled "Problems of Respiration in Naval Medicine." On May 24, 1939, the morning following the *Squalus* accident, the S.P.A.D. contacted General McIntire and offered resuscitation equipment for the relief of submerged victims. This equipment, in charge of Lieut.-Commander A. R. Behnke, remained on board the S.S. *Falcon* throughout the period of rescue.

On June 28, 1939, General Ross T. McIntire did the Society the honor of personally presenting a paper on Asphyxia on the occasion of the opening of the Division of Pneumatology of the Medical Department of the New York World's Fair.

The matter which follows is a condensation of the contributions of Surgeon-General McIntire, Captain Brown, and Lieut.-Commander Behnke, supported by the work of Fraser, Schilling and Johnson. The material has been read by the Medical Department of the United States Navy.

Asphyxial accidents under the jurisdiction of the U. S. Navy may be grouped according to environmental circumstances in which they occur. They take place ashore, afloat, or in connection with submarine operations.

Accidents Occurring Ashore

These accidents are identical with those occurring in military operations and in civil life. Base hospitals suffering bombardment present the type of asphyxial casualties described in Chapter 24. Civilian causes of asphyxia are noted on Frontispiece.

Accidents Occurring at Anchor

While a ship is peacefully at anchor or wharfed, unexpected asphyxial accidents peculiar to the sea are likely to occur.

From Decomposing Grain or Fruit. For instance, Brooke describes the case of the S.S. *Jacob* containing a cargo of oranges in her hold for a prolonged period, in which 2 men were killed; the air contained 22.5 per cent carbon dioxide and 1.8 per cent oxygen. It is recorded in the British "History of the World War" that 3 men perished in the hold of the S.S. *Arabic* in the process of removing a cargo of sodden and decomposing grain. This ship had been wrecked and sunk but was subsequently salvaged. It was concluded that oxygen deficiency was the cause, induced by absorption of oxygen by the grain.

In Closed, Unventilated Spaces. Typical of these hazards is the ship's "blister" and the submarine pontoon. The so-called blister is a characteristic of the modern battleship developed since World War I and designed for torpedo defense. It consists of a false hull overlying the original hull, reinforced by transverse and longitudinal frames and conforming to the longitudinal lines of the ship. The maximum depth of the blister is about 40 feet from top to bottom. The blister is subdivided into sections of two compartments each on both sides of the ship, with manholes provided for communication. There are a total of 80 blister compartments. The interior is painted and kept hermetically closed to insure watertightness, except for inspection and possibly renovation at 6-month intervals.

A description of an accident in a blister compartment on the U.S.S. *New York* in 1929 follows: "The manhole cover was removed and an officer and three men entered without the precaution of preliminary ventilation. Just as the officer entered the lower compartment of the blister he fell to the bottom apparently overcome. The three men with him immediately descended the ladder to his assistance and all three collapsed. Two additional persons reaching the bottom were also incapacitated and two fatalities resulted, *i.e.*, the officer and one man. The symptoms of those recovering were diagnosed as due to carbon monoxide. The blood of the two fatalities was positive for carboxyhemoglobin."

A disaster incident to entering a compartment of a submarine pontoon in December, 1931 at a naval station is noted: the pontoon is a metal drum designed for the raising of sunken submarines: the compartment was about 12 feet in height and of 1540 cubic foot capacity. It was ventilated for a very brief period, but not adequately, as is required by naval regulation. Three men descended and were immediately overcome. Three additional men were incapacitated in attempting to rescue their co-workers. The first three men were dead when finally removed. An analysis of the air of the compartment taken three days later was reported as showing 0.1 per cent carbon monoxide, the pontoon having been closed in the interim. The percentage was undoubtedly greater when the pontoon was first opened 4 days before. A concentration of 0.1 per cent would not have induced immediate unconsciousness in air otherwise normal. It appears more probable that oxygen deficiency was the direct cause.

The Hazard of Paint Applied to Unventilated Compartments. Belli* by experiments in the Italian Navy showed that the oxygen in a ship's

* All references in this chapter, see bibliography.

double-bottom can be reduced to 3 per cent by the use of red lead paint. Gardner pointed out that when linseed oil or similar drying oils are spread in thin layers, the absorption of oxygen which takes place on drying is accompanied by the evolution of carbon dioxide and organic substances, carbon monoxide also being evolved in small amounts. However, Klein questioned the validity of the technique by which these results were obtained. King studied the subject and confirmed the finding of Gardner that carbon monoxide was evolved in relatively small amounts in the drying of paint, but the absorption of oxygen from the air was not considered.

Newington reported, as a result of extensive experiments, that carbon monoxide was evolved in the process of drying of linseed oil, boiled or unboiled, and whether alone or admixed in an oxide of iron paint. Gas in a drum which had contained boiled linseed oil and remained sealed for 5 months contained 0.21 per cent oxygen, 3.28 per cent carbon dioxide and 0.35 per cent carbon monoxide. There was actually more carbon monoxide than oxygen in the atmosphere. The air of a blister of a British battleship sealed for 2 years showed 1.3 per cent oxygen and .03 per cent carbon monoxide. Newington concluded that with an excess of linseed oil present, as must always be the case in a sealed compartment coated internally with paint, the maximum content of carbon monoxide to be expected would be of the order of 0.3 to 0.4 per cent. He also pointed out that the large deficiency of oxygen produced by absorption of the linseed oil, together with the appreciable concentration of carbon monoxide formed from the linseed oil during drying, emphasizes the necessity for insisting on the observance of effective measures of ventilation before the entry of personnel into painted confined spaces. Corroborative evidence of the formation of carbon monoxide in the study of Newington was afforded by a biological study with small animals conducted concurrently by Dudding, Dudley and Frederick.

Before entering an unventilated compartment the following precautions should be taken:

(1) The space should be tested for oxygen content with a safety lamp.

(2) It should be thoroughly ventilated in all parts before entering.

(3) A lifeline should be attached to a person entering.

(4) An oxygen apparatus should be worn.

(5) A rust-proof coating which does not absorb oxygen should be used on all surfaces in place of ordinary linseed-oil paint.

Fire Hazard Aboard Ship. As has been previously stated, oxygen deficiency may occur as a result of fire in confined spaces in a ship, and the presence of carbon monoxide and carbon dioxide adds greatly to this danger. It is found in badly ventilated compartments such as in the holds and bunkers of commercial ships.

Extinguishers. A rather common cause of asphyxia is the fumes from fire-extinguishing apparatus. Apart from the risk of encountering carbon monoxide and possibly nitrous gases in fighting fires in confined spaces in wartime, an additional danger has arisen from the toxic gases evolved by chemicals, such as carbon tetrachloride when it is sprayed on fire. The chief decomposition products are phosgene, chlorine, and hydrochloric acid. Attention should be called at this point to the risk involved in using carbon tetrachloride as an ordinary cleaning fluid. Many accidents have occurred from its excessive use and from allowing it to be exposed to heated surfaces.*

The Hazard of Fumigation. The hazard of hydrocyanic acid gas is confronted whenever a ship is fumigated. Various methods have been used to determine the presence of this gas, which has a tendency to linger in the ship's hold or in closed chambers, particularly in warm, damp weather Canary birds and guinea pigs were first used to test the presence of the gas. These have been supplanted by a special test paper, which is normally a copper color. Upon exposure to hydrocyanic gas it rapidly turns pink. If this change is slow, the concentration of the gas is low; if rapid, the concentration is high. The presence of hydrocyanic acid gas may be detected by its odor. It therefore does not present the same hazard as CO_2 and low oxygen. Hydrocyanic acid gas clings to bedding and mattresses in quantities which may prove lethal. Care must be exercised in thoroughly ventilating such materials.

Accidents Occurring on the High Seas

In addition to the asphyxial hazards which may occur while at anchor there are (1) the danger of carbon monoxide and nitrous fumes in gun-fire and from high explosives, and (2) the hazard from gasoline engines from planes aboard airplane carriers.

Carbon Monoxide and Nitrous Fumes from Explosives. *Poisoning by Carbon Monoxide from Gunfire.* It is of course a familiar fact that gas poisoning is frequently associated with gunpowder accidents. The great bulk of gassed cases in the British Navy during World War I were due to carbon monoxide and nitrous fumes. Chemical warfare agents were not employed by sea forces in that conflict. In the British Army very few of such cases occurred, and these were found mainly in connection with mines the subsequent formation of gas pockets in the surrounding ground. The comparative rarity of such cases following powder explosions in the Army is explained by the fact that the explosions usually occur in the open, whereas in the Navy the gases evolved are confined by the decks or turrets.

It has been calculated that a 15-inch gun with its charge of 400 pounds of smokeless powder will give off 2500 cubic feet of carbon monoxide each time it is fired. If for any reason any portion of the gases are permitted to blow back into the turret in which the gun is mounted the carbon monoxide will naturally produce symptoms. This is normally prevented in the U. S. Navy by means of an automatic air blast which instantly clears the barrel of all traces of explosion gases.

* See Henderson, V., and Haggard, H. W., "Noxious Gases," pp. 137 and 206, New York, Reinhold Publishing Corp., 1943.

The toxic hazard of carbon monoxide must, however, be considered as a possibility, as shown by an accident aboard the H.M.S. *Iron Duke* in 1930. Three men were overcome by fumes blown back into a turret during the firing into a headwind of 6-pounder sub-caliber guns after approximately 80 rounds. These cases, all of which recovered, were definitely diagnosed as carbon monoxide poisoning. As the men were overcome within 10 minutes, the concentration of carbon monoxide must have been high, but it was probably pocketed in the turret, since the remaining men escaped. It would probably have required 0.5 per cent to account for the collapse of these men in view of their strenuous muscular activity during the 10-minute period of exposure.

Similar cases of gassing have been reported on other British ships when carrying out sub-caliber firing into a headwind. With this type of gun the air blast was not automatic on beginning to open the breech, in contrast to the full-caliber rifle, the result being a tendency at times for some of the combustion products to blow back into the turret before the operation of the clearing air blast.

Poisoning by Nitrous Fumes from Gun-fire. When detonation of an explosive, such as Navy smokeless powder, is incomplete, or when burning is produced from some cause, nitrous gases as well as carbon monoxide are generated, the amount of each varying with the nature of the combustion. The gas danger, therefore, from normal detonation does not compare in gravity with the hazard resulting from incomplete detonation or from the burning of an explosive charge. The nitrous gases consist of a mixture of the oxides of nitrogen. NO is formed first, and is rapidly oxidized to NO_2, which is reddish brown in color; this in turn rapidly changes to nitrous acid. The toxic effects on inhalation are probably due to the action of a mixture of nitrous and nitric acids.

Under conditions of the ignition of a powder charge in a confined space, such as a turret or compartment, the gases and the heat of the partial explosion will drive the normal atmosphere out, and the resulting reduction in oxygen will add to the toxic effect of the gases by causing increased respiration. The toxic action of the gases will also be increased by their high temperature. To personnel nearby, the force of the partial explosion and the heat evolved will be the primary factors in the injury, although among these asphyxia or lung injuries may also occur. At a greater distance from the point of explosion the factor of gas poisoning becomes more prominent. The possibility of the inhalation of flames or overheated inert gases must also be considered and, of course, the shock effect of extensive burns.

Fairlie has reported a study of powder-gas casualties in World War I. Eleven casualties, two of which were fatal, resulted from nitrous fumes in the Battle of Jutland. Seventeen cases, of which 14 expired, occurred in 1916 aboard H.M.S. *Russell* as a result of fire and explosion incident to contact with mines. Fifty-six cases were reported from H.M.S. *Britannia*, 12 of which failed to recover, following torpedoing of the ship and a subsequent fire in one of the magazines. The clinical reports of these disasters emphasize the period of latency, which is an outstanding aspect of poisoning with nitrous fumes as it is after poisoning with phosgene, a chemical warfare gas. The initial symptoms of irritation may be comparatively slight and pass off readily; they are followed by severe or even fatal pulmonary edema after a variable interval. Many of the cases from the *Britannia* were landed apparently in normal condition, which persisted for many hours before serious signs developed; the latent period of these cases averaged 10 to 12 hours.

The *Mississippi* disaster in our Navy in 1924 will be recalled by many. This resulted from the accidental ignition and burning of a powder charge in a turret mounted with 14-inch guns during target practice. Forty-eight men were killed, only two persons in the turret surviving. The head and hands of practically all the dead showed signs of burns, but in the majority of instances not sufficient to account for death. Evidence of asphyxiation was present, although no blood examinations for carboxyhemoglobin were conducted.

About four months later, a similar accident with burning powder occurred aboard the U.S.S. *Trenton* involving the forward twin gun, with a total loss of 14 men, only 5 of those exposed surviving. The blood was examined for carboxyhemoglobin in all these cases with negative results; with reference to the survivors they did not appear consistent, although the period of exposure to the fumes was short. Death was generally ascribed to extensive burns and shock. Shortly after this disaster Knight and Walton undertook a study at Edgewood Arsenal to furnish data on the effects of burning smokeless powder in a semi-closed space simulating a turret. The tests were conducted in a horizontal steel cylindrical tank having a capacity of 98 cubic feet. The burning took place in two stages: first the combustion of the powder itself, resulting in a temperature of about 750°C. and a heavy blast of flame issuing from the opening of the chamber; and secondly, after the chamber had cooled sufficiently to draw in fresh air, burning of the combustible gases present. At the end of 2 minutes 2.2 per cent NO_2, 16.8 per cent carbon dioxide, 8.4 per cent carbon monoxide and 3.4 per cent oxygen were found by analysis.

Guinea pigs were employed as animal subjects. Although the high heat produced a large number of casualties, the tests with animals protected from the flames by special arrangement indicated that under certain conditions it may be possible to survive the heat effect of the blast. The authors point out that even if the marked lack of oxygen, in some exposures persisting for 5 minutes, did not cause death from asphyxia, it would cause an enormous increase in the breathing rate, which would be still further accelerated by the high carbon dioxide and the high temperature. The casualties among the animals from the gas standpoint were as follows:

(1) Those killed immediately by carbon monoxide and lack of oxygen.

(2) Those brought out unconscious from carbon monoxide and revived by fresh air or oxygen treatment.

(3) Those brought out unconscious from carbon monoxide and revived by fresh air or oxygen only to die later of pulmonary edema from nitrous fumes.

(4) Those brought out seemingly little affected at the time but dying later of pulmonary edema due to nitrous fumes. (See pages 224, 295.)

Carbon Monoxide in Relation to Aircraft. The possibility of carbon monoxide as a hazard from the exhaust gas of the gasoline motor of the airplane in the U.S. Navy was first suggested in connection with the death of Captain A. H. Page, U.S. Marine Corps, as a result of a crash at the National Air Races in Chicago in 1930. Captain Page was an outstanding flyer of the Marine Corps and this loss of control of his plane in view of the attendant circumstances could not be accounted for at the time. The finding of carbon monoxide in the blood was later reported, although it was of so low a percentage saturation as to render this factor of doubtful significance in the crash. As a result, however, the question of the possible hazard of carbon monoxide in relation to aircraft was raised and exhaustively studied by the Bureau of Medicine and Surgery in conjunction with the Bureau of Aeronautics. The findings of this study have been presented by White.

The first series of tests gave positive results for carbon monoxide in the blood of pilots and passengers in three types of planes, *i.e.*, observation, bombing, and amphibian—10 per cent in the observation and bombing planes and 15 per cent in the amphibian. The tests were repeated, with similar results. Analyses of the air were then conducted in the cockpits of various types of planes by means of a new type of carbon-monoxide indicator developed for this purpose. As typical results the following are taken: OL-8 plane, 0.01 per cent in rear cockpit but below breathing level; 02C-1 plane, 0.02 per cent in both cockpits; O2C-1 plane, 0.02 per cent in front and 0.01 per cent in rear cockpit; O3U-1 plane, 0.03 per cent in rear cockpit; OSu-2 plane, 0.02 per cent in front and 0.04 per cent in rear cockpit. On the other hand, tests with many of the planes yielded no evidence of carbon monoxide.

It was therefore concluded that in some types of planes, depending upon the arrangement of exhaust leads, fuselage, etc., carbon monoxide is carried back to the cockpits in sufficient concentration to result in as much as 15 per cent saturation of the blood of the pilot and other occupants of the plane. Although the highest saturation of blood obtained during the study would not be sufficient to render a person unconscious or to be a direct cause of a crash, the absorption of even minute amounts of carbon monoxide is highly undesirable, due to the possible adverse effect on the efficiency of the pilot. It was evident that a definite hazard existed in this regard. Immediate steps were taken to eliminate the carbon monoxide by a simple and inexpensive modification of the exhaust leads of those planes which yielded positive tests for the gas. In some instances one modification was sufficient; but in other cases several modifications were essential before the carbon monoxide was entirely eliminated. As a result of this research the specifications now prescribe that all new airplanes in the future shall be tested at the factory for carbon monoxide; if present, it must be eliminated before final acceptance by the Navy.

Accidents Occurring in Submarine Operations and Deep-Sea Diving

Submarine Ventilation. The modern submarine naturally presents many difficult problems of physiological hygiene not encountered on surface ships. The greater number of these have now been satisfactorily solved, but there are certain aspects of the air conditions, particularly in the tropics, which have not yet been controlled.

The submarine is propelled on the surface by a Diesel engine and when submerged, by an electric motor. The electric current is furnished by storage batteries of which there are 120 to each vessel. Submersion is accomplished by first filling the ballast tanks to the point of slight positive buoyancy, followed by the action of the diving rudders; emersion by emptying these tanks by means of compressed air. A compressed-air plant is therefore part of the equipment of the vessel. The submarine is divided into compartments, the number depending upon the type.

The designer of a submarine is confronted with certain difficulties. Within the weight alloted he must include machinery for surface propulsion. *i.e.*, Diesel engines and the necessary supply of fuel; machinery for submerged propulsion, *i.e.*, electric motor and storage batteries; there must be a heavy hull to withstand the crushing pressure of the sea when submerged and a vast amount of special piping, valves and high-pressure air tanks for the sole purpose of enabling the boat to submerge. When he has finished providing these absolute essentials, there is little weight or space left for anything else; the small remaining margin is distributed to the best advantage between safety features and measures for the habitability of the crew, which varies from 10 to 60 men. The crew must be carefully chosen to eliminate men sensitive to oxygen deprivation and to pressure effects. Johnson states that only 11 out of 100 are accepted and that 25 per cent of these are later eliminated.*

Air Purification in Submarines. Purification of the air is required for two reasons: (1) to maintain the military efficiency of the personnel submerged, and (2) to maintain respiration while awaiting rescue after a disaster. Owing to definite limitations of weight and space, air purifying equipment must be reduced to the minimum. It is therefore impracticable to maintain the atmosphere of a submarine at the normal atmospheric concentration of 20.9 per cent oxygen and 3 per cent carbon dioxide. As a result of experimental work, a standard of 3 per cent is prescribed as

* Johnson, Capt. Lucius, *Marine Corps Med. J.*, **40,** 1065.

the upper limit of carbon dioxide and 17 per cent as the lower limit of oxygen. Anoxemia becomes evident at 15 per cent oxygen concentration. The physical efficiency of the personnel is not materially impaired when submerged under these conditions, although the depth of respiration is considerably increased with muscular work. Carbon dioxide is removed by soda lime of the type employed in pneumonia oxygen chambers, and oxygen tents, the chemical being contained in canisters connected to the ventilation system. One-half pound of soda lime is required per man per hour. Soda lime presents a definite disadvantage in that the absorptive capacity is ineffective if the air temperature falls to approximately 50°F. Oxygen is supplied from cylinders, one being carried in each compartment. Average requirements of oxygen are 250 cc. per minute, or 750 cubic feet per hour.

In submarines of the S-class, which includes the majority of those in the U.S. Navy, the carbon dioxide will ordinarily not reach 3 per cent until after 17 hours, and in the larger cruiser class, 19 hours. Air purification therefore is not used unless longer periods of submersion are contemplated. Under ordinary conditions the period of submersion is so short that purification is not required. It is, of course, a comparatively simple matter to calculate the time of submersion required to reach the limiting figure of 3 per cent carbon dioxide on the basis of the average volume of carbon dioxide exhaled per man per hour and the capacity of the air contained in the vessel.

The standard allowance of air-purifying equipment for submarines is sufficient for 72 hours. The maximum period of submersion thus far attained in the Navy is 96 hours, but additional materials were carried. In a disaster such as that of the S-4, which was sunk off Provincetown in 1927, the electric power is practically always out of commission and forced ventilation therefore stops. Under these conditions, with the crew trapped in a compartment, it is directed that the soda lime be spread in thin layers over mattress covers, relying on natural currents of air for contact with the chemical. Unpublished naval research has shown that absorption is nearly as efficient under these conditions as in canisters with forced ventilation. The early effects of CO_2 are excess drowsiness and headache. This is accentuated by cold.

The question arises as to the lethal concentration of carbon dioxide for men in a submarine compartment without means of air renewal. In the S-4 disaster six persons were trapped in the torpedo compartment without provision for carbon-dioxide removal. The last signal received by divers from the survivors was 75 hours after the collision. It is estimated from the volume of the compartment and the approximate hourly output of carbon dioxide at rest for six men that a concentration of approximately 9 per cent was reached. Life at least existed under these conditions. Facilities are now in operation to completely condition submarine atmosphere. The air is deodorized and decontaminated, the temperature is reduced to 75°F. wet-bulb, and excess moisture is removed.

Noxious and Explosive Gases in Submarines. The accidental access of salt water to the storage batteries of a submarine causes evolution of chlorine gas. Casualties of this nature have occurred on our submarines. A special type of gas mask designed for the specific purpose of protecting against chlorine is provided for all personnel.

A number of serious epidemics of arsenic poisoning occurred in British, French, and Italian submarines during World War I. Dudley has reported some of these outbreaks in British submarines. The toxic agent was the gas arsine, which was traced to the presence of arsenic as an impurity in the plates of the storage batteries; the gas was evolved through reaction with the free hydrogen in the battery. It has been detected in the air of certain American submarines, but never in sufficient concentration to induce symptoms. The protective measure consisted in the replacement of contaminated batteries by units free from arsenic.

A number of hydrogen explosions due to deficient ventilation of the storage batteries under conditions of charging have occurred in submarines. Under these circumstances, hydrogen had accumulated to an explosive concentration, and ignited from spark formation. Adequate ventilation has practically eliminated such accidents.

The Rescue of Submarine Victims by Surface Craft

A few physical and biophysical facts are to be noted in connection with under-water operations.

Physical considerations. At sea level the pressure of the atmosphere is 14.7 pounds per square inch. As a body submerges in salt water the pressure upon it increases at the rate of 0.44 pound per foot of depth, to which the atmospheric pressure at sea level must be added. At 33 feet the pressure is therefore $14.7 + (33 \times 0.44) = 29.2$ pounds, or approximately two atmospheres; at 50 feet approximately $2\frac{1}{2}$ atmospheres; at 240 feet approximately eight atmospheres; and at 560 feet seventeen atmospheres. It has been stated that human protoplasm can tolerate a (compensated) pressure of sixteen atmospheres (Behnke).

The volume of air under pressure varies inversely as the pressure. The volume of air in a given space, therefore, at a depth of 33 feet is approximately one-half that at the surface. Compressed gases maintain their partial pressures (proportions), *i.e.*, nitrogen 79.1, oxygen 20.9, or a ratio of 4 to 1. Both nitrogen and oxygen are soluble in fluids; the solubility of nitrogen is .01 per cent. The molecular weight of nitrogen is 28, of oxygen 32. Air weighs 76.38 lbs. per 1000 cubic feet.

The use of helium was suggested in 1919. The molecular weight of helium is 4; its density is therefore $\frac{1}{7}$ that of nitrogen and $\frac{1}{8}$ that of oxygen. A thousand cubic feet of helium weighs only 10.54 lbs. Its solubility is only .008 per cent. Helium is entirely odorless, tasteless, colorless and chemically inert. It is twice as easy to move as air. Not only is it much less soluble in fluids (.008 against .01), but because of its low molecular weight it diffuses much more rapidly, and can be compressed and decompressed more quickly. Helium can be liquefied or solidified.

Biophysical considerations. At sea level man's internal and external pressure environment is balanced at 14.7 pounds per square inch. If the chest cavity were a perfect vacuum, the pressure upon it would be approximately two tons (Fraser).

Variations in the pressure of the external gaseous environment are rapidly balanced by internal adjustment, first, in the open cavities—middle ear, sinuses, intestines, etc.—secondly, in the tissues. These variations are referred to as pressure and decompression. Pressure is produced rapidly during submersion, but decompression is produced slowly during emersion.

Sudden and great increases of pressure unevenly applied, as when a diver in an undecompressed uniform falls overboard, may result in fatal crushing injuries. A diver in a diving suit with an internal atmospheric pressure of one atmosphere who falls overboard to a depth of 33 feet will be compressed to one-half his normal volume. It has been necessary to dig such a man out of his diver's helmet. The naked diver exposed to a much greater depth, uniformly compressed, suffers no injury. On the other hand, a diver who drops suddenly from a depth of 250 feet to 283 feet will experience greatly reduced pressure effects owing to the fact that his total volume has been reduced one-eighth instead of one-half.

It is obvious, therefore, that in order to escape the crushing effects of deep water pressure, the man to be so exposed must be adjusted to a pressure corresponding to the depth at which he is to begin his emersion. Such pressure compensation is developed as a preliminary to escape (from a sunken submarine) by placing the man about to be released in a watertight compartment filled with air. The sea water is then allowed to enter slowly until the air pressure which the man respires is equal to the water pressure to which he is to be exposed. At the maximum depth of 560 feet the air would be reduced to $\frac{1}{16}$ of its volume, if pressure was begun at one atmosphere. If begun at two atmospheres it would be reduced to $\frac{1}{8}$ its original volume. When compression has been accomplished and internal and external pressure balance, the man is free to leave the submarine for surfacing. Man's body is 80 per cent water, 15 per cent fat and 5 per cent solids. Gases are soluble in water and in fat.

The solubility of a substance in fat is a measure of its narcotic power (Myer and Overton hypothesis). As has been noted, the solubility of nitrogen is .01 per cent and that of helium is only .008 per cent. Therefore, the solubility coefficient of nitrogen is 5.2 to 1, and that of helium is 1.7 to 1 (Behnke).

The effects of compression depend upon its degree, its duration and the rate of its release (Schilling). Tables in which the required time for decompression appear have been prepared.

Returning to the man about to emerge from a sunken submarine, the

PNEUMATOLOGICAL (GAS THERAPY) CONSIDERATIONS IN THE UNITED STATES NAVY

Altitude (ft.)	*Pressure (mm. Hg)*	*Levels*	*Remarks*		
50,000	87.3	Extreme upper limit of ascent.	Partial pressure of CO_2 and H_2O occupy all space. $(39 + 47 = 86)$ O_2 crowded out. (Puncture-proof pressure cabin required.)		
40,000	140.7		54 mm. $(140 - 86 = 54)$ available for O_2 and N. Pre-flight washing out of N by O_2 for 5 hours allows 2 hours' safe flight.		
37,000	158.0	(Never more than 38,000 ft.)	3 hours pre-flight O_2 allows 2 hours' flight; 5 hours allows 6 hours' flight. (Behnke)		
30,000	225.6	Limit for ordinary flying.	Pressure cabin plane the answer to most problems.		
25,000	281.9	Hazardous level with O_2. (Armstrong)			
20,000	349.1	Silent bubble level. (Behnke)			
15,000	428.8	Ordinary extremes for flying without oxygen.			
0	760.0	ON LAND OR ABOARD SHIP	O_2 or O_2-He. Recreation room for flyers.		
Depth (ft.)	*Pressure lbs./sq. in.*			*Nitrogen*	*Helium*
50	22 (+14.7)	Helium decompression begins.	Mol. wt.	28	4
100	44 (+14.7)	Nitrogen narcosis begins.	Sol. per ccm.	.01	.008
			Decompression	slow	rapid
150	66 (+14.7)	Helium-oxygen delivered.	DIVER—He-O_2 supplied; CO_2 absorption practiced; heated garments used. Air line takes 2000 lbs pressure. Breast line 1300 lbs traction.		
200	88 (+14.7)	Limit for naked diver.			
240		SQUALUS — BOTTOM N narcosis increased by CO_2	MOMSON LUNG—N and O_2 (air) pressure in escape chamber at 8 atm. N decompressed by O_2 in lung at knot levels; decompression completed on shipboard. SUBMARINE—O.K. if found in 72 hours. Must be air conditioned for fighting efficiency and to save life until rescue. Sufficient O_2 stored for 72 hours if quiet crew, for 15 hours if active. Gas hazards: Cl from sea water in batteries, free Hydrogen from batteries, Arsenous acid from batteries. Excessive CO_2, more than 3% or deficient O_2 less than 17%.		
300	132 (+14.7)				
440	176 (+14.7)	Limit for uniformed diver. (Behnke)			
500	220 (+14.7)	DIVING BELL (p. 328)			

gas which has been compressed in his tissues will require removal or decompression as he emerges from the depths. In order that decompression may be gradual, he is required to stop at a given interval at depths indicated on his escape guide line.* At each stop he runs pure oxygen into his "lung" in order to eliminate the decompressed nitrogen or helium, as the case may be. This so-called "stage decompression" was developed by Haldane (Fraser). Should the exhaust valve in the "lung" fail to operate and oxygen distend it, the man may be blown to the surface. The compressed gases in his tissues, unable to escape slowly from his lungs, will form bubbles* in his muscles, joints [complicated by traumatic emphysema (Johnson)] or in his blood vessels, producing circulatory emboli (aeroembolism) which may induce severe asphyxia or cardiac failure. This is also known as bends, caisson disease, aeropathy, and diver's paralysis and was first described by Paul Bert in 1860.†

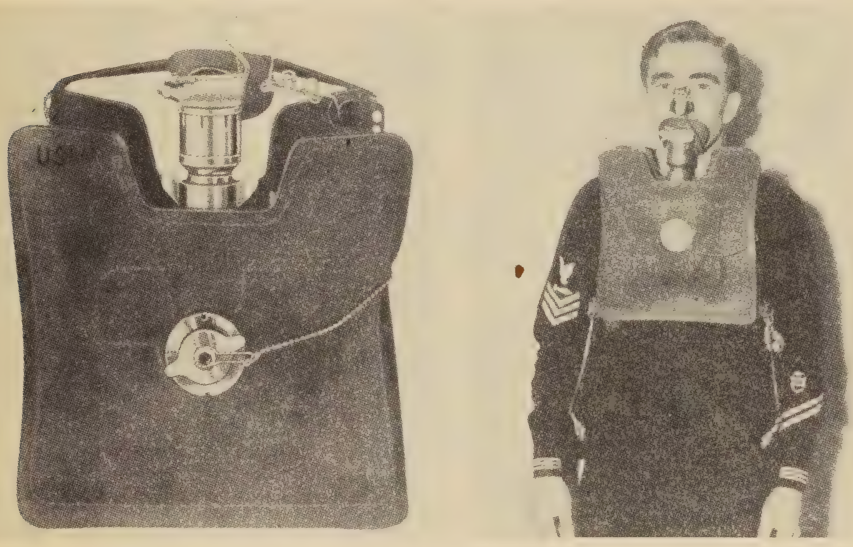


Fig. 165. Submarine escape apparatus (Momson Lung).

Because of the solubility of nitrogen in body fats, symptoms of early narcosis appear at a pressure of 150–300 feet. These symptoms increase with the depth and with the concentration of accumulating carbon dioxide. In order to meet this hazard, helium-oxygen mixtures (He 75, O_2 25) are substituted for air. The Momson Lung is illustrated in Figure 165, which indicates clearly its mechanism and method of attachment. The low solubility of helium and its rapidity of diffusion have served to dispose of the

* See Alexander, J., "Colloid Chemistry," Vol. 5, Paper on cavitation (decompression) by Piccard (Reinhold Publishing Corp.) 1944.

† See Henderson, Y., and Haggard, H. W., "Noxious Gases," pp. 151–157. Reinhold Publishing Corp., 1943.

narcotic effects of air under pressure. Furthermore, because of its rapid elimination, it greatly hastens the completion of decompression.

Experience indicates that, while pure oxygen at atmospheric pressure may be breathed safely for 48 hours without interruption, irritation takes place when it is under pressure. Pure oxygen breathed at a pressure of $2\frac{1}{2}$ atmospheres is irritating (Behnke) and may lead to pulmonary edema.

Notes on Deep-Sea Diving. Dives varying from 80 to 100 feet are frequently made by naked sponge and pearl divers, and penetrations to a depth of 200 feet have been recorded. Professional divers in diving uniforms have descended to 440 feet. Dr. Beebe in an observation unit (bathysphere) built to withstand high external pressure has attained a depth of 3028 feet.*

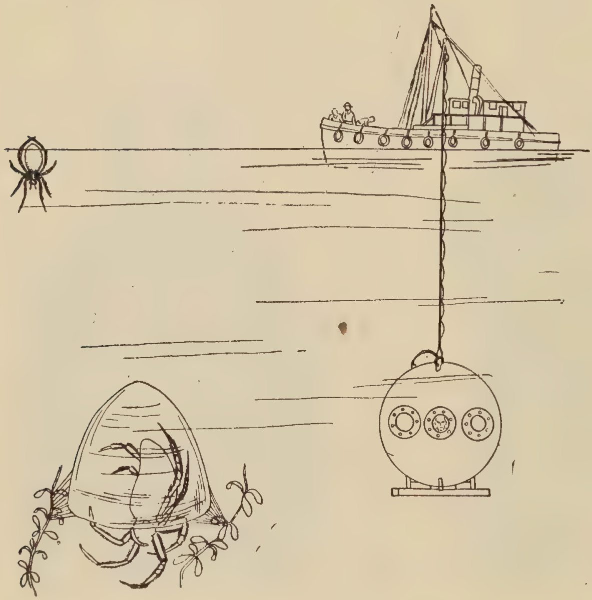


Fig. 166. Millions of years before a human-made bathysphere was ever thought of, water spiders filled their silken bells with air and in this artificially assembled atmosphere lived, ate, courted, and sheltered their eggs.

From Wm. Beebe's "Half Mile Down" (Harcourt, Brace and Co.)

Diving uniforms are of two types. One of these permits freedom of movement and consists of a helmet attached to a steel breast-plate. The balance of the uniform extends to the waist and is closed at the wrist. It is made of flexible, water-resisting material. These uniforms are attached to the surface ship by an air line capable of withstanding an internal pressure of 2000 pounds per square inch. A breast-line is attached which will sustain a lifting strain of 1300 pounds. The air supply line consists of alternating sinking and floating sections which will give the diver freedom but avoid the danger of excess line in the field of operation.

* "The famous bathysphere used by Dr. William Beebe for the study of deep sea marine life has gone into war service. The odd-looking underwater observation post is used to test depth charges to determine the most effective use of these weapons against the submarine." *New York Times*, Jan. 14th, 1944.

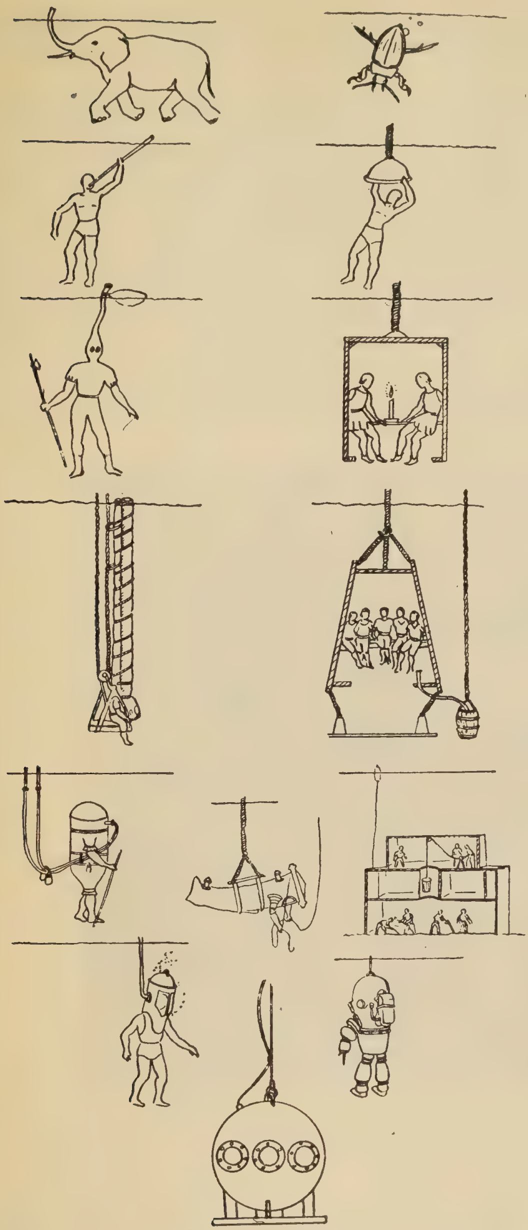


Fig. 167. The Evolution of Human Diving. The left column shows the various attempts at drawing air down from above to the diver, from the elephant's trunk to the modern helmet and hose. The right column illustrates the gradual attainment of success in actually conveying a supply of air beneath the surface. The water beetle does this, and points the way, from the inverted vase of Aristotle to the self-contained bathysphere.

From Wm. Beebe's "Half Mile Down" (Harcourt, Brace and Co.)

Air is supplied by deck pressure pumps in series, so that a constant and adequate volume and pressure may be maintained. The pressure gauge at the supply source indicates the depth reached by the diver. A self-contained telephone within the helmet of the diver permits constant contact with the deck. Diving suits are electrically warmed. Air is supplied to the diver in such a manner that it will pass over the visibility ports, removing respired moisture and operating a Venturi valve (see Fig. 18), which causes the exhaled CO_2 to pass through soda lime before being rebreathed.

Water is prevented from entering the waist and wrist joints of the suit by air pressure within the uniform. If an accident should prevent the escape of air through the release valve, the diver may be blown to the surface, in which event he may strike the bottom of the vessel and be injured or he may experience the effects of sudden decompression. Since recent experience (Behnke) has demonstrated that the narcotic effects of nitrogen pressure may be prevented by the use of a 75–25 mixture of helium and oxygen, when a depth of 100 feet has been reached, diving operations below this level are now carried on by means of this type of gas therapy. The use of helium furthermore permits the diver to be returned

Fig. 168. The Bathysphere is surfaced after submersion. *From Wm. Beebe's "Half Mile Down"* (*Harcourt, Brace and Co.*)

rapidly to the 50-foot level, where helium which he has absorbed may quickly be decompressed by inhalations of pure oxygen, the time of decompression being determined by the depth and duration of the dive.

The second type of diving suit is armored; it consists of a pressure-proof metal container, built to withstand depth pressures. These uniforms are for observation purposes, and are not adapted to salvage operations. The respired atmosphere is self-contained. The necessary oxygen is supplied

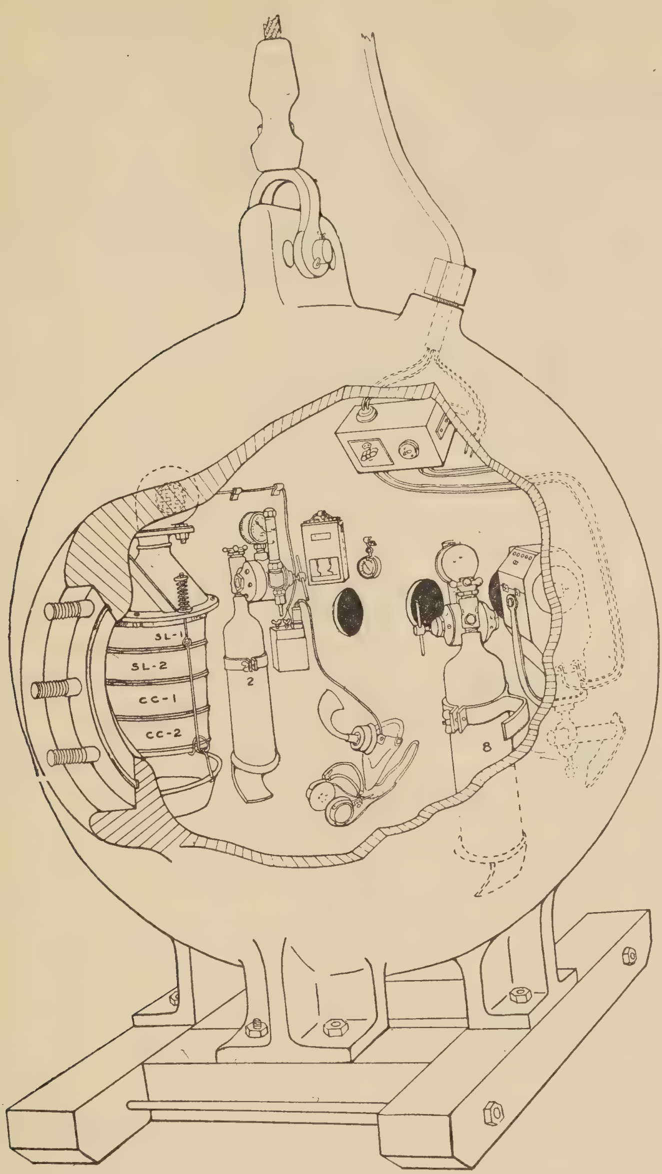


Fig. 169. Internal Arrangement of the Bathysphere of 1934. From left to right: Chemical apparatus with its blower, four trays and pan; oxygen tank and valve; telephone coil and battery box; thermometer-humidity recorder, and below it the left hand sealed window; barometer; switch-box at top of sphere; central observation window, immediately below switch-box; oxygen tank and valve; searchlight. *From Wm. Beebe's "Half Mile Down" (Harcourt, Brace and Co.)*

by compressed oxygen. The CO_2 is absorbed. The respired atmosphere within the suit is at sea-level pressure; consequently there is no decompression problem.

The Pressure Chamber. The pressure chamber is a rescue device for the safe removal of several victims at one time from a sunken submarine. It is ten feet high and about seven feet in diameter, and will accommodate from seven to nine men (Figure 170). It is horizontally divided into two compartments. The upper compartment is a high-pressure chamber with its contained equipment. The lower compartment, water-filled, is made

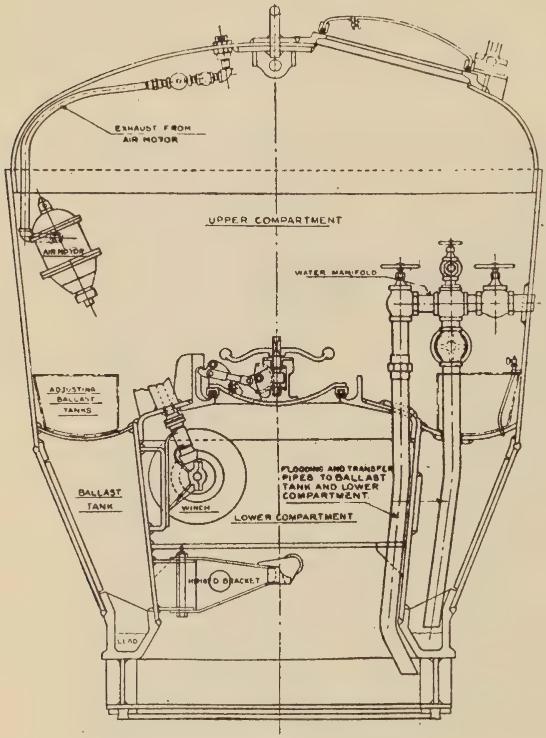


Fig. 170. Cross-section of rescue chamber.

Courtesy Medical Society of New York.

to fit over the escape hatch of the submarine. The rescue chamber is equipped with a motor device for taking up the anchor line attached by a deep-sea diver to the sunken submarine's hatch. The chamber is lowered from the ship by a cable and directed to the hatch of the submarine by the reel line referred to. When it is over the hatch, the water is blown out of the lower compartment by compressed air. The passengers then open the hatch and enter the compressed air compartment from which the water has been excluded by the compressed air chamber of the submarine. The whole is then drawn to the surface. At the 50-foot level the helium in the circulation of the victims rescued is decompressed by pure oxygen administered by inhalers; this may take from 30 minutes to 2 hours. A chamber full of compressed oxygen would present a fire hazard. Conditions permitting, the chamber may be placed aboard the rescue vessel and decompression carried out under the ideal conditions which exist there.

As a stirring example of the practical application of the principles just discussed, the dramatic rescue of the *Squalus* victims, as described by Behnke, is given below.

Rescue of the *Squalus* Survivors. On the morning of May 23, 1939, preliminary preparations were already under way at the experimental diving unit of the Navy Yard at Washington, D. C. for diving tests in the vicinity of Portsmouth, N. H., beginning about June 7. For a period of 20 months the personnel of the diving unit under the supervision of Lieut.-Commander Momsen had been conducting laboratory experiments and tests in a pressure tank to ascertain the value of helium-oxygen mixtures in deep-sea diving. At about 11:30 on the morning in question, a critical experiment in a series designed to determine the level of maximum nitrogen elimination from the body during oxygen breathing was interrupted by the announcement: "The *Squalus* is down off Portsmouth; stand by to leave within 2 hours." Shortly after 2 p.m. the personnel of the unit, supplied with diving apparatus and tanks of helium left by airplane from Anacostia field. Arriving at Portsmouth in the early evening, they prepared in a cold, drizzly rain to rescue survivors of the disaster.

By means of temporary telephone communication, and then by tapping signals it was learned that the *Squalus*, flooded aft of the control room, was lying on a fairly even keel at a depth of 240 feet, 16 miles east of Portsmouth. In the control room and forward, 33 men were alive. The pressure in this part of the boat had built up to 13 pounds per square inch. Aft of the control room in the flooded portion of the ship, the fate of 26 men was unknown.

Diving operations were contingent on the arrival of the U.S.S. *Falcon* from New London. In the meantime, a submersible decompression chamber was sent to the scene of the disaster aboard a Coast Guard cutter to provide recompression for possible survivors escaping from the after end of the boat by means of the Momson lung. Aboard the sister submarine *Sculpin*, which first sighted smoke bombs released from the sunken *Squalus*, Rear Admiral Cole and staff officers were making plans for the rescue of survivors.

Early the next morning, May 24, the *Falcon* arrived with the rescue chamber, and by 9:30 a.m. was moored over the sunken submarine. Then occurred a series of remarkable operations, characterized by calm, faultless execution. All hands, conscious of the momentous task of rescue, worked in perfect unison. Sibitzky, the first diver down, landed forward on the submarine about 8 feet from the torpedo room hatch, where the downhaul cable of the diving bell was to be attached. His dive successful, the rescue chamber was started on its way to the submarine.

In the early afternoon, the first group of survivors was brought to the surface. These men appeared calm and relaxed. There was no evidence of hysteria. All were cold and some were in a condition of mild shock. About one-third of the survivors suffered from headache, undoubtedly the result of increased carbon-dioxide concentration. The development of bends, although the survivors had been subjected to an excess pressure of 13 pounds for over 30 hours, was extremely remote. The recompression chamber, however, was utilized to supply warmth, and if necessary, for oxygen therapy. Medical treatment was directed toward maintaining absolute rest, and supplying heat and fluids, consisting of coffee and malted milk, to which liberal amounts of sugar were added. Particularly effective were the hot towels placed over the upper abdominal and hepatic areas. Within a few hours, the survivors had recovered sufficiently so that they could be safely transported to the Portsmouth Naval Hospital.

Shortly after midnight, or about 15 hours after the start of operations, the fourth and last group of survivors was brought to the surface. This last ascent of the diving bell was marred by the jamming of the downhaul cable and for more than 4 hours the survivors were trapped at a depth of 240 feet, unable to surface. In the meantime, strands of cable leading from the rescue chamber to the *Falcon* began to part. Two divers failed to attach a new cable, and the task now resolved itself into severing the downhaul cable leading from the chamber to the submarine. In successfully cutting it, Squire, the diver, had to descend to a a depth of 220 feet in cold water (39°F), enveloped by complete darkness. Moreover, breathing air at this depth induces in divers a condition of intoxication so that coordinated activity requires intense effort. By the time that the downhaul cable was severed, the rescue chamber was suspended by a weakened safety cable. For fear of breaking this controlling wire by hauling with power machinery, officers and men under the direction of Commander McCann and Lieut.-Commander Momson actually pulled the rescue chamber to the surface.

Rescue operations ended on the morning of May 25, when Badders and Mihalowski, descending in the rescue chamber to the after hatch of the submarine under a pressure of 108 pounds per square inch, reported that the torpedo room was flooded.

Meanwhile, all the survivors had been sent to the hospital and with the exception of three, were in good condition. Hospitalization served not only to prevent complications, particularly the development of pneumonia, but also to keep the survivors in a single group under naval surveillance. Of conditions in the submarine prior to rescue, Lieutenant Naquin, the commanding officer of the *Squalus*, made the following comments:

"Every effort was made to conserve the energy of the men, who spent a great deal of time sleeping. The men were instructed to remain calm, as excitement would increase oxygen consumption and carbon dioxide output. The oxygen supply and available carbon dioxide absorbent were adequate for about 72 hours. The carbon dioxide concentration probably reached about 3 per cent. One tank of oxygen was used in the control room (containing about one-half of the survivors) and another tank in the torpedo room. The intermediate battery compartment was not inhabited since it was feared that chlorine gas might be generated as a result of entrance of sea water into the storage batteries. After a number of hours the odor of chlorine was detected in this compartment, and the men wore "lung" appliances converted into chlorine protectors en route from the control room to the torpedo room, where escape into the diving bell was effected. Carbon dioxide absorption was facilitated by spreading absorbent throughout the compartments. A noticeable improvement in respirability followed each fresh addition. Except for the men engaged in communication with surface vessels by tapping signals, there was no activity on the part of any of the survivors, who remained in the same positions throughout the period prior to rescue (28 to 40 hours).

"With respect to food, the emergency ration of beans was eaten by only a few and in small quantity. The men particularly relished and ate almost exclusively canned pineapple, tomatoes, and peaches, which were available in the commissary storeroom. Fluids were derived entirely from the canned goods, as the fresh-water supply in the control and torpedo rooms, although potable, had an unpleasant taste.

"The atmosphere in the submarine was dark, cold, and moist. The men suffered acutely from cold, which was only partially relieved by eating. It is apparent that the survivors while awaiting rescue consumed a minimum amount of oxygen. The remarkable discipline present under trying conditions certainly prevented the early occurrence of oxygen lack and high carbon-dioxide increase. The atmospheric conditions in the submarine were, however, not conducive to effort. The men existed in a dark atmosphere, saturated with moisture at a temperature between 45° and 55°F. Moreover, they were under a pressure of 13 pounds per square inch. It was impossible to keep warm even with blankets, as the body heat was rapidly lost through conduction in the moist atmosphere.

"The communicators especially were taxed severely in their efforts to send and to receive messages. In fact, any exertion caused great discomfort. The maintenance of an adequate oxygen concentration and the limitation of carbon-dioxide content were so well directed by the commanding officer that life in the compartments could have been maintained for at least 72 hours."

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Chapter 24

Asphyxia as a Problem of Civilian Defense

The author has made repeated inquiries of physicians who have attended the stricken in bombed areas of England. He has asked, "What about your asphyxial accidents? How do you deal with them?" Invariably the reply has been, "Asphyxia is not frequent or important." Abashed by the response of experience, the author's first reaction was, "Strange. How could I have been so mistaken?" Reflection, however, has brought back the conviction that he was right, for if the factors which produce anoxia are present, asphyxia is inevitable. The conditions producing asphyxia are not only frequent—they are pandemic. Incapacitating, killing asphyxia is present in bombing, as surely as is the invisible carbon monoxide.

"Three feet from home and lost, by gracious!" often describes the abdominal surgeon confronted with an eye operation or a brain lesion. We may be even more emphatic in the censorship of the otherwise well trained medical man who fails to anticipate and diagnose asphyxia. Asphyxia consciousness in the average medical man is yet to be achieved. The prevention of asphyxial death has just begun.

Four factors seem necessary to bring home the existence and the urgent need of meeting this major medical problem.

(1) The imagination to anticipate asphyxial accidents.
(2) Knowledge of the phenomena of asphyxia.
(3) Trained senses to see cyanosis and to hear respiratory obstruction.
(4) Manual dexterity to correct the abnormal with one's own hands.

The reader may acquire a basic knowledge of asphyxia by a careful perusal of this book. Interest thus aroused will, it is hoped, induce him to put his knowledge into practice, to train his senses, to educate his hands. The purpose of this chapter is to kindle the imagination so that the mind may be enlightened to deal with these hazards promptly and effectively.

A City Is Bombed

The bombers spiral heavily into the star-lit sky. With increasing altitude, the pressure cabins are sealed, and oxygen is brought into play: 15,000 feet, pressure 428 mm, oxygen reduced to 54 per cent; 25,000 feet, pressure 288 mm, oxygen reduced to 37 per cent; 35,000 feet, pressure 178 mm, oxygen only 25 per cent of normal. The decompressed pilots are comfortable. The new man, sitting over the bomb rack, recalls his air sickness of last week, and wonders if he will soon begin to feel the "butterflies." The ground moisture oozes out of the wing surfaces. The water vapor in our own exhalations becomes a factor of importance. On the ground it was 40 mm in 760 mm; now at 30,000 feet it has become 40 in 225. The nitrogen escapes from our tissues into our blood, into our alveolar air, into our breathing bag. If it comes too fast, it will add up with the water vapor and crowd out our oxygen.

In steady formation, with full fighter protection, we roar out of the west into the dawn. The tiny patch-work target is rimmed with a thin line of breaking surf. The defense awakens; angry hornets circle from the field. In 3 minutes they will be up to meet us. We're there! Here comes their flak. The co-pilot's mask is shot off. With it goes all the research of the laboratory boys. He is physiologically naked now. Pressure 225 mm, oxygen 7 per cent. How long will it take him to collapse? Two minutes oxygen supply in his own blood; after that he's got to have at least 300 cc. a minute (p. 226). The lad is sick at his stomach. He's having trouble with his breathing. Which will get him first, low-tension oxygen or obstruction from his lunch? If he can't get oxygen into his blood at once, he'll lose consciousness; his swallowing and his cough reflex will stop; then he'll choke (p. 236) or drown (p. 155). Think of drowning 30,000 feet above water! (*Asphyxia*?)

Over the target—bombs away!

A warden emerges from his shelter and picks his way cautiously through the ruins. That man with his hand on that wire! Look out, he's charged; don't touch him, that's a live wire! What's the matter with those men at the station? They had orders to cut the circuit during air raids. Take that broom, pry away that wire; maybe his grounding was light. He may have a chance! (p. 211). (*Asphyxia*?)

There, under that plaster—see that foot? I just saw it move. Get your shovel. Hurry up, or he'll suffocate! (p. 235) (*Asphyxia*?)

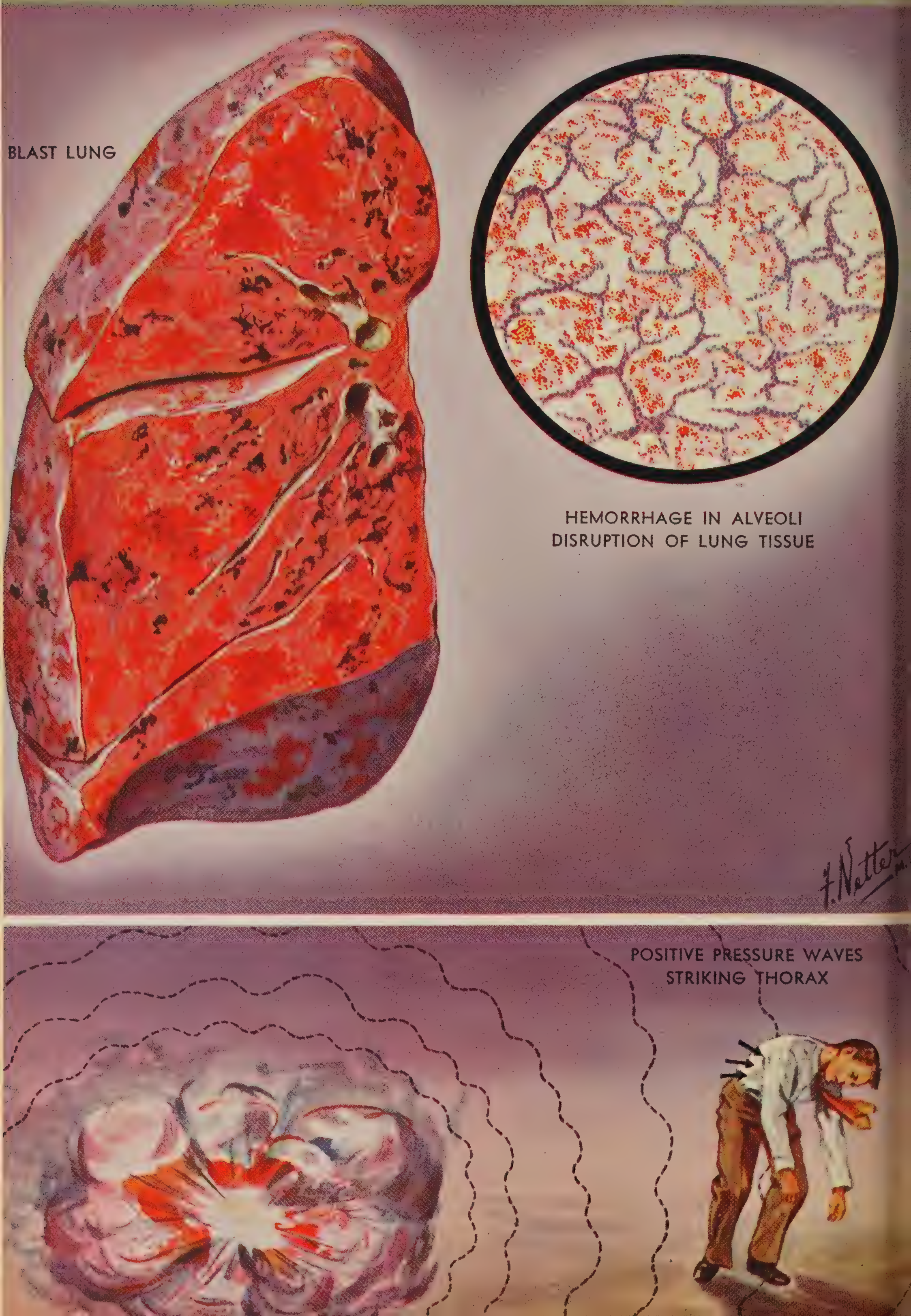
Come on, watch these stairs. There's the bed-room. Over there in that corner, see that woman? Get that two-by-four off her chest! Still breathing? Easy on the stretcher. What did you say? Nail on the beam in her chest? That explains her panting, her color! Only one lung working! (p. 28) (*Asphyxia*?)

What have you got there, boys? Is he conscious? Looks as if his jaw's smashed. Get him over on his side. Don't you see his mouth is full of blood? Never mind the teeth, but keep them out of his airway or he'll choke to death (p. 237) (*Asphyxia*?).

Watch out for that smoke! We can't go in there without a closed oxygen inhaler. Same old story, smoke poisoning. Smoke poisoning my eye! that's what they said about Cocoanut Grove. What difference does smoke make when you have twenty times the lethal dose of CO_2, CO, H_2S, ammonia and hydrocyanic acid (p. 225) (*Asphyxia*?). The victims are lined up for the ambulance. The surgeon gives them all a quarter grain of

BLAST INJURY

Courtesy Ciba Pharmaceutical Products, Inc



morphine from a common syringe. The man with the broken jaw sobs off into oblivion. Respirations become more shallow, his color darkens. The woman with the chest injury breathes more easily. She doesn't seem to have to make much effort now. She was a bit white before; now her lips are gray. Can't you see what you are doing, helping these people to die from asphyxia!

Now stretcher bearers arrive. Look at these two children. How pink and well they look. "Found them in a kitchen," explains the warden, "bad gas leak. I thought the order was to shut off all gas mains in a raid? Wonder if any one else has noticed these children, with cherry-red lips and cheeks; they look so peaceful. They breath so quietly." All the carbon dioxide has been ventilated out by their initial carbon monoxide anoxic dyspnea. (p. 148) (*Asphyxia*?)

Into the bus, back to the casualty center; bad going. The man with the broken jaw starts bleeding. The woman with the injured chest vomits. Every one is too busy to pay much attention. The patients don't mind—a quarter grain of morphine makes anyone comfortable. But when the ambulance arrives the woman has stopped breathing. She is dead. "Chest injury and shock," says the tag. Asphyxia, say we!

The man with the injured jaw is in shock. Systolic pressure 80. Pulse running and thready. Plasma is what he needs. He gets it. His blood pressure goes up and he becomes restless. Give him another hypo. He is going to surgery. "A little intravenous pentothal should control him," says a young Captain. The technique is good. The anaesthetist is out of the way. The surgeon can have the field to himself. Everything goes beautifully. No excitement on induction—he just goes to sleep. But suddenly his respiration stops. He stays asleep. The pentothal got in its punch a minute after it was given. Too bad. Officially he died of jaw wound and shock; actually, of sedation and anaesthetic.

In the recovery ward is a man who was pulled out of a fire. He seemed pretty well at first, but now his respiration has gone up. He is getting blue, and spitting red foam. Pulmonary edema. "Careful how you use oxygen," says the chief. His lungs are already inflamed. "Do a venesection; it will relieve his right heart and improve his color." "What did you say?" "Give him plasma; give him oxygen under pressure?" (p. 296).

In the morgue, several bodies are set apart. No burns, no injuries, no blood. But there are areas of lividness and pallor around the mouth and nose, where soft but firm continued pressure has occurred. They were found in a damaged shelter, crowded with panic-stricken people. Gas, smoke? No, just suffocated. (p. 231) While they were struggling, the oxygen must have dropped below 10 per cent (p. 226) while the carbon monoxide increased. What was done for them when they were pulled out? Why nothing—they were asphyxiated.

We are told that asphyxia is not a problem in bombed areas. What miracle, it may be asked, is expected to preserve us from the results of the accidents just noted?

That the need for organized resuscitation is recognized by the British War Office is evident from the Army Orders issued in July, 1941.* That 90 per cent of these orders deal with the treatment of shock by intravenous fluids and 10 per cent by inhalation therapy, or the direct control of asphyxia, suggests that resuscitation, as it is understood in America and in England should be coordinated into a fully effective service for Civilian defense.

Personnel Medical

Physicians trained in the care of the unconscious patient and in the use of gases for the control of pain (anaesthesia) are well qualified to be placed in charge of resuscitation activities. While it is obviously absurd to term such activities anaesthesia, suitable authority and terminology should be provided the directors of these services to permit them to become fully effective.

Non-Medical. The bulk of First-aid treatment during transportation, preoperative care, anaesthesia, post-operative care and ward protection is in the hands of trained nurses, pharmacist's mates, etc. The practical success of First-aid groups in artificial respiration, the success of nurses in administering anaesthetics, in firmly established routines under intelligent supervision, suggests the desirability of intensively training such groups with due regard for their natural limitations. It is constantly to be borne in mind that a skilled technical act can be performed by any person with a fast reaction time who possesses manual dexterity. The medical judgement to *time* to *select,* and to *modify* this act is something entirely different. This is the practice of medicine and cannot be delegated to a lay person.

To assist the reader in providing such simple basic instruction to the non-medical personnel under his authority, a section (p. 401) has been devoted to First-aid formulas. This basic information may be elaborated by additions from appropriate sections in the text, *i.e.*, First-aid Preventing electric shock, p. 408 see chapter on Asphyxia from electrocution, p. 209.

Integration of Treatment

It has come to be understood that one of the best ways to be saved in an accident is to escape transportation. Such hazards are discussed on p. 103, where inattention to the respiratory needs of the patient, posture and sedation are stressed. It is highly desirable that the sequential care of the asphyxiated patient be closely integrated, from location, through transportation, receiving ward, operating room and post-operative care.

At this writing the treatment of anoxia, if recognized, is intensive at the

* #26, publ. 5120.

site of the accident. During transportation, the speed of the ambulance is supposed to take up the slack in the care of the patient. Unless the surgeon or attendant has been instructed in the simple technique of treatment en route (p. 108), nothing whatever is done. It seems plain that prone-pressure Schaefer, in an ambulance, is a mere gesture. At the receiving ward a new personnel takes over, who know little and often care less for the precise details of the early state of the patient. During the preoperative and operative period still another group assumes responsibilities. After operation a fourth group, the recovery ward personnel, is in charge. By this time it is practically impossible to locate those who first attended the patient. An organized service will reduce or prevent the interruption of treatment. The man who picks the patient up and first attends him will continue through to recovery, at least in an administrative capacity.

Lastly, the public should be informed, first, of the expected incidence of asphyxia, and secondly of simple methods for relief and care. Instruction to date has left the public with the impression that if the First-aid squad is summoned, that is fine. If oxygen is supplied by the First-aid group, that is better. If a chrome-plated machine can be brought to the accident, that is perfect. Should the patient recover, he has been saved; but if he fails to recover, nothing else could possibly have been done. A physician on the scene is viewed with mild curiosity. In large cities he is legally barred from giving treatment. Authority runs from police, to rescue squad, to ambulance surgeon to hospital receiving ward. When the ambulance surgeon takes over, all that he is expected to provide is hypodermic stimulation—and when the time comes, to pronounce the patient dead (p. 392)!

In all likelihood this situation will continue until the prevention of anoxia is recognized as the first and most urgent need of the patient about to die from an accident which embarrasses respiration.

Chapter 25
Asphyxia in Dentistry

The chief anaesthetic for general anaesthesia in dentistry is nitrous oxide and oxygen. The hazard of asphyxia which the dentist faces appears to turn upon one basic factor: the duration of the time of exposure to the anaesthetic. A few short references support this contention.

As an example of the brief exposure required for the simple extraction of teeth, the author submits at the conclusion of this chapter the result of a recent survey in which the anaesthetic deaths from N_2O and oxygen are reported as three in approximately 300,000 anaesthesias. The post-mortem findings in these three deaths were: enlarged thymus with typical general anatomical characteristics, pulmonary embolus, and heart block. In a recent article* Dr. Albert Miller refers to the mortality under short N_2O-O anaesthesia as one in a million.

While the anoxia which so frequently accompanies the administration of N_2O and oxygen appears to be innocent over very brief periods, even when the critical oxygen deprivation of 6–13 per cent is reached (see p. 138), prolonged exposure is an entirely different thing. "Prolonged administration of nitrous oxide-oxygen for major surgery is quite a different proposition from the brief administrations of dentistry" (Freeman Allen). Mayo wrote, "Nitrous oxide in general hands is more dangerous than chloroform." Baldwin referred to it as the most dangerous anaesthetic. The physiological principles underlying anoxia stated in this book confirm the clinical opinions noted above.

The dentist who appreciates the vital factor of *the duration of the anaesthesia* when administering N_2O and oxygen, will forestall mortality and morbidity. Let the dental surgeon recall that a candle will be extinguished when oxygen falls below 17 per cent, that the effects of an altitude of 40,000 feet (6 to 13 per cent oxygen) will operate just as effectively in a dental chair as in a high-altitude plane and that the low oxygen concentration in a blazing house (6–10 per cent) which helps to overcome the fireman will overcome his patient in the same manner.

As in other forms of asphyxial accidents three stages may be recognized: depression, spasticity, and flaccidity (p. 124).

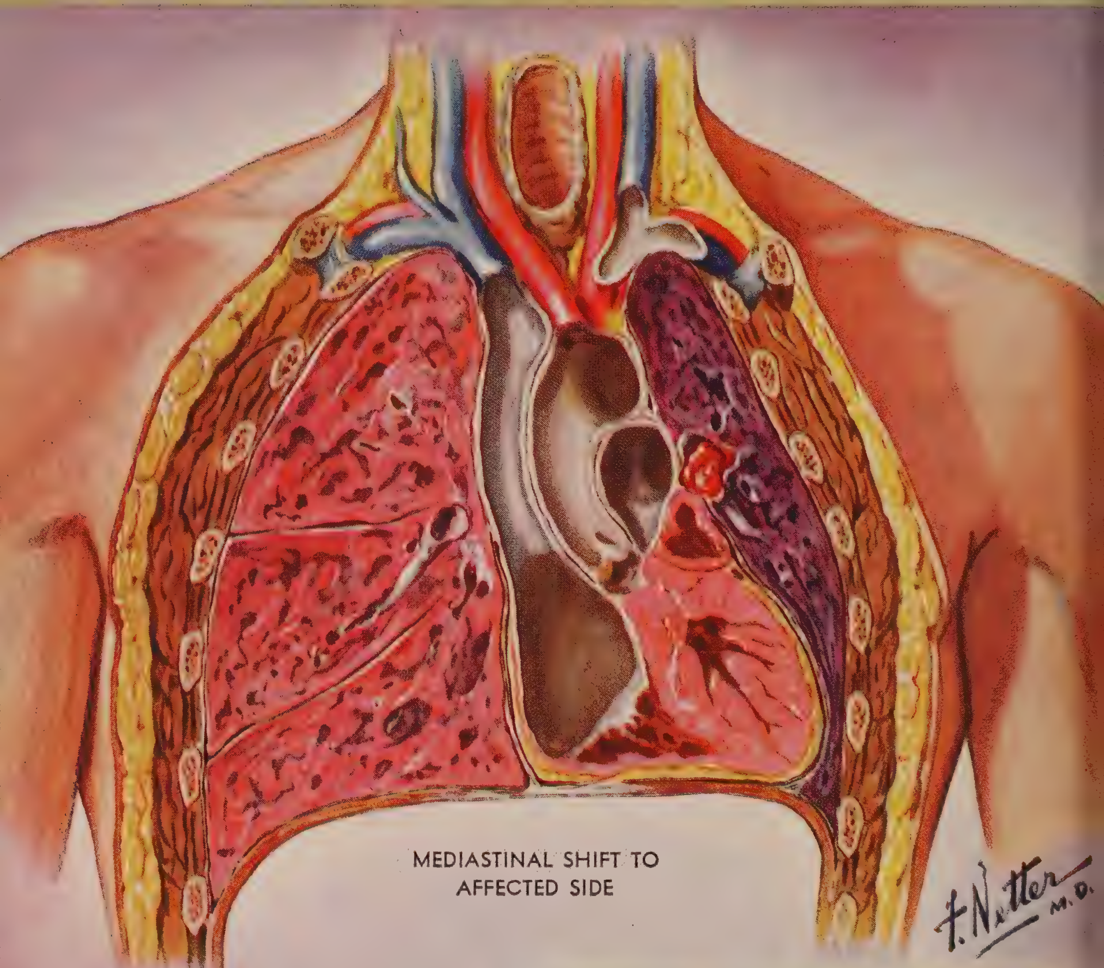
Depression

Into your office walks a pale little middle-aged woman. As you examine her, you note her pallor and the fine, cool perspiration on her forehead.

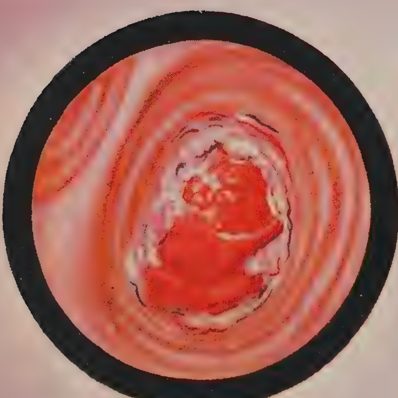
* Miller, A., "Technical Development of Gas Anaesthesia," *Anaesthesiology*, **2**, No. 4 (July) 1941.

MASSIVE COLLAPSE (ATELECTASIS)

Courtesy Ciba Pharmaceutical Products,



MEDIASTINAL SHIFT TO AFFECTED SIDE



BLOOD PLUG OCCLUDING BRONCHUS
(bronchoscopic view)

Without further warning, her arms relax, her body slumps into the chair and her head rolls to the side. What has happened? What is likely to follow? What are you to do for this patient?

A large, heavy-set man introduces himself. He has little to say. His face is red. He is breathing rapidly and occasionally sighing. He is restless and fidgety. His finger tips are yellow. You proceed with your work. As you test for pain in a discolored tooth you note that the florid color has been replaced by a pallor. Suddenly the jaws close on the mouth prop, displacing it; the patient becomes rigid, his eyes roll upward. His pupils dilate. What has happened? What will follow? What will you do for him?

These accidents are not uncommon, and need not cause serious embarrassment. They may be readily and successfully treated, if the dentist is informed and prepared. It is customary to ascribe these reactions to emotional stress, to a psychological disturbance. Because of the pallor exhibited, the hazard is regarded as primarily cardiac. The head is lowered; stimulants, such as spirits of ammonia, amyl nitrite, a hypodermic of strychnine, caffeine citrate, camphor in oil, metrazol or coramine is administered.

After a variable period of unconsciousness, usually brief, the patient recovers. The pale woman may simply open her eyes in mild wonderment, ask where she is and what has happened, and then lapse into semi-conscious depression. The man may break out into a violent fit of laughter. He may try to get to his feet. The restrained excitement which he brought with him is no longer under the control of his will. It expresses itself as an emotional outburst. The question is raised, Are the hypothetical reasons (emotional strain, fear, etc.) advanced to explain the cause of these accidents correct and complete? These patients do not always react quickly and favorably. Sometimes the depressed patient suffers repeated relapses which give the dentist much anxiety. Is there any way of anticipating or cutting short these unpleasant incidents?

The purpose of this book is to bring together similar phenomena occurring in what have been looked upon as widely separated fields. In this instance, let us consider for a moment a curious reaction seen among passengers in airplane travel. The following is quoted from Boothby.*

"The symptoms of hyperventilation, namely, dizziness, faintness, tingling sensations, and involuntary muscular spasms, especially of the hands and feet, have been described frequently. Most normal persons can experience these symptoms on the ground by voluntary forced breathing. Fear, airsickness and neurasthenia commonly evoke unconsciously deep breathing among passengers, especially those taking their first trip on an airplane

* Proceedings Staff Meetings Mayo Clinic, **16,** 209. 227 (April 2 and 9) 1941; **17,** No. 27 (July 15) 1942.

and particularly if the flight is bumpy. All personnel on air liners, therefore, should be instructed to recognize the early signs of hyperventilation and to institute treatment early. The effects can be overcome readily.

"Any normal person readily can experience the symptoms of hyperventilation by voluntary forced breathing. It is only necessary to force maximal inspiration and maximal expiration at an approximately normal rate. Within thirty to sixty seconds an odd sense of faintness and dizziness is noted. True rotary vertigo is not present, but an unsteadiness described as a 'lightheaded' feeling is experienced. Within a minute or two a characteristic numbness and tingling type of paresthesia will be noted in the extremities and a sense of panic and palpitation are evident. The mind becomes clouded: surroundings take on an appearance of unreality and the person borders on unconsciousness. If the forced breathing is continued for five minutes or more, the muscles may become perceptibly tense and eventually the tetanic contractions of carpopedal spasm will ensue. In later stages a sense of air hunger may be experienced even during hyperventilation, tending to produce a vicious circle. If forced breathing is continued, a state of vasomotor collapse resembling shock may be produced. The pulse is weak, rapid and irregular and the skin is pale, cold and moist. The patient is in a state of extreme anxiety and often lapses into semiconsciousness.

"These more violent manifestations are rare in clinical practice and occur only among frankly hysterical patients; but the milder sensations are common. Patients describe a feeling of constriction in the thorax, a sense of inadequate ventilation associated with dizziness, faintness and anxiety. When prolonged, the highly characteristic tingling and numbness of the extremities are present. Usually the patient does not know that he is hyperventilating, but in the more severe instances even an untrained witness may observe and report it.

"The recognition and treatment of the effects of hyperventilation are extremely simple and should be included, it would seem, in the instruction of every attendant on airplanes; the symptoms are induced by airsickness and fear (sometimes subconscious fear) and may be associated with, or aggravated by, a state of emotional tension.

"The methods of treatment are (1) administration of oxygen with the oxygen mask which provides some rebreathing; (2) encouragement of slow respiration; (3) holding the breath for a few moments from time to time and (4) breathing into a paper bag held over the nose and mouth."

There is also the possibility that the clinical syndrome of depression and syncope may be related to certain physiological phenomena of the autonomic nervous system. Carr conducted two interesting studies in clinical research, both related to an investigation of the variations in blood pressure under conditions of the emotional strain of dental operations.

One study was entitled "Clinical observations of the physiological effect of emotion (fear, anxiety syndrome) upon the circulatory system as observed by variations in blood pressure." This study was suggested by the clinical experience of patients developing depression symptoms and syncope immediately following the injection of procaine (1 to 2 per cent) with a mixture of suprarenin for either operative dentistry or dental surgery. It was suspected that, whereas a certain number of these patients may have had an allergic idiosyncrasy to the suprarenin, there might also have been other factors, particularly a fear-anxiety syndrome. Accordingly, a group of patients was selected for observation who had previously developed syncope when injected with the local anaesthetic solutions. During the experiment these patients were injected with 1.5 cc of sterile normal saline solution instead of the local anaesthetic which had previously induced the symptoms.

Observations were recorded in each case before injection, *i.e.*, rate and rhythm of the pulse, respiration, temperature, and blood pressure. In a large percentage of the patients thus studied, there was a fall in blood pressure, the pulse became rapid and weak, respiration became shallow and syncope developed, with dilation of the pupils and unconsciousness attended by pallor and cold perspiration.

In a similar study conducted by Carr,* "The effect upon blood pressure of paralyzed subthreshold afferent stimulae of the tregeminus nerve in the production of syncope simulating shock", patients were observed to develop depression and syncope as the result of pre-dental operations for cavity preparation and the filling of teeth. In this series of cases no local anaesthetic was used. In each instance the same observations were recorded in the development of syncope as in the first series.

Turning to the experiences of Yandell Henderson, we find the following. "Operating upon dogs with the chest open, it was necessary to blow air into them so as to keep them distended, as well as to maintain respiration." Henderson found that if this artificial respiration was vigorous, the dogs promptly went into collapse and died, demonstrating that simple overventilation may cause death by loss of CO_2 in the circulation.

A man at rest by merely breathing deeply and rapidly for a sufficient period may become unconscious. The reason for this is that when the CO_2 in his circulation drops below the necessary level, the oxygen in his red blood cells will not pass into his tissues. There develops what is known as asphyxia without cyanosis (p. 27).

Let us apply this hypothesis to our two patients in the dental chair, as well as to travelers in an airplane. Anxiety from impending fear of operation or fear of flight hazards accelerates the respiration with its tendency to overventilation, without the compensating factor of CO_2 production by working muscles (vigorous exercise). The loss of CO_2 induces anoxia without cyanosis, resulting in loss of muscle tone, further anoxia and temporary

* Carr, M. W., Unpublished report.

collapse. During the period of unconsciousness and depressed respiration, the CO_2 re-accumulates in the circulation, consciousness is regained and the patient recovers. In the case of the pale woman however, the stimulation of recovery again accelerates her respiration, she again loses her CO_2 and goes into relapse. The man, however, produces CO_2, by his muscular excitement, compensating for the loss through his increased breathing.

Whether or not this hypothesis of pre-operative collapse is tenable may easily be proven by the practical application of the very simple treatment indicated for depression. This treatment consists of the application of an inhaler and the administration of oxygen and CO_2, (90 and 10 per cent). In the absence of CO_2, the patiênt may rebreathe oxygen alone back and forth into a bag until the respirations are throughly stimulated and his color becomes pink. The dentist will find that even the color of a pale woman will be improved under this treatment. It is quite practical for the experienced dentist to use pure CO_2 from a sparklet or other container sprayed near the mouth and nose. If the patient's respirations are very shallow it can do no harm. As soon as stimulating effects are produced, such therapy must of course be discontinued. Following the administration of high concentrations of CO_2 there is a lapse of 10 or 15 seconds before the peak of the stimulation is reached. CO_2 stimulation by this technique should be discontinued as soon as the respirations begin to pick up. The use of pure CO_2 is dangerous in inexperienced hands. Oxygen 90 and CO_2 10 is a perfectly simple, safe treatment. It may be administered as a preventive as well as a cure for these cases of syncope.

As the reader has noted, emphasis has been placed upon gas therapy in the treatment of depression. Little or nothing is said of stimulation by medication. This is as it should be. Efficient respiration, natural or artificial, is the best possible cardiac stimulant. The mechanical act of respiration helps to relieve the congestion of the right heart and to overcome the anoxic depression of the heart muscle. Without respiration cardiac stimulation is futile. You may step on the starter and run the battery down, but the car will not travel without gasoline, which in this case is oxygen. Every effort of the surgeon should be directed to getting oxygen into the circulation. While these efforts are in progress, hypodermic stimulation with camphor and oil, strychnine, caffeine, etc., may be administered by an assistant or the office nurse.

Spasticity

The picture presented by spastic asphyxia is well known. The jaws are clenched, the muscles of the throat are in spasm, the respiration is obstructed, and rapidly progressive cyanosis develops in the mucuous membranes and skin. The color of the blood turns from brick red to claret; bleeding from the wound increases; the pupils are eccentric and are either dilated or contracted.

Spasticity often follows obstruction to the respiration, especially nasal. The mere addition of oxygen to the respired gases may relieve it. If, as often happens, the mouth prop has become dislodged and a full set of upper and lower teeth is tightly clenched, nasal respiration becomes inadequate. The usual procedure is to attempt to separate the teeth by force with wedge, wooden screws, or a so-called jaw forceps, to permit ventilation through the mouth. At this point the dentist asks himself two questions: (1) Is my objective to relieve the asphyxia, irrespective of whether or not the patient comes out of the anaesthetic? (2) Do I propose to keep up the anaesthesia regardless, accepting the asphyxia as a nuisance, to be controlled only as it interferes with my operative work?

If the first position is assumed, asphyxia may quickly and quite certainly be relieved by passing air or oxygen into the posterior pharynx. Stopping the flow of N_2O and giving pure oxygen by the inhaler should first be tried. This will often provide relief if the obstruction is not severe.

The next simplest and least traumatic method of accomplishing relief is to pass a 26 or 28 French catheter, lubricated, into one nostril to a depth of a distance equal to the space between the nostril and the ear of the same side. The tip will then reach back of the soft palate but will not pass into the esophagus. The catheter will be in the correct position when respirations pass through it. Oxygen may be blown into this tube, or in an emergency the dentist may blow into it himself. As anoxia passes off, the spasm will disappear. General anaesthesia may then be resumed, before the recovery of *complete* consciousness. Patients who are not entirely re-oriented have no memory of such incidents, associating them as a rule with their dreams. This method is strongly recommended, first because it is promptly effective, secondly because it avoids damage to the teeth and tongue.

The third method of relieving spasticity is that ordinarily recommended. The teeth are pried apart by a wedge or mouth gag. When they are sufficiently separated, an airway (Fig. 59) is introduced over the surface of the tongue. The airway used should be of heavy-gauge metal so as to afford a non-cutting edge. It should be lubricated. In an emergency the patient's saliva will serve as a lubricant. At this point pharyngeal suction should be thoroughly applied (Fig. 60). The relief of respiratory obstruction is not to be considered complete until suction has been carried out. Adequate mechanical suction should be available wherever unconscious patients are to be cared for. There is but one initial cost. For a description of various types of suction the reader is referred to page 59.

Flaccidity

To return to the second question, *i.e.*, the policy which the dentist elects to pursue in the event of spastic asphyxia, if the dentist considers that the spastic asphyxia which he encounters is merely an incident to be dealt with only insofar as it interferes with his work, he may soon find that the spasm will disappear, that the respirations will become more quiet, and that his field is much more comfortable. The onset of relaxation, dark blood, dilated pupils, and reduced rate and depth of the respiration following spasticity is not only a red light—it is like driving off a pier into the river. If one does not stop instantly and devote every conceivable effort to resuscitation, death may be expected as a matter of course. The mystery is that deaths do not occur more frequently following flaccidity. In the event

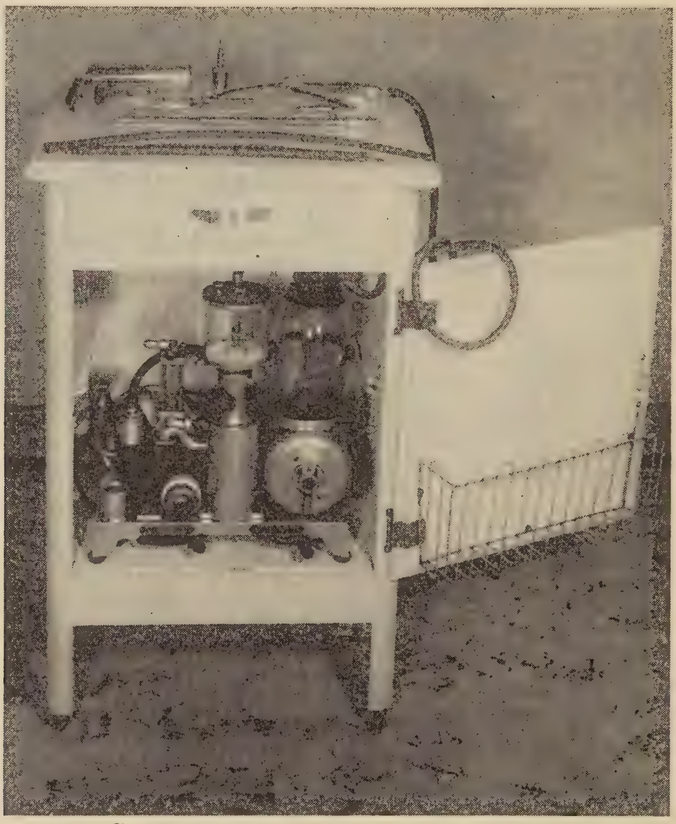


Fig. 171. Carr's insufflation suction assembly for oral surgery.

of such an accident the following steps should be taken at once.

Stop all operative work.

Remove pharyngeal packs or other foreign matter in the mouth. (blood, mucous, etc. by suction)

Lower the patient's body.

Insert an airway.

Apply an inhaler with oxygen 90 CO_2 10. If a sparklet is available or some other source of carbon dioxide this may be used for a few breaths. If respiration is still functioning nothing is to be gained by artificial respiration, as the patient will promptly pick up the necessary oxygen-CO_2 from the breathing bag. If respiration has ceased, the chest should be compressed by an assistant every five seconds.

The ideal resuscitation technique is described on page 79, the model of which is the asphyxiated new-born. It would seem that a technique practiced in a delicate infant could scarcely be regarded as traumatic in an adult. Insufflation of oxygen-CO_2 should continue until the color is normal. The patient should be placed between warm blankets and kept in the recumbent position for at least an hour. He should be taken home by a friend or attendant and not turned loose into the street.

Increasing the Margin of Safety of Nitrous Oxide and Oxygen.

The addition of dilute ether vapor to the nitrous oxide and oxygen administered for brief dental operations greatly widens the margin of safety and efficiency. Strangely enough it is seldom noticed by the patient. If a little ether is added, the oxygen concentration used may be increased and spasm from anoxia reduced. Ethylene or ethylchloride is preferred by some, and the asphyxial complications which may be encountered are dealt with as described above. Cyclopropane has not proved practical because a stricly closed technique must be employed in its administration. It is not unlikely that the following cases passed through the spastic and the flaccid stages just noted.

Medical Examiners Report of Deaths under Anaesthesia in Dentists' Offices

Case 1. *Synopsis.* The deceased was referred for dental extraction. He had been under treatment for cellulitis of the right jaw. External film revealed all lower teeth on the right side to be abscessed. Due to evident toxicity the patient was requested to come to the clinic for incision of abscess of the jaw. He was given gas-oxygen anaesthesia, and the incision was made. The pulse of the patient became weak, so the anaesthetic was discontinued. Coramine was injected, artificial respiration was performed, and CO_2 oxygen was given. The deceased seemed to rally for a short time, but expired in about an hour.

Findings. A generalized cyanosis is present which is more marked in the dependent portions. The right jaw reveals a recent incision along the lower angle of the right mandible, from which blood serum is exuding very slowly. The whole right side of the tongue reveals a necrotic new growth extending to the soft palate and the pharynx. The lumen of the right bronchi is found to contain much fluid similar in appearance to that which appeared in the linear incision. This material extends through the bronchial tree. The pleural surface of the right lung shows a slight peripheral atelectasis in the right lobe. A large necrotic growth lies between the root of the tongue and the lower jaw. The periosteum of the mandible is involved. The new growth extends into the pyriform-fossa lying close to the epiglottis. The mucosa of the epiglottis and the ary-epiglottic fold is edematous. The vocal cords are free. Cause of death: Edema of the glottis.

Case 2. *Synopsis.* Patient came to have five teeth extracted. Dentist reports having used a 92–98 per cent mixture of $N_2O + O_2$(?) for about 2 minutes. One tooth had been extracted when patient ceased to breathe; artificial respiration was performed, and a hypo of caffeine was injected without result.

Examination: Patient lying on the floor beside the dental chair and gas machine. A plaster cast is over the body, to the lower ribs for a fractured leg.

Autopsy: Multiple fracture and dislocation of the cervical vertebrae, with laceration of the spinal cord. Bilateral congestion of lungs, with intra-alveolar hemorrhage. Lymphoid hyperplasia, oropharynx and thymus atrophic.

Intravenous Anaesthesia

It is hard to see why those responsible for the popularity of intravenous Pentothal and Evipal should fail to follow the admonitions of the manufacturers and to emphasize the fact that these agents are not suited to dental surgery. Comfort to the patient has apparently outrun ordinary caution, and numerous dentists in the fear of being charged with conservatism have found themselves in a blind alley.

The risks of intravenous anaesthesia by the barbiturates are not mysterious, but are inherent in the drugs. A consistently safe technique for their use is not practical in dental surgery, since the hazards faced constitute a violation of basic physiological principles. These hazards are:

(1) An extremely narrow margin of safety (low index).
(2) Consistent and specific respiratory depression.
(3) Stimulation of pharyngeal and laryngeal reflexes (a typical barbiturate reaction) which precipitates cough spasm and respiratory obstruction.
(4) Because of the depressed respiration it becomes mandatory to supply oxygen by artificial means (through a field obstructed by operative procedures)

Allergy and Anaphalactic Shock

It is difficult to understand how a few drops of a solution (diluted to 1/10,000) (local anaesthestic or other foreign substance) injected into the circulation may cause sudden syncope, serious prostration, or even death. The following quotation from Vaughan* is enlightening: "Capillaries of the muscles of the average man have a total area of approximately 6,300 square meters, or 3000 times the area of the body surface. In the absence of restraining forces, the entire plasma volume could pass from the capillaries to the body tissues in 10 seconds."

* Vaughan, W., "Practice of Allergy," St. Louis, C. V. Mosby, 1939.

In susceptible patients any degree of edema may be encountered. In extreme cases there is a vast outpouring of blood plasma into the tissues. The specific treatment is the administration of adrenaline. One-half cc. of 1–1000 solution should be given subcutaneously, preferably at the site of the dental injection, which may have precipitated the attack. The symptoms of shock should disappear within a minute or two. If sudden and severe edema of the tissues is seen to occur and the patient becomes dysponeic and cyanosed, oxygen should be given immediately under a pressure of 5 mm., in conjunction with the adrenaline administered by hypodermic.

In perhaps no field of surgery has careful adherence to conservatism and a consistent technique proved of greater value than in the use of N_2O and oxygen in dentistry. The results of the following questionnaire tend to confirm the author's opinion that a hundred thousand administrations of the same anaesthetic by a technically skilled personnel with a carefully regulated routine will yield a lower mortality and morbidity than the present practice of employing a great variety of agents by a personnel constantly shifting and of indifferent skill. The questionnaire was directed to thirty leading oral surgeons throughout the United States. Three deaths from N_2O and oxygen are reported in practices which aggregate more than 300,000 anaesthesias for oral surgery.

Deaths Occurring in Dental Office Practice: Questionnaire

Causes of Death. *General:*

(1) Emphasis on comfort, instead of safety and efficiency.
(2) Experimental use of new agents.
(3) Use of advertising material of manufacturer as guide.

Personnel

(1) Lack of training in observation of signs of depth of anaesthesia, of obstruction and of asphyxia.
(2) Dependence upon mechanical measuration, rather than upon physiological signs.
(3) Unfamiliarity with the anatomy and physiology of the "death zone" (epiglottis to vocal cords) combined with a technical inability to care for this region by inspection, suction, and intubation.
(4) Lack of care for obstruction on other portions of the respiratory tract.
(5) Lack of knowledge of how to force oxygen into the lungs with an anaesthetic gas machine or other apparatus.

Equipment

(1) Faulty equipment.
(2) Poor preparation (such as tanks on wrong yokes, empty oxygen cylinders, etc.)

Principles to Be Advocated

(1) Selection of anaesthetic agent invariably based upon safety, efficiency and comfort in the order stated.
(2) Simplicity of equipment. Physiological rather than mechanical indications in variations of dosage.
(3) Intimate knowledge of the appearance of and ability to care for the "death zone"

Comment No. 1:

I have never had a death in my office from nitrous oxide. In this area I have been lucky, as I have had situations in my practice in which it was I who nearly died, due to the patient's respiratory spasms, from which they recovered.

The closest to a death in my office was some twenty-one years ago. A sixty-eight year old woman appeared at my office for the removal of four lower anterior teeth. The work was done very quickly. Nitrous oxide was insisted upon by her son-in-law, a physician, who was with her and who stated that she was diabetic. She walked from the chair to the rest-room. Shortly after I heard her retching and found her lying on the cot under great stress from pain around her heart. Her son-in-law was administering a sedative hypodermically and told me not to worry, that she had had a similar attack three weeks previously. About one hour later she was carried out of my office in a chair, conscious and cheerful, placed in an ambulance and taken to the hospital. She died at six o'clock that evening (eight hours after the administration of nitrous oxide). Her bladder was aspirated and found to be loaded with sugar. She died in a coma and the death certificate stated she succumbed to diabetic shock.

I have been administering nitrous oxide for forty-one years, extensively since 1917, as a specialist in exodontia, and I honestly believe that I have given more of it than any living man in America. Last year I used 68 3200- gallon cylinders of nitrous oxide.

Comment No. 2:

I have been an advocate of N_2O and oxygen for 20 years. No deaths have occurred in my practice. I believe that death results from ignorance of the signs of anaesthesia, incorrect or inadequate use of oxygen in respiratory failure, incorrect use of mouth props and mouth packs and attempting general anaesthesia where it is contraindicated.

Comment No. 3:

Have been fortunate after fifteen years, and have never had a fatality.

Comment No. 4:

We have given over 12,000 administrations of N_2O and oxygen in our office for surgery without a fatality.

Comment No. 5:

The only information I can send at this time concerns a dental office case resulting in the death of a young woman in the chair under pentothal sodium.

Comment No. 6:

I feel that most of my asociates are competent, well trained and experienced in the general requirements of anaesthesia. Accurate preoperative diagnosis in my opinion is the best way to avoid trouble. I have no office deaths to report.

Comment No. 10:

The profession as a whole is unfamiliar with the anatomy and the physiology of the death zone. Obstruction is often overlooked. The profession should know how to resuscitate with oxygen.

Comment No. 11:

In 31 years of specialized practice in oral surgery after ten years of general practice in which I gave anaesthetics, I was never so unfortunate as to have an anaesthetic death, nor have I ever been present when a patient has died under an anaesthetic. I have had some "narrow squeaks" and I have seen patients shortly after they have died in other offices.

Comment No. 12:

I realize that perhaps even before you receive this letter I might have such an unfortunate occurrence, but at this time I have none to report.

I specialized in the practice of oral surgery in 1916 after using nitrous oxide-oxygen anaesthesia in my practice for three years. For the past 30 years I have used this method of anaesthesia practically exclusively. I'll admit that I have had a few border-line cases. I average approximately from 1,800 to 2,000 nitrous oxide-oxygen anaesthesias per year and possibly have administered 50,000.

I am very much opposed to pre-medication, especially with the barbitals. In the cases of death in oral surgeons' offices I have been told that in 90 per cent of the cases a barbital sedative had been administered and this, not the anaesthetic, I believe resulted in death.

In a few cases, the very heavy alcoholic types, I find it advisable to premedicate, but with $\frac{1}{2}$ gr. of codeine and $\frac{1}{150}$ gr. scopalamine.

We are extremely cautious in keeping our patients out of the cyanotic stage. I prefer a light anaesthetic even if there is some slight movement. Also, I am careful of the position of the patient in the chair—that the head is not too far back or forward too much, so that the airway would become congested. Also, we use packs and a suction to control the blood and mucous in the throat. If the blood becomes very dark or the pupils dilate, full oxygen is administered at once. Also, if there are signs that the patient has gone too far, we administer through the nose-piece and mouth the one, two, three oxygen, then artificial respiration, and inject 1.5 cc of Coramine and 1 cc Metrazol. If the patient shows signs of shock he is placed in a prone position with a nurse in constant attendance with blankets, so that there will be no chill.

Death I

I have had one anaesthetic death in a busy exodontia and oral surgery specialty practice of 25 years. It occurred in 1926. Here are the facts.

Patient, male, city fireman, age 32, weight about 150, sallow complexion, presented for removal of lower right bicuspid root which had been attempted two or three months previously by referring dentist. X-ray revealed considerable pathology of surrounding alveolar bone, and slight swelling of gingival tissues was present. It was apparently a very simple case requiring three to five minutes' anaesthesia.

Induction was uneventful; no excitement stage or struggling. Nitrous oxide-oxygen used. I had just picked up a scalpel to incise for a gum flap when my trained nurse-anaesthetist and I both observed that the shallow respirations had ceased. At no time was any cyanosis present. Pure oxygen was forced into the lungs; manual artificial respiration was applied in conjunction with intermittent oxygen. No difficulty whatever with airway. Intravenous injection of heart stimulants. Dr. — was with us in 12 minutes and direct injection into heart of adrenaline solution was given. Patient simply had no pulse after the two minutes of induction. At autopsy the next morning at —, the only outstanding abnormality (and indeed, quite sufficient) was a very large thymus gland. Patient had practically no hair on his body except sparsely on the scalp. — was present at the autopsy.

I would emphasize shallow, slightly yellowish color of patient which did not change in the slightest. I do not know how this could have been avoided or foreseen.

Death II

Pre-operative findings: infected teeth.
Pre-medication: None.
Anaesthetic agent: N_2O and oxygen.
Operation: Removal of two teeth.

Signs preceding and accompanying fatal issue: all normal anaesthetic signs present before patient stopped breathing.

Color: pink.

Pulse: 80.

Relaxation: yes.

Eye signs: oscillating eye, small pupil.

Pharyngeal and laryngeal reflexes: quiet.

Post-mortem findings: Pulmonary embolus.

Active T. B.; should not receive inhalant anaesthetic.

This is the only death (anaesthetic) experienced in the dental surgery department of —. We have given around 200,000 general anaesthesias ranging from $\frac{1}{2}$ minute to an hour. Practically all were N_2O and oxygen. I was not present and would not have given it at this case. No deaths in private practice, specializing in exodontia and oral surgery since 1925.

Death III

Pre-operative findings: Cardiac.

Pre-medication: None.

Anaesthetic agent: N_2O and oxygen.

Method of administration: Nasal.

Operation: Removal of teeth.

Signs preceding and accompanying fatal issue: Became obstreperous and then collapsed.

Methods used for Resuscitation: Forced oxygen under pressure. Post-mortem findings: Heart shock.

The low mortality resulting from a carefully supervised routine is an enviable record for any professional group. The foregoing report is valuable because it suggests an ideal attainable by all members of the dental profession who are willing to submit to the necessary training and who can attain the required technical skill.

Unlike anaesthesia for general surgery, which tends to settle into a steady rhythm as the operation progresses, dental anaesthesia is usually so light that the hazard, as has been pointed out, is proportional to the operating time. It is therefore recommended that when other than brief gas-oxygen anaesthesia is desired, the dentist avail himself of the endotrachael anaesthesia method described on p. 190.

Chapter 26

Asphyxia as an Economic Problem of Public Health*

With the vision and the good judgement which have always characterized his views, Dr. Haven Emerson offered the following ideas as to the prevention of asphyxia as a public health problem.

"It is quite apparent that every new movement that concerns us with the extent and quality of deaths reveals an inadequacy of our fundamental information.

"I should like to use the terms that are proposed to us by the biologists in discussing cellular survival, namely, reversible and irreversible processes. We may consider death an irreversible process. But death by drowning is, at a certain point, a reversible process. So we are discussing in all these categories of deaths those that are reversible by means of technique now at our disposal, those which may be postponed, and those which are inevitable.

"When I speak of deaths that may be postponed, I refer to those that occur in poliomyelitis. It is not uncommon in the course of an epidemic of poliomyelitis to find a person suffering from a respiratory paralysis which is not necessarily permanent. If he is relieved of the embarrassment of respiration for a period while recovering from the congested stages of the lesions in the spinal cord and bulb, that person may recover and carry on a reasonably normal life afterward. But if the damage is of a structural kind and permanent in its nature, affecting the anterior horn cell, then we have a postponement of death with the aid of artificial respiration, but no recovery. So the very approach to this problem demands that we look at our potential deaths from various points of view: those that are impossible to prevent, and those that are preventable if within easy reach of the skills and resources we now command in certain conditions, or that are almost automatically preventable if the patient is left alone under reasonable and favorable conditions at favorable ages of life.

"It is recognized that there are now resources at the Bellevue Alcoholic Wards which have never been used and which may be increasingly available, that is, devices for assisting the alcoholic, intoxicated person to recover his capacity of elimination of the drug by artificial respiration methods. That has been shown to be a valuable resource. I think we must not blind ourselves to the fact that the per capita consumption of alcohol in England has run very closely parallel with the incidence of deaths from overlying [of infants]. That isn't an expression of the alcoholism that we have had in

* The matter in this section is adapted from the transactions of the S. P. A. D., *J. Am. Med. Ass'n.* (July 22) 1933.

this country. Overlying seldom occurs here, because mothers rarely sleep in the same bed with their infants. They seldom take beer, porter and ale to increase their milk, and they do not succumb to the week-end and bank holiday drunkenness which was formerly an expected experience of the nursing mother in England, and to some extent in Ireland. The most immediate response to war-time reduction in alcohol consumption in England was the reduction of deaths from asphyxiation from overlying of infants. That was the most immediate and striking change in the mortality picture.

"The ratio of asphyxial to all other deaths, according to Federal experience, checks quite closely with that of the City of New York, and constitutes approximately 18 per 100,000 per year. That is, if we assemble all these different several cases of deaths from asphyxiation into a single category, it would appear that approximately 18 deaths per 100,000 in a year are due to one or another of these causes. Among those the largest element is submersion, and of these the largest number are cases of suicide.

"But how are we to break up into its possible preventable elements this mass of 18 deaths per 100,000 per year? Of course the only way is by the report of the individual physician, and the listing of each case as to its cause and origin. Find out whether by education or official action those cases can be reduced, and whether, when the physician is faced with the particular type of asphyxial death that threatens, he has the necessary skills or can make them available.

"Dr. Wynne has estimated that 400 lives a year might be saved in New York City from asphyxial accidents occurring in little children. Realize that this is twice the number of deaths we now have annually from diphtheria, and see the expense to which we are quite properly going, because we have a considerable knowledge in the matter, to prevent those 200 annual deaths from diphtheria. A little intelligent organization and knowledge of the resources will give us as much quantitatively in the saving of life in asphyxial cases as in diphtheria. We are sensitized, as it were, to the moral responsibility of removing diphtheria. But we feel that the death of an infant from asphyxia is the will of God, an unavoidable accident, something unpreventable and unforeseeable. We must give up that attitude and determine the resources that are now available through the teaching of physiology, through the ingenuity of chemists, physicists, and mechanics, which can be brought to bear at the time and place where these deaths may be expected.

"These are the things that strike one in an attempt to develop prevention. After all, there are only two problems of preventive medicine: one is a knowledge of the expected prevalence, the location, distribution, time, place, age, and occurrence of the disease that we think is preventable. The other is the specific employment of preventive resources. We are further along in the analysis of the specific means of prevention than in a knowledge of the distribution or prevalence of these conditions. As a student rather by proxy and by association with others better informed, I feel that a lack of self-respect affects the medical and statistical interests, and that we have allowed experimental sciences to precede our exact knowledge of a cause of death which may reach to 50,000.

"I share with Mr. Whitney a considerable skepticism as to the certainty that these 50,000 deaths can be prevented. But it is a working model. It is a theory to go on. We may be able to save them. We may not be able to hand over to our successors $250,000,000 of life values a year by our present knowledge, but we can go a long way, even if we don't consider the child in the womb as a value. There are many persons unnecessarily sacrificed in a year because of lack of use and availability of technical and lay knowledge.

"I would like to call your attention to a time when we shall all have our minds on the power of government, the transfer from the individual to government of great and very broad powers. We have not used the power of the Health Department, the authority of the Sanitary Code, or the laws of the state to require, as we might conceivably think it worth while to do, that any institution offering general medical and surgical diagnosis and treatment to the public shall have certain minimum equipment for emergency recovery. We don't require that. It is to my mind very important that we should not interfere with the proper development of individual initiative by setting up a pattern of what the state or a city law might require. We must make it customary for hospitals that offer to meet any emergency to have certain equipment available, but to require that would, I believe, be going beyond our present medical and scientific knowledge.

"The State Department of Health can require that no laboratory offering diagnostic service should be without certain facilities for diagnostic aids in infectious diseases; that their technique must be of a certain quality; that they must have personnel of a certain skill. If we should attempt to translate that theory, that practice, into the operation of our hospitals I believe we would do damage at this stage. We are in the experimental stage. We don't know just what instruments or skills or materials are going to be always necessary and must therefore be required. It is rather for us to make it common for institutions to have certain minimum essential conveniences with which to meet what we can show them are rather frequent emergencies. From the statistical point of view we can say for every 10,000 patients that come to you in a year, for every so many people coming either on foot or on wheels to your institution, there are going to come in the ordinary course of experience so many persons with this and that asphyxial emergency, and that to care for those it is suggested that you have this and that kind of equipment.

"The final stage in any movement such as this is initiating will be expressed, of course, when the actuaries, underwriters, and financially interested concerns attempt to express as a fixed human hazard these particular forms of accidental death as a basis of insurance or individual protection. That is a stage which I am sure, when the time comes, the Association of Underwriters will be interested in taking up. The insurance companies quite aware of these deaths have, you might say, lost the significance of them, because the international list of causes of death has stressed etiological factors and anatomical types of causes of death rather than having deaths from a particular cause assembled under one title. It will take a considerable rearrangement of all the statistical material now available to answer the kind of questions they have put to those of us who have rationally entered the field of discussing the vital statistics of asphyxial deaths. I might end by saying, "There is no such thing as vital statistics of asphyxial deaths in the United States." This is a matter for the future. First, the incidence of cases. Secondly, the number of deaths properly attributed to them. That will give us the case mortality per cent. Of course we can then say how many were unnecessary or avoidable, and how many were inevitable or irreversible. I like the idea of considering death as an irreversible phenomenon.

The Economic Value of a Human Life

"In 'The Money Value of Man' Hublin and Letka give the present worth (at a discount rate of 4.5 per cent per annum) of future net earnings (that is, earnings less living expenses) for the income class of $1,000 a year as $750 for age 0 and $9,400 for age 30. They actuarially compute the value of the future earnings of a new-born baby at $1,000 a year. This, of course, includes the probability of death at these various ages. If one computes that on an actuarial basis and deducts living expenses each year, one finds on that basis, at discount rate of 4.5 per cent, assuming ordinary mortality, that the value of a baby's life is $750. In other words, the probable value of the difference between the earnings and the living expense for a new-born baby would be $750. For a man aged 30, it would be $9,400. If it is assumed that these figures are applicable to the present case, the economic value of 50,400 lives will be $207,824,400.

The Point of View of Compensation Insurance

"Institutions that insure the obligations of the employer for loss due to work accidents are interested in methods of prevention and in the restoration of the worker to life and health. Education is the foremost method of accident prevention. Insurance carriers hold to the doctrine that all accidents are preventable through education and proper supervision. The state labor department is one of the agencies for establishing rules as a basis for education. In dealing with the subject of accidents due to asphyxiation, the Industrial Code provides the following rule:

Rule 741. All dust, fumes, vapors, gases, fibers, fogs, mists, or other atmospheric impurities that are emitted or created in quantities tending to injure the health of employees in or in connection with any process of manufacture which involves the formation or use of substances. . . . that can be detected by a recognized standard method of chemical analysis shall be removed by means of suction devices as far as practicable at their point of origin.

"Carbon monoxide, chlorine and cyanogen compounds are among the harmful substances which produce injuries unless proper precautions are taken against asphyxiation. Rule 724 of the industrial code provides:

Every workroom in which carbon monoxide is emitted or created in or in connection with any process of manufacture shall be provided with such ventilation that the carbon monoxide shall not exceed two parts in ten thousand volumes of air in any occupied part of such workroom.

Moneys Paid Out in Compensation Insurance for Asphyxial Deaths*
(a comparatively narrow field)

Year	Cause	Cases	Deaths	Amount
1931	CO and CO_2	51	3	
	Illuminating gas	32		$184,000
	Hydrocyanic acid	4		
	Electric shock	647	60	555,853
1936	CO and CO_2	41	2	15,022
	Ill. gas	15		12,584
	Hydrocyanic acid	4	2	5,289
	Electric shock	287	21	246,102
1939	CO and CO_2	36	8	52,811
	Ill. gas	10	12	27,144
	CO	11		21,639
	Electric shock	261	29	299,761

Author's note: Why separate CO poisoning from illuminating gas poisoning?
In 1939, CO appears twice as a separate item.
The figures on electricity would have to be broken down into those which might have been saved and those which could not have been.
It would seem that these losses would justify an annual budget provided by the insurance carriers for the purpose of reducing them, *e.g.*, through intensive education.

"In mines, black damp is formed by the combustion of nitrogen and carbon dioxide. Fire damp is formed by an explosive gas called methane. Acetylene mixed with air becomes an asphyxial hazard in industry. Blast-furnace gas contains from 24 to 30 per cent of carbon dioxide. The extraction of gold from ore requires the use of chemical salts which, when in contact with air, form the deadly asphyxiant hydrocyanic acid.

"I† have encountered numerous cases of preventable losses due to asphyxia. About four years ago two men were killed in a plant located at Corlears and Water streets, New York City. All employees had been

* Special Bulletin #210, N. Y. State Dept. of Labor, Pages 98–100.
† Leon Senior.

warned to vacate the plant on Saturday afternoon, at which time it was to be fumigated by releasing cyanogen gas on the premises. Two of the workmen either neglected to comply or forgot the warning and went to the basement to sleep. Both were found dead on the following Monday after the factory had been cleared of gas. A thorough inspection of the premises might have saved the lives of these two men. In another plant in New York City an employee, while admitting chlorine gas into a tumbler of starch in order to bleach it, loosened the coupling that closed the chlorine gas container. He was overcome, was rushed to a hospital, and died of asphyxiation. The accident might have been prevented if the employee had been thoroughly educated in the danger of his occupation. On May 22, 1933, the newspapers, carried an account of a catastrophe in Newark, N. J., in which five men lost their lives in a gas pit of the tannery. A review of the case leaves one astonished at the contempt for the danger shown by the workmen and the firemen who attempted rescue efforts.

"Prevention measures lie partly in the field of safety engineering and partly in the field of medicine. Industrial safety engineers have given much time to the study of accident prevention and to educational work. The time has come for the medical profession to undertake the second part of the problem; namely, the study of methods for restoring the injured after the occurrence of an accident. There is an economic aspect of huge importance to saving the lives of men in industry, most of whom have dependents who have no other resources than the wage earned by the workman. Workmen's compensation is but a poor substitute for that wage, and yet in this country it represents a cost of at least $150,000,000 per annum. This cost paid for insuring compensation benefits under the laws of our several states is the economic contribution, the economic loss, if you please, paid by society for work accidents, many of which are preventable. This economic loss takes no account of the physical and mental suffering, of broken homes, of injured workmen, of widowed mothers and orphaned children. Society has become, so to speak, acclimated to these results of industry's using deadly implements to accomplish its purpose, to the point at which most persons accept the situation as beyond repair. It is the duty of technicians in public economy and in safety engineering and of physicians to arouse the public conscience, to dramatize the facts, and to encourage the men and the organizations that seek by research and study to reduce the social and economic loss resulting from the use of dangerous implements or substances in industry."

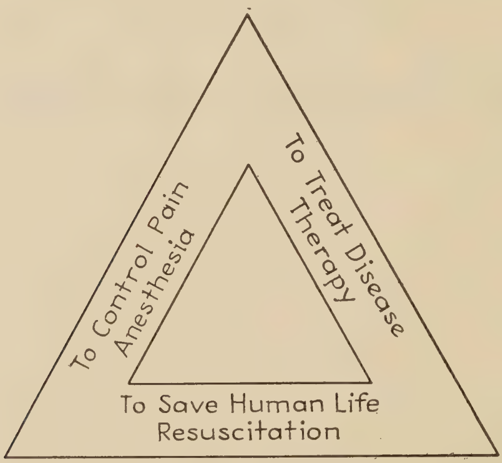
Part VI

The Coordination of Gas Therapy

PNEUMATOLOGY

Definition: "The science dealing with air or gases, their physical and chemical properties and their therapeutic application for the control of pain *ANAESTHESIA*; saving of life *RESUSCITATION*; and the treatment of clinical disease *OXYGEN THERAPY*". "Stedman's Medical Dictionary", P. 878, Williams & Wilkins, 14th Ed., 1939.

"The science of respiration, the science of gases, also their use as therapeutic agents." "Gould's Medical Dictionary", P. 1114, 5th Ed.



PNEUMATOTHERAPY

Definition: "The treatment of disease by compressed or rarefied air. The therapeutic use of gases for the control of pain for anaesthesia, resuscitation, pneumonia, etc." "Gould's Medical Dictionary", P. 1114, 5th Ed.

PNEUMATOLOGICAL TECHNICIANS

Definition: All non-professional personnel, trained nurses, pharmacist's mates, corps men, members of first aid squads of the Police and Fire Departments, the industries and the utilities, engaged in the technical aspects of Pneumatology, *i.e.*, anaesthesia, resuscitation and inhalation therapy." (Author's suggestion.)

Chapter 27

Pneumatology

In the fall of 1929 a meeting of the American College of Surgeons was held in Boston. A group of anaesthetists present were addressed by Dr. Yandell Henderson of Yale. Dr. Henderson stated, "You gentlemen are more than anaesthetists, because you use gases for numerous purposes; you are in reality gas therapists." The spark struck at that instant, smoldered for a period, to kindle three years later in the formation of a society with the avowed purpose of preventing deaths occurring from asphyxiation. Interest became increasingly active in many quarters in the problem of the use of gases for the saving of life and for the treatment of clinical disease.

Exactly 140 years before Dr. Henderson made his remark in Boston, pneumatology* as a special branch of medicine was established at Clifton, near Bristol, England, through the opening of the Pneumatic Institute of Sir Thomas Beddoes.

Beddoes and his associates were enthusiastic over the results achieved by the use of oxygen and other gases in the treatment of pulmonary and other diseases. Enthusiasm for this special field, however, outran the actual results achieved and a reaction set in which extended over a period of years, during which the value of pneumatology was questioned in well-

* In a search for such a term, among those responsible for the nomenclature of disease, the following terms have been suggested: inhalational therapy, asphyxial medicine, apnotherapy, aeriotherapy, aerotherapy and pneumatotherapy.

The term selected should have a sound derivation, a certain familiarity in appearance and lend itself to an elaboration to cover the science, the technique and the technical personnel.

"Inhalation therapy" and "asphyxial medicine" do not lend themselves to the acceptance of the suffix, "ology," or science. "Aerotherapy" and "aeriotherapy," while descriptive of the use of gases for therapeutic purposes, savor too much of aeronautics. "Apnotherapy", signifying breathlessness, lacks familiarity. "Pneumatotherapy" is suggestive; it is derived from the correct roots and suggests inclusiveness. "Pneumon," lung, and "pneumatics," the science of gases, is covered by the scientific term, "pneumatology", which is readily expanded into "pneumatotherapy" and "pneumatological technician."

Should "pneumatology" be found acceptable, it would include as a generic term, the specific activities referred to as anesthesia, resuscitation and oxygen therapy. The objection that anesthesia includes also the use of local, conductive, rectal and intravenous methods may be met by the fact that at least 80 per cent of all anesthetics administered are produced by the use of such gases as nitrous oxide, oxygen, cyclopropane, ethylene, ether and chloroform, the vapors of the last two being regarded as gases in practice.

informed quarters. Toward the middle of the nineteenth century, however, the discovery by Long and Morton of ether, a new gas for the control of pain, yielded such dramatic results that Oliver Wendell Holmes was constrained to give this practice a new name. He called it "anaesthesia." Overnight, the pneumatologist became the anaesthetist, limiting his activities thereby for three-quarters of a century to the administration of gases for the control of pain. The new terminology, *anaesthesia*, was unfortunate because it restricted the use of gases for therapeutic purposes to the field of surgical procedure, retarding for more than half a century active development in the improvement and popularization of the use of gases for the saving of life, *resuscitation*, and the treatment of clinical disease, or *inhalation therapy*, including other new valuable gases such as carbon dioxide and helium.

It is to be expected that the personnel engaged in the field of anaesthesia, which covers the larger volume of patients, and provides routine material as well as a wide experimental background, should provide the personnel from which the pneumatologist will emerge. The pneumatologist will radiate instruction and control over the non-medical personnel now engaged in the administration of anaesthetics, in oxygen therapy, and in resuscitation. The pneumatological technician will fall into his natural relationship to medical direction and control. Owing to the scope of the field, the present competition existing between the technical and the medical group is automatically eliminated.

The historical background referred to, indicating the normal scope of pneumatology, is now being confirmed in an extraordinary manner in ultramodern activities. The New York *Sun* of August 14th, 1936, released the following news item:

"Scientist who inspired Col. Lindbergh in the creation of the artificial heart for the culture of organs operated through the use of gas, is an anaesthetist and a specialist in combating asphyxiation."

That the air-conditioning of high-altitude passenger planes involves an understanding of the behavior of gases and the prevention of asphyxiation in rarefied altitudes has been confirmed by a letter received from the president of one of the largest airplane corporations. In the *Medical Officer*, official organ of the United States Public Health Service, an article appears on asphyxiation as it relates to public health problems (November, 1937). On February 28th, 1939, a communication was received from the Surgeon General of the United States Army, Dr. Reynolds, referring to the extreme importance of gases in warfare.

The problems of submersion, of carbon monoxide poisoning and of the seventeen other causes listed in an editorial in the *American Journal of Surgery* for November, 1939, indicates the scope of the situation which is faced.

Pneumatology may well be looked upon as a man's job, demanding the widest experience and skill, and the best personnel which the medical profession has to offer. Where, we may ask, is this personnel to be found, and how shall it be developed? It is natural to turn to the physicians now administering anesthetics as a nucleus for such a group.

While it is insisted that anaesthesia is of vital importance, on a par with the surgery itself, the fact remains that the art is a contributary one; it complements the work of the surgeon and is subject to him. It does not function *per se*. Surgery may be done without anaesthesia, but there is no excuse for anaesthesia without surgery (except in occasional nerve blocks). As a consequence of this relationship the anaesthetist is, by the nature of his work, assistant to the surgeon; by and large he must do as he is told. This relationship is bound to result in more or less of an inferiority complex which affects his relation to fellow physicians—unless his field of activity is extended to include independent action. As an anaesthetist, he may claim numerous other activities as lying within his field. Such claims are but wishful thinking, however, for unless he employs a terminology which includes such fields his title fails to describe his knowledge or limitations. The general practitioner may give an anaesthetic and be a respected general practitioner. An anaesthetist who does general practice remains an anaesthetist, and in the minds of many is but a glorified technician.

The qualified pneumatologist must be acquainted with the use of gases for the control of pain, and the less frequent but important regional, basal and intravenous methods. He is an anaesthetist but much more as well, for pneumatology in many of its phases implies an art which is separate and distinct and not subject to the direction of another specialty. Whoever applies and directs resuscitation is in charge of the situation; whoever controls problems relating to high-altitude flying or submarine disaster, with its complicating factors of compression and decompression, acts as an independent directing physician in charge of the patient under treatment; whoever directs instruction to large groups is a physician practicing independently.

In spite of the fact that the anaesthetist has served as assistant for 75 years we believe that, given the opportunity, the moral support, and the financial subsidization for institutional activities which this field implies, he will bring to bear his unique training and experience in the integration of a specialty second to none in its professional, public, and military importance.

The popularization of pneumatology along the lines which we have discussed will immediately draw into the specialty highly qualified men who have been unwilling to enter anaesthesia because of the unfortunate confusion which has existed in the popular mind between the physician who administers anaesthetics and the nurse anaesthetist or anaesthetic technician.

It is greatly to be doubted whether any nurse, however skillful in the administration of anaesthetics, was ever desirous of assuming the professional obligation of diagnosis and prescribing treatment necessitated by unforeseen emergencies. Well-trained, non-medical persons make excellent anaesthetic technicians, and when operating in a carefully supervised professional circle carrying on consistent routine they may frequently show a record of low morbidity and mortality. This condition, satisfactorily carried on for years by the many large medical centers, leaves little to be desired as to safety to the patient and a satisfactory field for the surgeon. It must be admitted, however, that such a service is static, depending altogether upon the vicarious interest of the operating surgeon in the subject of anaesthesia. Its development turns upon qualified supervision by a trained pneumatologist.

On the other hand, when such technicians are transplanted to the environment of the small suburban institution, the morbidity and the mortality which might be expected from unsupervised technical work promptly results.

Viewed from the field of pneumatology, the nurse anaesthetist remains the anaesthetic technician. The rescue squads of the Fire, the Police, Consolidated Gas, Red Cross, and other groups fall naturally into the position of technicians in resuscitation.

Men and women in charge of oxygen equipment, such as tent and catheter services, are automatically classed as technicians in therapy.

These technicians in anesthesia, resuscitation and inhalation therapy, both intramural and extramural, should fall under the direct supervision and direction of the pneumatologist in charge of the administration of gases for therapeutic purposes.

It has been argued, Granted the importance of the administration of gases in anaesthesia, what is to become of the skilled administration of spinal and basal anaesthesia, and intravenous techniques? This question is answered in the statistical frequency with which these methods of anaesthesia are used. A survey of all of the general anaesthesia administered throughout the United States would doubtless indicate that between 70 and 80 per cent of it is induced or completed by the use of anaesthetic gases. Spinal and other methods, therefore, fall under the control of pneumatology in the same manner as a transfusion of a cardiac stimulant would be in the hands of the pneumatologist in charge of resuscitation.

Conclusions

Present trends demand the logical integration of all activities having to do with gases used therapeutically, *i.e.*, for the control of pain, the saving of life, and the treatment of clinical disease. In order to coordinate these fields it is suggested that the restrictive term "anaesthesia" be dropped and the comprehensive and scientific terminology "pneumatology" be employed.

In adopting the title "pneumatologist," the anaesthetist claims and controls a field in the practice of medicine which began with Beddoes in 1789—155 years ago—and which has come to include problems of a most vital character in public and military affairs. Evolving from the status of a surgical assistant, the pneumatologist provides a greatly-to-be-desired direction over the technicians in anaesthesia, resuscitation and inhalation therapy. He is prepared to serve with equal skill the surgeon, the hospital, the city, the state and the nation.

Wartime Needs

The mass demand for medical service implicit in war emphasizes the need for simplicity in medical procedures. Routines recommended by the Army, Navy and Public Health Services stress this point.

Anaesthetists in particular must adopt simplicity and resourcefulness as cardinal principles. Under combat conditions or in a major civilian disaster they will have to dispense with many of the newer anaesthetics, which require complicated apparatus for safe use, and return to basic agents and techniques. The physician-anaesthetist should not limit himself to the control of pain. He should invoke the whole broad field of pneumatology, not only for anaesthesia but for resuscitation and therapeutic purposes.

Never before in its 155 years of history has there been as great a need, or as great an opportunity, for broad application of pneumatological principles. The physician trained in them can help to combat poison gases and assist in the solution of the opposed pressure problems presented by submarine air-conditioning and escape and high-altitude airplane flights. Unfortunately, at the present time there is no coordination of pneumatological research and practice in the Army, Navy, Public Health Service, Veterans' Administration and Public Health Aeronautical Authority. Problems of high and low pressure, poison-gas control and the use of gases for anaesthesia, resuscitation and treatment of clinical disease are handled as separate fields.

Coordination of these intimately related activities should be promptly effected, both to foster continued scientific development of pneumatology and to insure proper application of its teachings. There must be pooling of scientific material, standardization of equipment, instruction of the public in the prevention of asphyxia, and proper training and supervision of technical workers in this field.

The nation is fortunate in having several physician-anaesthetists who are outstanding pneumatological experts. Under their direction its resources in material and personnel could be welded into an effective instrument of teaching, research and cure.

Chapter 28

Pneumatology in the Hospital

On December 15, 1933, there was a meeting of the N. Y. Academy of Medicine of the Hospital Information and Service Bureau of the United Hospital fund. This meeting was for the purpose of drawing up questionnaires concerning medical specialties within the hospital which would bear, not only on their equipment and work, but on their relation with other branches of medicine. The author, representing anaesthesia and resuscitation, submitted the following questionnaire with a view to the coordination of the various fields of gas therapy.

"The rapidly increasing use of gases for the purpose of saving life, for the control of pain and for the treatment of disease suggests the necessity of coordinating the activities of those interested in their use for the purpose of providing well-trained personnel and adequate equipment. It is only by such organization that a sequence of instruction may be provided and records of value secured.

"The Department of Gas Therapy provides skilled medical care for *emergency* accidents, such as drug poisoning, carbon monoxide poisoning, asphyxia neonatorum, anaesthetic accidents, etc. It also directs and employs the *routine* use of gases and vapors for inhalation anaesthesia and the oxygen therapy commonly used for acute respiratory affections.

"The questionnaire which follows indicates the elementary requirements of this Department relative to (1) personnel (2) equipment (3) instruction and (4) records."

Personnel

Director

Name
Medical School
Date of graduation from medical school
Preliminary experience in the use of gases
Do you require regular attendance of the Director?
In his absence, who covers acute emergencies?
What authority is delegated to the Director?
Has the Director any other duties in the hospital?
Does the Director hold an appointment as gas therapist in any other institution?
What is the nature of the Director's compensation?

Assistants

Number
Names
Training of each in gas therapy
Are interns appointed as assistants?
Is a trained assistant always in attendance?

Are his whereabouts known at all times to the hospital telephone operator?
Are your assistants capable of employing and directing the use of the following:
(1) The Schaeffer prone-pressure method of artificial respiration;
(2) The use of gases by inhalation and insufflation, with inhaler, nasal or oral tubes;
(3) Can your assistants laryngoscope and intubate an unconscious asphyxiated patient with safety and dispatch?
(4) Are your assistants familiar with the indications, the use and the care of negative-pressure cabinets?
(5) Are your assistants familiar with the use of anaesthetic vapors and gases, ether, chloroform, ethyl chloride, nitrous oxide, carbon dioxide, etc.?
(6) Are your assistants acquainted with the fundamentals of oxygen therapy, and are they familiar with the more commonly used types of oxygen chambers and tents?

Technicians

How many do you employ?
What is the training of each?
Are your technicians familiar with the Schaeffer prone-pressure method?
Do they understand the use of inhalators and insufflation tubes for the emergency treatment of the acutely asphyxiated?
Have they been properly instructed in the behavior of gases as related to the question of pressure, explosibility, care of tanks, etc.?
Are they familiar with the use of pressure gauges such as water and mercury manometers and flow meters of the various types?
When administering gases for anaesthetic purposes who, in the absence of the Director of Gas Therapy and his assistants, is responsible for the choice of the anaesthetic and the method of administration?
Is it customary for the technicians to induce anaesthesia in the absence of a physician?
Is the patient accompanied by a House Officer in transference from operating room to bed?
Is it customary for your technicians to instruct interns?
Do your technicians order medication or change the gases used during anaesthesia at their own discretion?
Do your employ technicians for oxygen therapy? What is their number and training?
Are these technicians capable of making a reliable gas analysis?
Are they capable of reporting the physical condition of the patient?

Equipment

What equipment have you provided for resuscitation?
In what form do you supply oxygen and carbon dioxide?
Are inhalators, nasal tubes and oral tubes available for resuscitation, or are these part of the usual anaesthetic equipment?
At what regular interval and by whom is your equipment inspected?
Is equipment cleaned and sterilized immediately after use, and is it put away in sterile towels?
Is this equipment always available, or is it under lock and key?
How many laryngoscopes do you provide and where are they located?
How frequently are the lights inspected?
Have you facilities for intubation and insufflation of infant and adult?
How frequently do you carry out resuscitation drills to develop emergency teamwork?
Are your technicians familiar with the operation of resuscitation apparatus?
What equipment is available for the administration of ether (cyclopropane, etc.)?
What equipment for nitrous oxide and oxygen?
Have you suction available in each operating room? Is this electrical, central or steam?

What equipment have you for ether anaesthesia about the head and neck; for gas anaesthesia about the head and neck?
Is the equipment which you use in contact with the patient's face and for rebreathing purposes, sterilizable?
Do you recommend the use of gloves by the anesthetist?
Have you facilities for grounding static sparks?
Do you heat blankets for operating room use in a warmer?
Is it your practice to supply a change of operating gown before removal from the operating room?
Is a mouth gag and airway carried with the patient on his return to his room?
Have you suction in rooms used for the recovery of post-operative nose and throat cases?
What facilities have you for the administration of oxygen to asphyxiated pneumonia and cardiac cases?
What equipment have you for nasal delivery?
With regard to the air-conditioning of oxygen tents which you employ:
  What is their cubic capacity?
  What is their temperature control?
  What means are employed to regulate the oxygen-CO_2 concentration?
  What degree of humidity within the tent do you allow?
  Is the tent equipped with thermometer and hygrometer visible from the outside?
  How frequently are the contents of the tent changed?
  What percentage of oxygen do you ordinarily employ?
  Are fire danger signs conspicuously placed?
  Are oxygen gauges marked?
  Of what composition is the tent window?
  What is the basis loss of oxygen in the tent you employ?
  How do you sterilize your tent after use?

Instruction

Does your Director deliver lectures to interns and the general nursing personnel?
How many lectures were given last year; what subjects were covered?
What instruction is given interns by assistants?
Is there a record of instruction and demonstration available?
Do technicians instruct visiting medical students?
Is a departmental conference held, and how frequently?

Records

When and by whom made?
  Please submit a sample of the record form which you employ for
    (1) Resuscitation
    (2) Anaesthesia
    (3) Oxygen therapy
Do you employ a nurse-secretary to keep records of equipment, check losses and report equipment out of order?
What plan of organization have you regarding attendance and duties of personnel?
What sequence have you in filling vacancies?
What compensation do you pay your assistants; your technicians?

At the request of the Bureau, the following supplementary matter was submitted.

While the anaesthetist, because of his familiarity with the phenomenon of unconsciousness, is called upon to relieve acute asphyxia from its many causes, and because of his knowledge of the behavior of gases is likewise expected to be familiar with the indications and use of oxygen therapy, an important question of terminology and departmental organization must be considered.

A. It may be conservatively estimated that between eighty and ninety per cent of all anaesthesias are brought about by the use of gases.

B. Certainly the major factor of resuscitation is the administration of oxygen and carbon dioxide.

C. Oxygen therapy for pneumonia and its allied anoxemic states is entirely a question of gas therapy.

As suggested by Yandell Henderson at a meeting held in Boston in 1929, the physician who administers gases for anaesthesia, who practices resuscitation and who employs oxygen therapy is in reality practicing more than anaesthesia. He is a gas therapist, and the work in which he is engaged may be referred to as gas therapy.

The practical application of such terminology may readily be seen in the following references:

The gas therapist (anaesthetist) is called to treat a case of submersion.

The gas therapist (anaesthetist) is called to relieve carbon monoxide poisoning.

The gas therapist (anaesthetist) is called to set up equipment for acute lobar pneumonia with a high degree of anoxemia.

The gas therapist (anaesthetist) is called to relieve cardiac decompensation.

The gas therapist (anaesthetist) is called to treat a case of electric shock.

The term *anaesthesia* implies the artificial production of unconsciousness.

The term *gas therapy* implies not only the production of unconsciousness, but also the treatment of such a state with a view to its recovery.

Objection: It is argued that the term *anaesthesia* is well established and its replacement implies a great deal of unnecessary resistance.

Answer: This is granted, but since the questionnaire under consideration is forward-looking, implies revaluation and an attempt to set an ideal, the objection does not hold.

Objection: Anaesthesia is brought about through local and conductive methods as well as by basal anaesthetics. This is not gas therapy.

Answer: Eighty to ninety per cent of all anaesthesia is brought about by the use of gases and vapors. The ten to twenty per cent exception noted above offers no more reason for objection than hypodermic medication in resuscitation and in oxygen therapy.

The secularization of anaesthesia by the increasing employment of technicians during the last fifteen years has destroyed morale. This state of affairs not only offers resistance to the entrance of new graduates of medicine into the field, but reduces the professional status of those at work. The reduced number and quality of medical men devoting themselves to anaesthesia hamper the adequate care of cases requiring resuscitation and oxygen therapy.

The proposal of an up-to-date and extensive field of professional endeavor, offering as it does the substantial, constant, familiar specialty of anaesthesia, serves to attract a well-trained, competent medical personnel.

The term "pneumatology (the use of gases for therapeutic purposes) is recognized in medical nomenclature. It is suggested that it be generally adopted.* The chief reason for urging the adoption of this terminology is to provide an organized service for all types of asphyxial emergencies both within and without the hospital. While the medical anaesthetist should be in charge, his present designation is restrictive and limits his activity in the wide field of gas therapy, which extends beyond the control of pain.

The movement to prevent asphyxial death is two-fold: medical education over a ten year period and a present emergency problem. The hospital and general practitioner must meet the emergency during the next decade.

The prevention of asphyxial death by medical organization, *i.e.*, establishment of pneumatology, is an answer to the question of how to prevent asphyxial death occurring in the hospital and outside it.

The activity is recognized as necessary. (See comments of a number of the 335 hospital superintendents representing over 100,000 beds of the Committee on Hospitals of the S.P.A.D., page 17).

It is granted that medical education will take up the slack in the next ten years. The beginning has already commenced in an interesting correspondence from sixty professors of obstetrics in as many medical schools in the United States and Canada (page 121). In the meantime, the prevention of asphyxial death, largely in the hands of technical groups, is quite at the mercy of commercial propaganda†.

There is no intention of bringing about "rigid uniformity of method." The intention is to point out the pathologic physiology underlying the adoption of all reasonable methods.

It is maintained that these principles are not generally understood, but are self-evident when pointed out, *i.e.*, stages of asphyxia are not generally recognized, and the indications for inhalation or insufflation treatment are not generally known.

Specific Considerations

(a) To attract attention to the need of a specialized knowledge in the field.

(b) To place hospital routine and experimentation in their proper relation, *i.e.*, reduce the morbidity and the mortality resulting from "the individual trial and error technique."

(c) By pointing out a path based upon fundamental, generally accepted principles, waste may be reduced in the purchase of equipment likely to be dangerous or to become obsolete.

* *The Health Officer*, p. 292 (November) 1937.

† *Science*, **99**, 469.

(d) To accumulate statistical material for the purpose of strengthening a rational approach to a new but major medical problem, *i.e.*, the loss of 50,000 lives a year by asphyxiation.

(e) To provide direction through medical channels to lay groups, functioning as rescue units in the municipal, industrial and utility fields.

The foregoing principles apply to resuscitation, and depend upon the qualified medical anesthetist for their proper functioning. The hospital anaesthetists become automatically responsible for the emergency care of all classes of asphyxial emergencies covered by the Department of Pneumatology. Because of his acquaintance with anoxemia, the Director of the Department of Pneumatology will frequently be called upon to cooperate or direct the use of gases (oxygen, helium, etc.) for the treatment of clinical disease, so-called oxygen therapy.

In view of the above facts, details of which are available, the writer urges the establishment of Departments of Pneumatology in all City Hospitals. As a means of bringing about and assisting such establishment, the operation of the post as Pneumatologist to the Department of Hospitals of the City of New York was requested.

In Feb. 15, 1936 an invitation was extended by the Society for the Prevention of Asphyxial Death to all hospital superintendents in the United States conducting institutions with 100 beds or more to serve on a National Committee on Hospitals for the following purposes:

To bring to a focus hospital administrative experience with the problem. To point out the incidence, or the relative frequency of death from the various causes of asphyxiation which take place within the hospital.

To formulate a policy which will enable the hospital to reduce this mortality in an effective and economical manner.

The hospital superintendent is in a position to help make the hospital "asphyxia-conscious" by:

(1) Requiring detailed reports of asphyxial death occurring in the various hospital services.
(2) By causing to be posted and distributed suitable literature relative to the problem of asphyxia.
(3) By requiring the instruction of house officers in modern medical methods of resuscitation.
(4) By encouraging the integration of activities within the hospital through which the control of asphyxial death may be directed and carried out, for example, anaesthesia, oxygen therapy and resuscitation.
(5) By calling the attention of the Nurses' Training School to the problem.
(6) By extending his good offices to secure the interest and the support of the lay groups associated with the hospital.

In answer to the 1700 personalized and signed letters of invitation sent out, acceptances were received from 335 hospital superintendents, representing 46 states. Twenty of these institutions had bed capacities of 2,000 or more. As evidence of administrative interests in the prevention of asphyxial death, the following comments are quoted:

Illinois: "I shall deem it not only a privilege, but a pleasure to serve on this committee." . . . "I believe that there are great possibilities for accomplishments by your Society. Unfortunately, many physicians and even hospitals are not competent to deal with the many problems involving asphyxial death."

Indiana: "I deem your cause a most worthy one. Please count on me for anything that I or my Institution may be able to do."

Kansas: "I will outline our experience (in epilepsy) which is rather unusual. The circumstances surrounding these cases make our problem a different one."

Kentucky: "Please call upon me for anything that I can do at any time."

Maine: "I have noticed the organization of your activity, and believe that it is of much importance to the administration of an Institution of this kind." . . . "I will be glad to do what I can to ward off preventable death."

Maryland: "I appreciate the opportunity to have the name of this hospital connected with your organization."

Massachusetts: "I will be glad to cooperate with you in every possible way, and wish you success in this very fine work."

Michigan: "I can see, if your organization is to prosecute actively, the functions that could be expected to issue from such an organization. I could learn much from the opportunity to read your issuances. In that sense, I am glad to accept the membership."

Missouri: "I shall be pleased to serve in the capacity of cooperating with you in every way to obtain information concerning asphyxial death."

New Hampshire: "The names which appear on your Advisory Board makes me know that your Society is one of the highest possible standing, both ethically and professionally. I am happy to accept your invitation."

California: "I am naturally interested in such prevention. I gladly accept, and will do what I can through correspondence."

Connecticut: "I cannot refuse! I accept your invitation." . . . "While a State Tuberculosis Sanatorium would not seem to present this type of death as one of its chief problems, it is possible that a pulmonary hemorrhage and a clot formation might be responsible for such a condition."

Colorado: "The subject is of considerable importance to State Hospitals, for quite frequently cases of asphyxial death are seen by staff physicians."

Delaware: "I assure you that I will do everything in my power to assist you in your work."

District of Columbia: "I am very glad to serve as a member of this Committee, and to assure you of the moral support of this Institute."

Florida: "I think it is an excellent project and I shall be glad to cooperate with you in any way that I can."

New Jersey: "It will be a pleasure to be of service in any way possible." . . . "I am not sure what assistance I can give, but naturally I am in sympathy with the project pointing in the direction of preventable death."

New York: "I consider the subject of great importance."

While the interest aroused by this contact with medical superintendents has been of the greatest possible benefit, subsequent contacts became impossible because the Society was unable to secure funds to carry on the project. It may be pointed out that there is little reason to believe that the interest exhibited in 1936 exceeds that which now obtains (1944).

Chapter 29

Pneumatology in The Medical School

The coordination of gas therapy is a problem of medical education. The need for such coordination has been indicated in the preceding pages; the method by which it may be effected is now briefly described. Two approaches are available: (1) segregation, in which relevant matter in the various specialties is stressed, and (2) integration, in which such relevant matter is assembled, to form a new department.

(1) *Coordination by segregation* is a method of expediency; it depends upon creating interest among departmental heads and success in soliciting the cooperation of each. It may be effected informally by contact during social visits, luncheons, teas, etc. In the course of these conversations emphasis is placed upon various aspects of the specialty bearing on the problem; for example, instrumentation of the airway, in relation to the orientation and measurement of the structures involved, may be discussed with the professor of anatomy; the practical effects of the presence or absence of the various reflexes in anaesthetic and resuscitative procedures can be talked over with the professor of physiology; the behavior of gases involving partial pressures, inflammability, and compressibility as these apply to gas therapy, can be taken up with the professor of physics; the composition of gases and the manner of their elimination in anaesthesia and resuscitation may be discussed with the professor of chemistry; the practical differences between intrapulmonary fluid and intrapulmonary edema, the effect of instrumental trauma, regeneration following injury, etc., will interest the department of histology; the very large and important field covering the neurological lesions will appeal to the professor of neurology; the pathologic physiology of the various causes of asphyxia is of interest to the department of internal medicine; the effects of gas pressure, oxygenation and its control, massive collapse of the lung, atelectasis, etc. are of great importance to the department of thoracic surgery.

(2) *Coordination by integration* may be achieved by enlarging departments of anaesthesia and operating these under appropriate and descriptive terminology. (Experience has clearly demonstrated that it is both illogical and impractical to attempt to expand anaesthesia to include the extra-operative field of resuscitation and inhalation therapy. A part cannot be greater than the whole. See page 171.) This term should be both inclusive and scientific. The prospectus which follows is offered for the use of the forward-looking medical school.

Pneumatology

This specialty coordinates the use of gases therapeutically employed for the control of pain (anaesthesia), the saving of life (resuscitation), and the treatment of clinical disease (therapy). It considers within its scope the less frequently employed methods of pain control—local, regional, basal, and intravenous—as well as the use of narcotics, sedatives and transfusions employed in resuscitation and therapy.

The Department of Pneumatology offers a three-year curriculum leading, upon its satisfactory completion, to the degree of Master of Science and Medicine (Pneumatology). This curriculum embraces a year of college work devoted to a study of the basic sciences and fundamentals of Pneumatology, and two years of residence in an acceptable hospital. This three-year period of study is divided for convenience into three academic sessions of approximately one year each. The theoretical course corresponds to what is frequently referred to as a "refresher course" in the fundamental background of the therapeutic use of gases. Based upon this, the technical problems associated with the administration of gases are intensively studied and put into practice in order to prepare the pneumatologist for the position which he will shortly fill as instructor and director of the large technical groups in hospitals and in the fields of municipal and public health who are now responsible for the administration of gases for the saving of life, the control of pain, and the treatment of clinical disease.

In addition to the completion of the three years' course, a research problem in resuscitation, in anaesthesia, and in therapy must be submitted to the departmental faculty and approved before the candidate may be recommended for the degree. For information regarding admission requirements, administration, fees, etc., see General Statement.

First year: **Theoretical Approach***

During this period of approximately nine months the student physician serves as a non-resident fellow in pneumatology in the medical college. Systematic progressive courses of instruction are scheduled in the basic sciences applicable to this specialty, as indicated; courses in theoretical and practical pneumatology are also available.

Anatomy. A course in anatomy is given, with special reference to relations, measurements, and general orientation through complete exposure by oral endoscopy, as well as by post-mortem sections, of the structures of the upper airway. Comparative anatomy of the infant and adult cadaver is stressed. Emphasis is placed upon the anatomical location of accessible blood vessels for purposes of transfusion, medication, and intravenous

* Acknowledgment is made to the "Department of Post Graduate Study, New York Medical College," from which material in this section has been adapted.

anaesthesia. A course in regional anatomy with special reference to nerve blocking for regional anaesthesia is provided.

Physiology. A study of infant, child, and adult physiology with special reference to reflexes, muscle tone and impaired function from the point of view of anaesthesia, resuscitation and therapy is stressed. The purpose of this course is to emphasize the difference between living and dead tissue and structures as these are encountered in the field of pneumatology.

Biochemistry. Demonstrations covering the chemistry of tissues, metabolism, and hormones, the respiration, water balance acid balance, and functional tests as these apply to anaesthesia, resuscitation and therapy are carried out.

Chemistry and Physics. The chemical makeup of anaesthetic and other gases used for therapeutic purposes with special reference to their elimination is studied, together with the physical behavior of gases with special reference to partial pressures, compressibility, explosive factors, and portability.

Pharmacology. Laboratory work dealing with the chemistry of gases and drugs as well as agents employed for controlling pain, saving life, and treating clinical disease is required.

Pathology. Special consideration is given to pathological lesions of the pulmonary and circulatory system as these relate to anoxemia. Special emphasis is placed upon the destruction of nerve tissues by anoxemia. Reference is made to blood diseases that affect the action of anaesthetic gases and drugs, such as the anemias, purpuras, and leukemias, as well as to the pathological effects of (1) poisonous gases which frequently result in asphyxial death—for example, gases used in warfare, toxic gases in industry (carbon monoxide, ammonia, carbon dioxide) and gases for fumigation; (2) of drugs such as hypnotics, narcotics and sedatives, including acute alcoholism; (3) of overdose of anaesthetics, (4) of submersion, and (5) of electrocution. Special reference is made to the pathological lesions from asphyxia neonatorum, suffocation, strangulation, as well as from tumors and foreign bodies in the airway. The pathology of terminal poliomyelitis is also discussed. The microscopic anatomy of the respiratory tract is studied with special reference to gas exchange, regeneration following trauma, and inflammation. The special requirements of thoracic surgery in pneumatology, the effects and the control of gas pressure, oxygenation, the problem of bilateral surgery and the treatment of extensive tuberculosis and its complications are considered. Special attention is accorded to types of pathology which have recently been referred to the pneumatologist for relief; traumatic or surgical collapse or shock, abdominal distention, headache following encephalography, certain types of migraine, profuse pulmonary edema, massive collapse of the lung, pulmonary embolism, angina pectoris and other cardiac conditions, infections due to anaerobic organisms, such as those of gas gangrene and tetanus, and certain infections possibly due to partially anaerobic organisms.

Clinical pathology. The indications for blood transfusions and a review of the techniques usually employed, as well as reference to hemo-radiation and its effects on the oxygen-carrying power of the blood are presented.

Medical jurisprudence. This includes the relation of the law to death from asphyxia; local rulings relative to operating-room deaths; national death returns as these refer to asphyxial accidents; and a careful survey and record of all asphyxial deaths under the supervision of the university hospital.

Medicine. Physical examination with special reference to pre- and post-anaesthetic examination; examinations which have special reference to the treatment of clinical disease, and its treatment by pneumatology, such as massive collapse of the lung, atelectasis, laryngeal obstruction, pneumonia, asthma, and advanced tuberculosis. Familiarity with the physical signs and diagnosis of death, as these are determined by the absence of heartbeat, by the intra-cardiac needle, by blood color during oxygen insufflation, and by the eyegrounds in terminal asphyxia.

Radiology. The interpretation of radiographs with special reference to pathology of the airway, *i.e.*, collapse of the lung, atelectasis, pneumonia, malposition of the trachea from pressure and tumors; interpretation of foreign-body obstruction involving respiration in the employment of gases for anaesthesia, therapy, and resuscitation.

Supplemental Information. Library and research technique and Journal Club. Stated meetings of the Journal Club for the study of current literature dealing with pneumatology.

Clinics. This course includes operating-room work, ward work and ambulance service emergencies in which pneumatology is called upon to function, and covers also the evaluation of operative risks, choice of anaesthetic, and method of administration, through the stages of induction, maintenance and recovery, and post-operative ward care of the patient. This overlaps the field of post-operative clinical disease, and the use of oxygen or helium employed by catheter, inhaler or tent. The relative value of liquid and compressed oxygen is stressed, as are also the supervision of technical attendants upon such cases, attendance and demonstration upon accident cases brought into the emergency room, and attendance en route by ambulance.

Laboratory. Laboratory work and experimentation are done on problems illustrating reflex irritability, the factors altering metabolic rate and methods of controlling them, and the effects of anaesthetic gases and drugs upon normal physiology.

Extramural activities. Emphasis will be placed upon the fact that pneumatology is by no means limited to operating-room activity or ward supervision. Emphasis is to be placed upon the obligation of providing a thoroughly modern resuscitation service not only within the hospital for asphyxia neonatorum, drugs, hypnotics, narcotics, acute alcoholism and pulmonary conditions, developmental abnormalities, anesthesia overdosage, mechanical obstruction from foreign bodies in the airway and trachea, allergy, terminal polio, polyneuritis, and facial diplegia, but in every ambulance servicing asphyxial accidents which occur in public places, (gases used industrially, carbon monoxide, refrigerants, chemical fumes, fumigation, submersion, fire-fighting, electrocution, strangulation, hanging, suffocation from soft materials and from external pressure of moving objects, *e.g.*, automobiles, elevators, laundry machines, from collapse of buildings, earth mounds, sand, coal, landslides in manholes and declivities).

The Pneumatologist will understand that his work has a bearing on public health scarcely second to his hospital activity. Opportunities are offered for reference to the United States Army, Navy and Public Health Medical Departments as these national bodies offer occasion for Pneumatological service.

Reference is made to the airplane industry, with special attention to the air-conditioning of high-altitude planes and the effects of high-altitude anoxemia; to the Navy, with special emphasis on escape equipment and air-conditioning of submarines; and to industry, including a visit to construction employing positive pressure and decompression techniques.

The student will be advised of his responsibility to instruct, upon request, fire, police and industrial First-aid groups, as well as those sponsored by the Red Cross, private camps, and other organizations. Acquaintance with the instruction provided will greatly help to put an end to the present dearth of information and the total lack of supervision in many fields now employing gases for the saving of life, the control of pain, and the treatment of clinical disease.

Second Year

The course in pneumatology provided in the first year is a per-requisite for admission to the second session. Selected students are appointed as residents in pneumatology for a period of approximately one year. During this period of residence each student physician is on hospital duty and carries out all assignments of the faculty and staff in the operating room, wards, out-patient departments and ambulance service. Intensive guided training is thus provided in case workups, indications and contra-indications for various anaesthetic agents and techniques, resuscitation techniques and the treatment of clinical diseases by the use of gases. The normal integration of these fields is emphasized by the practical application of pneumatology. In addition to hospital and dispensary work, participation at clinical conferences, departmental meetings, journal clubs, and the extramural activities noted above are required.

Upon the satisfactory completion of this session appointment as resident in pneumatology may be continued for a third session. Before the work of the second session may be considered satisfactory, the student must have selected and have had approved by the departmental faculty a subject for research in anaesthesia, resuscitation and therapy to be completed during his third session.

Third Year

The courses provided in the first and second years are prerequisites for admission to the final year. This comprises approximately one year's work as resident in pneumatology and is a continuation of the second year. Special emphasis is placed upon the administrative function—the supervision, instruction, and direction of technicians in anaesthesia, as these are found in the operating room; in resuscitation, as organized in the wards and ambulance service, as well as in municipal and other groups which may wish to take advantage of these modern opportunities for guidance; and in therapy, as it applies to technicians in the wards and the ambulatory groups delivering and maintaining inhalation-therapy equipment. In addition to this administrative activity the student must satisfactorily complete three research theses, and have these approved by the departmental faculty.

Note: A single thesis bringing to a focus the integration of the fields of pneumatology in anaesthesia, resuscitation, and therapy will be accepted in lieu of three separate investigations in each of the subdivisions of this major field.

Chapter 30

Color in Pneumatology

The importance of color in pneumatology may be looked upon as a fundamental factor of environment. The interpretation and the measuration of the color of the patient as he enters and emerges from the anoxic state is of first importance to the physician engaged in gas therapy. "Pink patients don't die", but unchanging cyanosis in a breathless patient under oxygen insufflation means death.

The Color of the Blood as a Sign of Death*

The scientific background of the sign of death to be offered for consideration is broad in its scope. Its practical application, however, is of the utmost simplicity. Failure to employ this sign, not only as a measure of viability but to save life, might well be interpreted as an act of gross negligence.

The sign of death to be described is based upon a new means of proving that the heart has stopped, as evidenced by an unchanging cyanosis of the blood, observed through the skin and the mucous membranes in the presence of intratracheal oxygen under pressure.

The conclusions to be drawn from this sign are based upon an integration of anaesthetic experiences accumulated during the last twenty years.

(a) It was first observed that the color of the blood varied almost instantly with conditions which impeded or favored free respiration, *i.e.*, the free interchange of atmospheric and blood gases.

(b) That in the presence of a free respiration the color of the blood, that is, the quality, not the quantity of the color, varied directly with the percentage of oxygen offered for respiration.

(c) That while ordinary conditions are satisfactorily met by the 20% oxygen content of the atmospheric air, many conditions occur in which reduced rate or volume, as well as reduced absorbing surface, demand 50 to 100% concentration to produce full oxygenation.

(d) It is assumed that the color of the circulating blood is directly dependent upon its relative oxygenation; that 100% oxygenation yields the typical cherry red; and that 0 oxygenation yields a deep claret color, which

* Read before the Society of Medical Jurisprudence at the New York Academy of Medicine, February 13, 1933.

when seen through the skin or the mucous membranes appears as a dull blue or slate gray.

(e) Furthermore, this color is due exclusively to a change in the iron (hemin) content of the red blood cells in which the loose combination known as oxyhemoglobin is formed.

(f) A device for measuring this color was reported before the Society of Experimental Medicine and Biology in the spring of 1922. This measurer of cyanosis is known as the Oxyhemoglobinometer.

(g) In the asphyxia which precedes and accompanies death the oxygen of the circulation is gradually decreased until at death the oxygen saturation approaches 0.

(h) The color of the blood as seen in the skin and lips closely indicates the oxygen content of the circulating blood.

(i) A high percentage of oxygen thrown directly into the lungs in the absence of fluid or edema is instantly absorbed by the capillary circulation.

(j) According to Lowery,* the absorptive power of the respiratory mechanism and its consequent sensitiveness will be seen in the following facts: area, 1,000 square feet; thickness of alveolar membrane .004 mm; under ordinary conditions 6,083 cc of oxygen can be absorbed in one minute, only one-eighteenth being required for the oxygenation of the organism.

(k) If the heart is beating, however slowly or feebly, this oxygenated blood is carried to the capillaries beneath the lips and the skin at a rate proportionate to the vigor of the heart's action.

(l) Normally, full oxygenation will occur in 5 to 10 seconds. If there is no change in color in 10 minutes, proof becomes available that the heart has ceased to beat during this interval and that the patient is dead.

It is a routine experience in the practice of anaesthesia and in the resuscitation of the asphyxiated that obstruction of the respiration by vomitus, blood, or sea water, and relaxation or distortion of the soft parts are major causes of suboxidation, and that the removal of these causes of obstruction is indicated as a preliminary to any other treatment. One of the most remarkable and spectacular phenomena in physiology is the prompt change in the color of the patient when obstruction is removed. This phenomenon is seldom seen in the conscious patient because of the effective struggle for self-preservation and the activity of his protective reflexes.

The airway having been freed, the color becomes normal, provided the air breathed contains a sufficient amount of oxygen. As has been noted a marked reduction results in distress, cyanosis, and finally in loss of consciousness.

In the event of depression of the respiration in which both rate and volume are reduced, as in drug poisoning by morphine, etc., the patient will demand a higher than atmospheric oxygenation to maintain his color. Oxygen with CO_2 is then given with the double motive of stimulating the

* *Principles of Human Physiology*, Philadelphia, Lea and Febiger, 1915, p. 907.

respiration and increasing the blood oxygen. When the absorbing surface of the lung is reduced by pneumonia, a higher concentration of oxygen must be given to maintain color. A constant atmosphere of 50% oxygen is in common use in the practice of oxygen therapy in pneumonia.

The color of the blood is due to the red blood corpuscles floating in a comparatively colorless (slightly yellow) serum. The coloring element hemoglobin has a strong affinity for oxygen, with which it combines to form oxyhemoglobin, which, however, exists in a loose combination. This combination is more easily broken up in the presence of CO_2 when present in due concentration (in accordance with the phenomenon described by Bohr). Normal blood quickly clots when released from its intravascular environment. Potassium oxalate prevents this formation of clots and permits us to study the reaction of fluid blood to various gases.

Imagine that we are looking at three flasks, each containing some oxalated blood. Through the first we bubble oxygen; through the second a mixture of oxygen 90% and carbon dioxide 10%; through the third hydrogen, a neutral gas which serves to wash out the dissociable oxygen in the blood. Soon the first flask will contain 100% oxygen saturation, the second will show the effect of an addition of 10% CO_2 to the oxygen and, the third will be practically free from dissociable oxygen.

We therefore have a measure of cyanosis scientifically correct, in practice approximate, by which we may determine changes in the oxygen content of the blood.

A point of extreme importance in the physics of color must here be noted. The color of an object depends upon the color in the light which illuminates it. Daylight contains the full spectrum and shows the full color value of an object. The ordinary electric Mazda light will show the value of red. On the other hand, the so-called daylight bulb is deficient in yellow, which contains red, and is a continual source of annoyance to the student of color.

The color of the blood as seen under the mucous membrane of the lips and under the skin indicates quite closely the state of the oxygen content of the circulating blood. Variations in the translucency of the skin, capillary stasis in the florid patient and stasis due to cold, mask the true color of the systemic circulation.

Oxygen treatment, to be effective, must reach those membranes through which it is to be absorbed, *i.e.*, the lining membrane of the lung (applied in the face of respiratory obstruction it cannot be so absorbed). If the patient is breathing, oxygen by a mask or by a throat tube will suffice. But in the apneic, apparently dead patient, oxygen so applied will not reach the absorbing surface. To accomplish this end a technique now approved by the American College of Surgeons was devised.*

Let us refer once more to the specimens of oxalated blood through

* *American J. Surgery*, **8**: 1204 (1930). *Archives of Otolaryngology*, **12**: 23 (1930).

which we have been bubbling oxygen, oxygen and carbon dioxide, and hydrogen. It will immediately be seen that the oxygen specimen is brilliant red, that there is no appreciable difference between the oxygen and the oxygen-CO_2 specimen, but that the hydrogen specimen is a dark claret or maroon. If we were to examine this specimen chemically we would find that the only change which has occurred has been a washing out of the oxygen. Let us now suppose that this deoxygenated blood, instead of being contained in a flask, was present in the small superficial blood vessels. Seen through the skin or the mucous membranes this maroon looks pale blue or gray. Let us further imagine that we insufflate such an apparently dead patient. The heart, is still beating so slowly and feebly that it cannot be heard by a stethoscope. However, it is still capable of moving the blood from the lungs to the heart and from the heart to the periphery. We therefore observe the change which is apparent when oxygen is blown through the flask. The color of the blood changes, the skin becomes less blue and gradually pink. Should the heart have ceased to function no change will be observed regardless of the pressure or the duration of oxygen insufflation.

Clinical Application of the Color Sign

The color sign is merely a corroborative sign. It is by no means intended to take the place of other signs. This sign is one which may be elicited under favorable circumstances. These circumstances are brought out very vividly in almost every daily paper at least once a week, in the detailed description of the life-saving squad working for eight or ten hours over a patient with an inhalator. They work very hard for ten hours, and the patients still die. In other words, they were very dead ten minutes after they started, and there was no way of determining that fact even though all the facilities for intratracheal insufflation were present. Under these conditions the sign means saving of man power and oxygen. I found myself upon just such a case at Coney Island. This patient was an old woman who had been taken out of a kitchen in which she had been overcome by gas; the police squad were doing the prone-pressure method with the inhalator. We exposed her larynx without the least difficulty. I demonstrated her larynx to the policemen. They were delighted, because they had never seen a larynx before; we used oxygen and CO_2, and carried on intratracheal insufflation without any difficulty—all the time she was perfectly dead, and we knew it. The corneal and all other reflexes were absent—there was, of course, no response whatever and no change in color.

Another case was one in which a well known surgeon, who was operating on a patient, died during the course of the operation. I happened to be present—the only other medical man in this team. The surgeon was feeling rather out of sorts, and the nurse suggested he go out and get a little air. He said he would continue, and the first thing I knew, I saw him put his head down on the patient, and roll over on the floor. Within thirty seconds I was to him, and I laryngoscoped him and put a tube in his glottis and blew into it. This was all in less than a minute. He was completely relaxed. I blew into his chest, there being no oxygen available. At the end of my insufflation, he gave a little catch, but did not breathe. This was evidently a clear case of cardiac failure. I felt, however, that he had had everything possible in the way of treatment. It was out of the question to resuscitate his cardiac failure; respiratory obstruction, however, had been eliminated.

In regard to children, if you can laryngoscope them, you will observe a phenomenon that is rather common in cases that are deprived of oxygen, but not completely asphyxiated (you would call them apparently dead), that is the tone of the glottis

—a very slight tone of the vocal cords on irritation by intubation. One sees this frequently in the newborn. If a newborn baby who is laryngoscoped shows no response whatever to an intratracheal tube, that baby is very sick, and there is not much chance of its coming back. If there is any tendency for the cords to grasp the tube, resuscitation is simple.

There is a phenomenon which comes out in the respiratory response of these resuscitated patients. In a patient who has been completely asphyxiated, whose heart is still beating, we find the respirations return not by a deep respiratory effort, but by a very shallow panting, which increases in depth as the O and CO_2 affect and stimulate the center. Another point which is very important is the effect of CO_2 on the respiratory center as a direct stimulant. I was called to see this patient who was operated on in the morning, and I found her in her bed in a sanitarium, surrounded by her family, and the house staff trying to administer oxygen by funnel. She was dusky, breathing about once a minute, and she was on her way out. I laryngoscoped her without any difficulty. When I put the tube in place, I thought there might be a clot present which she had inhaled, but her airway was clear (most of the foreign bodies which are inhaled can be taken out by suction or forceps). This patient responded immediately to the oxygen insufflation, the color changed from a dusky one to a pink, and then I heard the respiration coming up. It was so rapid and shallow that another observer thought it was the pulse. Soon she was quite all right, and then the oxygen ran out. I sent down for more, and they brought up a tank, which I connected up. The patient started to go bad. In other words, the color sign warned us that something was wrong. I looked down and found that I had been given a tank of nitrous oxide. We disconnected this very promptly, and put her back on the oxygen which remained in the room—oxygen from the low pressure tanks. The color picked up, but the respirations did not. We got our CO_2 tank and the respirations promptly came up, and she was joking with the surgeon within fifteen minutes.

The color sign is merely complementary to everything else we have to prove death. The question of resuscitation in asphyxia turns on the popularization of about two inches of airway—two inches between the glottis and the vocal cords. If we can make that area of the respiratory tract popular, we will promptly save at least 10 and perhaps 25 per cent of asphyxiated patients. There is probably not one medical man in five hundred who has ever seen a glottis, or who has any idea of being able to see it. The reason is that there is no differentiation made between the asphyxiated subject and the ordinary patient who is laryngoscoped. It has been assumed when you use a laryngoscope that you are going to meet a certain set of difficulties which you cannot avoid. These difficulties, the muscle tone and reflexes, disappear with the asphyxial state. If every medical man could be instructed, as he should be, to expose that two inches of airway we would save a great number of patients.

Background of Use of Color*†

What is the scientific background of the use of color? It is believed that a consideration of color involves three generic factors: (a) the object, (b) the illumination, (c) the observer. If any one of these three factors is eliminated, the conclusions will be incomplete.

While these three factors are to be considered in a generic sense as any object anywhere, any illumination anywhere and any observer anywhere, the principle may be restricted in the present instance to a particular object (wound or skin color of the patient's ear), illumination (the illumination under control) and observer (the surgeon and the operating personnel).

The ear is well supplied with blood. The quantity and the quality of its color as this varies with blood pressure and oxygenation may be conveniently observed. The color of the ear varies with the quantity of the

* *The Modern Hospital*, June, 1939.

† Excellent article on color, in color, appears in "Life" for July 3, 1944.

blood in the capillaries, as measured by the volume of the pulse or by means of the blood pressure apparatus and by the *quality* of the coloring matter in the red blood cells determined by the amount of oxygen present.

In judging the color of the object, therefore, quantity and quality of color are basic considerations. Scientifically speaking, variations in quantity are represented by the same hue (shade) but have variations in value and chroma intensity; variations in quality are represented by changes in hue (light or dark) as well as in value and chroma.*

If one bears in mind the facts that light contains color, that color is reflected light and that the color of the field depends upon the light that falls upon it, it is almost impossible to overemphasize the importance of illumination.

As is the case with the object, correct illumination also turns upon the question of intensity and quality. Intensity of illumination creates the problem of reflection with its special type of ocular fatigue affecting the muscles of accommodation. Quality brings up the question of spectral value and its association with color fatigue which results in reduced visibility of the structures in the field.

The findings of the committee on operating theater lighting of the Canadian Hospital Council summarize the problem of *quantity* of illumination as follows:

"Adequate illumination of the immediate field of surgical work which the surgeon has in hand: sufficient illumination of surroundings; absence of glare, direct or reflected, and sufficient diffusion to avoid dense shadows."

It is further stated that "since the contrast factor in the immediate field of surgical operation is low, a high intensity of illumination is absolutely essential for the best results . . . but too much stress is placed on the shadowless feature. Shadowless illumination of a field having three dimensions is neither practical nor desirable. It is by virtue of variations of lighting of an object that we gain a knowledge of its contour. The shadows in any field of operation should not, however, be so dense that detail is lacking, the smaller the source of light the denser will be the shadows, but with well-diffused light from a large area, the shadows are soft and transparent and maximum visibility is thereby secured." As to the volume requirements of light, it is stated that an illumination of 640 foot candles at the field of operation, of 175 foot candles at the patient's head and of 16 foot candles on the instrument shelf is adequate general illumination.

The *quality* of the illumination turns upon its spectral value. It is generally recognized that since the human eye is accustomed to the spectrum of daylight this spectrum should provide the criterion for the artificial illumination of the operating room. Since the color of an object depends upon the quality of the light that falls upon it, it follows that a light, the spectral value of which is deficient in red and yellow when falling upon the field of operation, will produce a decidedly abnormal effect.

* Munsell System of Color Notation, Baltimore, Md.

Distortion of the color value of the operative field by deficient illumination is a constant source of anxiety to those who have the lives of patients in their hands.

Fortunately the empirical standards noted above have been recently reduced to a more accurate measuration of the quality of color necessary for operating room illumination.

It has been stated that the color of an object depends upon the spectral value of the light that illuminates it. Exceptions to this broad principle occur only in those bodies that are in themselves luminous, through heat or fluorescence or through sensitiveness to ultraviolet stimulation.

Heat becomes light when the object heated reaches 500° to 1000° K. As the temperature rises the color changes from red to the white of the noonday sunlight, passing to the blue of the northern sky at 9000° K. Beyond the blue and violet, vibrant energy passes into the range of the ultraviolet outside of the visible spectrum.

For practical purposes and in our present objective we are concerned with light that corresponds to our ordinary daylight. Ordinary daylight, however, has many variations.

Instead of creating the variations that occur by changing the temperature of the radiant filament, this being impossible because the filament is melted at temperatures higher than 3600° K., translucent filters are used which screen out the longer red, orange and yellow waves allowing the blue end of the spectrum to come through as an illuminant. This screening is done at the expense of the intensity of the light which is, in turn, compensated for by an increased wattage in the lamp used.

The problem of matching gradations of light as delicate as those that occur throughout the day was largely empirical until the development of the recording spectrophotometer. This extraordinary instrument accepts a sample of color be it light, textile, tile or any other material, analyzes its spectroscopic pattern and records this pattern as a graph as shown in Fig. 172.

The ordinary spectroscopic reading, formerly used to value color, is thereby translated into its temperature value and is permanently recorded as a curve, as illustrated in Fig. 173.

The basic curve for all light and color is on file in the U. S. Bureau of Standards at Washington.

The problem to be met in illuminating the operative field, therefore, is to obtain a light that will give the sharpest contrast between structures within the wound and a correct appraisal of the oxygen content of the blood.

Given an operating field adequately illuminated as to intensity and quality of light, the reaction of the individual surgeon and others who may be gazing into the field of operation or upon the skin of the patient constitutes the final judgment in the determination of color. An intense illumination while revealing the depth of the wound, if reflected from its periphery by white drapes, results in glare and color fatigue. Intense illumination reflected from the white drapes about the field of operation in due time will cause eye-strain and headache, with general irritability. To overcome

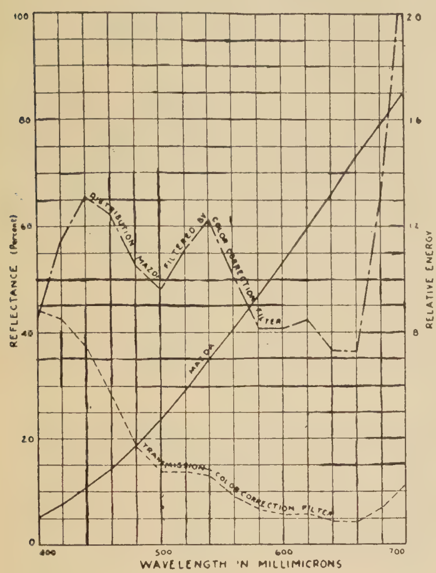


Fig. 172. Graph showing the energy distribution curve of artificial light.

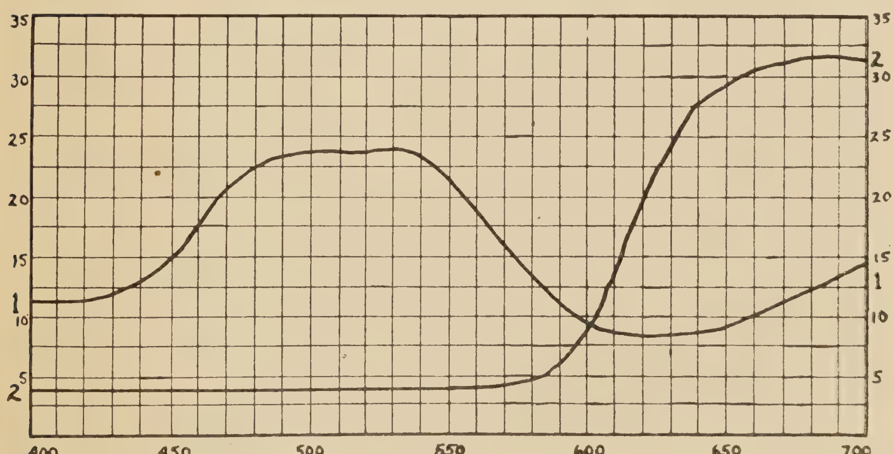


Fig. 173. This graph records the spectroscopic patterns of (1) four thicknesses of green cloth and (2) hemoglobin scale A-100 backed by black velvet.

this effect, color of sufficient chroma to absorb the greater part of the reflecting rays should be used in the drapes. For this particular purpose, any dark color is serviceable. Black, gray or green are frequently used.

Color fatigue, however, is a problem that is separate and distinct from glare. The nerve endings on the velvet black surface of the interior of the eyeball select and register the various colors of the spectrum.

Reaction of Eye to Color

Color impressions are received by three groups of cones in the retina. One group is hypersensitive to red; another, to green; another, to blue. Each, however, is also sensitive to a lesser degree to the other hues of the spectrum.

When true complementary (orthochromatic) colors impinge upon the retina—for example, orange, red and blue green (minus red)—the result is the impression of pure white. Pollution with other colors gives gray.

White, therefore, does not exist except as a synthesis of true complementary colors. A full spectrum provides complementary colors that balance each other within the visual range yielding white.

The absence of color yields an impression of black. To gaze intently upon one portion of the spectrum for a period of time is to paralyze the response of the nerve endings for the particular portion. The color thus viewed loses its sharpness and its hue.

In order to obviate color fatigue the true complement to the red of the operative field should appear in the drapes that surround it. Drapes of this complementary hue, steam resistant and durable, are now available in the open market. When these are employed, the brilliance of the field is increased, differentiation of the tissues is enhanced and one can see more easily into the depth of the wound.

Proof of the complementary color is readily available. This may be determined by spinning a circular card on which are exposed the red color of the blood and its complementary green. The resulting neutral gray cannot be obtained unless these colors are true complements.

Color complementation turns upon hue or what is ordinarily spoken of as the "color," as red, yellow, green, blue, purple or their intermediates. Complementary hues may be used through many grades of value, or brightness, and saturation, or chroma, to meet individual needs.

It is through these varying degrees of brightness or saturation that we are enabled to meet the psychologic needs of many individuals. Through this selection we are enabled to meet the radical and conservative tastes while still retaining the basic principles of our approach.

The practical applications, therefore, of the principles just stated as they apply to the object, the illumination, and the observer are: (1) all three factors must be considered simultaneously; (2) drapes alone will not provide relief; (3) light alone will not provide relief, and (4) the observer cannot be considered apart from the object, the illumination and the quality of color. Illumination must be viewed in its intensity and color value; observers must be protected against glare and color fatigue.

What constitutes resistance to the general acceptance and employment of color?

It is the author's conviction that the following are the principal reasons for the resistance to the widespread use of color in the operating room:

(1) Color involves an emotional reaction. This subjective factor in choice complicates decision.

(2) The scientific reasons for the use of color are not generally understood.

(3) The acceptance of the principles underlying the use of color and the desire to put these into practice are complicated by the fact that there is no commonly accepted designation to cover the correct hue in textile, light, paint, tile and glass that will enable the purchasing department to complete its transaction with precision and simplicity.

Chapter 31

The Signs of Death

Death may be molecular or somatic. Molecular death is the biochemical disintegration of tissues or organs. Somatic death is the final catastrophic disintegration and dissolution of the organism as a whole.

The effects of this disintegration become quickly apparent. The silent heart and respiration are preludes to the chilling of the face, hands, feet and trunk. The disrupted Oxygen-CO_2 tissue tension results shortly in a pH whose increasing acidity gives rise to the rigor of death. The terminal stagnant anoxia shows itself as superficial lividity or suggillations in the dependent parts. Out of the dry cornea look pupils whose irregularity reflects the rigor mortis of the ciliary muscles. The arteries become empty tubes. The veins contain oxygen-free blood, which does not coagulate. The ever-present bacteria, finding themselves in this non-resistant nutrient medium, quickly multiply in vast numbers, distending the closed cavities with carbon dioxide, hydrogen sulfide and methane. Finally come liquefaction, saponification or mummification—to dust.*

*Dr. Howard Neaile: Do any of us know the cause of death? Having done thousands of autopsies, I find that we do not and cannot recognize the cause of death in from two to four per cent of the cases autopsied. There is no apparent reason why the individual is dead. Here we have a case of asphyxia, carbon monoxide, that the individual has been exposed to for some twenty or thirty minutes, or hours, and has died, and we have another individual who has had an equal number of hours' or minutes' exposure, and has lived. We have an individual with a certain heart condition. He has died. On autopsy we find his heart condition of rather a trivial nature—slight coronary sclerosis, or a slight heart lesion. Another individual that we autopsy presents a heart with a pathology that has lasted for years. Do we know the cause of death? I question it. We say an individual dies of pneumonia. Does he die of pneumonia? What happens in that particular individual to cause his death? We know certain facts about life and death. We know that man lives. His culture, of course, was very limited five hundred thousand years ago. But we know that he lived to about the same age we live, because we have found his remains, and can tell from his teeth that that individual lived approximately so many years. It seems it has been decreed that man shall live a limited number of years. So is it with all other living things. As a class, they have a certain number of years allotted to them, but different families of that class, curious to say, live longer than their cousins or other distant relatives. Take birds, for instance: why should a goose live to be a hundred years old, and a chicken die at the age of fifteen? It is a peculiar phenomenon, and the question has arisen in my mind many times whether or not there are laws, complicated and complex as they may be, that control our destiny—the individual destiny of a man. Every science has such laws, physics and chemistry, and so forth, but undoubtedly due to the complexity of the thing, I have not been able to satisfy myself what happens in a particular individual to cause his death.

* Dr. Le Wald: Many of you saw in the newspapers, the heading "Hospital Morgue Gets a Living Boy." That appealed to me particularly because I have seen two cases

* *Long Island Med. J.*, **61**: 205–208 (1933).

Herold states, "It is the rarest thing in the world, not excepting anything, for the physician to be placed in doubt as regards the actual presence of death." "The danger of live burial is reduced to the barest possible chance." "Science has certainly supplied us with signs of death sufficient in number, when taken together, to brand him who does not know enough about them to guard against the possibility of burial alive, as ignorant and culpable."

And yet there is an increasing insistence among many lay persons and among morticians that greater care be exercised in establishing proof of death. As an evidence of this anxiety the following is submitted.

94th General Assembly
Regular Session
1941–1942 — H. B. No. 442

Mr. Donovan

A Bill

"To enact supplemental section 204-1 of the General Code, to provide for the complete determination of deaths.

Be it enacted by the General Assembly of the State of Ohio:

Section 1: That supplemental section of the General Code be enacted to read as follows:

Sec. 204-1. The body of a person whose death occurs in Ohio State shall not be removed to any autopsy room nor to any place where embalming is done, nor shall any mutilation of any kind be permitted, until the body of such deceased has been subjected to scientific life tests to determine whether or not death is complete. The nature and character of such life tests shall be ascertained from year to year by the head of the State Public Health Service from the tests advocated by the American Medical Association and national selected morticians.

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come to life, or rather, they were not dead, in a morgue, and it is not too far-fetched occasionally to say that such a circumstance may happen. I do not know anything about the final outcome of the case reported in the newspaper of a sixteen year old boy in a sanitarium who lived two days after he was supposed to have been dead. These cases very often are *in extremis*, and it may be only a short prolongation of life is possible, but of course it is one's duty to do that much. This was true in one of the two cases I saw, a patient who had been ill with a cardionephritic condition. I have no doubt that the patient was *in extremis* when last seen by the nurse, and was then sent to the mortuary, but about six hours later was found to be alive, and was returned to the hospital, finally succumbing after about 24 hours. The other case I know of was one of new-born twins, attended by a midwife. The first one born was truly stillborn, and did not recover. The second twin was also considered to be dead, and the father was directed to take the two infants to the morgue. About six or eight hours later it was discovered that the *second* one of the twins was alive. I was present myself, and, in fact, made the discovery. The patient was immediately taken to the hospital and placed in an incubator, under the most favorable conditions, but did not survive long. However, the procedure described by Dr. Flagg was not done, and I think if it had been instituted, the baby might perhaps have survived, in spite of the exposure, but those are rare exceptions, of course. Nevertheless, they are true cases.

Such tests shall be administered by a physician, or by a Public Health Service medical employee no sooner than four hours after death.

The examiner's report of the findings of these tests shall be made out in triplicate in the presence of the body, two copies of which shall be given to those nearest the deceased, one copy to be retained by them and one copy to be given to the attending mortician. The third copy shall be sent to the Public Health Service. If objection is raised to these tests, the body shall be removed only upon the pronouncement by a physician or Public Health Service medical employee that the signs of death are unmistakable after the body has been kept in an unmutilated condition for at least eighty (80) hours after death has been reported."

The following is a quotation from a communication received from the same state: "The local mortician who told me of receiving an average of about ten bodies a year not ready for him, now is beginning to carry on. He has an oscillating bed and an electric blanket arranged for his use in a reviving room to care for patients he brings to his establishment" (Nov. 24, 1940).

This fantastic condition of affairs in these United States would be amusing if they were not so serious in their basic implications. These implications are the necessity of proof of death by a properly qualified physician. This necessity is accentuated by the fact that certain religious sects do not call a physician.

The above matter is reproduced to confirm the fact and the necessity that "the fact of death" be pronounced by the physician. What then are the signs of death, "ignorance of which brands the physician as ignorant and culpable." Quoting Herold, these signs are as follows:

(1) Cessation of Respiration
- (a) Mirror test
- (b) Feather test
- (c) Water or mercury test
- (d) Stethoscopic test
- (e) Rhythmic traction of the tongue

(2) Cessation of the Circulation
- (a) Stethoscopic test
- (b) Ligature test
- (c) Scarification or cupping test
- (d) Opening an artery
- (e) Needle test (Cloquets)
- (f) Fluorescine test
- (g) Injection of ammonia (Monte Verdes Test)
- (h) Diapharous test (Carrier's)
- (i) Roentgen test

(3) Changes in the Eye
- (a) Test by bright light

(b) Test by mydriatics
(c) Test by ophthalmoscope
(d) Test by ophthalmometer
(4) Loss of Animal Heat
(a) Temperature test
(5) Loss of Sensation and Motion
(a) Electric test
(b) Heat test
(6) Muscular Flaccidity and Contractibility
(7) Cadaveric Echymosis, Lividity or Hypostasis
(8) Cadaveric rigidity, cadaveric spasm, rigor mortis
(9) Putrefaction

Mirror test: Small, cold mirror held before nose and mouth for an hour. Breath condensing upon it suggests invisible respiration, but escaping gases produce the same effect.

Feather test: Feather or wisp of cotton over nose or mouth is moved by respiration. Also moved by other currents of air.

Water or Mercury Test: Water or mercury placed in a glass and allowed to rest on the abdomen give rise to movements of the abdominal wall. Intra-abdominal gases also give rise to movements of the abdominal wall.

Stethoscopic test: Heart silent when death is present.

Tongue (Laborde's): Traction stirs up pharyngeal or laryngeal reflexes when present.

Cessation of circulation: Said to occur in traumatic shock, catalepsy or syncope. But audible heart sound is always present in life. Fifteen minutes silence in auscultation means death. (Stethoscopic test)

Ligature test: String tied about finger or lobe of ear. If there is any circulation in the part there is always distal swelling and white mark of string. If no change in fifteen to thirty minutes the part is without circulation.

Scarification and cupping: Venous blood may flow eight days after death.

Opening of an artery: When the heart has ceased to beat the artery is empty. If the temporal or radial artery is opened and found empty this is an infallible sign of death. Blood in veins does not coagulate.

Needle test: Clean, bright needle thrust into muscle should oxidize and tarnish in life, remains bright in death. But oxidizing has been demonstrated to occur in death.

Fluorescine test: Hypodermic injection under skin, 1 mg. If alive, no color; if dead, color remains. A corroborative sign.

Monte Verdes Sign: Hypodermic solution of ammonia under skin, port wine color if alive, no color if dead. Not reliable.

Diapharous Carrier's Sign: Translucency of fingers and hand before bright light in life, opacity in death. Not reliable.

X-ray: Moving structures, heart diaphragm, etc. give a hazy line in life; if dead, outlines shown are clear.

Eye signs: Loss of tension, loss of tone, flaccid battery 12–14 h post mortem. But may be larger and firmer if drowned.

Light reaction: Disappears with reflexes, gone in deep anesthesia.

Mydriatics: No reaction in death expected, but exceptions occur.

Ophthalmoscope: Cornea must be clear, which is rare. Yellowish red fundus in life becomes yellowish white in death. Arteries empty, beaded appearance of veins (pneumatosis) due to gas bubbles. The difficulty is to secure a clear cornea in order that these signs may be visualized.

Ophthalmometer: Normal tension 18–21 Gm. At death 12, in half an hour 1–3, in 2 hours 0.

Sensation and motion: Unimportant; imply active reflexes.

Electric test: Absence of response of galvanic current in death. Contractibility lost in 8–12 hours. The electric current offers an almost certain method of ascertaining whether life is extinct or not by its success or failure in producing muscular contractions. But this must be skillfully used. Of 402 cases, 107 were irritable in ½ hour, 67 in 2 hours, 4 in 3 hours; balance not irritable.

Heat test: Reaction of skin to hot needle, giving rise to blister. Test doubtful as it will appear in amputated limb.

Caustic test: If alive, black or reddish eschar; if dead no eschar.

Flaccidity and contractibility: Occur in ordinary deep anaesthesia.

Conclusions: Justin Herold states, "Putrefaction is a certain sign of death. The most certain test to be applied without possibility of error I hold to be the prompt and immediate opening of the temporal or radial artery".

Herold makes it clear that each and every one of these tests turns upon the complete cessation of the respiration and the circulation. He believes that immediately after somatic death, two signs are conclusive. Stethoscopic auscultation and the opening of the radial or temporal artery. In death, arteries are empty. Putrefaction subsequently confirms death.

Ganzales for practical purposes reduces the foregoing signs to stoppage of the heart and respiration, loss of body heat, post-mortem lividity or suggillations, rigor mortis, eye signs and decomposition. The following very brief references amplify these signs.

Stoppage of heart and respiration, elicited by stethoscope over heart and larynx. Slightest heart or breath sound is audible. Cataleptic trance or traumatic shock is ruled out by this test. Complete silence of cardiac area means death.

Loss of body heat depends on environment, and the covering and size of the body. Average, hands and face are cold in 3–4 hours, trunk in 10–12 hours. (Herold reports the following average temperature change, with the bulb of the thermometer on the abdomen: 2–3 hours 77°; 4–6 hours 72°; 6–8 hours 69°; 12 hours 67°). *Infectious diseases* occasionally give a post-mortem temperature rise.

Lividity (Suggillation) appears on dependent parts, especially the neck, shoulders and back, 30 minutes to 2 hours after death in plethoric cases, in 4 hours in anemic. Complete in 12 hours. Color cherry-red in CO poisoning, chocolate or coffee brown in chlorate of potash or analine (methhemoglobin).

Rigor Mortis. Muscles stiffen and shorten after death. For a short time after death reaction of muscle sarcoplasm is slightly alkaline, and the muscle is then flexible. In 2–6 hours sarcolactic and phosphoric acids give rise to rigor mortis; it begins in the face, then spreads to the jaw and upper extremities, then lower; complete in two hours. Rigor persists for 12–48 hours. The onset is hastened by heat or cold; heat accelerates disappearance. Rigor disappears when muscle becomes alkaline again.

Eye signs. Dried secretions give lackluster cornea. Rigor of ciliary muscles give unequal pupils. Open eyes, reddish brown sclera.

Decomposition. Blood is culture medium, especially for gram-positive bacilli. Result: CO_2, methane and hydrogen sulfide. In 48 hours green areas appear on the abdomen (iron sulfide). This action is rapid in warm climates, and faster when the body is lying on its face. Post-mortem vessel rupture and hemorrhage are common.

Mummification results from water loss in dry sandy soil, cool dry plains or dry cemetery vaults.

Adipocere occurs in submersion or due to wet soil; tissue is transformed to dirty white opaque material (soap) in 6–7 weeks.

Embalming. Body fluids are removed by aspiration and replaced by formaldehyde compounds injected into the empty arteries. It delays bacterial decomposition and induces mummification in whole or in part.

The author's sign of death are as follows: the quality of the color of the circulating blood depends upon its oxygen concentration. Blood (in bulk in test tube) saturated with oxygen is brilliant red; completely free of oxygen it is a deep claret or maroon. Seen through the skin of mucous membranes the color is pink, changing to deep blue or grayish green according to the thickness of the overlying tissue. When oxygen is insufflated into the lungs under pressure, the alveolar capillaries immediately absorb it. This blood passing to the heart by the pulmonary veins is brilliant red. Delivered through the left side of the heart to the arterial system, this brilliant blood is immediately reflected in the pink color of the mucous membranes of the lips. (There is only one exception and that is in the new born with congenital heart disease, *i.e.*, open foramen ovale). In the presence of oxygen under pressure in the lungs, regardless of whether or not there is voluntary respiration, the heart picks up and circulates bright red blood. The color of the mucous membranes must be pink, *as long as the heart acts.* Heart failure is indicated by a gradually increasing cyanosis *in the presence of oxygen insufflation under pressure.* If an apparently dead person is insufflated with endotracheal oxygen under pressure for a period of ten minutes with no alteration in the color of the mucous membranes, one may safely assume that the heart has ceased to beat. The routine use of this sign will automatically save life and prove death.

Conclusion

Somatic death may be said to have occurred when the respirations and the circulation have failed and cannot be reestablished over a period of 15 to 30 minutes. If the heart continues to beat, life may be maintained for many hours without any respiratory effort if oxygen is made available to the alveolar capillaries by endotracheal insufflation. The diagnosis of death therefore turns upon the cessation of the heart action.

Three signs are offered as proof that the heart has failed.

(a) Auscultation of the heart with a stethoscope.

(b) In the presence of oxygen endotracheal insufflation under pressure there is no change in the color of the mucous membranes, over a period of 10 minutes of intermittent pressure at 25 mm. Hg.

(c) If, upon opening the temporal or radial artery, it is found to be empty of blood.

The last sign is conclusive evidence that the arterial circulation has finally and permanently passed into the venous blood. The foregoing signs are shortly confirmed by loss of body heat, suggillation, progressive rigor mortis and biochemical disintegration.

Resuscitation may fan the spark of life into a living flame. It cannot create the spark. The pathologic physiology of asphyxia indicates the limit of viability in experimental animals, which Coryllos has shown to be four minutes in the terminal period of apparent death.

Part VII

Miscellaneous

Appendix I

Instruction of Technicians

This chapter is intended for the instruction of and used by the layman who is unfamiliar with the principles and the practice of Pneumatology, *i.e.*, the First-aid worker, members of the rescue squads of municipal and industrial services: Fire, Police, Gas Companies, etc.

Pneumatology

The term *pneumatology*, its derivation, scope and field of practical usefulness are briefly described as follows: *Pneumatology* is an old term which should be revived because its common usage will save many lives now lost through asphyxial accidents. It provides a direct contact between the patient and the specialist trained in expert treatment. From the Greek Pneuma, meaning *air* or *gas*, "pneumatology" is scientific in its derivation and usage.

In Steadman's, Lippincott's and Dorland's Medical Dictionaries pneumatology is defined as "the use of gases for medical or therapeutic purposes." Many new editions of medical dictionaries in this country and in England have accepted the term in the sense in which it was employed in the Medical Department of the New York World's Fair, namely, the therapeutic use of gases for the control of pain (anaesthesia) the saving of life (resuscitation) and the treatment of clinical disease (oxygen therapy). Pneumatology for the treatment of such clinical diseases as asthma and tuberculosis was first introduced by Sir Thomas Beddoes in 1789. The Pneumatic Institute, established by Beddoes at this time in Clifton near Bristol, was the cradle of this specialty.

In the middle of the nineteenth century the introduction of the gases nitrous oxide and ether for the control of pain provided such dramatic interest that a name was suggested by Oliver Wendell Holmes to describe the state of unconsciousness produced. This state he called **anaesthesia.** Overnight, the pneumatologist became the anaesthetist. Unfortunately, the restriction imposed by this term, limiting the use of gases to the control of pain delayed for more than half a century the development of the use of gases for the saving of life and the treatment of clinical disease. It is therefore suggested that the anaesthetist adopt the name *pneumatology*, to the end that those who suffer from disease or who are about to die from suffocation may be aware of his special skill and secure the benefit of immediate assistance.

Gases for the Saving of Life and Indications for Their Use

Oxygen, discovered by Priestley in 1774, is now generally available in every community and institution. An understanding of its special usefulness and effective administration is by no means so universal. The technique of transferring oxygen from the container in which it is stored to the iron or hemoglobin of the red blood cell should be the common knowledge of all physicians and familiar to laymen responsible for the First-aid treatment of *acute* asphyxial accidents.

While oxygen is the fuel which permits the continued functioning of the human mechanism, carbon dioxide is the ignition which regulates and times the vital process. Without oxygen the brain cells promptly die; without

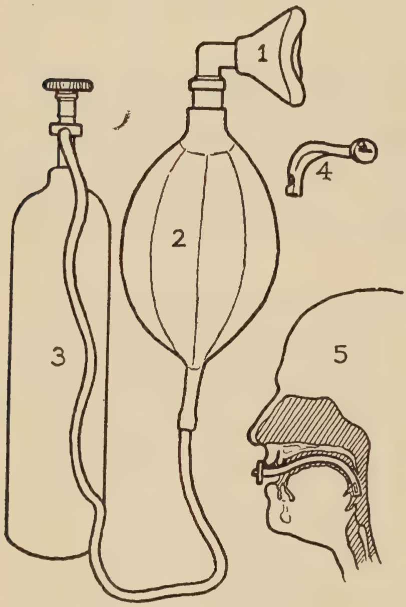

Fig. 174. (1) Inhaler, (2) rebreathing bag, (3) tank of oxygen or O and CO_2, (4) metal or rubber airway, (5) airway in position. R. M. Waters, *J. Am. Med. Assn.*, **123,** 559 (1943).

adequate CO_2 the active respiration ceases and the circulation fails. For practical purposes these two gases are mixed in the proportion of oxygen 90 parts, CO_2 10 parts. Cylinders containing these mixtures are plainly marked and generally available.

In order to treat an asphyxial accident effectively one must have a clear understanding of the degrees or stages of asphyxia. Three stages are easily recognizable; the depressed, the spastic, and the flaccid.

The *depressed* patient, as the term suggests, is suffering from a mild degree of shock as the result of the accident to which he has been subjected. His vital functions, that is, his respiration and his circulation, are intact. His color is but little affected. Most important, he may be roused and will respond when stimulated. This patient will be revived and reoriented, and will recover if he is allowed to breathe a mixture of oxygen and CO_2. A paper bag would serve the purpose, but a simple inhaler is more practical.

The *spastic* patient differs sharply from the patient who is *depressed* because he *cannot be roused.* Muscle spasm is common in the muscles of the jaw and extremities. Spasm is frequently accompanied by bleeding from the nose or mouth and occasionally by vomiting. The vital functions still operate, but they are in grave danger of serious embarrassment, owing to the artificial obstruction to the respiration which exists. The

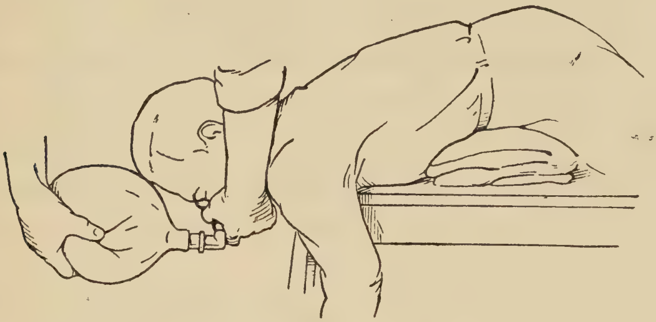

Fig. 175. Position suggested for transpharyngeal insufflation. Pad to prevent distension of stomach; fluid drains. (Secretions draining into face piece must be removed before insufflation is done.) (R. M. Waters.)

Fig. 176. Relief of tongue swallowing and aid in passing airway (R. M. Waters).

color will vary according to the severity of this obstruction, which must be satisfactorily met, or the patient will pass promptly into the terminal stage, or *flaccidity.*

The *flaccid* patient is entirely unconscious: the coughing, swallowing and other reflexes are in abeyance. The respiration may or may not be present and the circulation is much depressed. This type of patient requires and should receive expert medical care if he is to survive.

Unconsciousness in *any* patient requires the immediate attendance of the physician. While First-aid measures should always be employed, they should not take precedence over summoning a physician.

Gases for the Control of Pain

Oxygen is necessary to life. A reduction in the amount of oxygen ordinarily found in atmospheric air results in loss of consciousness and death. While pain as a symptom disappears with loss of consciousness, the reflexes and the muscle tone remain until oxygen loss is so extreme that it destroys nerve cells.

It has been found in practice that loss of consciousness may be secured and that reflex activity and muscle tone may be controlled by gases which are added to the blood through the respiratory tract. The first important gas that was found to produce this effect was nitrous oxide, reported by the pneumatologist Priestley in 1774. Since then, the gases of chloroform, ether, ethyl chloride (it may be noted in passing that vapors are inhaled as gases), of ethylene and more recently, cyclopropane, have been added to the armamentarium of the anaesthetist. It is to be observed that the control of pain brought about by the use of these gases is not due to oxygen limitation, but to other causes. These effects are imperfectly understood and vary in toxicity.

In practice, the gases of ether, chloroform and ethyl chloride are often employed alone. Nitrous oxide, ethylene, and cyclopropane are used in combination with oxygen. Anaesthetic gases are also commonly used in various sequences, such as nitrous oxide, oxygen, ethylene, oxygen, chloroform, nitrous oxide, ether. While carbon dioxide and helium are used in anaesthesia, they are not, strictly speaking, anaesthetic gases. Of the gases, ether is safest, yet, on the other hand, it is the most difficult to administer.

Many institutions employ local, regional, basal and intravenous anesthesia. While these methods are valuable adjuncts to the gases in the control of pain, their use throughout all institutions constitutes certainly less than twenty-five per cent. Furthermore, these methods cannot be used efficiently as exclusive routine. If all the anaesthetic gases were removed from the field of pain control, surgery as we now know it would become paralyzed.

Given ordinary operating-room conditions, each anaesthetic gas, with the exception of nitrous oxide and chloroform, presents a fire hazard, which varies with the physical characteristics of the gas. Some gases burn, others explode. Ether forms explosive mixtures with oxygen, ethylene and cyclopropane. Ether used with air may be ignited; it burns, but rarely explodes. The fire hazard of any gas depends upon the degree of accuracy and the fidelity with which the demands of its physical characteristics are met.

The Use of Oxygen and Carbon Dioxide in Artificial Respiration

Artificial respiration is for the purpose of bringing oxygen into contact with the blood. Oxygen exists in atmospheric air, constituting about onefifth of its volume. As one climbs a high mountain or goes up in an airplane the *percentage* of oxygen remains the same, but the gases at the high altitude are so diluted or thinned out because of the low pressure at such heights that there is not enough oxygen in the mixture for normal breathing. Therefore, for both artificial respiration and high-altitude flying it is highly desirable to increase the percentage of oxygen in the mixture.

Artificial oxygen is supplied in steel cylinders under pressure. It is released by special valves or what an electrician might term transformers. The gas may then be administered to the patient at low pressure. In addition to oxygen, carbon dioxide has come into popular use in recent years. Carbon dioxide gas, or carbonic acid gas, is used in "soda" fountains for the charging of beverages; it provides the sparkle in ordinary soda water. Carbon dioxide has been found to exert a stimulating effect upon that portion of the brain which regulates the respiratory act, that is, inspiration and expiration. If there is too little of it in the air, breathing will stop; if there is too much, breathing will become very deep and rapid, finally leading to exhaustion. The unconscious patient, whose respiration has been shut off, needs the stimulating effect of a little carbon dioxide gas mixed with the oxygen administered. This proportion is usually between five and ten per cent and is dispensed in cylinders containing both gases as a mixture.

Pure oxygen administered to the patient instead of air is five times as effective in oxygenating his blood. The problem in artificial respiration is to place as high a concentration of oxygen as possible in contact with the patient's blood. One hundred per cent oxygen may be administered continuously for as long as two days without irritating the lungs. The simplest way to increase the oxygen administered to a patient is to place a mask delivering oxygen over his face, while he is receiving prone-pressure Schaeffer treatment. If the oxygen mask is well applied and the patient's air passages are open, this treatment will be five times as effective as when air alone is used.

Improved resuscitation methods developed by physicians all depend upon means which will permit the introduction of higher and higher percentages of oxygen within the lungs. If the patient is breathing even in a shallow manner, a well applied mask is sufficient. If the respiration is obstructed, a rubber tube may be passed into one nostril. If the patient is profoundly asphyxiated, pure oxygen gas may be introduced directly into the lungs by apparatus especially designed for the purpose.

Artificial Respiration over Long Periods

Prolonged artificial respiration is indicated where the normal respiratory act fails because of profound depression of the respiratory center from drugs, or where the muscular mechanism carrying on respiration becomes paralyzed, notably in ascending poliomyelitis (infantile paralysis.) The distinction between *emergency* resuscitation, whose immediate object is to prevent impending death from a profound but temporary lack of oxygen, and *prolonged* respiration whose object is to prevent the effect of prolonged lack of oxygen, should be clearly understood. The first requires a fast, snappy technique, with every unused moment a liability. The second should be performed with a routine involving the deliberate functioning of trained personnel. As a consequence of these fundamental considerations, facilities for emergency resuscitation should always be available, and when necessary they should function as a preliminary to the establishment of methods designed for prolonged artificial respiration.

Artificial respiration by endotracheal insufflation has been maintained for as long as fifteen hours. Conditions requiring artificial respiration for hours and days, however, should be carried on by methods less likely to result in injury. Insufflation bridges the gap between death and safe transference to a negative-pressure cabinet.

Artificial respiration by the prone-pressure method produces its effects indirectly, that is, the entrance of air into the lungs is accomplished by squeezing air out of the chest and depending upon the elastic recoil of the chest walls to cause air to enter. Such technique is harmful over very long periods and ineffective when the elasticity of the chest walls has disappeared in cases of deep asphyxia. By placing the patient's body in a cabinet with his head outside and by reducing the air pressure on his body within the cabinet, air may be sucked into the mouth each time the pressure on the body is reduced. This technique is a direct method of bringing about inhalation and is entirely non-injurious. Certain conditions must be present, however, before this method will operate. The passage-way from the outside to the interior of the lungs must be entirely free from foreign matter, such as water, blood or vomitus. Obstruction caused by flaccid structures such as the tongue and epiglottis or by the position of the head must be relieved. The lungs must be free to distend by suction applied to the chest wall. They should not be collapsed or unexpandable. Obstruction may be relieved by suction and the use of mechanical airways. Failure of the lungs to expand, or massive collapse of the lung may be helped by oxygen insufflation synchronized with movements of the respirator.

The Iron Lung

The iron lung is designed for maintaining artificial respiration over long periods. It is not well adapted to emergency resuscitation. A clear understanding of this fact will serve to place its general use on a more favorable basis. The prone-pressure Schaeffer resuscitation technique seeks to produce artificial respiration indirectly, by depending upon the recoil of the chest following pressure applied against the lower ribs, the iron lung on the contrary produces its effects directly. It eliminates the necessity of the elastic recoil, (expansion) of the chest.

To understand the operation of the iron lung, it is necessary to consider a few facts relating to air pressure. The atmosphere in which we live extends about five miles upward. At sea level a column of air one inch square and five miles high weighs about fifteen pounds. The reason that we can tolerate this great pressure on our bodies is because it exists inside them as well as on the surface.

When we compress the chest wall by the prone-pressure Schaeffer technique, we squeeze out air. When we let go, the chest wall springs back, creates a reduced pressure inside the lungs, and causes air to be sucked into them. If we were to remove some of the atmospheric pressure, for example, one quarter of a pound to the square inch on the outside of the body, leaving the pressure on the nose, mouth and throat normal, the same effect would be produced. Air would be sucked into the chest. When the pressure on the chest returns to normal the air which has been sucked in escapes.

The iron lung operates on this principle. A patient's body is placed inside an air-tight box with the head outside. Pressure is reduced within the box over the surface of the patient's body by means of an electrical suction pump. When the pressure is reduced, the cavity of the chest enlarges, and air is sucked through the patient's nose and mouth. As the pressure within the box returns to normal, the air escapes. For example, reduced pressure on body, patient breathes in; normal pressure on body, patient breathes out. Instead of place, push, rise, release, rest, (Schaeffer method) we have suck, rest; suck, rest.

It is plain that should entrance of air into the lung be blocked, which occurs, for example, when the tongue is allowed to drop back into the throat or when there is blood or water in the mouth, two complications will immediately arise: (1) little or no air will enter the lungs, and (2) matter in the throat may be sucked into the lungs causing pneumonia. It is important, therefore, that such matter be removed from the nose and throat and that a free passage for air be guaranteed at all times.

The Use of Suction in Artificial Respiration

The employment of suction is so important in artificial respiration that everyone should be familiar with its general principles, understand its application, and how to effect it.

Whenever a difference in atmospheric pressure is suddenly brought about, a vacuum results. This vacuum, or space in which the pressure has suddenly been reduced, may be either partial or complete. Thunder, which we hear after a lightning flash in a summer storm, is the result of a vacuum produced in the atmosphere by the electricity; it is caused by great walls of atmosphere rushing together. The filament in an electric-light bulb glows because it exists in a vacuum. When an electric-light bulb breaks, glass particles do not fly away from the bulb, but collapse owing to the atmospheric pressure on the outside walls. The ordinary automobile engine produces a partial vacuum when the piston within the cylinder is raised. The result is a flow of gas and air into the cylinder. The partial vacuum or suction produced by the pistons of the engine of your automobile is also used to operate the windshield wiper; and this type of vacuum brings us very close to the problem of artificial respiration. If, instead of a piston, a stout-walled rubber ball is squeezed, upon recovering its shape it forms a partial vacuum exerting a force which may be used to suck in air or water. The patient's chest in the prone-pressure technique resembles the rubber ball in sucking atmospheric air into its interior. (If the patient's mouth were under water when prone pressure was applied, water instead of air would be sucked into the lungs.) In artificial respiration we employ the power of suction to bring oxygen in contact with the circulating blood. In order to prevent accidents such as pneumonia we also use suction to remove water, vomited material and blood from the air passages. The need of suction for the removal of foreign matter from the air passages is obvious.

It is strange that its general use has been neglected for such a long time. No operating room would today be considered complete without some form of suction, produced by electricity or steam. Dental offices and other professional locations are well supplied with suction. It is now available in the more modern resuscitation outfits. A convenient form is that of a metal plunger pump. In an ambulance, suction may be readily provided by attaching a tube to the windshield wiper, whose source of suction is the intake manifold. Every ambulance should be so equipped. Of late years it has been recognized that one of the most important features in connection with the resuscitation of the newborn is suction applied to the mouth and throat.

Become familiar with suction; see that it is available; and use it freely when necessary.

First Aid Resuscitation

Electric Shock. The electrical worker who is called upon to render first aid to a fellow workman who has been shocked should act immediately as follows. He should:

(1) Break the circuit.

(2) Perform Schaeffer prone-pressure artificial respiration.

(3) Contact police for medical help.

(1) In breaking the circuit, which has been accidentally established with the patient's body, it is important to avoid any direct contact with the patient. If he is still attached to the source of the electricity you may receive the shock yourself. Contact with the loaded wire should be broken by a long, dry stick or wooden pole, such as a broom handle, never by a wet object or one containing metal. A folded dry coat with no metal in the pockets may be used if no wood is available. If the patient is lying

on the source of electricity, a leg or arm farthest from the source of current should be grasped through a dry coat. Always be certain that the circuit has been broken before touching the patient.

(2) Perform artificial respiration as follows: Place your man on his face with both hands extended above his head. Turn the face to the right, make a pillow of the left arm and lay the man's head on it, pushing the forehead well back to extend the head. Straddle the patient so that your knees are opposite his waist. Place the flat of each hand against the lower ribs at the small of the back. With the elbows held stiff and straight push down on your hands against the patient's back. Remove pressure, remove hands, rest. Continue this act, repeating as you do so; place, push, rise, release, rest; place, push, rise, release, rest. If you have assistance, have the patient's teeth separated and a finger passed into the mouth to remove any foreign matter such as false teeth, a wad of tobacco or chewing gum. Have covering placed over the patient's legs and do not expose his chest. Continue artificial respiration until the arrival of medical help. Every unconscious patient who cannot be roused is entitled to the services of a physician. You are called upon to face a desperate medical emergency. You have not done your duty if you have limited your care to First-aid methods, as these, while excellent in themselves, go only part way.

(3) For medical help send a messenger immediately to the nearest policeman or have Police Headquarters called, which in turn will establish immediate and authoritative contact with the ambulance and the physician nearest the site of the accident.

How Does Electricity Kill?

Electric shock or electrocution is one of the many causes of death which may be charged to suffocation or asphyxiation. It is classified with drowning, carbon-monoxide poisoning, smoke poisoning, and suffocation from foreign bodies in the throat, because the first medical treatment applied in each of these accidents is artificial respiration. Without adequate artificial respiration the patient dies from asphyxiation; with it, his life may be saved. Unlike other causes of asphyxiation, such as drowning and carbon-monoxide poisoning, the heart is usually first to suffer in cases of electric shock. Stoppage of the breathing follows. If the shock has been severe the patient will die instantly from heart failure. As we can never be sure that a fatal shock and a complete heart stop has taken place, we must always give the patient the benefit of the doubt and carry on artificial respiration in the hope that the heart will recover.

As has been pointed out, artificial respiration brings the oxygen in the air into contact with the blood circulating in the lungs. This blood oxygenates the brain, which acts as the timer or the regulator of the respiratory act. If oxygen does not reach the brain cells because of the temporary failure of the respiration, the effect of the electrical shock upon the heart is increased and recovery from even mild cases of shock may fail to take place. The First-aid worker faced with this desperate emergency can do much to save the life of a shocked patient. First, he can shorten the period of exposure by breaking the electrical circuit. Secondly, he can help control the asphyxiation and bridge the gap between heart recovery and death. Thirdly, by sending in an immediate call for the medical assistance he will give the patient the benefit of the administration of pure oxygen, which is five times as efficient as air, and of carbon-dioxide, which will stimulate normal breathing. This is accomplished either by an inhalator, or by a tube in the nose or throat, as the case may be. First-aid resuscitation methods should be familiar to all. A knowledge of what the physician can do to supplement First-aid treatment is scarcely less important.

Emergency medical services throughout the country are now turning their attention to the newer methods of preventing asphyxial death. The apparatus required is relatively simple, and the method of its application is easy. The results are what might be expected through the greatly increased mechanical and physiological advantages of precision methods.

Why Call a Physician?

The technique and team work which have been developed by police and fire departments, the Consolidated Gas Co., and other groups have not infrequently resulted in a call from a hospital, or even from an operating room for the services of the squad. It is quite natural, therefore, that in a case requiring artificial respiration—be it drowning, electric stroke or carbon-monoxide poisoning—the question be raised, Why call a physician? We would answer this question as follows.

The high mortality from the many causes of asphyxial accidents or suffocation has only recently been recognized as an important medical problem. Improved methods for the medical care of the asphyxiated patient are also of recent origin and acceptance by the medical profession. Because of these facts the prone-pressure Schaeffer method of artificial respiration, the use of the simple inhalator, the addition of hypodermic medication and application of heat to the body have been looked upon by the average physician as a satisfactory and complete technique for the treatment of the asphyxiated.

Specialists in medicine referred to as pneumatologists, who are familiar with the use of gases for medical purposes, have developed simple procedures which are recognized and which are being generally adopted by the medical profession. If you call a physician he will be prepared to diagnose the degree of asphyxiation which is present. He will recognize at once simple depression, spasticity or flaccidity. He will help the First-aid team by the correct application of an inhaler supplying oxygen and carbon dioxide to the depressed patient. He will overcome the obstruction to the breathing of a spastic patient by the use of portable suction and by passing a tube carrying oxygen and carbon dioxide into the back of the throat. He will be prepared to examine the throat of a flaccid patient with a pocket flashlight, to put a tube into the windpipe, to suck the lungs free from fluid, and to blow oxygen gently into them. These procedures are so simple in the conditions requiring their use that physicians are rapidly acquiring these methods. In addition to the benefits to be derived from them, the medical treatment outlined permits the patient to be removed immediately and treated en route. Conditions for continued treatment in a well equipped ambulance are much more favorable than at the site of the accident. The correct care of the patient upon his arrival at the hospital is carried out in the Department of Pneumatology under the direction of the hospital anaesthetist. This physician is trained in the care of the unconscious patient.

Whenever you encounter a person unconscious from accidental causes, be sure to call a physician.

What to Do for the Unconscious Patient

The idea of unconsciousness is terrifying. We dread losing control of those faculties which have protected us so long and so well from bodily harm. Unconsciousness, however, may mean more than losing voluntary control of the mental faculties. It may go further and involve the protective usefulness of what are known as voluntary reflexes: the lid reflex which causes the eye-lid to wink and keep out dust; the cough reflex, often called the watch-dog of the respiratory tract; and those profound mechanisms which make the heart beat regularly, and control the rhythm and the depth of the respiration. Transportation of the unconscious is concerned with these deep reflexes, as well as with one's ordinary ability to protect oneself against harm by the use of reason and judgment.

We ordinarily think of unconsciousness as due to sudden illness or accident. This sometimes comes in the course of diabetes or kidney diseases; from hemorrhage in the brain; so-called stroke; as the result of shock due to injury; as a result of excessive heat; from a sudden epileptic seizure, and not infrequently from heart failure. We think of unconsciousness too in connection with the many causes which may end in asphyxial death, such as drowning, electric stroke, suffocation, etc.

There is, however, a large group in which unconsciousness is deliberately brought about by the use of gases and drugs which we call anaesthetics. Under these conditions, unconsciousness is encouraged, so that the patient will not feel the pain of the surgery about to be performed. With good reason, we fear accidental unconsciousness, but are very likely to welcome the unconsciousness which will protect us from pain.

We fear the first and welcome the second, not because of any great difference in the state which is induced, but because we realize that unconsciousness deliberately brought about by the carefully trained medical man is backed up by a knowledge of accurate dosage, accompanied by expert supervision and control. We also feel at ease because the means of causing such unconsciousness is always accompanied by the knowledge and by every modern means of returning us to consciousness should we go further than expected.

In order to protect the unconscious, it is necessary to be somewhat familiar with just what takes place when unconsciousness occurs. As in many other medical conditions, there are degrees or stages of unconsciousness, each of which may be readily recognized.

The first degree of unconsciousness consists of a complete, if temporary, loss of the mental faculties, accompanied by depression of, but no serious interference with, the reflex protective reactions which ordinarily function without the direction of the will. In this first degree, which we speak of as the stage of depression, the eye-lids will wink, the patient can swallow, he can cough, his heart action is steady and regular, and his respirations are quite normal; but the patient is stuporous and can be roused only with difficulty for brief intervals.

In the second stage, that of spasticity, we have the complicating effect of spasm or rigidity of the muscles, affecting the extremities and particularly the face, mouth and throat. The result of this spasm is a tendency to clench the teeth, which interferes seriously with the freedom of the patient's breathing. If the teeth bite the tongue, bleeding may result. Not infrequently vomiting occurs at this time. It is easy to see how vomitus caught in the throat and prevented from escaping by the clenched teeth will result in obstruction to the breathing, resulting in cyanosis and a very serious condition. If the stage of spasticity is not relieved by suitable means, the third stage of asphyxia, *i.e.*, flaccidity, promptly follows.

In the stage of flaccidity, the protective reflexes, which are vital functions, are rapidly dissipated and soon disappear altogether. The obstruction caused by spasm and foreign matter in the throat accompanied by asphyxiation, or suffocation, profoundly affects the center governing respiration, and in turn the action of the heart. Unless suitable means are available to introduce oxygen directly into the lungs to overcome this asphyxial effect, the patient will die. In this connection it is extremely important that the intimate relationship between the respiration and the circulation should be understood. These two functions cannot act separately. Relief and stimulation of the respiration will relieve and stimulate the circulation. Stimulation of the circulation alone without relief of the respiration, however, is useless.

Unfortunately, the degrees of asphyxiation just referred to are by no means generally understood. The average unconscious patient, who is asphyxiated, is approached without regard to the degree of asphyxia or the special indications for treatment. There has been entirely too much tendency to fit such desperately dangerous accidents into routine technique and routine apparatus without regard to the special needs of the case. It is obvious that such needs can be determined only by the trained physician.

Every unconscious patient is entitled to, and requires the immediate services of a physician. First-aid measures are excellent and necessary, but they do not replace this need.

The factors complicating unconsciousness may, therefore, be summarized as follows: Complete loss of voluntary control, a gradually increasing depression of the vital functions, due to spasm of the muscles, and obstruction to the breathing. Body heat is rapidly lost. Unless particular care is taken, injuries may occur to the extremities or to the skin, to the eyes, to the nose, or to the ears.

Recognizing these complications we should know how to meet them. Injury to the extremities is most likely to occur in transit as the result of an arm or a leg dangling loose as the patient is carried. Injury to the skin occurs from heat or from abrasions; and if the eye-lids are open, the lid reflex being sluggish or absent, injuries to the eyes may occur by contact with foreign objects. Owing to the fact that the automatic center governing the heat of the body is often seriously interfered with, chilling occurs easily. The help of direct sunshine or warm blankets will do much to obviate this. Obstruction to the breathing may be prevented by placing the patient on his side and separating the teeth with a pencil wrapped in a handkerchief. When the patient is placed on his side, his hands should be clasped before his face and the knee in contact with the ground should be bent upward as far as possible, the other leg remaining straight. The chin should be lifted away from the chest by placing the hand on the forehead and pushing the head backward.

It is a mistake to believe that speed is vital in the transportation of the unconscious patient. Speed in transportation is entirely secondary to a sensible handling of the immediate conditions.

Occasionally, one encounters patients subject to fainting attacks. These attacks are usually preceded by symptoms well recognized by the patient, such as dizziness, disturbances of vision, etc. When such a patient is aware of the onset of such an attack, she should immediately be seated, place her head as low as possible between her knees and hold it there for at least two minutes. Fainting of this character is caused by sudden withdrawal of blood from the head. When the head is lowered and bent forward the blood remains in it and the attack passes off. Patients who are to be transported by man power should be carried with the head low, so as to maintain as free breathing as possible. When the head is bent on the chest, breathing is interfered with. When it is lifted away from the chest, breathing improves.

Strangely enough, modern ambulance developments have been directed to appearance and to securing the effect of a hotel bedroom rather than offering the necessary facilities for the emergency treatment of the unconscious patient. Not long ago, the speaker transported an anaesthetized patient for a long trip in an ambulance on a cold winter day. While the ventilation of the interior of the car was entirely unsuited to the removal of the anaesthetic vapor, this hazard was nothing compared to the danger from the exhaust back suction, containing carbon monoxide, which found its way into the car through windows which he was obliged to open.

Modern facilities are now available for adequate closed-car air-conditioning at low cost. There is no excuse for a failure to make use of these. Such closed-car air-conditioning must embody the following principles: uncontaminated air must be taken from outside of the car, passed into the car body, and heated by water control, never by the exhaust manifold. The volume of such air must be sufficient to meet the needs of the passengers with all windows closed, except an exhaust vent in the rear to complete the ventilating cycle. A satisfactory type of equipment embodying these principles, known as the Evenair Conditioner, manufactured in Detroit, Michigan, is making an important contribution to the prevention of carbon-monoxide poisoning in closed cars (also Studebaker, p. 109).

The second important requirement for the unconscious patient in transit is "suction." Suction is easily available in every automobile through connection with the intake manifold of the engine; the suction thus produced is powerful and operates in the same manner as that which controls the windshield wiper.

Adequate illumination should be available for direct and continuous inspection of the patient. Such illumination is not satisfactorily provided by a pocket flashlight, but should consist of a powerful swivel ceiling light.

Facilities should also be available to meet the varying degrees of asphyxiation which may develop in the unconscious patient. These facilities include a supply of oxygen and carbon dioxide with the incidental instruments necessary to meet the indications presented by each stage of asphyxia.

It is no longer necessary or desirable to treat the patient at the site of the accident. Facilities should become generally available for treatment en route.

An understanding of the principles underlying the various degrees of unconsciousness with simple mechanical means of meeting these principles will do much to reduce complications which are now so likely to occur during the transportation of the unconscious patient.

Appendix II

Asphyxia Neonatorum*

As a result of the request and two follow-up letters addressed to fifty and twenty-nine universities respectively, sixty-eight replies were received. Five of these sixty-eight universities offered only a two-year theoretical course, reducing the total available response to the obstetrical services of sixty-three medical schools.

Since the outline submitted offers a common ground for discussion, it will be of interest to break this down as follows:

General approval and disapproval by states.

Reaction to specific details by states including matter suggested by correspondence not included in the outline and deserving of emphasis. (Key number noted below refers to particular state.)

The following special references are noted and quoted references follow:

Use of Carbon Dioxide.
Use of Aspiration.
Use of Heat.
Use of Intravenous Alpha Lobelin.
Lumbar Puncture and the Use of Whole Blood Injection.
Mouth to Mouth Insufflation.
Drugs for Respiratory Stimulation.
The Position of the Baby After Delivery.
The Use of Intracardiac Injections.
The Use of Indirect Intubation and Suction.
Prochownick's Method of Artificial Respiration.
The Use of Direct Intubation and Insufflation.
The Relation of Intracranial Pressure to Asphyxia.
The Post-Operative Observation of the Baby.
The Causes of Atelectasis.
The Circulation of Information to the General Profession.
Obstruction by Pressure on the Baby's Face.
The Use of Mechanical Devices—E & J, Flagg, Kreiselman.
Research Now Under Way.
Reference to Euthenasia.
The Use of Sedatives in Obstetrics.

* Continued from page 125.

States and Key Numbers

California, 4, 5, 6
Colorado, 7
Connecticut, 8
Washington, D. C., 9, 10
Georgia, 12, 13
Illinois, 15, 18
Indiana, 19
Iowa, 20
Kansas, 21
Louisiana, 23, 24
Maryland, 25, 26
Massachusetts, 27, 29
Michigan, 30, 31
Minnesota, 32
Mississippi, 33
Missouri, 35
Nebraska, 38
New York, 40, 41, 42, 43, 46, 47
New Mexico, 4, 8
North Carolina, 50, 51
North Dakota, 52
Ohio, 53, 55
Oklahoma, 56
Pennsylvania, 59, 60, 61, 62, 63
South Carolina, 64
South Dakota, 65
Tennessee, 67, 68
Texas, 69
Utah, 71
Vermont, 72
Virginia, 73, 74
Wisconsin, 76
Delaware, 77
Alberta, Canada, 78
Winnipeg, 79
Halifax, N. S., 80
Kingston, Canada, 81
London, Canada, 82
Toronto, Canada, 83
Montreal, Canada, 85

General Approval and Disapproval of Outline

Eight universities objected to the outline proposed for the following reasons:

Considered incomplete.

Raised the question whether first and second stages were asphyxia.

Maintained that cerebral hemorrhage occurred before asphyxia.

Attention should be directed to prevention rather than treatment.

Attention should be directed to causes, sedation and anesthesia.

Suggests that the outline be limited to two stages.

No practical advantage.

Seven universities made no comment.

Forty-eight universities specifically approved the outline as submitted:

"Thoroughly in accord with your classification and treatment. I follow the same treatment in teaching." 62, Penn.

"A good summary." 50, N. Carolina.

"I am in complete accord with treatment as outlined." 48

"Simple, sane, conservative." 12, Georgia.

"Concur in idea presented. Seems extremely sound. Would like reprint." 13, Georgia.

"Data as outlined meets in every detail with observation and treatment carried out in our institution." 23, Louisiana.

"Have gone over whole outline with members of my staff and we feel that it is entirely correct. I am taking the liberty of retaining a copy for the information of our various residents and internes." 24, Louisiana.

Certainly the Importance of Establishing Generally Accepted Principles Covering the Routine Treatment of Asphyxia Neonatorum is Urgently Necessary.

"I thought so highly of your outline for the rational treatment of Asphyxia Neonatorum that I am keeping it in my files on this subject." 38, Nebraska.

"We feel that the brief outline suggested as a fundamental approach to the treatment of Asphyxia Neonatorum is a good one, especially because of its simplified classification." 43, N. Y.

"I agree with your questionnaire as a whole. I think the dissemination of such information in uniformity of treatment will be of great benefit in combating fetal mortality." 51, N. Carolina.

"A very good statement of present day knowledge referable to asphyxia of the newborn." 79, Canada.

"I would personally compliment you on this simple and very rational classification and method of treatment which you have outlined for the asphyxia of the newborn. I like your classification so well that I think I will adopt it. The form of treatment outlined is very similar to what we have been using for the past few years and have been highly delighted with the results. Again let me congratulate you on this splendid contribution on such an important subject." 82, Canada.

Reaction to Specific Details—
The Use of Carbon Dioxide in Resuscitation.

A difference of opinion exists regarding the benefit to be derived from the use of carbon dioxide as a respiratory stimulant.

Since objection to the use of this gas is voiced by a relatively small but important minority, these opinions are quoted.

Ten of the sixty-three universities responding definitely object to the use of carbon dioxide. The following statements are on file as opposed to the use of CO_2:

"We do not believe in the use of CO_2 mixtures in the resuscitation of newborn infants, since the asphyxiated infant is more than saturated with CO_2 and is suffering from oxygen lack." 1

"I am dubious as to the wisdom of using CO_2." 9

"I agree heartily with the above (outline) except for the use of CO_2. We feel that pure oxygen is preferable when artificial respiration is necessary. There is much experimental evidence to support this condition." 10

"We do not believe in the use of CO_2." 18

"If the infant is apneic, it is felt that CO_2 is contraindicated, regardless of whether the depression of the respiratory center may be due to drugs, anoxia or to excess CO_2 alone or together." 13

"In hearty accord with your efforts if we except the CO_2 question." 25

"I note that you include in your recommendations the employment of carbon dioxide and oxygen; and to the use of the former gas, as you know, I cannot agree." 25

"I still feel there is some question whether oxygen alone is not better than oxygen CO_2." 42

"Only comment we had to offer was whether CO_2 should be administered with oxygen in the state of flaccidity." 43

"We do not make use of the combination of oxygen and CO_2." 76

On the other hand, fifty-three universities or eighty-four per cent specifically approve or voice no objection to the outline approving the use of carbon dioxide.

The Use of Aspiration or Suction

If there is one point in the treatment of Asphyxia Neonatorum in which there is almost universal agreement (one exception reported) this is the removal of fluid or other foreign matter from the airway of the newborn infant.

Not only is there unanimity of opinion relative to the value of suction, eighteen universities having called attention to this, but a variety of methods are employed to accomplish this purpose. A widely used method of removing fluid from the trachea and pharynx is the so-called Stripping technic in which the baby is suspended by the feet and the trachea is massaged towards the mouth. Suction applied by the surgeon's mouth through a catheter fitted with a glass receptacle is common practice.

"We make sure that the air passages are cleared before any attempt is made at artificial respirations. As soon as the baby is born the escape of mucus and liquor amnii is encouraged by suspending the baby by its legs. At the same time the pharynx is cleared by suction, using a rubber ear syringe which is sterilized and included in the delivery kit." 1.

"We use gentle cleansing of the mouth and posterior pharynx by oral suction with a soft rubber tube." 13.

"Our management of asphyxia neonatorum includes immediate emptying of pharynx of mucus and fluid by tracheal catheter." 18.

"We use a built-in suction apparatus attached to the bassinet which can be adjusted for weak suction making possible thorough aspiration of mucus without danger of damaging the delicate epithelium of the posterior pharynx and larynx." 25.

"We aspirate mucus from the nose and mouth with ear bulb syringe as soon as head is visible, milk down the trachea as soon as the head is born. If necessary, introduce tracheal catheter and aspirate mucus from trachea placing child on table with head held over edge. We feel that plugging with secretion of the bronchial tree and aspirated mucus is one of the commonest causes of respiratory difficulties, and we use every effort to relieve this condition." 38.

"We consider among the procedures of greatest value in the therapy of asphyxia neonatorum clearing the mouth and throat of mucus." 67.

"The general emphasis on suction and tracheal catheters bothers me. All of these methods, including the use of carbon dioxide and oxygen, doubtless have a very valuable place, but I foresee new difficulties on the basis of their promiscuous use." 30.

"As the clinical procedure for resuscitation I stress the freeing of the air passages of all mucus and secretions. I use a blowing technic that I have perfected through many years rather than the aspiration catheter, though some of my students prefer the latter." 55.

Use of Heat

Obstetrical clinics over the country are becoming thoroughly heat conscious and employ this in the treatment of asphyxia neonatorum. Twenty-two universities consider the application of heat to the child's body of sufficient importance to stress this point in their replies. Among these comments we quote the following:

"We have a very high premature death rate here. We cut it last year over the year before from sixty-three to fifty-two per cent, mainly I think by maintaining the body heat from time of birth to that of arrival at the hands of the pediatricians." 13.

"It would seem advisable to stress more thoroughly the need for maintaining body heat. . . . In severe cases we rely largely on preserving body heat through the use of hot tubs." 20.

"Of the two single procedures which we feel are of greatest value in the therapy of asphyxia neonatorum we would select aspiration of mucus and the application of heat." 67.

"The maintenance of body heat cannot be overemphasized." 5.

"In your outline no statement is made as to how heat is to be applied to the body or what the degree of temperature should be." 1.

"Promptly after delivery the baby is wrapped in a sterile, dry warm blanket." 69.

"Every endeavor is made to keep the child as warm as possible. We do not use the hot and cold bath but sometimes the baby is put in a warm bath, leaving only the face exposed while CO_2 and oxygen or occasionally mouth to mouth insufflation is tried." 79.

"We should handle a baby with extreme gentleness and maintain the baby in a warm temperature." 76.

"The maintenance of body heat is often forgotten until too late." 40.

"Wherever I have any foreknowledge of possible asphyxia I have a warm bath ready so that the baby can be put in it the moment it is born. This means in effect that whenever I put on forceps—no matter how low—wherever I have a breech, or any other abnormal presentation —whenever the labor has been protracted in a tight-fitting pelvis— wherever there are signs of a failing fetal heart, the bath is ready and waiting to receive the baby. I feel very strongly that this is the most vital part of my treatment.

"I do not see the sense in resuscitating a baby that is going to die a few hours or a few days later because its body heat has been allowed to fall too far in the first half hour after birth, and I therefore feel that my main criticism of the scheme you lay down is the neglect to stress this most important point. I would not only put it before all your other steps, but I would put it in the largest capitals you have. And what is more, I would like to stress the point that the bath should be ready and waiting when the baby is born." 80.

Intravenous Medication

Reference is made by fourteen correspondents to experience in the use of intravenous medication with special reference to alpha lobelin.

"Am persuaded that alpha lobelin into the umbilical vein is of a valuable help. There seems to be some difference re use but I am convinced that after ten years' use of it that it does the trick in a certain percentage of cases." 80.

"We resort to the administration of oxygen CO_2 under controlled pressure for a measured period. The percentage of CO_2 being five per cent. Should this method fail to resuscitate the infant, alpha lobelin is then administered, one twentieth grain injected into the umbilical vein as prescribed by Dr. Robert A. Wilson of New York City." 23.

"We are under the impression that alpha lobelin is of value in the second and third stage." 6.

"If the baby is doing badly, 1 cc. of alpha lobelin is injected into the cord vein before the baby is cut off." 74.

"In those cases in which there is respiratory failure we use alpha lobelin." 15.

"In cases of delayed respiration we inject alpha lobelin or coramine directly into the vein of the umbilical cord and milk it towards the baby's abdomen. We are not sure that these drugs are of any value but we are of the opinion that they do help in initiating respiration." 24.

"I believe that there may be some value in the addition of alpha lobelin or metrazol in certain cases." 31.

"We give alpha lobelin in the hope of stimulating respiration." 38.

"Occasionally alpha lobelin is injected into the umbilical cord and then gradually milked into the circulation as needed." 1.

"Would urge caution in the use of hypodermic or intravenous respiratory stimulants unless cardiac stimulants are also available. This applies particularly to alpha lobelin." 52.

"Most, if not all, drugs used to stimulate respiration are valueless. Certainly we have found this true of alpha lobelin." 40.

"I have yet to see a single baby benefited by the intramuscular or intravenous injection of drugs." 21.

"I do not believe that intravenous medication is practical except through intra cardiac injection or into longitudinal sinus." 18.

"Between the years 1925 and 1927 I used alpha lobelin routinely in the treatment of asphyxia neonatorum but stopped after having lost two infants from convulsions and I have reason to believe that they were the result of alpha lobelin. Autopsies showed no organic causes for these convulsions. You will note that even enthusiastic authors admit that alpha lobelin produces opisthotonus." 25.

"I am not convinced that some of the respiratory stimulants, such as alpha lobelin, are valuable." 18.

Use of Lumbar Puncture; Injection of Whole Blood to Reduce Cerebral Hemorrhage

"If we suspect intracranial hemorrhage we usually employ lumbar puncture with the administration of whole blood into the buttocks of the baby." 76.

"The clotting time on the baby is taken and if over five minutes by our method the baby is given an injection of 10 cc. of blood to try to control cerebral hemorrhage because that, we believe, is a very common cause of asphyxia." 74.

"At times we practice a spinal or cistern puncture hoping by that to relieve pressure on the vital centers. There is some difference of opinion particularly on the part of the pediatrician as to the advisability of this procedure but I personally feel that at times a baby has been saved by spinal or cistern tapping at this time." 24.

Mouth-to-Mouth Insufflation Is Well Established and Generally Recognized

This is especially useful where mechanical methods have not been adopted or are not available.

One of our correspondents refers to this technic in no uncertain terms.

"Mouth to mouth insufflation properly used is much more valuable than any machine yet devised." 40.

"If simple apparatus is not available we employ mouth to mouth insufflation covering the baby's mouth with gauze, holding the nose to prevent reflux and extending the head in order to establish a free airway." 24.

"We are still employing mouth to mouth insufflation. I am acquainted with several drawbacks of this method but it possesses a great advantage of requiring no apparatus and being available on a moment's notice. Our staff are given instructions in regard to the force of breath equivalent to 14 cm of water and are urged never to exceed it." 25.

"I lay more stress than you apparently do to mouth to mouth breathing and to artificial respiration by compressing and relaxing the thorax with the babe held in the inverted position, the head resting on the table to prevent extension." 55.

"It might be noted that when pure CO_2 is not available mouth to mouth respiration is a substitute of some value if properly done." 83.

A Number of Institutions Employ Drugs for Respiratory Stimulation

"Metrazol may help. We are proceeding slowly with our experiments with this drug." 40.

"Coramine and caffeine are occasionally used." 8.

"I have not seen any wonderful results by hypodermic injections." 27.

"Our experience with drug medication has not been very good and consequently we use it rarely.

"Adrenalin, alpha lobelin and coramine are rarely employed except in very severe asphyxia where the results of our administration are not particularly good." 20.

"We employ alpha lobelin and adrenalin injected directly into the musculature of the heart as a measure of last resort." 67.

"We rarely use hypodermics as respiratory stimulants." 69.

The Position in Which the Newborn Baby Is Placed after Delivery Is Considered of Importance by a Number of Our Correspondents

"It would seem advisable to define correct posture." 20.

"I would state definitely what you believe to be the correct posture in which these infants should be placed." 46.

"I feel that the correct posture should be explained for those unfamiliar with it." 51.

"The child is kept on its right side immediately after suction." 59.

"Immediately after delivery the infant is maintained in a posture with the head slightly below the level of the body." 61.

Intracardiac Injection for Resuscitation Is Recommended as a Last Procedure

"Adrenalin is injected into the right auricle if cardiac arrest is present." 13.

"Intracardiac administration of adrenalin is reserved for desperate cases." 18.

Spanking, Tubbing in Hot and Cold Water and Holding by the Feet

"We still conform to some of the older methods, thumping of the soles of the feet gently and a mild or brisk rubbing of the back of the child while in the vertical position." 23.

"Avoid suspension by the feet if cerebral hemorrhage is suspected." 38.

Indirect Intubation

Indirect intubation by manual palpation accompanied by suction according to the method of de Lee is widely practiced as may be noted from the fact that it is referred to by seventeen universities. We quote a number of these comments:

"If cardiac action does not improve, tracheal catheter is introduced and suction made, removing all fluid from the upper respiratory tract." 18.

"We also aspirate mucus from the pharynx but rarely resort to tracheal catheterization." 20.

"If respiratory efforts are made and it is apparent that air is not entering the lungs, the trachea is cleared with an intratracheal catheter according to the de Lee method." 24.

"We do not have any instruments except the tracheal catheter for clearing the passages." 27.

"The creation of a clear air passage, if possible, is absolutely necessary and can be accomplished in the great majority of cases without the use of a tracheal tube. The use of the latter by other than the most experienced is dangerous. Rarely do we find it necessary." 40.

"We recommend blind passage of intratracheal catheter with an index finger. Laryngoscope never found necessary." 67.

"We use a catheter attached to a glass mucus trap for sucking the mucus out of the air passages." 69.

"We have a tracheal catheter if ordinary suction does not seem sufficient." 79.

"I have found that my house staff can be easily taught to introduce a soft rubber catheter into the trachea under the sense of touch." 1.

"The general emphasis on the tracheal suction and catheters bothers me. I am inclined to believe that inexperienced and unqualified individuals will in time do considerable harm if they become enthusiastic about such procedures." 30.

Prochownick's Method of Artificial Respiration (referred to by two universities although the method is undoubtedly practiced by many schools).

"I would suggest the inclusion of Prochownick's method of resuscitation in the first and second stages." 48.

"In most cases we find that cleansing the throat of mucus and applying gentle rhythmic pressure over the chest with the newborn held by its feet and the head extended (Prochownick's method) will resuscitate most of the asphyxia cases in the stages of depression and spasticity." 67.

Direct Intubation

Direct exposure of the larynx by laryngoscope according to the Chevalier Jackson method of intubation and suction and insufflation as recommended by Flagg are both condemned and approved.

"I believe that examination of the pharynx and glottis which requires the use of a suitable piece of apparatus is beyond the ability of the average physician, largely because he does not possess such a piece of equipment." 1.

"I fail to see when an inspection of the glottis has anything to do with the treatment of these cases. While we do not have one, there is a bronchoscope in use for premature infants that is inserted by sight due to the fact that the premature baby's throat is so small that it is almost impossible without doing a great amount of harm to insert the tracheal catheter in the usual manner. After insertion of this bronchoscope the catheter is passed through it into either bronchii. This is exceptionally useful in the treatment of premature infants. It requires no great amount of training for its use." 15.

"I do not believe inspection of the larynx and the vocal cord are practical in the average delivery room during delivery by an interne, general practitioner or obstetrician." 18.

"We tried the bronchoscope but on the recommendation of the pediatric section we have discontinued anything which would cause an inflammatory action." 27.

"Close at hand in each delivery room are the E & J respirator and the Flagg respirator. The Flagg apparatus is used routinely when spastic obstruction is present and the E & J respirator and Flagg apparatus are applied when necessary and air-way is often used with the E & J." 59.

"All treatment is stopped when the reflexes return and when respiration is established and in those which falter, constant observation and administration of CO_2 and oxygen are given at measured intervals. In addition, I am giving our figures compiled by the Chief of the Pediatric Staff over a period of seven years. These were 15,488 deliveries and 944 baby deaths, a fetal mortality of 2.6%: there were 4.062 private cases and 98 baby deaths or 2.4% with corrected fetal mortality of .98%." 59.

"I personally am convinced that it is seldom necessary or advisable to invade the trachea with a metal tube and apply strong mechanical suction." 63.

"We use the Flagg apparatus and like it." 7.

Asphyxia and Cerebral Hemorrhage

There is much difference of opinion concerning the sequence in which asphyxia and cerebral hemorrhage take place. The opinion is repeatedly expressed that intracranial hemorrhage occurs independently of, or is the cause of, asphyxiation.

"One of the most important factors to determine in this whole subject is the relative importance of the asphyxia as the cause of death or some other condition, such as cerebral hemorrhage, which produces the asphyxia. The present statistics on asphyxial death in the newborn are absolutely worthless. We are showing that twenty-five per cent of the neonatal deaths, if carefully autopsied, are due to cerebral hemorrhage. It is probable that no amount of management of the asphyxia will greatly influence the outcome in these cases." 18.

"Any discussion of asphyxia in my opinion should not fail to take into account the fact that many babies delivered instrumentally are really suffering from intracranial hemorrhage which may be the main factor in the case. This is particularly true, of course, as regards difficult forceps delivery or breech extraction. Of course premature babies are particularly liable to intracranial hemorrhage. We have even noticed this complication in some babies delivered by caesarian section. Some of these can be explained by the fact that the mother had been in labor for a considerable time before the section was performed, with pressure of the fetal head above the pelvic brim. For others, no other explanation seems to be forthcoming." 24.

"I might say that I have become intensely interested in the late effects of asphyxia resulting in cerebral anoxemia. Our interest has been particularly aroused by work of Schriber, Journal, Michigan State Medical Society, Feb., 1938, as to the possible late end results in infant suffering from this condition." 31.

"The pallid, flaccid type of asphyxia will rarely respond to treatment even if it does, the eventual outcome is doubtful since most of these babies have suffered intracranial hemorrhage." 40.

"The most difficult problem is to determine whether one is dealing with asphyxia alone or intracranial hemorrhage." 42.

"I am convinced that the picture described under stage three is in the vast majority of instances associated with an underlying intracranial hemorrhage." 46.

"In the few cases in which asphyxia does occur we believe that the first efforts should be made to determine whether there is intracranial damage or not. If we suspect the former we usually employ lumbar puncture and the administration of whole blood into the buttocks of the baby." 76.

"It is my opinion that some cases of still birth following the flaccid type and not coming to autopsy are really due to intracranial injury." 83.

"Experience in numerous autopsies have demonstrated that the cause of asphyxia is a large or a small cerebral hemorrhage." 85.

Post-Operative Observation

An important factor in the treatment of neonatal asphyxia is continuous post-operative observation of the baby. This point is referred as follows:

"Would suggest specific instructions for observation for some hours after birth." 50.

"Constant observation is recommended."

Atelectasis

A reference dealing with atelectasis, an extremely interesting and important problem in connection with asphyxia is noted.

"One group of cases of asphyxial deaths which particularly concerns me are those babies born spontaneously or by caesarian section which appear normal at birth, cry lustily, are a good color when sent to the nursery, who within a few hours develop a cyanosis and respiratory difficulty and who die apparently from this cause. Post-mortem in these cases shows marked atelectasis. This degree of atelectasis probably did not exist shortly after birth. Why it developed I have not determined. I am sending a group of newborns to the X-Ray Department for lung pictures in order to throw some light on the expansion of the air sacks in the first few hours of life." 18.

General Information to Be Disseminated

A number of our correspondents feel that it is desirable that certain general information should be widely circulated. Specific recommendations are noted as follows:

"To spread broadcast the technic of some relatively simple program such as you recommend is most commendable; and efforts along this line should be pushed, it seems to me, as much as possible." 25.

"Would like to see you eliminate:

1. Ancient resuscitation methods from modern textbooks.
2. Those awful resuscitation basins from delivery rooms.
3. Haphazard hypodermic cardiac stimulation.
4. Intracardiac injection.
5. Routine holding up of babies by feet.
6. Alpha lobelin." 41.

"Suggestions:

1. I feel that the correct posture should be explained for those unfamiliar with it.
2. Call attention to certain don'ts; such as, Do not manipulate or strike the baby too vigorously in attempting stimulant or respiration.
3. Use tracheal tubes with caution." 51.

"The reader is specifically referred to the results of a questionnaire suggested by the Chicago Board of Health." 67.

"Frankly it seems to me that in such a report the emphasis should be placed on prevention rather than cure. I am convinced that if proper obstetric diagnosis and care employed together with good judgment in the use of analgesic and anesthetic agents there would be very little need for the active treatment of asphyxia neonatorum. Furthermore, auscultation of the fetal heart in the second stage of labor with judicious control of the oxygen and carbon dioxide content of the mother will render the number of asphyxiated babies so small that there will be little opportunity to put into practice means of treating asphyxia. . . . I am firmly of the opinion that this is where the emphasis should be placed and it seems to me one of the most neglected phases of modern obstetrics." 76.

Pressure on Lower Jaw

The importance of obstructing the baby's airway by making pressure upon the lower jaw with a mask is noted.

"We emphasize as you do that artificial respiratory obstruction may be easily induced by the slightest pressure on the baby's face which tends to depress the lower jaw." 5.

"Keep the baby's lower jaw well foward." 15.

"If the note under Indications for Relief of 2nd Stage implies that artificial respiratory obstruction is frequently induced by the slightest pressure on the baby's face which tends to press the lower jaw, I must disagree, since in my experience the overwhelming majority of these cases respond to the passage of a tracheal catheter in the usual manner which necessitates inserting the finger in the mouth and pressing the lower jaw. I feel that the danger in attempting not to clear the upper pharynx is greater than the danger of producing artificial respiratory obstruction in these cases." 18.

Specific Apparatus

Since specific references to apparatus known by name are made in a number of cases we note these references:

"We had one of the first Drinker respirators that was made but have disposed of it, since we did not find it of any benefit. The difficulty seems to be that the machine operating at a regular rate of speed could not be adjusted to the respirations of an infant which, as you know, are extremely irregular at birth." 1.

"The use of the small Drinker respirator has been helpful in the relief of the third stage of asphyxia." 6.

"The using of various mechanical respirators, such as the Drinker apparatus and others may prove of value when available." 18.

"In some cases especially as regard premature babies we have employed the Drinker apparatus and feel that we have occasionally saved a baby's life in this way." 24.

"As to the use of methods of resuscitation, permit me to say that in addition to the usual methods of artificial respiration we also have the Drinker respirator. I am sorry to say that the mechanical respirators which we have used, and we have probably used each one of them as they have appeared and been discarded, we have not been greatly impressed by them. We have found these machines valuable only in maintaining the respiration after breathing has been established." 32.

"We have practically given up the Drinker respirator and use it only for those cases which continue to have respiratory difficulty, namely atelectasis and weak, puny infants whose respiration falters while in the nursery and in the early days of their existence." 59.

"If breathing does not begin immediately the infant is placed in a Drinker respirator and is given artificial respiration continuously until the normal rate of respiration is inaugurated or until death takes place. This is indicated by periodic examination of the heart sounds. During the treatment in the respirator a face mask covers the infant's face and it is permitted to breathe a mixture of carbon dioxide and oxygen. The treatment is the same regardless of the apparent degree of asphyxia." 61.

"We recommend the Foregger or Drinker respirator for artificial respiration in the hospital." 67.

"We have been using the E & J resuscitator for the past six years. We feel quite certain that we have saved one life and possibly two or three others during that time." 21.

"Close at hand in each delivery room are the E & J respirator and the Flagg respirator." 59.

"We use the E & J resuscitator which has given excellent satisfaction." 63.

"In extreme cases the Kreiselman machine is used." 1.

"The Kreiselman apparatus is obviously suitable only for hospital use." 25.

"We have been using the Kreiselman apparatus for the past two years in the treatment of asphyxia neonatorum. It seems to us to supply the four main desiderata, proper posture, aspiration of mucus, the delivery of oxygen under measured pressure and warmth." 25.

Researches in Asphyxia Neonatorum Now under Way

"Our assistant pediatrician is conducting a somewhat extensive piece of research on exchange of gases through the placenta." 1.

"Research under way: Determination and comparison of maternal oxygen tension to fetal oxygen tensions when Barbiturate and Hyoscine Amnesia employed in delivery." 13.

"Studies in intra-uterine respiration and the effect of various types of anesthesia on this phenomenon. We have been hoping to get under way a study dealing with the efficacy of various methods of insufflation in producing expansion. This would have to be carried out on stillborn infants and presents certain technical difficulties which we have thus far not been able to circumvent." 25.

"At present we are undertaking an investigation bearing directly upon the problem of asphyxiation. Earlier in the year, however, we carried on some unpublished clinical experiments on the effect of morphine administration to the mother in labor. As a result of this work, we are more than ever persuaded that morphine rarely has any harmful effects and that it has unjustly developed a bad reputation as an agent likely to produce fatal asphyxia. It is likewise the opinion of the staff that any possible morphine effect on the baby is rarely, if ever, fatal." 20.

"Children whose mothers have had some form of analgesia during labor seem more prone to develop cerebral anoxia. We think that possibly this may be due to a lowering of the metabolic rate during labor under that which we would normally expect in a woman without analgesia, and we are attempting now to set up a normal by taking metabolic rates on patients actually in labor without analgesia." 31.

"A report on infant autopsies." 46.

"Euthanasia" in Asphyxia Neonatorum

"Some times I wonder how justified we are in stressing procedures for resuscitating babies. If we assume that the more severe grades of asphyxia are commonly associated with intracranial lesions there is certainly some doubt as to whether these babies should be saved. Certainly when there is definite evidence of intracranial hemorrhage it may be better for everyone concerned if they are permitted to die peacefully, rather than to make them burdens to themselves and to their community.

"No doubt you have given thought to the hard-boiled philosophy in such a suggestion." 20.

Sedatives to Mother

Indirect reference to sedatives given the mother have already been noted. However, the following comments stress the situation directly.

"We feel that it is important not to give morphine to the mother in the last three or four hours of labor, fearing that it will depress the baby's respiratory center. Consequently we do not employ it previous to the performance of caesarian section. We do not feel that the barbiturates which are so widely employed at the present time when properly administered give any particular difficulties in regard to the resuscitation of the baby. Ether when given to the surgical degree does appear to effect the baby to some extent but it has never done so to an alarming degree in our experience. We never employ it in an operative obstetric case except when we are unable to obtain a skilled anesthetist or when apparatus is not available, such as is the case in obstetrics performed in the home. Our preference is for ethylene oxygen as an obstetrical anesthetic." 24.

"First, while asphyxia neonatorum will always be with us, I wonder if we haven't got the cart a bit ahead of the horse in emphasizing asphyxia when perhaps we might be placing the emphasis on the factors which produce asphyxia with particular reference to over-sedation." 30.

"I am impressed by the fact that the conduct of the labor and the use of drugs for relieving the pains of labor are large factors in asphyxia. I feel that there should be a special investigation of the cases in which the various drugs have been applied in the production of so-called painless labor. I think that one of the objections to the indiscriminate use of such drugs for the relief of pain is reprehensible, and in any research on this problem this should be a prominent feature." 32.

"It is my belief that the two predominant causes of asphyxia neonatorum are: First and foremost, injudicious and unwise attempts at analgesia and anesthesia; and secondly, the inordinate amount of operative deliveries that are done in the country as a whole. The two subjects are so intimately interrelated that it is usually impossible to separate them. In other words, excessive drug therapy of various kinds is being employed which so interferes with the normal process of labor as to render operative delivery more or less the rule. For example, just recently, I heard a prominent obstetrician connected with a well-known medical society, in which he advocated large doses of nembutal. He admitted that the dosage was so great that it was necessary to complete the delivery by forceps in almost every instance. I cannot help but believe that these two factors are responsible for the large incidence of asphyxia neonatorum.

"Our practice here is to give our patients as much relief in labor as we can consistent with the normal progress of labor and this we accomplish in the first part of the first stage by the use of heroin; in the latter part of the first stage and throughout the second stage the use of intermittent nitrous oxide oxygen, maintaining the oxygen at a high level, especially for the few moments before the birth of the baby. Of course, we have to vary our technic with the individual patient, but I have outlined the management of the average case on our hands. The result is that labor is not unduly prolonged, operative incidence is not increased, and rarely is it necessary for us to use artificial means to establish normal respiration.

"I would not have you believe from the above that we are entirely satisfied with our methods of pain relief in labor. While I wish we could afford more relief to our patients than they now obtain, and I believe that the day will come when that will be possible, I am satisfied that we are not jeopardizing the baby's welfare and neither are we increasing our incidence of operative deliveries." 76.

"I am also impressed with the fact that the conduct of the labor and the use of drugs for relieving the pains should be a special investigation of the cases in which the various drugs have been applied in the production of so-called painless labor. I think that one of the objections to the indiscriminate use of such drugs for the relief of pain is reprehensible, and in any research on this problem this should be a prominent feature." 32.

"We use no pituitary extract during labor. We use no scopolamine and no barbiturates. For twenty-five years it has been well accepted that 30% of all babies born in twilight sleep labors show some degree of asphyxia. Fritz Irving's article on the barbiturates some two years ago showed exactly the same figures (30%). Without making a careful survey of our own results, it is my opinion that we do not have more that 3%, including the complicated as well as the uncomplicated asphyxia. I think it is high time that the American Medical profession should receive a scathing denunciation of twilight sleep and all similar procedures. It seems to me that your committee is in an excellent position to render a real service of this sort. The only defense that I have ever heard a physician give for the use of such procedures is that they were "Business getters." 21.

Note: Endotracheal intubation and suction may be said to be practiced by two distinct schools. The first employs palpation and blind intubation. It corresponds to the technic popularized by Joseph O'Dwyer in his treatment of diphtheria fifty years ago. The second employs direct vision by the use of a laryngoscope and corresponds to the technic employed by Chevalier Jackson in per oral endoscopy. Endotracheal suction is indicated and should be practiced only in advanced spasticity and flaccidity. In spasticity suction may be necessary to relieve glottic obstruction from mucus or amniotic shreds. In flaccidity tracheal fluid may demand removal. It is difficult to remove shreads of mucus or pools of fluid which cannot be seen. It is an entirely non-traumatic procedure to laryngoscope and intubate a flaccid baby.

Exposure of the field and treatment under direct vision in the case of asphyxia neonatorum will very likely follow the course pursued in the case of diphtheria where blind intubation has given way to direct methods as familiarity with the structures involved and the technic employed becomes generally understood.

Appendix III

Judicial Electrocution

The man offers the factor of resistance to current (in ohms). This is unknown until read from ammeter when the initial shock is thrown.

Electric current is supplied by special dynamo and carried by a special circuit to floor of death chamber. The voltage to be delivered to the victim is set on voltmeter at 2000 volts. This figure has been developed from experience. Lower voltage produces delayed effects, higher voltage burns.

The Amperage or amount of current delivered is determined by the resistance offered by the body. Thin men offer more, fat men less resistance. The amperage used varies from 8–13. The voltage used governs the amperage delivered. As the initial shock of 2000 volts is thrown the amperage used to overcome the resistance of the body is immediately indicated on the ammeter and recorded. L. required 10 amps, C. 11. L. therefore presented a resistance of 200 ohms. $I = \frac{e}{r}$ or $a = \frac{v}{r}$, $10 = \frac{2000}{X}$ or $10\,X = 2000$, $I = 200$ ohms.

Execution: Prior to execution the victim's hair is clipped short, for better contact. Before the arrival of the victim the executioner selects a helmet conforming to the shape and size of the man's head. This helmet or anode contains a circular metal band which runs around the inside of the helmet like a hat band. Radiating metal strips connect it to the apex which terminates in a connection for the wire terminal bringing the current to the chair from the cement floor. The metal cage is covered by a wire mesh. This in turn is covered by a sponge-like cap kept wet with a brine solution until just before the victim is admitted to the chamber. The cathode emerges from the cement floor through a metal post near the right front leg of the chair. To it is attached a wire terminating in a metal band with a brine soaked lining. This band is attached tightly to the right leg just below the knee.

The man enters, is seated in the chair and strapped in place by the guards. The executioner meanwhile tightens all electrical connections. He then places the helmet firmly upon the man's head attaching it by a strap going under the chin. The anode terminal is then attached to the helmet. Meanwhile the guards apply a rubber face mask over the face. This has a slit to conform to the mouth to permit drooling. The cathode is firmly attached to the leg, the right trouser leg having been slit to accommodate it. The executioner then steps quickly to his booth. At a signal from the P. K. (Principal Keeper) he throws the main switch which has been set to deliver an initial voltage of 2000v. He immediately reads the ammeter and notes the reading which varies from 8–13 amps. He then reduces the voltage until two amps are being delivered. At the end of thirty seconds the voltage is raised to 1600 volts and again reduced to deliver two amps. This is repeated five times in all (initial shock, at 30 sec., at 1.00 min., at 1.30 min., at 2.00 min.). Two amperes have been flowing continuously with the above noted variations in voltage and amperage. At the end of two minutes the main switch is released. The victim remains in the chair until pronounced dead by the prison physician. The straps are then released and the man is transported to the morgue for immediate autopsy. High degrees of heat are developed at the anode and cathode. The temperature of the brain has been noted at 120°F. A guard who lifted a victim from the chair to the stretcher with a hand on the leg burned by the cathode received himself a third degree burn from the residual heat in the tissues. *Heart action.* Auricular and ventricular fibrillation is frequent. L.'s, heart showed fibrillation of the auricle and fibrillation ten minutes after removal from the chair, after the chest wall had been removed and the pericardium had been opened. Death is thought to be due to permanent paralysis of the respiratory center, resulting in fatal asphyxia.

It is estimated that shock produces loss of consciousness with twice the rapidity that pain is perceived. Therefore death is entirely without pain.

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