3 with sulfanilamide, and (e) 1 with sulfanilamide, serum and neoarsphenamine.

2. Neoarsphenamine gave the best results in selected cases.

3. The dictum "hands off the local lesion" should be strictly adhered to.

Front and Luzerne streets.

ARTIFICIAL RESPIRATION AND INHALATION

THE PRINCIPLE DETERMINING THE EFFICIENCY OF VARIOUS METHODS

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In the thirty odd years since Schafer [1] introduced prone pressure artificial respiration, this method of resuscitation has come to be used in all English speaking countries to the virtual exclusion of all other manual methods. In this country the Red Cross and the United States Bureau of Mines, through their field agents and by enlisting the collaboration of others, have trained many millions of persons—police, firemen, seamen, miners, boy and girl scouts, college students and others —in this method of resuscitation.

Outside the English speaking countries, however, the Silvester [2] and other older methods are still extensively used, and various other manual methods—generally modifications of that of Schafer—have in recent years been proposed. All these methods, old and new, were demonstrated by means of photographs and respiratory measurements at the International Congress on Resuscitation and First Aid at Zurich in August 1939.[3] One of us participated, and in this paper we shall draw in part on the evidence there presented.

THE ELASTIC RECOIL OF THE CHEST

In all manual methods, expirations are induced by the operator compressing the chest of the patient with his hands, or by pressing the diaphragm up against the bases of the lungs. The Schafer method differs from the others in the fact that this is all that the operator attempts to do. To induce inspirations he merely removes his hands or quickly releases the pressure, it matters not which. The inspirations are therefore wholly due to the elastic recoil of the chest in the intervals between compressions. But in order to permit this expansive force or recoil to come into play the more easily, the patient's arms are once and for all placed as far forward as possible; one of them is bent at the elbow with the head resting on it. The chest is thus brought initially, as far as possible, into the inspiratory position —a feature introduced many years ago by one of us (Y. H.) in work for the first resuscitation commission [4] and now long since universally adopted as part of the prone pressure method. The technic is therefore extremely simple; it requires much less exertion on the part of the operator than any other method, and it can be maintained longer without fatigue. The one requirement for maximum efficiency is that the tempo shall be slow enough and the intervals between compressions long enough to allow the chest to expand all that it will. In patients with broken ribs prone pressure is the least harmful form of manipulation.

All, or nearly all, the other manual methods attempt to contribute actively to the expansion of the chest by pulling the arms, rolling the body or lifting the shoulders, as Nielsen [5] does. These methods raise the question whether such manipulations actually do aid in artificial respiration. In other words, is it really possible by any such means to increase the expansion of the chest? To these questions the experiment to which all investigators have resorted has been that of applying artificial respiration to normal men.[6] The men are told to make no effort to breathe but to submit themselves passively to whatever manipulation the operator applies. Some investigators have measured only the volume of the first few breaths and have claimed an increase by their technic. But this increase is really due only to the discomfort induced in a conscious subject and is followed by a few seconds of apnea. Other investigators [7] have measured the amount of pulmonary ventilation per minute by means of a gas meter and have found that, if the experiment is continued for several minutes or longer, the volume of air pumped in and out of the lungs is essentially the same under all methods, no matter which one is used and no matter whether a large or only a moderate force is applied.

The fact that the volume of pulmonary ventilation is the same, in spite of wide variations in the technics and exertions of the operator, is at first somewhat surprising. But its correctness is attested by Henderson's observations in work for the first resuscitation commission [4] nearly thirty years ago. It was then found that, when the volume of air which a normal man breathes in natural respiration and then the amount of artificial respiration that can be induced by the Schafer, Silvester of any other manual method are measured, all these volumes—that of natural breathing and those under the various forms of artificial respiration—are always essentially the same in liters of air per minute.

Similarly, in all the new technics shown at Zurich,[3] it was clearly to be seen that the amount of pulmonary ventilation induced was always so near the normal that the patient had no tendency or desire to breathe for himself. But on the other hand—and this is the crucial point—no manual method tested in this laboratory or demonstrated at Zurich, no matter how vigorously applied, has ever produced any considerable degree of overventilation of the lungs, as demonstrated by the fact that when the artificial respiration is stopped the subject, after apnea of only a few seconds at most, immediately begins to breathe again for himself.

From the Laboratory of Applied Physiology, Yale University.

1. Schafer, E. A.: Description of a Simple and Efficient Method of Performing Artificial Respiration in the Human Subject, Especially in Cases of Drowning to Which Is Appended Instructions for the Treatment of the Apparently Drowned, Med.-Chir. Tr. London **87**: 609, 1904; Artificial Respiration in Man; Harvey Society, New York, 1907-1908, p. 223.
2. Silvester, H. R.: A New Method of Resuscitating Stillborn Children and of Restoring Persons Apparently Drowned or Dead, Brit. M. J., 1858, p. 576.
3. International Congress on Resuscitation and First Aid (transactions not yet published); Dr. H. Schaer, president, University Klinik, Zurich; Dr. C. J. Mijnlieff, general secretary, Wodanstraat 24, II, Amsterdam.
4. Report of the Committee on Resuscitation from Mine Gases, Technical Paper 77, United States Bureau of Mines, Washington, D. C., 1914.
5. Nielsen, Holger: An Oplivningsmetode, Ugesk. f. læger **94**: 1201 (Dec. 15) 1932.
6. Anderson, N., and Ekström, T.: Die Ventilation bei künstliche Atmung am Menschen mit der Methode von Schäfer und Holger Nielsen, Skandinav. Arch. f. Physiol. **83**: 211 and 225, 1940 (full literature).
7. Henderson, Yandell: Adventures in Respiration: Modes of Asphyxiation and Methods of Resuscitation, Baltimore, Williams & Wilkins, 1938, p. 273. Drinker, C. K.: Carbon Monoxide Asphyxia, New York, Oxford University Press, 1938, p. 172. Drinker, C. K., and Shaw, L. A.: A Modification of the Nielsen Method of Artificial Respiration, J. Indust. Hyg. **17**: 243 (Nov.) 1935. Waters, R. M., and Bennet, J. H.: Artificial Respiration: Comparison of Manual Maneuvers, Anesth. & Analg. **15**: 151 (May-June) 1936.

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THE PRINCIPLE DETERMINING THE VOLUME OF VENTILATION

Evidently, then, in a normal man under experimental artificial respiration it is the man's own metabolism that determines how much pulmonary ventilation he needs, and this is the amount that can be, and is all that can be, induced by any method of manual artificial respiration. Evidently also it is under essentially the same control as natural breathing, the control exerted by the carbon dioxide and oxygen of the blood acting on the respiratory center. Through the work of Hess [8] it is now recognized that the influence of the respiratory center on the diaphragm and thoracic muscles is largely that of controlling tonus, and it is the tonus of muscles that affords their elasticity, so called—not a mere mechanical elasticity but really an entirely vital property. It is largely for this reason that inhalation of carbon dioxide increases both the volume of natural breathing and the volume of pulmonary ventilation under manual artificial respiration both of a normal man and of one who is unconscious and apneic. Conversely, if a man has overbreathed and has thereby decreased the carbon dioxide in his blood just before he receives manual artificial respiration, very little pulmonary ventilation can be induced. The tonus of the muscles is decreased, and after the chest is compressed it does not expand again with normal vigor. In deep asphyxia a similar decrease occurs. A few minutes after death the chest does not expand at all; tonicity and elasticity are gone. On the other hand, as has recently been shown by Lougheed, Janes and Hall [9] in their important studies on resuscitation from drowning, the efficiency of manual artificial respiration is much increased and the patient is more rapidly resuscitated if the artificial respiration is supplemented by the simultaneous inhalation of carbon dioxide and oxygen. The influence of carbon dioxide on muscle tonus is potent also for the support of the venous return of the circulation and the efficiency of the heart. Continuance of this inhalation for a time after resuscitation should tend also to prevent pneumonia, as it does after carbon monoxide asphyxia.

The principle determining the efficiency of all methods of artificial respiration is thus found to be essentially the same as the principle controlling the volume of natural breathing. It is the influence of the blood gases on the respiratory center that largely determines the tonus of the respiratory muscles; and this tonus is a principal factor in the volume of lung ventilation alike in natural breathing and in artificial respiration. Because of this principle, no manual method—neither prone pressure nor any other—can induce any larger volume of lung ventilation than the tonic elasticity of the body at the moment permits. The reason is that, if any degree of overventilation is induced for even a few seconds, the decrease of the carbon dioxide in the blood induces a decrease in the tonic elasticity of the body with a corresponding decrease in the amount of ventilation that can be produced. All attempts to invent some new method or some modification of an old method that will cause more ventilation than the prone pressure method, which is best merely because it is simplest and easiest to apply, is—and in our opinion will always be—wasted effort. In order to bring this point to a conclusive test, one of us, at the close of the International Congress on Resuscitation and First Aid, placed in the hands of the executive committee the sum of 100 Swiss francs to be offered as a prize (or rather a challenge) to any one who can devise and demonstrate a manual method which, after application for fifteen minutes, is followed by apnea for three minutes.

BREATHING MACHINES

In physiologic laboratories animals under experiment, after being deprived of the capacity to breathe normally, either by some drug or by section of the vagus nerves, are kept alive by means of artificial respiration. It is generally administered with an air pump or bellows. Similarly in cases of respiratory paralysis at the hospital the patient is kept alive for long periods by means of the Drinker respirator.[10] Neither in the laboratory nor in the hospital, however, are such appliances used for the purpose—strictly speaking—of resuscitation. They do not induce a rapid recovery of the capacity in the animal or man to breathe for himself; indeed, they are not so intended. They maintain life but do not restore the animal or man to normal condition—at least not quickly—as for instance does resuscitation from drowning or electric shock by manual artificial respiration.

It is a surprising fact, yet quite certainly true, that even a rather crude pump or bellows, provided it induces only inspirations under any moderate pressure, always administers almost exactly the amount of pulmonary ventilation that the respiratory metabolism—the exchange of oxygen and carbon dioxide—of the animal requires. With the Drinker respirator also no considerable degree of adjustment is needed; yet the lungs of the patient are neither overventilated nor underventilated. The same protective reaction comes into play as that just described in relation to manual artificial respiration. Under the influence of the blood gases the respiratory center adjusts the tonic elasticity of the diaphragm and thoracic muscles and thus the amount of the recoil of the chest, so that just the required amount of pulmonary ventilation is obtained—no more, no less.

(Note on revision of proof: From evidence presented in the British report on "Breathing Machines," quoted at the end of this paper, it appears probable that, for the maintenance of life under artificial respiration for prolonged periods, it is essential that some parts of the respiratory muscles shall have escaped paralysis and have retained some degree of contractility and tonic elasticity. The report states (pages 55 and 65) that "when poliomyelitis is so severe as to damage the mechanism of respiration" . . . "the results of treatment in a Drinker Respirator are disappointing—in round figures, of every five patients treated, only one is alive eighteen months later.")

SUCK AND BLOW RESPIRATION

Clear and simple as is the principle here involved, nevertheless few seem to have realized it. Just as normal breathing is automatically and accurately regulated to meet the body's respiratory needs, so also artificial respiration, if it is to be effective for resuscitation and not harmful, must be of a form that takes account of this physiologic regulation and respiratory needs. If, for instance, these needs are exceeded, serious injury, or even death, may result. For the study of this harmful

8. Hess, W. R.: Die Regulierung der Atmung, Leipzig, Georg Thieme, 1931.

9. Lougheed, D. W.; Janes, J. M., and Hall, G. E.: Physiological Studies in Experimental Asphyxia and Drowning, Canad. M. A. J. **40**: 423 (May) 1939.

10. Drinker, Philip, and McKhann, C. T.: The Use of a New Apparatus for the Prolonged Administration of Artificial Respiration, J. A. M. A. **92**: 1658 (May 18) 1929. Drinker, Philip; Shaughnessy, D. J., and Murphy, D. P.: The Drinker Respirator, ibid. **95**: 1249 (Oct. 25) 1930.

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form of artificial respiration and its effects, Henderson and Haggard [11] employed such apparatus as that shown in figure 1. Instead of a rhythmic succession of inspirations alternating with pauses to allow the elasticity of the chest to produce expirations, this apparatus induces both inflation of the lungs by positive air pressure and also deflation by negative pressure. And the forced deflation—unless the negative pressure is kept very low indeed—may induce overventilation, injure the lungs and do serious harm. Dogs thus overventilated with positive and negative pressures of 31 cm. water column, in 3 cases for twenty-two, one hundred and ten and one hundred and ninety minutes respectively, in the experiments of Henderson and Haggard,[11] exhibited a subsequent progressive fall of arterial pressure and death within a few hours thereafter. The carbon dioxide of the blood, both the content and the capacity or alkali reserve, suffered a great decrease. After briefer, but even more forcible, periods of suck and blow respiration with a double pump, Henderson [12] found that natural breathing failed to return, and the animals died in apnea.

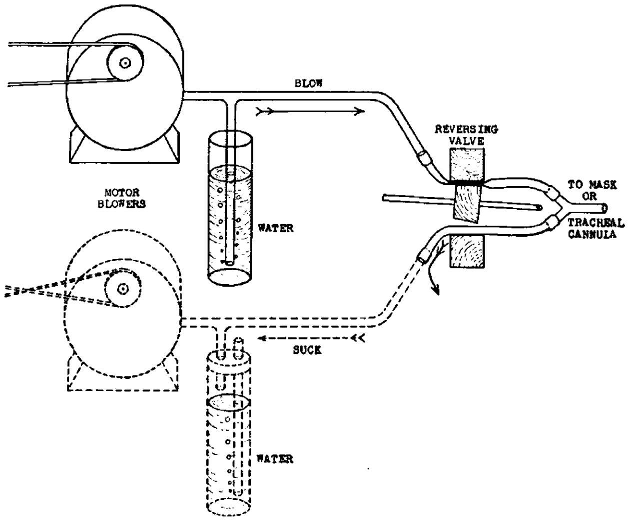

Fig. 1.—Suck and blow artificial respiration apparatus used to apply positive and negative pressures of 15 cm. water column in experiments summarized in columns 1, 2 and 3 of figure 4; used also in previous investigations, with much higher pressures, for the study of the ill effects of forced mechanical ventilation. With this apparatus a man or animal of any size from that of a horse down to that of a mouse can be respirated at any desired rate and with any desired positive and negative pressure. No apparatus on the pulmotor principle—no matter what its construction—can do more. If the parts indicated by the broken lines are omitted, the apparatus becomes a proper single phase respirator such as is commonly used in physiologic laboratories. (A small motor blower convenient for such use is the Eastern laboratory pump, model B, made by the Eastern Engineering Company, New Haven, Conn.)

In some of the experiments on asphyxial dogs that we have here to report we also have employed the suck and blow apparatus of figure 1, but with positive and negative pressures of only 15 cm. water column. With such pressures no serious harm nor any appreciable relief of the asphyxia was induced.

THE PULMOTOR CONDEMNED

As interest developed in regard to resuscitation from the acute asphyxias, the question presented itself whether artificial respiration by means of some mechanical device might not be more efficient than that induced merely by the pressure of the two hands of the operator on the back of an apneic patient. Among the attempts to exploit this possibility the best known is the pulmotor.[4] In the pulmotor oxygen from a cylinder flowed through a reducing valve and then through an injector to a face mask, under a sufficient pressure to inflate the lungs. As soon as the lungs were filled sufficiently to resist further distention, the pressure tripped a valve, reversed the direction of flow and sucked out a part of the contents of the lungs—after which the direction of flow was again automatically reversed. At least it was so intended. By this alternation of mechanically induced inspiration and forced expiration it was supposed that any foreign gas would be rapidly sucked out of the blood and replaced by oxygen. The pulmotor made a rubber bag or doll "breathe" quite realistically.

The pulmotor and similar devices were first investigated by the Committee on Resuscitation of 1912-1913,[4] again by the commission of 1917 [13] and again by that of 1922.[14] Not only the pulmotor but also the principle of suck and blow artificial respiration were condemned on the grounds, first, that expiration forced by negative pressure is unnatural and may be injurious; second, that such apparatus, instead of operating at a rate approximating that of normal breathing, responds to any obstruction in the throat, or to relapse of the tongue when the patient is on his back, by reversing its sucking and blowing phases so rapidly that no appreciable movement of the lungs is induced, and, third and most important, that the first essential for resuscitation from drowning and electric shock is the immediate application of artificial respiration. No apparatus can be applied as quickly as can the manual method.

It was the unanimous opinion of all the commissions that, in all groups in which such apparatus is introduced, training in the manual method tends to be neglected, time is lost while the apparatus is being brought to the patient and—most important of all—even if it is on the spot, the loss of a few seconds, while the mask is being applied and the apparatus is set in operation, may in cases of severe involvement mean the loss of a life which would have been saved by the immediate application of manual artificial respiration. This opinion is now receiving strong support in the resuscitation from electric shock by the "pole top" [15] method of artificial respiration. It avoids the loss of time required to lower the patient to the ground. The investigations of Lougheed, Janes and Hall [9] on resuscitation from drowning also indicate the supreme importance of immediate artificial respiration and the efficiency of the manual method.

THE PROBLEM OF CARBON MONOXIDE ASPHYXIA

The problem which the first two resuscitation commissions faced was mainly that of how best to bring an apneic—i. e. nonbreathing—patient back again to a state of natural respiration. In drowning and electric shock the period of complete anoxia—deprivation of oxygen—is usually brief, only a few minutes at most. If it is complete for more than four or, at most, five minutes, resuscitation is never effected. But, in the large majority of cases, once the patient is breathing again his life is safe. (In regard to drowning: if the victim can swim even a little, he may last much longer than 5 minutes. But all scientific evidence indicates that in a very few minutes complete anoxia causes irreparable damage to vital centers in the brain.)

In cases of carbon monoxide asphyxia, on the contrary, the main problem is essentially different; yet down to 1922 its nature was scarcely realized. A large proportion of patients in this group are still breathing, although feebly, when removed from the gassing cham-

11. Henderson, Yandell, and Haggard, H. W.: Respiratory Regulation of the CO_2 Capacity of the Blood, J. Biol. Chem. **33**: 355 (Feb.) 1918.
12. Henderson, Yandell: Acapnia and Shock: IV. Fatal Apnea After Excessive Respiration, Am. J. Physiol. **25**: 310, 1910.
13. Third Resuscitation Commission, Science **48**: 563, 1918.
14. Final Report of the Committee on Resuscitation from Carbon Monoxide Asphyxia, J. Indust. Hyg. **6**: 125, 1923.
15. Osterreich, E. W.: The Pole Top Method of Resuscitating Linemen After Electric Shock, Bull. Edison Elect. Inst. **7**: 121 (March) 1939.

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ber. For them artificial respiration with air, or even with oxygen, is of little benefit as compared with its importance in drowning and electric shock. It does not by itself alone directly produce a sufficient ventilation of the lungs to induce a rapid elimination of the monoxide from the blood. In many cases the anoxia is not so complete as to cause a quick death; yet in these cases, unless the asphyxiation is quickly terminated by elimination of the monoxide from the blood, the brain is irreparably damaged. The patient then remains unconscious; and he may die many hours after the last trace of the monoxide has gradually diffused out of his blood. The problem of resuscitation from carbon monoxide asphyxia is therefore mainly not one of artificial respiration, although artificial respiration is an important aid in starting the inhalation of a resuscitant mixture of carbon dioxide and oxygen. The prime object is generally rather that of stimulating the already existing natural breathing by means of carbon dioxide and thus inducing so large a volume of respiration that the mass action of the inhaled oxygen may displace the monoxide from the blood as rapidly as possible. The period of cerebral asphyxiation is thus cut short, life is saved and postasphyxial damage is prevented.

For this purpose, in 1922, the inhalation of a mixture of carbon dioxide and oxygen was introduced by Henderson and Haggard.[16] It is now generally used in all civilized countries. In large cities it has decreased the mortality from asphyxia by 50 per cent; and among the patients who receive early treatment the postasphyxial nervous defects that were once common have been almost entirely prevented, as has also pneumonia.

RETURN OF THE PULMOTOR IN THE FORM OF RESUSCITATORS

Americans have great faith in machinery and ingenuity in inventing it. Many suppose that artificial respiration acts mechanically like the cranking of a stalled motor instead of chemically, as it does, like a renewed supply of oxygen to a smothered fire. Accordingly, as soon as it was demonstrated that an increased volume of breathing, under the influence of carbon dioxide mixed with oxygen, is a prime factor in resuscitation from carbon monoxide asphyxia, a number of inventors, acting quite independently of one another, undertook to combine inhalation of the mixture of carbon dioxide and oxygen with mechanical artificial respiration. The various pieces of apparatus developed for this double purpose are generally called "resuscitators." They are intended to induce a more effective artificial respiration than any manual method. A recent advertisement of one of them states that it "eliminates all need for manual artificial respiration."

A number of resuscitators are now offered. Several —particularly the McKesson resuscitator and inhaler and the E & J resuscitator and inhalator—have received some support from clinicians; the latter device has been accepted for trial by the Council on Physical Therapy of the American Medical Association.[17] We have examined all of them. They differ in details of construction but are all ingeniously designed and well constructed. With those matters we are not concerned but solely with the action that they apply to a patient. This consists of suck and blow artificial respiration essentially like that afforded by the pulmotor. They inflate and deflate a rubber bag exactly as does the pulmotor, and in their action on a patient they are therefore essentially reversions to the pulmotor principle. None of them afford the kind of artificial respiration that is commonly used in physiologic laboratories or that supplied by the Drinker respirator as now used. When first introduced, the Drinker respirator afforded both an active inspiration and a forced expiration, but as experience accumulated the forced expiration was discarded.

The only experimental study on a resuscitator that has been reported is that on the E & J apparatus by Coryllos.[18] He was primarily interested in maintaining artificial respiration during surgical operations in the open thorax; this he found that the apparatus would do. But resuscitation in carbon monoxide poisoning and asphyxia of the newborn were not investigated. Some experiments were made on drowning. Death was found to occur after four or five minutes, as in the experiments of Lougheed, Janes and Hall.[9] If the E & J resuscitator, supplying oxygen and carbon dioxide, was immediately applied at the fourth minute, resuscitation resulted. A delay of another minute was fatal: a point which Coryllos admits; yet he fails to recognize that the use of artificial respiration apparatus on human victims necessarily involves delays often much longer than one minute.

As regards the use of the E & J resuscitator for prolonged artificial respiration, Coryllos reports that 7 healthy dogs, anesthetized with sodium amytal, but subjected to no other operation nor to any form of asphyxia, were submitted to the action of the apparatus for periods of one to eight hours continuously. Various degrees of shock, acapnia and depression of body temperature were induced, except in 3 which were kept warm with electrical heating pads: they recovered. The other 4 died between twelve hours and five days, and autopsy showed areas of consolidation in the lungs with moderate edema. Although the experiments were performed at the ordinary laboratory temperature, Coryllos considered the chilling, rather than the damage to the lungs and shock, as the fatal factor. We, on the contrary, should regard the fall of body temperature as rather a feature of the shock induced by the damage to the lungs and overventilation of the blood. But no matter whether the fall of temperature was a cause or an effect or both, this much is clear: These experiments by Coryllos again demonstrate, as did the earlier experiments of Henderson and Haggard[11] and of Henderson,[12] that prolonged forcible suck and blow artificial respiration may cause injury to the lungs, shock and death.

Coryllos also reports use of the E & J resuscitator on patients who collapsed during surgical operations in the thorax. As the lungs are essentially elastic bags which deflate when the chest is open, there is obvious need in thoracic surgery for some means of keeping them inflated, or reinflating them. For this purpose moderate positive air pressure is needed; but negative pressure is certainly contraindicated. In regard to clinical use of the E & J resuscitator we have received reports of patients with poliomyelitis who were removed for a time from a Drinker respirator, were treated with the E & J apparatus and did not long survive.

16. Henderson, Yandell, and Haggard, H. W.: The Elimination of Carbon Monoxide from the Blood After a Dangerous Degree of Asphyxiation and a Therapy for Accelerating the Elimination, J. Pharmacol. & Exper. Therap. **16:** 11 (Aug.) 1920; The Treatment of Carbon Monoxide Asphyxia by Means of Oxygen and CO_2 Inhalation, J. A. M. A. **79:** 1137 (Sept. 30) 1922.

17. E & J Resuscitator and Inhalator Acceptable, report by Council on Physical Therapy, J. A. M. A. **112:** 1945 (May 13) 1939.

18. Coryllos, P. N.: Mechanical Resuscitation in Advanced Forms of Asphyxia: A Clinical and Experimental Study of the Different Methods of Resuscitation, Surg., Gynec. & Obst. **66:** 698 (April) 1938.

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As regards the use of resuscitators in asphyxia of the newborn, Martinez[19] has reported favorably. He fails, however, to offer any evidence that suck and blow artificial respiration is more effective than the administration of oxygen and carbon dioxide by the gentle positive pressure induced by merely squeezing the breathing bag of a simple infant inhalator without any negative pressure. From the personal communications of competent witnesses we are inclined to believe that in cases of asphyxia pallida the best method of resuscitation is afforded by the administration of oxygen, either alone or with carbon dioxide, by means of intratracheal insufflation.[20] (The technic, once acquired, is very simple.) It certainly does not seem like sound therapy to apply negative pressure to infant lungs that are still atelectatic or that, if already partially inflated, may be again collapsed. Equally unjustifiable is it to apply the volume and positive pressure of the "blow" phase of a resuscitator to lungs in which the adherent walls of the little air sacs are easily torn under even a low dilating force. That they are sometimes torn is reported to us by several competent witnesses who have seen such injuries at autopsy. As a number of hospitals now have resuscitators, it is important that additional evidence on this matter should be obtained by more frequent autopsies on nonresuscitated babies.

THE PROS AND CONS OF RESUSCITATORS

No feature of the E & J, McKesson and other resuscitators has done more to gain consideration for such apparatus than the demonstration that with them, as with the pulmotor, a rubber bag may be rhythmically inflated and deflated and a rubber doll made to "breathe" realistically. Yet this demonstration is essentially specious. The bag and doll offer no resistance to inflation until full nor to deflation until empty. A conscious man can indeed generally adjust his breathing to the rhythm of the apparatus, or induce the apparatus to time its phases of suck and blow with his expirations and inspirations; but in unconscious men and animals such cooperation rarely occurs. The tonus of the chest produces sufficient resistance to inflation and deflation to throw the switch, and the apparatus goes into a rapid succession of reversals. In the first case of carbon monoxide asphyxia that one of us saw under treatment with the pulmotor, as in other cases reported later, the comatose patient was breathing at one rhythm and the apparatus was operating at another rhythm—about twice as rapid. It was impeding instead of aiding respiration. With resuscitators this has frequently been seen on asphyxial dogs.

Resuscitators, as now used, differ from the pulmotor in that the latter apparatus administered oxygen diluted with air, while resuscitators generally administer a mixture of oxygen and carbon dioxide. But the use of this mixture does not improve the cooperation of patient and machine. On the contrary, the stimulant action of the carbon dioxide tends to render the respiration of a man or animal even more recalcitrant to mechanical control than if oxygen alone, or mere air, was administered. That such is the case is indicated by the following experiments in which varied gas mixtures were used:

MECHANICAL RESPIRATION IN COMA

The experimental observations here referred to were made on healthy dogs weighing from 7 to 10 Kg. Each animal was first kept for from twenty to twenty-five minutes in a gassing chamber containing air to which 0.35 per cent of carbon monoxide had been added. At the end of that time every one of the animals had passed through the usual stages of the development of asphyxia and was in coma—completely unconscious and either convulsive or limp. In six of these experiments the asphyxiation was continued until breathing had completely stopped. Three of these animals were then treated with the E & J resuscitator, but unsuccessfully: owing, presumably, to the fact that a few seconds were necessarily lost in removing the animal from the gassing chamber and in adjusting and starting the apparatus. The other 3, on removal from the gassing chamber, were instantly treated with manual artificial respiration and inhalation of carbon dioxide and oxygen. All 3 were revived; and these 3 and a number of others that had not been quite so deeply asphyxiated were then used for the following experiments:

As soon as the risk of immediate death was past and the animal was breathing, although feebly, mechanical artificial respiration was started. Under the influence of the mixture of carbon dioxide and oxygen thus supplied to the lungs the animal's own respiratory efforts quickly became vigorous; but in the large majority of cases—indeed, with only one exception—the rate of natural breathing and the rate at which the apparatus operated were entirely different. There was conflict, instead of cooperation, with the result merely of the waste of a large amount of the resuscitant gases. In a succession of experiments with the E & J resuscitator we recorded the simultaneous rates of natural breathing (N. B.) and those at which the apparatus operated (E. J.) as follows: N. B. 60, E. J. 120; N. B. 30, E. J. 60; N. B. 22, E. J. 120; N. B. 30, E. J. 30; N. B. 8, E. J. 50; N. B. 16, E. J. 72; N. B. 16, E. J. 74.

All forms of suck and blow respiration apparatus—pulmotors, resuscitators, and so on—are, it is claimed, adjustable to the patient's rate of breathing. On normal conscious men such is the case, although more of the adjusting is effected by the subject than by the apparatus. On comatose subjects, on the contrary, both dogs and men, synchronization is much more difficult. The directions which come with the apparatus are to the effect that, after the patient is breathing, the inhalator attachment alone should be used. But in view of the inadequate supply of the resuscitant gas mixture afforded by the inhalator attachment, the patient is more likely to recover if the larger supply afforded by the suck and blow device is continued, even though it conflicts with the natural breathing. In the E & J resuscitator the maximum flow of the resuscitant gas mixture on the inhalator side is only 7 liters a minute, which is about the volume of normal resting respiration; whereas a man under inhalation of carbon dioxide and oxygen will breathe 20 or even 30 liters a minute and needs all of it for the rapid elimination of carbon monoxide from his blood.

This discordance between natural breathing and mechanical artificial respiration is shown graphically in figure 2. The graphs here reproduced were obtained by means of a body plethysmograph (a large glass specimen jar with holes bored for the resuscitator tubes) in which the animal was placed, and a Connell compensator bag [21] (in place of a tambour) connected with

19. Martinez, D. B.: The Mechanical Resuscitation of the Newborn: A Report of 500 Cases, J. A. M. A. **109:** 487 (Aug. 4) 1937.

20. Flagg, P. J.: The Treatment of Postnatal Asphyxia, Am. J. Obst. & Gynec. **21:** 537, 1931.

21. Dr. Karl Connell supplied this instrument, which can record accurately much larger volumes than any tambour can take.

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the plethysmograph and writing on a smoked drum. In graph *A* the large waves are the animal's natural breathing; the small waves are the artificial respiration produced by the resuscitator. In graph *B* is shown one of the rare instances in which the resuscitator, applied between the arrows, operated in step with the natural breathing. But comparison of the middle part of the graph with the first and last parts, in which the

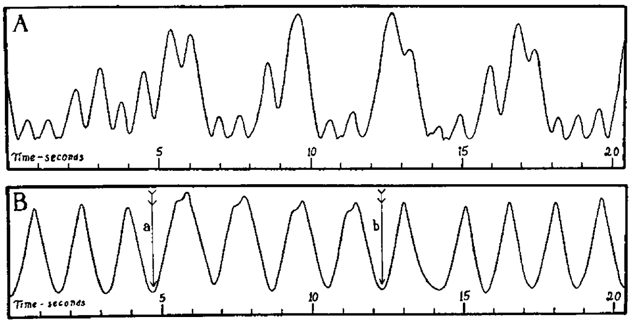

Fig. 2.—Graphic records of the respiration of 2 dogs by means of a body plethysmograph during treatment with the E & J resuscitator administering carbon dioxide and oxygen. In *A* the large waves are natural breaths; the small waves show the conflicting action of the apparatus. In *B* the four respirations between the arrows show the best cooperation between natural breathing and the E & J resuscitator that we were ever able to obtain in any experiment. Yet the unaided natural breaths before arrow *a* and after arrow *b* afford as much ventilation—slightly smaller breaths at a slightly faster rate—as with the assistance of the resuscitator. The inhalator part of the E & J resuscitator affords a flow of gas just large enough for a moderate sized dog under stimulation by carbon dioxide and oxygen but much too small for a man inhaling that mixture.

animal received mere inhalation, indicates that, although the artificial increment rendered breaths slightly larger, it also rendered breathing slightly slower. The amount of pulmonary ventilation per minute was not increased; in fact, as the next section will show, under mechanical respiration it is generally diminished.

THE ELIMINATION OF CARBON MONOXIDE

The prime object of any treatment for carbon monoxide asphyxia, as shown by the investigations of Henderson and Haggard[16] already referred to, is the elimination of the asphyxiant gas from the blood as rapidly as possible. The injury to the brain is determined not merely by the length of time the patient has been in the gassing chamber but even more by the time before elimination is effected. That method is therefore best which will effect the elimination most rapidly. How effective is the method now in general use is shown in figure 3, in which are compared the rates of elimination of carbon monoxide from the blood in deeply asphyxial dogs under four different treatments.[16] The animals had been gassed in a chamber into which enough city gas had been introduced to make a concentration of 0.35 per cent of carbon monoxide. Thereafter one group breathed merely fresh air (i. e. no treatment); a second group received an inhalation of pure oxygen; a third received an inhalation of carbon dioxide diluted with air, and a fourth group received an inhalation of carbon dioxide and oxygen.

The essential feature of the inhalational method is the administration of the stimulant mixture of carbon dioxide and oxygen by means of an inhalator which can supply the mixture up to the largest volume of breathing that the patient may reach: 20 or even 30 liters a minute. The volume should be sufficient and the valves on the mask should be so arranged that there is no rebreathing; for if any of the mixture of carbon dioxide and oxygen that has been inhaled and again exhaled is then again inhaled—as in the inhalator attachment of some resuscitators—the carbon monoxide that it has taken up from the blood prevents the blood from giving off more of that gas, and the mass action of oxygen on the blood is impeded. Such valves should be used on resuscitators also; they are not under patent or any other restriction but are free for any and all to use.

Using the experiments summarized in figure 3 as a standard of comparison, we now report an extensive series of resuscitations on dogs gassed as described in the preceding section. The object of these experiments was to compare the rates at which carbon monoxide may be eliminated from the blood and resuscitation effected by three different gas mixtures: (*a*) air, (*b*) air plus carbon dioxide and (*c*) carbon dioxide and oxygen. Each was administered by three different methods: (1) by simple inhalation initiated by a few seconds of manual artificial respiration to make sure the animal was breathing; (2) by mechanical respiration with the E & J resuscitator (we have tested three of these apparatus: two of the portable models and one of the hospital type), and (3) by means of the suck and blow artificial respiration apparatus shown in figure 1. This apparatus has the advantage that it can easily be made to operate in step with natural breathing, which the pulmotor and resuscitators rarely do. To attain this coordination, the operator watches the animal and applies positive pressure as the animal inspires and negative pressure simultaneously with expiration. If any breathing machine could augment the volume of natural breathing it would be some such device as this. The results—or lack of beneficial results—obtained with it show that no such augmentation is possible.

In all the experiments the animal's head was enclosed in a mask made airtight by a collar of sheet rubber fitting closely around the neck and tied over the edge of the mask. At the beginning and end of each resuscitative treatment, and at intervals between, blood was drawn from the jugular vein and analyzed for carbon monoxide by the Sayers-Yant method.[22] The carbon monoxide was made fresh from formic acid for each experiment.

The results of the administration of air, of air plus carbon dioxide and of the mixture of 5 per cent carbon dioxide and 95 per cent oxygen by simple inhalation

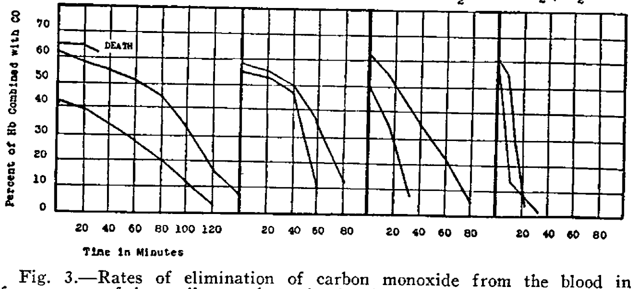

Fig. 3.—Rates of elimination of carbon monoxide from the blood in four groups of dogs, all severely asphyxiated with city gas: (1) untreated, (2) under inhalation of oxygen, (3) under inhalation of carbon dioxide diluted with air and (4) under inhalation of carbon dioxide and oxygen.

and by means of the suck and blow apparatus sketched in figure 1 are shown in the first three sections of figure 4; the results obtained with the E & J resuscitator administering the mixture of carbon dioxide

22. Sayers, R. R., and Yant, W. P.: The Pyrotanic Acid Method for the Quantitative Determination of Carbon Monoxide in Blood and Air, Technical Paper 373, United States Bureau of Mines, Washington, D. C., 1925.

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and oxygen are shown in the fourth section. Each line indicates a complete experiment. The solid lines show the rates at which carbon monoxide was eliminated from the blood by natural breathing under simple inhalation. The broken lines show the rates of elimination under the two types of mechanical artificial respiration.

Examination of figure 4 shows that mechanical artificial respiration, even when administered in close harmony of rhythm with natural breathing—first three sections of the figure—does not accelerate the elimination of carbon monoxide from the blood. And in the fourth section it is to be seen that when the artificial respiration is out of time with natural breathing it rather tends to impede the elimination. All the benefit afforded by treatment of an asphyxial animal—and so presumably of an asphyxial man also—by means of mechanical artificial respiration administering carbon dioxide and oxygen is due to the resuscitant gases and not at all to the mechanical method of administration. For the same gas mixture is equally effective or is

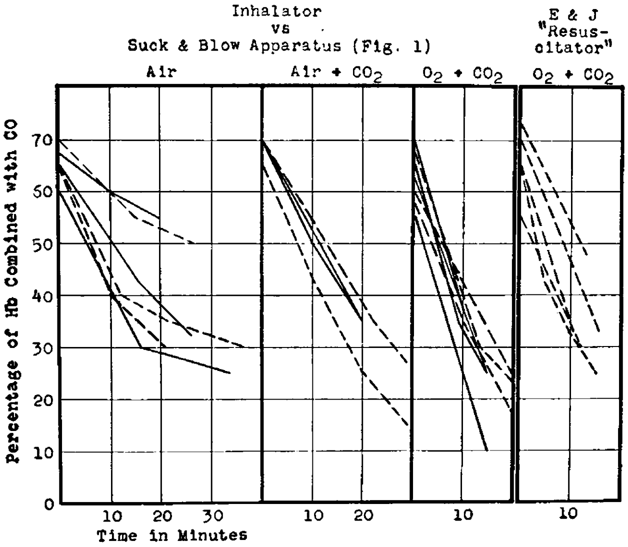

Fig. 4.—Rates of elimination of carbon monoxide from the blood in dogs severely asphyxiated by from twenty to twenty-five minutes in an atmosphere of 0.35 per cent of pure carbon monoxide in air. The solid lines indicate the experiments in which inhalational treatment was given. The broken lines indicate the comparable experiments in which suck and blow artificial respiration was administered either by means of the apparatus shown in figure 1 (first three groups) or by means of the E & J resuscitator (right hand group).

even more effective when it is administered by inhalation. In brief, a resuscitator is merely an inhalator which tends to impede natural breathing by sucking and blowing.

It is now well recognized that inhalation of 7 or 8 per cent carbon dioxide in oxygen induces more rapid resuscitation than does 5 per cent. But, as resuscitators are generally supplied with the 5 per cent mixture, it seemed best to use it throughout our experiments. With the stronger mixture the resuscitator would make an even worse showing; for the more respiration is stimulated to activity by carbon dioxide, the more resistant it becomes to outside mechanical control. In order to administer carbon dioxide and oxygen efficiently the pulmotor, E & J resuscitator and similar devices should be modified to eliminate the suction phase, decrease the pressure of the blowing phase and greatly increase its volume. They would then become efficient resuscitation apparatus, which at present they are not.

SUMMARY

Resuscitation is often thought of as if it were the restarting of a machine that has stopped. Actually, if the vital machine has fully stopped it cannot be restarted; it is not like an automobile motor to be started by "cranking." What resuscitation does—for example in the case of drowning—is to prevent the machine from coming to a full stop. For this purpose the essential is a renewed supply of oxygen while the body still retains some of its tonus and the heart is still beating. Hence the importance of immediate artificial respiration.

The volume of pulmonary ventilation that can be induced under any form of manual artificial respiration is here shown to be controlled by a physiologic principle: the same principle as that which regulates the volume of normal breathing under the influence of the blood gases acting through the respiratory center on the tonus of the respiratory muscles. The prone pressure method of Schafer produces all the pulmonary ventilation that this principle permits. In spite of claims for other manual methods, old and new, none of them can do more. The prone pressure method is the simplest to learn and the easiest to apply; it is therefore the best. It can be started more quickly than any mechanical device—a point of vital importance in resuscitation from drowning and electric shock.

For resuscitation from carbon monoxide asphyxia the point of vital importance is the rapid elimination of the asphyxiant gas from the blood by means of carbon dioxide and oxygen. The experiments here reported were therefore performed on deeply asphyxiated dogs to determine the most effective method of administering the resuscitant gases: whether by mechanical respiration or by simple inhalation. The results show that the volume of pulmonary ventilation which can be safely induced by mechanical artificial respiration is limited by the same physiologic principle as that for manual artificial respiration and is not more, but rather less, than under simple inhalation. The deaths resulting from suck and blow respiration applied to dogs in the experiments of Henderson and Haggard,[11] of Henderson[12] and of Coryllos[13] deserve serious consideration. Mechanical respiration has therefore no advantage over inhalation but rather defects that must tend to decrease the saving of lives.

CONCLUSIONS

When natural breathing has stopped, it is restored more effectively by manual artificial respiration than by mechanical respiration.

Mechanical respiration, unless so forcible as to be harmful, does not increase the volume of natural breathing.

Inhalation of carbon dioxide and oxygen increases the efficiency of manual artificial respiration but increases the antagonism between mechanical respiration and natural breathing.

In brief, the best method of resuscitation from drowning and electric shock is prone pressure artificial respiration supplemented by inhalation of carbon dioxide and oxygen. The best method of resuscitation from carbon monoxide asphyxia is inhalation of carbon dioxide and oxygen, initiated in cases of severe involvement by prone pressure artificial respiration.

ADDENDUM

After our investigations were completed there came to us a report by the British Medical Research Council, Respirators (Poliomyelitis) Committee, on "'Breath-

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ing Machines' and Their Use in Treatment."[28] As its conclusions are essentially the same as those to which we have been led, a few quotations are appropriate:

"There is a natural tendency among doctors as well as laymen to credit a machine with greater powers than the manual method of artificial respiration and to delay or suspend manual methods pending the arrival of apparatus. . . . No development or multiplication of apparatus for such treatment can compare in usefulness with the training of all members of the community in methods of manual artificial respiration" (page 43).

"Excessive pulmonary ventilation removes carbon dioxide from the blood and thus depresses respiration and circulation. . . . Large negative pressures, if used over a period of time, tend to produce emphysema" (page 48).

"Breathing machines are required only for protracted failure of respiration" (page 64).

"The commonest causes of asphyxia are drowning and gas poisoning, in which treatment in breathing machines is not required. The prognosis in these cases is directly dependent on the duration of the asphyxia and the speed with which treatment is begun. Manual respiration, combined if necessary with inhalation of oxygen or oxygen plus carbon dioxide, does all that is wanted in such cases" (page 65).

440 Prospect Street.

Clinical Notes, Suggestions and New Instruments

ACCIDENTAL DEATH FROM ABSORPTION OF HEATLESS PERMANENT WAVE SOLUTION

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On March 19 a healthy young married woman, mother of three children, drove her car, accompanied by a woman friend, from her home to Atlanta—a distance of 40 miles—where she had an appointment for 10:30 a. m. at a beauty shop for a heatless permanent wave. She arrived a little late, had her hair cut by the proprietor, an oil treatment and her hair prepared for the heatless permanent wave.

Her friend went by the beauty shop about 2:30 p. m. She told her friend to go to a movie or do anything else she had to do, since the treatment would require more time than she had anticipated. A few minutes later, after the preliminary preparations had been completed, the treatment was started by a trained operator. He had planned to continue it for seven minutes. The procedure consisted in spraying the wave solution through a closed circuit of tubes into a rubber cap which had been sealed tightly over the head and covered all the hair, which had been previously wrapped in rows on curling tubes.

The operator had been spraying the solution for about four times a minute for about three minutes when the patron said "Does this ever make one feel faint?" These were her last words. The operator stopped the treatment, applied the neutralizing solution and called for help. A physician was summoned, an ambulance called and artificial respiration begun, since the patron had turned a dark purplish. The physician and the ambulance arrived quickly. She appeared dead but was rushed to Crawford W. Long Memorial Hospital, where she was pronounced dead on arrival about 3 o'clock.

One of us (A. H. B.) had examined and treated members of the family for several years. The patient, aged 39, had had a complete physical examination, roentgen examination of the sinuses, chest and abdomen and analyses of the blood and the urine some time previously. None of these showed any gross abnormality. She had a physical check-up very recently, at which time she appeared to be in excellent health.

Shortly after the arrival of the body at the hospital the patient's husband arrived from their home and readily consented to a postmortem examination to determine, if possible, the cause of death. This examination was begun in the hospital at approximately 4:15 on March 19 with the following data noted:

The body was well developed and well nourished. Rigor mortis had not set in and the body was still warm. Examination of the surface showed numerous small hemorrhagic abrasions covering almost the entire scalp. These had the appearance of areas in which the surface epithelium, down to the dermis, had been destroyed. They were arranged in parallel rows, apparently one row associated with each curl of hair. The largest of these were about 1 cm. in diameter and the smallest were approximately 2 mm. Their outlines were irregular, and in some areas they had a punctate appearance. There was an area on the left side of the chin somewhat similar in appearance. An old bruise about 7 cm. in diameter was present on the right knee. The skin generally, and particularly about the face, had a dusky hue suggestive of cyanosis. There were no other surface markings of note. The body was opened by a cross incision.

The lungs were free in the thoracic cavities. They were light colored and showed some areas of moderate atelectasis but little congestion. There was no evidence of pneumonia or specific infection. No infarcts were present.

The pericardial surfaces showed no abnormality. Approximately 100 cc. of blood was removed from the right ventricle before the circulatory system had been entered elsewhere. This was put aside for subsequent chemical examination. The pulmonary artery was opened in situ. No embolus was present.

The heart was somewhat small. There were no grossly demonstrable abnormalities of either the musculature or the valvular structures. The coronary system was intact and showed no evidence of obstruction at any point. There was a moderate degree of atherosclerosis of the aorta, but this was not at all extensive.

The liver was normal in size but was a peculiar purplish red before removal. This was apparently due to extreme congestion, since the color became appreciably lighter when the organ had been removed and the blood drained from it. A cut section of the liver showed a brownish discoloration which was not normal. The consistency of the tissue appeared to be poorer than is normally seen and suggested diffuse degeneration. The liver was saved whole for possible chemical examination.

The gallbladder showed an occasional small deposit of cholesterol on its inner surface. Otherwise the biliary tract was normal.

The spleen was enlarged to about three times its normal size. There appeared to be considerable hyperplasia of the reticuloendothelial elements. There were irregular areas which were light pink and were definitely softer than the surrounding tissue. These appeared to be areas of degeneration.

The kidneys were in their normal position and were normal in size. The capsules stripped with ease, leaving a smooth cortical surface. A cut section showed an extreme degree of congestion, both organs being deep purplish red. No other gross abnormalities were noted in the urinary tract.

The adrenal glands appeared grossly normal. The pancreas was normal. The gastrointestinal tract showed no particular abnormality in any part. The whole tract with its contents was saved for possible chemical examination.

The uterus had been suspended in the past and was in an abnormal anterior position. There was a single leiomyoma measuring approximately 5 cm. in diameter on the superior, posterior portion of the fundus. The tubes showed no abnormalities. The ovaries showed a few small cysts with some fibrosis.

There was no enlargement of lymph nodes demonstrable at any point.

The cranium was opened in the usual fashion. When the scalp was reflected there were a number of perfectly round, slightly reddish, discolored areas on the under surface. These did not correspond in position to the abrasions previously described on the outer surface of the scalp. The calvarium came away easily, leaving the dura intact. Removal of the dura

From the Crawford W. Long Memorial Hospital, Emory University School of Medicine.

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