tion of Potassium Iodide, N. F. (saturated solution), may conveniently be used, starting with 1 cc. in a glassful of milk three times daily after meals and gradually increasing the dosage until the limit of tolerance is reached as indicated by coryza, acne, digestive disturbance, the phenomena of hyperthyroidism or the development of cachexia. The treatment should continue for at least one month after apparent cure, with immediate readministration at indication of relapse.

Roentgen irradiation may be used in a dosage of 140 kilovolts, with 0.25 mm. of copper and 1 mm. of aluminum filtration for relatively superficial lesions to 0.5 mm.

PRESCRIPTION 8.—*Iodine Solution*

℞	Iodine	1.00 Gm.
	Potassium iodide	10.00 Gm.
	Distilled water	500.00 cc.

M. and Label: Use on gauze as moist dressing.

of copper and 1 mm. of aluminum for deep involvement. A sufficient number of portals should be used to secure 300 roentgens at the site of the lesion. Such treatment may be repeated every two weeks for at least six months.

Surgical intervention should be confined to the evacuation of pus, excepting only in cases in which complete extirpation of the lesion is possible. Curettage should be avoided, as it may lead to dissemination of infection. Indeed, evacuation of the pus may be accomplished by mere puncture and aspiration, and the cavity filled with a 1 per cent solution of sodium iodide. Ulcerated lesions may be painted with Tincture of Iodine and dressed with an iodide solution, 1 : 500 (prescription 8).

Council on Physical Therapy

THE COUNCIL ON PHYSICAL THERAPY OF THE AMERICAN MEDICAL ASSOCIATION HAS AUTHORIZED PUBLICATION OF THE FOLLOWING ARTICLE.
H. A. CARTER, Secretary.

RESUSCITATION

YANDELL HENDERSON, PH.D.
NEW HAVEN, CONN.

(Concluded from page 754)

ASPHYXIA NEONATORUM [20]

There is no good reason to look, as many writers have done, for any reactions in the neurorespiratory system of a baby, either before or after birth, essentially different from those of an older child or adult. The normal baby starts to breathe under essentially the same stimulus that causes an adult to breathe again after holding his breath. If in the adult the breath holding has been aided by a preliminary period of voluntarily forced ventilation, the analogy is even closer. The reason that the fetus does not breathe in utero is that its blood is too well arterialized to stimulate the fetal neurorespiratory system, which is rather inexcitable so long as the lungs are atelectatic. If respiratory efforts do occur, the thoracic muscles, which are barely strong enough to dilate the lungs with thin air after birth, fail to draw in more than a minute amount of the much less easily inspired amniotic liquor. If some fluid is drawn in, it is quickly absorbed into the blood; for the lungs are by far the most absorptive organs in the body. The particles of meconium sometimes found in the trachea are probably drawn in from the mouth immediately after birth.

When cold water, or even cold air, is suddenly poured over an adult, the cutaneous stimulus causes him to "catch his breath" in a deep and prolonged or even repeated inspiration. Introduction to a cold world induces the same reaction in a normal baby. Its lungs are thus at least partially expanded. So long as they are atelectatic, the lungs send no impulses over the vagi to the respiratory center. But as soon as the lungs are even partially expanded the vagi carry impulses to the respiratory center that call forth the Hering-Breuer reflexes. These are the reflexes that determine the alternation of inspiration and expiration in normal breathing.

If during birth the head of the child is compressed and deformed, and particularly if an intracranial hemorrhage continues the compression of the respiratory center after birth, the blood supply to the center is diminished. If the umbilical cord is partially compressed, the entire body is correspondingly asphyxiated. A formation of lactic acid occurs, the p_H and alkali of the blood are diminished, and the carbon dioxide content, which at first is increased, is later diminished by escape presumably through the placenta. If these conditions are brief, the baby is livid; if they are prolonged, it is pallid. To whatever extent the sensitivity of the respiratory center has been diminished by lack of oxygen, a stronger stimulus in the form of increased pressure of carbon dioxide is needed to excite the neurorespiratory system to activity. Once its activity is induced, the renewed supply of oxygen gradually restores its normal sensitivity. Thereafter, respiration continues under a merely normal amount of carbon dioxide in the blood.

Atelectasis, however, is not so quickly overcome. Even in wholly normal babies the lungs are not fully expanded for hours, days, or even longer. A continuance of atelectatic areas provides conditions favorable for the development of pulmonary infections. The obvious correction is a routine roentgen examination of every baby a few days after birth, or else some dilating treatment without examination. For this purpose most textbooks still recommend that the baby should be made to cry. A much more effective means of accomplishing this end is afforded by repeated brief inhalations of from 5 to 7 per cent carbon dioxide. Herein lies an immediate possibility of a large decrease of the present high mortality of the first month of life.

ANESTHETIC DEPRESSION [20]

Few branches of medicine have made such great advances within the past three decades as has the art of anesthesia. Among these advances, the use of carbon dioxide is preeminent. Every anesthetist now knows that the hyperpnea of the excitement stage of anesthesia decreases the carbon dioxide of the blood and tends to induce failure of breathing under full anesthesia. He knows also that, on the contrary, a moderate amount of rebreathing deepens and steadies respiration. This knowledge and the inhalation of carbon dioxide, when needed, have almost entirely freed surgical anesthesia from what was formerly its continual imminent danger: failure of respiration.

It is not many years since it was a matter of course that after nearly every major operation the patient lay long unconscious, hypopneic and therefore cyanotic, then nauseated and tasting the incompletely exhaled anesthetic for hours. In part these conditions were due to acapnia induced by overbreathing and washing

out of carbon dioxide under the influence of anesthetic excitement and moderate oxygen deficiency. In part they were due also to the acarbia—diminished blood alkali—that asphyxia and acapnia induce. Simultaneously the volume of the circulation was subnormal, owing largely to the stagnation of the blood in atonic tissues.[24]

All these features of depression are now largely avoided by the increasing skill of anesthetists in preventing both anoxia and acapnia. But in extreme cases in which such ill effects do still sometimes occur they are rapidly and largely counteracted by means of inhalation of carbon dioxide. Full deep breathing returns; cyanosis disappears; the tonus of the muscles is recovered; the skin becomes pink as its vessels fill with blood from the previously atonic muscles; the jugular and other superficial veins are again distended, bringing a full supply of blood to the right heart; and with this restoration of the venous return, arterial pressure and a full pulse are reestablished.

Within the last few years yet another benefit accruing from the use of carbon dioxide and prevention of acapnia has come to light. Under acapnia and under any diminution of tonus the so-called vital capacity of the lungs is decreased. After nearly all surgical operations, especially those in the abdomen, the atonic diaphragm is relaxed headward by several centimeters. Under this condition the lungs are partially collapsed and some of the airways may be blocked. The air from the occluded parts of the lungs is soon absorbed and an area of atelectasis or even a massive collapse of one lung results. Prevention of the normal drainage of the lung through the airways permits the development of infection, resulting in what was formerly called "postoperative pneumonia." Now the etiology of postoperative pulmonary complications is understood; and the means of its prevention are available in the restoration of the tonus of the respiratory muscles and dilation of the lungs by full deep breathing.[25]

How far similar measures may aid in pneumonia of nonsurgical origin, particularly in bronchial pneumonia in children, is still undetermined. It appears, however, that the prevention of cyanosis by inhalation of oxygen is beneficial in lobar pneumonia in adults and that the patients do at least as well, perhaps better, if the carbon dioxide exhaled is allowed to accumulate up to at least 1 or 2 per cent in the oxygen tent.[26]

CARBON MONOXIDE ASPHYXIA[27]

The treatment of carbon monoxide asphyxia is now so effectively performed by the rescue crews of city fire and police departments and of gas and electric companies with their inhalators that the main duty of the physician is not to interfere with the artificial respiration. He should not even make a physical examination that requires cessation of artificial respiration, during which the patient may die. He should restrain his impulse to administer hypodermic, intravenous or intracardiac medication of any kind. The claims for methylene blue, lobeline and other drugs of similar properties have been completely disproved both in theory and in practice. Respiratory stimulants are generally cardiac depressants. When administered to a patient in profound respiratory depression they often afford an immediate and striking pharmacologic demonstration, but the patient is much the worse for it the next day.

Carbon monoxide forms only a loose and reversible combination with the hemoglobin of the blood. But until it is displaced, and the oxygen carrying power of the blood is restored, the asphyxial effect continues and is cumulative. The differences between asphyxiation by water—that is, drowning—and prolonged asphyxiation by carbon monoxide are important. Once the man who has been in the water is brought back to fairly normal breathing by means of artificial respiration, especially when supplemented by inhalation, complete recovery is almost certain to follow. In carbon monoxide asphyxia it is only for cases of brief and acute exposure that artificial respiration is an important factor in resuscitation. In cases of prolonged exposure inducing acarbic asphyxia it is only by a rare chance that the victim is found in the brief period—less than ten, or even five minutes—between cessation of respiration and fibrillation and standstill of the heart. After that he is irretrievably dead; before that he is still breathing spontaneously when removed from the poisonous atmosphere. Such cases are numerous, and it is for them that the advance in the technic of resuscitation has made its greatest contribution in the saving of life. They do not need and are little helped by artificial respiration, but they do need inhalational treatment; and in the acarbic or pseudo-acidotic cases it is needed even more than in the cases of apnea after short asphyxiation. To be most beneficial the inhalation must be immediate: on the spot, not after removal to the hospital.

In every large hospital a few years ago, before the city rescue crews were supplied with inhalators, cases of carbon monoxide poisoning were frequently seen in which, even hours after the patients were brought in by the ambulance, they were completely comatose. It was supposed at that time that their blood must still be largely combined with carbon monoxide, and for this reason bleeding and transfusion were sometimes done. Spectroscopic examination showed, however, that even without any treatment whatever most of the carbon monoxide was eliminated from the blood within a few hours. This fact demonstrated the uselessness of late transfusion and it demonstrated much more. Such of these patients as later recovered consciousness suffered acute and prolonged headache and nausea. Some suffered mental or physical impairment. In those who died, autopsy revealed asphyxial injuries in the brain. Thus it became evident that the continuance of the coma is not due to continuance of asphyxia but that it is due to the injury to the nervous system that was developed during the asphyxia. It became evident also that measures for the relief of asphyxia, when applied late, can at best be of relatively slight benefit. The primary object to be aimed at in the treatment of all cases of prolonged asphyxia is to apply inhalational

24. Henderson, Yandell; Oughterson, A. W.; Greenberg, L. A., and Searle, C. P.: The Third Major Mechanical Factor in the Circulation of the Blood, Science **79**: 508-510 (June 1) 1934.

25. Coryllos, P. N., and Birnbaum, G. L.: Bronchial Obstruction: Its Relation to Atelectasis, Bronchopneumonia and Lobar Pneumonia, Am. J. Roentgenol. **22**: 401-430 (Nov.) 1929. Brunn, Harold, and Brill, Selling: Observations on Postoperative Atelectasis: Consideration of Some Factors in Its Etiology, Prevention and Treatment, Ann. Surg. **92**: 801 (Nov.) 1930. Henderson, Yandell: Acapnia as a Factor in Postoperative Shock, Atelectasis and Pneumonia, J. A. M. A. **95**: 572 (Aug. 23) 1930.

26. Barach, A. L.: Personal communication to the author.

27. Henderson, Yandell: The Dangers of Carbon Monoxide Poisoning and Measures to Lessen These Dangers, J. A. M. A. **94**: 179-185 (Jan. 18) 1930.

treatment for the rapid elimination of carbon monoxide within the shortest possible time after removal of the patient from the poisonous atmosphere.

ASPHYXIA FROM MORPHINE, ALCOHOL, AND OTHER RESPIRATORY DEPRESSANTS

One of the principal effects of morphine is to decrease and, in sufficiently large dosage, finally almost to abolish the sensitivity of the neurorespiratory system to its normal stimulus, carbon dioxide. It is the resulting depression and final cessation of respiration that cause death from anoxia in cases of morphine poisoning. It is probable that many deaths among the newborn are due to the administration of morphine to the mother and the diffusion of the drug through the placenta to the child. The treatment of the resulting neonatal asphyxia has already been discussed.

For adults so long as pulmonary ventilation is maintained, either naturally or artificially, life continues and resuscitation results. If the narcosis is not too intense, it may be combated effectively by inhalation of quite high percentages of carbon dioxide with oxygen. For extreme cases artificial respiration by intratracheal insufflation of oxygen or air, interrupted ten or twelve times a minute, will maintain life. Artificial respiration with the Eve-Cornish tilting board will probably also be found effective.

Patients with alcoholic coma [28] also are rapidly revived and sobered under the increased respiration induced by inhalation of carbon dioxide. It would save trouble and expense if this treatment for alcoholic intoxication was utilized by the police.

CYANIDE ASPHYXIA

Cyanide is now extensively used for the fumigation of buildings, ships, greenhouses and even trees. The canister gas mask affords effective protection against inhalation. Yet moderate degrees of poisoning are fairly frequent, and severe or even fatal cases occur often enough to be important.

The action of cyanide in the body is to induce asphyxia by inhibiting the respiratory ferment in the cells of the tissues. As the poison is extremely volatile, it is not only rapidly absorbed but may also be equally rapidly eliminated through the lungs. The treatment of slight cases, as recommended by Dr. C. L. Williams [29] of the U. S. Quarantine Station at New York, is therefore "fresh air and plenty of it." In cases that have progressed to apnea, artificial respiration should also be immediately applied. As cyanide is a powerful respiratory stimulant, the victim, during the asphyxiation, will have developed some degree of acapnia. To counteract this condition and hasten elimination of the poison, carbon dioxide diluted in air or in oxygen should be given by inhalation.

Treatment by intravenous infusion has some advocates. It is based on the fact that methemoglobin combines with cyanide. The substances injected are therefore such drugs as are known to convert hemoglobin into methemoglobin. Hug [30] advocates alternating injections of sodium nitrite and sodium thiosulphate. It seems to me, however, that, especially when the poison has been absorbed through the lungs, it is better to promote elimination by active pulmonary ventilation than to attempt to fix it in a combination in the blood.

In adopting measures of resuscitation, it is important to choose those that themselves involve no subsequent ill effects.

PULMONARY EDEMA FROM IRRITANT GASES [31]

The edema of the lungs induced by irritant gases results in asphyxia. It is chiefly a problem of war. But in the industries of peace also many cases occur in which the victims have inhaled ammonia, sulphur dioxide, chlorine, the fumes of various acids, phosgene, or the oxides of nitrogen. The irritant effects of the first four manifest themselves immediately and thus give warning for escape. Phosgene, on the contrary, and especially the oxides of nitrogen, whether from the spilling of nitric acid on wood, from the fumes of explosives, or from burning celluloid, are of less immediate choking character but are of much more serious subsequent effect. Hours after exposure to these gases pulmonary edema may develop, and the victim may drown slowly in the fluid exuding into his lungs.

For the prevention and treatment of pulmonary edema after inhalation of any irritant gas, the first essential is absolute rest for from twenty-four to forty-eight hours. The patient, even though in no apparent danger of developing edema, should be kept in bed; and if any indications of edema have appeared, he should on no account be allowed even to sit up in bed. If asked to sit up for auscultation, he may do so and an instant later fall back dead.

The asphyxia from pulmonary edema may take either of two forms, or one may follow the other: a gray and shocklike form, and a cyanotic form with extreme venous congestion. The gray state involves deficiency of oxygen in the blood without retention of carbon dioxide; or there may even be some degree of acapnia. The congested state develops when the lungs are waterlogged to such a degree that not only oxygen cannot pass in but also the much more easily diffusible carbon dioxide cannot pass out. The resulting hypercapnia and the obstruction to the flow of blood through the lungs are the joint causes of the venous congestion and overloaded heart.

For both conditions oxygen should be administered continuously in a concentration sufficient, if possible, to overcome cyanosis. Carbon dioxide inhalation tends to revive the patient from the stuporous condition induced by asphyxia, but it does not prevent the further development of asphyxia. It may even increase the venous congestion and should therefore not be administered in the stage of venous congestion.

The venous congestion may be treated by bleeding, after which intravenous infusion of saline solution has been recommended.[32] But the chief point to be aimed at is absolute quiescence, for any severe degree of

28. Hunter, F. T., and Mudd, S. G.: Carbon Dioxide Treatment in Acute Alcoholic Intoxication, Boston M. & S. J. **190**: 971 (June 5) 1924; Treatment of Acute Alcoholic Intoxication, editorial, J. A. M. A. **83**: 199 (July 19) 1924.

29. Williams, C. L.: Fumigants, reprint no. 1473 from U. S. Treasury Dept. Public Health Reports **46**: 1013-1031 (May 1) 1931.

30. Hug, Enrique: Treatment of Hydrocyanic Acid Poisoning, Buenos Aires letter, J. A. M. A. **102**:552 (Feb. 17) 1934.

31. Henderson, Yandell, and Haggard, H. W.: Noxious Gases and the Principles of Respiration Influencing Their Action, American Chemical Society Monograph Series, New York, Chemical Catalog Company, 1927.

32. Underhill, F. P.: The Experimental Treatment of Poisoning by Lung Irritant or Suffocant Gases, Med. Dept. of the U. S. Army in the World War, vol. 14, Medical Aspects of Gas Warfare, chapter 19, pp. 680-712, Washington, D. C., Government Printing Office, 1926.

pulmonary edema generally results fatally in spite of any treatment.

Surgeons, who during war become accustomed to seeing huge physical injuries, are shocked by the appearance of patients gasping in pulmonary edema. Yet in fact such patients are so far anesthetized by asphyxia that they actually suffer far less than those with severe gunshot wounds.

HEMORRHAGE AS A FORM OF ASPHYXIA [33]

Hemorrhage produces its ill effects largely through asphyxia. This is tacitly recognized by surgeons in their preference for transfusion of blood over any mere infusion of a saline or gum solution. The victim of acute exsanguination exhibits air hunger. In less extreme cases the volume of breathing increases with the loss of blood in a manner closely similar to the progressively developing hyperpnea in a man or animal undergoing carbon monoxide asphyxia.

All these facts point to the loss of the red corpuscles as an important feature of hemorrhage. The effect is essentially like the abolition of the oxygen carrying power of the corpuscles by carbon monoxide. This statement does not deny that the diminution of blood volume also impairs the circulation. But simple decrease of blood volume, such as is induced by the diarrhea of cholera, is effectively combated by intravenous infusion of saline solution. Hemorrhage, on the contrary, is only partially and temporarily relieved by restoration of blood volume. For effective relief and the return of normal quiet breathing, restoration of the oxygen carrying power of the blood by restoration of the supply of hemoglobin containing corpuscles is essential.

If, then, hemorrhage is largely a form of asphyxia, the obvious first aid measure is to administer oxygen and to maintain this inhalation by means of an inhalator, a tent, a metabolism apparatus or an anesthesia apparatus supplying oxygen, until transfusion can be performed. Patients with hemorrhage, if left for even a short time without oxygen inhalation or transfusion, develop acarbia. But until the oxygen carrying power of the blood is restored, carbon dioxide is probably best not used. Exsanguinated animals were found by Henderson and Haggard [33] to react badly to inhalation of carbon dioxide. The exsanguinated man or animal cannot overcome even a pseudo-acidosis—that is, acarbia—without restoration of the red corpuscles. Carbon dioxide may prolong life but it cannot recall a normal amount of alkali to the blood except in the presence of an ample supply of oxygen to the tissues.

The main point to be emphasized is this: After a severe hemorrhage it is not enough to stop the loss of blood and to prepare for a transfusion. Until the transfusion can be performed, and even thereafter, oxygen should be continuously administered. Hemorrhagic asphyxia should not be allowed to continue for a minute longer than can possibly be avoided. In all forms of asphyxia, measures of resuscitation, in order to be most effective, must be immediate.

FAILURE OF THE CIRCULATION AS A CAUSE OF ASPHYXIA

Surgical and traumatic shock has long been recognized as involving depression and finally failure of the circulation. A condition results similar in appearance to that induced by hemorrhage, involving a deficient supply of oxygen to the tissues and the usual consequences of such deficiency: acapnia, acarbia and pseudo-acidosis.

In shock without hemorrhage there is, however, no loss of red corpuscles from the body, and the blood alkali is merely displaced. Both corpuscles and alkali may be recalled into use.[34] To effect such recall, both the asphyxia and the acarbia may be combated with inhalation of carbon dioxide and oxygen.

One of the oldest and also the latest of many conceptions of the underlying cause of the depression of the circulation in shock [24] and in illness is that the tonus of all the muscles of the body, both skeletal and visceral, is depressed, and that the blood stagnates in the atonic tissues. Stimulation of respiration with carbon dioxide increases the effective difference of pressure between the tissues of the body and the thorax and thus promotes the venous return to the heart.

In the large majority of all deaths, whatever the initial cause, the sequence is that of increasing weakness, decreasing tonus and failing circulation. The terminal gasps and "death rattle" express the final reaction of the neurorespiratory system to asphyxia.

SUMMARY

The conditions to which resuscitation applies are all essentially forms of asphyxia. They include drowning, electric shock, asphyxia of the new-born, carbon monoxide, morphine, cyanide and alcohol poisoning, anesthetic and postoperative depression, pulmonary edema and hemorrhage.

For brief complete asphyxia, involving failure of breathing, the principal measure of resuscitation is artificial respiration, reinforced by inhalation of carbon dioxide and oxygen.

For prolonged asphyxia, inducing coma with depression of breathing, the principal measure of resuscitation is inhalation of carbon dioxide and oxygen, initiated, when needed, by artificial respiration.

The various forms of apparatus for treatment of asphyxia are here evaluated. Artificial respiration apparatus of the laboratory type should be available in the operating room. But such apparatus is not suitable for general use by laymen. Outside the operating room and the hospital, reliance should be placed on inhalators and the Schafer prone pressure method of artificial respiration.

The theory of asphyxia now generally accepted in the medical sciences is inconsistent with the facts of resuscitation established clinically. If the condition now called "acidosis" were really acid poisoning, inhalation of carbon dioxide would further poison the victims of asphyxia. The fact is, on the contrary, that carbon dioxide combined with a supply of oxygen has proved to be the specific cure for asphyxial "acidosis." For further progress in resuscitation and in related problems of clinical physiology the development of a sound theory of asphyxia, and of "acidosis," or acarbia, is urgently needed.

33. Henderson, Yandell, and Haggard, W. H.: Hemorrhage as a Form of Asphyxia, J. A. M. A. **78**: 697 (March 11) 1922.

34. Henderson, Yandell; Haggard, H. W., and Coburn, R. C.: The Therapeutic Use of Carbon Dioxide After Anesthesia and Operation, J. A. M. A. **74**: 783 (March 20) 1920. Henderson, Yandell: Acapnia as a Factor in Postoperative Shock, Atelectasis and Pneumonia, ibid. **95**: 572 (Aug. 23) 1930. Kroetz, C.: Sauerstoff- und Kohlensäureatmung in ihrem Einfluss auf den Kreislauf bei Gesunden und Kreislaufkranken, Ztschr. f. Kreislaufforschung **22**: 641-645, 1930.