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Comparison of mechanical and manual methods of artificial respiration

Robert Herman Heise
University of Nebraska Medical Center

A COMPARISON
OF
MECHANICAL AND MANUAL METHODS OF ARTIFICIAL RESPIRATION

By Robert H. Helse

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The Rapid Progress True Science now makes, occasions my regreting sometimes that I was born so soon. It is Impossible to imagine the Height to which may be carried, in a Thousand Years, the Power of Man over Matter....O that Moral Science were in a fair way of improvement, that Men would cease to be Wolves to one another, and that Human Beings would at length learn what they now improperly call Humanity!

Benjamin Franklin, 1780

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INTRODUCTION

In the field of medicine there is a critical need for a comprehensive knowledge of the methods of artificial respiration. To be able to perform a successful revival of an asphyxiated patient it is necessary to know not only the method, correct procedure, etc., but to know also why they are used. Therefore, the aim of this article is to attempt to clarify the situation of resuscitation of the individual and to weigh the advantages and disadvantages of the different procedures.

This problem is somewhat neglected by some physicians. Many emergencies occur before his arrival and the laity or partially trained personel handle the unfortunate person. Yet, an asphyxial death, due to one of its various causes, occuring in the operating room is a deplorable situation if ignorance of treatment be its cause.

To emphasize the need for proficiency in the treatment of axphyxia the following classification of common situations in which artificial respiration is indicated, as reported by the Committee on Asphyxia of the American Medical Association (52) demonstrates the broad scope of this problem.

Situations in which artificial respiration is indicated

1. Asphyxia neonatorum.

2. Asphyxia from gases, used industrially.

   (a) Carbon monoxide from illumination 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.

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.

HISTORICAL SURVEY

It is quite probable that from time immemorial men tried to revive the apparently dead, and that for accomplishing this end employed all sorts of procedures. The Bible records in II Kings, Chapter 4, Verse 34, that Elisha, in his attempts to bring to life the dead son of the Shunamite woman, lay himself upon the child and put his mouth upon the child's mouth and breathed therein and the child was revived.

A second recording is in I Kings, Chapter 17, Verse 21, that Elijah revived the widow's son by stretching himself upon the child three times and asking God's aid in restoring the child to life.

In the early ages extremely barbarian methods of resusitation were used by those aiding in reviving an unconscious patient. Great noises were made and attempts to inflict torture were used, such as burning the bare skin with a red hot iron. Melted wax, pitch and oil were poured on the skin. Scarification or cutting the skin with sharp instruments was common. Drawing of one or more teeth seemed a favorite method. Screaming and shouting into the ears of the patient in attempt to call him back from the dead were freely practiced. Instilling into

the eyes burning or volatile spirits was another method. Boiling water was sprinkled on hands, feet and over the heart. "Moxa" was also burned on the stomach of the patient.

The veritiable history of resuscitation is only about 150 years old.

In 1909 Keith (39) reviewed the transactions of the Royal Humane Society in a series of Hunterian lectures. This comprehensive review has contributed much to the following historical survey.

In 1793 Dr. T. Cogan translated a booklet from a society in Amsterdam which delt with the resuscitation of the apparently dead. This booklet fired Dr. William Hawes with thedesire to form a society in London which would adapt a method by which they could revive these unfortunate people and establish the latest and most approved methods. He gathered about him the finest medical minds of England to establish and continue this society. This zealous man lectured through out his country teaching the laity and medical men his methods. The methods employed by the Dutch society at this time were; (1) Warmth. (2) Artificial respiration carried out in the following manner... the operator closed the patient's nostrils and applied his mouth to the patientis and by blowing the lungs were inflated and the belly and chest were expanded. By compressing these parts with

his free hand the operator brought about an expiratory movement. (3) Fumigation counted equally as important as the first two measures. By this means the great bowel was filled with tobacco smoke; sometimes all the passengers on a Dutch canal boat might be summoned to assist the operator in administering this treatment if the special instrument, the fumigator, was not at hand. (4) Rubbing the body -- friction. (5) Stimulants applied to the nose or skin. (6) Bleeding from a vein particularly necessary as life returns. And, (7) vomiting, sneezing, and internal stimulants were recommended as accessory and useful means. With the next 50 years these methods were all to be discarded by the Royal Humane Society.

The laity of England were also busy at resusitation during the 18th century. At sometime in the past by trial and error methods, it was found that by laying the patient face down on a barrel or any convient object of such construction and rolling the victim to wring the water from him, he often survived the trement and lived. Such methods were forbidden at this time by the Royal Humane Society but upon examination are similiar to the Shafer (53) method later acceptable to the Society. Also the laity would hang the victim up by his heels to let the water run out of him or else they would hang

the poor unfortunate over a table, strip him and rub him vigorously with salt. All of these attempts were successful at various times and still are used on occasion. The mouth to mouth method which was popular on the continent had little success in England.

In 1776 Dr. Hawes appealed to Dr. John Hunter to devise the best methods of artificial respiration. Dr. Hunter turned to some experiments done while working for his brother. He found that while working in the thorax of a dog, keeping the lungs active by means of a double acting bellows, one for inspiration and one for expiration, that when the bellows stopped the heart beat of the animal gradually slowed then stopped. He surmised from his experiment that restoration must therefore depend immediately on the application of air to the lungs. He further inferred that it was not the restoration of arterial blood to the nervous system but rather a reflex effect between the lungs and heart. He recommended; (1) that air be introduced into the lungs by properly constructed bellows and that the use of oxygen discovered in 1774 by Priestly would be of great benefit. (2) The gradual application of warmth. (3) The application of stimulants

to the nose, skin, lungs and to the stomach through the esophageal tube and lastly to the bowels by steam and enemata administered per rectum. He did much by condemning blood letting.

In 1772 A. de Haen, professor of Medicine at Vienna, found that water did enter the lungs in the process of drowning. This is contrary to the belief of the time and indeed contrary to the belief that persisted for a century after his discovery.

By 1782 the Royal Humane society recommended inflation by bellows in preference to the mouth-to-mouth method. Ingenious bellows were devised with valves so arranged that they could be used to exhaust as well as inflate the lungs. Usually the operator employed an assistant who applied pressure to the epigastrium when the chest was seen to heave after each inflation. The assistant also held the cricoid cartilage back to prevent the air passing down into the esophagus. That the tongue might fall back and occlude the opening into the larynx was discovered by E. Goodwyn This failing was overcome by Monroe by passing a catheter into the larynx by way of the mouth.

For the next forty years bellows were used throughout the

world as the best method of artificial respiration but in 1829 Leroy in France relegated the bellows to their proper place by the fireside. He found (1) that it was possible to kill an animal by suddenly inflating its lungs; and (2) that it was possible to produce emphysema of the lungs and pneumothorax in dead animals. This report alarmed the Society and in 1837 the bellows were no longer advisable. Sir Benjamin Brodie, in 1821, made an important contribution when he stated that artificial respiration should be started within two to three minutes after respiration had ceased because the heart would be stopped shortly thereafter and artificial respiration would be valuless. Following this statement more experiments were conducted and Ericson advised the use of warm air and using the bellows but it had little support and the bellows were speedily forgotten.

Historical manual methods

The report of the Royal Humane Society in 1812 recommended that if bellows are not present the surgeon should practise alternate compression and relaxation of the abdomen rather than to resort to mouth-to-mouth inflation. Since Leroy recognized the damage done to the lungs by the bellows method,

he recommended laying the patient face upwards and compressing the anterior walls of the abdomen and thorax, thus producing expiration; inspiration resulting from the elastic rebound of the chest wall. Dalrymple on reviewing Leroy's method suggested a side to side compression either by the direct application of the hands or, better, by a wide (18 inch) many-tailed bandage placed around the patient's body. The patient being laid on his back and the bandage passed beneath him, its ends being crossed in front of the chest. An operator on each side of the body compressed the thorax by pulling on the crossed ends, then relaxing their hold allowed the chest to recoil and inspiration occured. This method was used until Marshall Hall (27) introduced his method.

In 1856 Marshall Hall (27) introduced the first generally acceptable manual method of artificial respiration. While experimenting to perfect his new method of resuscitation he found that the tongue and larynx were apt to fall back and occlude the air passage thus confirming Goodwyn's statements of seventy years before. As a result he started his method by having the patient in the prone position and then applyed pressure to the back -- over the thorax and abdomen. The chest began

to recoil as pressure was withdrawn and inspiration was started. The completion of his method was for the operator to place one hand under the shoulder of the patient and the other under the hip and thus turn the patient and complete the inspiratory phase. When the shoulder is thus lifted every muscle passing to the sternal and lateral aspect of the chest becomes taunt and tends to expand the thorax.

Upon examing Marshall Hall's (27) method the possibility of carrying on artificial respiration by a process of compression depends on the resiliency of the thoracic walls -- on their tendency to resume their natural position when pressure is withdrawn. This property depends on: (1) the tension on the ligaments which bind the ribs to the vertebral column and limit the extent of the costal movement -- when the ribs are depressed beyond their functional extent a strain is thrown on these ligaments; (2) the bending of the ribs and especially their cartilages; (3) the stretching of all the muscles that are attached to the ribs; and (4) the compression of the liver, the heart, the spleen, and the intestines which in the asphyxiated may be regarded as sacs filled with semi fluid contents.

Following Marshall Hall (27), in 1861, Silvester (57) published a method of artificial respiration worked out

on quite a new principle. His aim was to imitate the natural respiratory movements. He selected supine posture, producing inspiration by expanding the chest wall and expiration by compressing it. The operator stands at the patient's head and seizing the arms above the elbows lifts them outward and then back towards the patient's head. The tauntness is much increased on the pectoral muscles according to Champney (39) in 1887 if the arms be everted or rotated outward as they are lifted. The humerus and shoulders are thus used as levers and the anterior wall of the chest is raised upwards and forward, expanding the part of the lung which lies under the sternal chondral wall. Expiration is produced by pressing the patient's arms firmly against the anterior wall of the chest. Unfortunately, it is a laborious and exhausting method and one which is effective only when applied expertly.

The efforts of Benjamin Ward Richardson (39) contributed no new method of artificial respiration but they did much to aid the understanding of and what should be considered in artificial respiration. From his experiments two inferences may be drawn: (1) Resuscitation is possible by artificial respiration so long as there is a pulmonary circulation; and (2) it is also

possible after circulation has ceased and before coagulation has begun if the blood can be drawn or forced through the lungs with sufficient pressure to enter the arteries of the heart.

In 1867 Pacini (39) introduced a new method in which the supine posture is used. In this manipulation the shoulders are seized with the thumbin front of, and the fingers behind, the head of the humerus; as the operator pulls and lifts the shoulders toward the patient's ears the pectoral muscles, passing to the front of the chest and the latissimus dorsi passing to its side, becomes stretched and inspiratory movement of the ribs -- chiefly the upper, is effected. Expiration is caused by the recoil which follows relaxation of the shoulders.

In 1868 Dr. W. P. Bain (2) modified Pacini's (39) method in such a manner that when grasping the shoulders the movements of the clavical might be made more effective. Also, he introduced an efficient but laborious method in which the operator stood at the head of the passive subject and then pulled and lifted the body by the hands toward him. The feet of the subject being held or fixed.

In 1871 Dr. Benjamin Howard (37) of New York taught the

New York police a simplified method of artificial respiration. The principles underlying his method are the same as were utilized by Leroy, Dalrymple, and Marshall Hall (27) - viz., expiration by compression and inspiration by the recoil of the chest wall. As a preliminary measure the body of the patient is turned face down and is compressed to expel the water, then turned face upward and artifical respiration is applied. The operator faces and kneels astride the patient placing a hand over each prominent subcostal margin so that the fingers occupy the furrows between the ribs above the margins and the palm below them. Pressure is then applied by theoperator placing his weight over his hands, producing expiration by a triple movement; (1) the lower sixth, or diaphragmatic ribs are depressed; (2) the abdominal contents, especially the liver and spleen, engorged and distended with blood, are compressed so as to force upwards the diaphragm and empty the lungs; and (3) the extension of the spine is partly undone. The over extention is produced by placing the subject, on the onset of artificial respiration, on any convenient support so as to make the subcostal margin prominent. Howard's (37) method differs from Silvester's (57) in its effect on the circulation because the pressure applied in this

method will force the blood through the pulmanary circulation.

In 1879 Max Schuller (39) reversed the movements used by Howard (37). He stood or knelt at the patient's head and bent over it to grasp the subcostal margins which are made prominent by placing a support under the patient's back; the operator inserts his fingers under the subcostal margins and then draws the ribs upwards and outwards in the direction of the patient's shoulders, thus producing inspiration. Expiration is produced by pressing the lower ribs downwards and inwards to force the abdominal viscera and diaphragm upwards.

THE MECHANICS OF RESPIRATION

AND

THE EFFECTS OF RESPIRATION ON THE BLOOD PRESSURE

Before birth the alveoli contain a small quantity of fluid; the thorax is unexpanded and completely filled by the airless lung. At this time only a small part of the blood from the right heart passes through the lungs. The remainder passes by the ductus arteriosus into the aorta and via the foramen ovale in inter-auricular septum to the left heart. Respiratory movements are made by the fetus in utero as was demonstrated by Snyder and Rosenfeld (59). These movements are readily inhibited, especially by anoxemia and the narcotics. They are depressed by a carbon dioxide deficit.

At the moment of birth the respiratory movements become more forceful, and diaphragm descends and the external intercostal muscles contract with the result that the diameters of the thoracic cavity are very considerably increased. A large proportion of the venous blood is now conveyed through the lungs.

The general enlargement of the capacity of the thorax -- a closed cavity -- tends to reduce the pressure on the outer surfaces of the lungs. The greater the degree of enlargement of the chest, the greater will be the reduction in the pressure upon the outer pleural surfaces on the lungs. The interior of the lungs,

however, is in direct communication, through the air passages, with the atmosphere. The visceral and parietal pleurae being inseparable, under normal conditions, the lungs follow the thoracic wall as it enlarges, and therefore must expand. The raretaction of the pulmonary air, because of the expansion, results in a flow of atmospheric air into the lungs. full expansion of the lungs is not attained until some few days after birth. The lung throughout the individual's life remains in the expanded position -- pressed, as it were, against the thoracic framework as a result of the greater pressure exerted upon the alveolar than upon the pleural aspects of the pulmonary tissue.

The pleural cavities

The lungs are invested by the visceral layer of the pleural membrane. The membrane is reflected from the root of each lung on to the inner aspect of the walls of the chest and upper surface of the diaphragm -- this is the parietal layer of the pleura. The two layers thus form a closed membranous sac, the pleural cavity, which surrounds the lungs. In health no actual space exists; the two membranes are in apposition except for a thin film of lymph which acts as a

lubricant to allow the surfaces to glide over one another during the respiratory movements.

Intrapleural pressures

It has already been mentioned that the pressure on the pleural surfaces of the lungs is less than that upon their alveolar surfaces, i.e., the intrapleural pressure is subatmospheric. How is this "negative" pressure produced? As stated above when the chest cavity is first expanded, the lungs are carried outwards by the inflow of air to fill the enlarged space. The expansion of the thorax, however, and the consequent inflation of the lung puts the pulmonary tissue upon the stretch. In other words, the closed thoracic box, as a result of the first breath, becomes too large for the lungs to fill by a simple unfolding of the walls of the air spaces. The elastic tissue of the bronchial tree, blood vessels, and of the air sacs themselves is put under stress and is constantly pulling against the stretching force. This pull of the elastic lung amounts, in the adult when the chest is about midway between inspiration and expiration, to a pressure of from -4 to -5 mm. of mercury. In the new born the lungs fill the cavity with comparatively little stretching. In later years the distensibility of

the lungs increases, since the thoracic cage grows more rapidly than the lungs; the elastic pull in consequence also increases and with it the intra-pleural negative pressure.

This "negative" pressure is increased during inspiration -- since the distension of the elastic lungs is greater -- and reduced during expiration. During the former phase of respiration it amounts to about -6mm. of mercury, during an ordinary expiration it is reduced to about -2.5 mm. of mercury. In midposition as stated above it is about -4.5 mm. of Hg. In forced inspiration the pressure with a closed glottis may amount to -40 mm. of Hg. and in forced expiration under the same circumstances it is abolished and a positive pressure of 50 mm. of Hg. is substituted.

The respiratory movements.

The foregoing paragraphs make it clear that the flow of air into and from the lungs depends entirely upon changes in the capacity of the thoracic cavity. The lungs play a purely passive role. During inspiration the thoracic cavity is enlarged in all diameters, vertical, anteroposterior and transverse. The enlargement is not equal in all directions. The upper part

of the thorax increases much less in capacity than does the lower; and since the position of the spinal column remains relatively fixed the increase in antero-posterior diameter of the thorax is due mainly to an expansion forwards. The increase in the vertical diameter is due, not to the upward expansion of the chest cavity but to the downward elongation resulting from descent of the diaphragm.

Unequal enlargement of the thoracic cage entails unequal expansion of the lungs. Keith (39) distinguishes three zones in the expanding lung.
(1) A non-expansile root zone containing the bronchus, pulmonary vessels and lymphatics and their main divisions.
(2) An intermediate zone in which the vascular and bronchial branches radiate outwards toward the lung surface. Between these rays is situated expansile pulmonary tissue. This zone therefore consists of tissue of varying degrees of expansibility, that lying near the periphery of the rays being more expansile than that situated more centrally.
(3) An outer or subpleural zone from 1 to 1¼ inches deep of maximal distensibility.

Those regions of the lungs lying in relation to the relatively immobile regions of the thoracic walls,

namely (1) the dorsal surface of the lung apex, (2) the posterior surfaces of the lungs in contact with the spinal column and attached segments of the ribs, and (3) the mediastinal surface lying in relation to the pericardium and other structures of the mediastinum, are expanded indirectly. The parts of the lung which are directly expanded during inspiration are those lying in contact with the freely movable boundaries of the thorax, namely (1) the sternum and ribs and (2) the diaphragm.

It is evident that those portions of lung in contact with practically stationary regions of the thoracic walls can only be expanded indirectly, that is, when other parts of the lung move out of the way. If the root of the lung were fixed this could not occur. Macklin ( ) showed by x-ray studies on the human that the lung root moves downwards, forwards and laterally during inspiration. Also he demonstrated that the bronchial tree becomes elongated during the inspiratory phase. The trachea becomes stretched and the apex of the lung actually descends as it expands. During expiration the highly elastic bronchial tree recoils to its previous length and the lung root ascends. If the root of the lung were fixed, little expansion of

a region such as the apex or other regions could result. At the same time only very moderate expansion of other regions (e.g. costosternal and diaphragmatic) occur if the bronchial tree could not lengthen.

The enlargement of the thoracic cavity during inspiration

According to Keith (39) there are four distinct mechanisms.

(1) The thoracic lid
(2) The upper costal series (second to the sixth rib enclusive.)
(3) The lower costal series (7th to the tenth rib enclusive.) and the diaphragm.
(4) The floating rib series and the muscles of the abdominal wall.

The thoracic lid or operculum

This unit is formed by the first pair of ribs and the manubrium sterni. It is jointed behind to the spinal column and in front to the sternum by the manubrio-sternal joint. During the elevation of the thorax in inspiration the thoracic lid moves as a single piece upon the body of the sternum, assuming a more horizontal position (by from one degree to sixteen degrees). That is, the manubrium is pushed upward and forward. Thus the upper part of the thorax is increased in its

anteroposterior diameter. The anterior portion of the lung apex is directly expanded to some extent by this mechanism.

The upper costal series

The second through the sixth ribs slope obliquely from behind downwards and forwards. Each rib is longer, its direction more oblique and it makes a fuller sweep outwards than its neighbor immediately above. During inspiration these ribs (with the exception of the second) assume a more horizontal position, their anterior portion moving upward and forward. That is, each rib rotates around an oblique horizontal axis parallel to its neck. The sternum is thrust forward and upward, executing a movement at the manubrio-sternal joint. These movements increase the anteroposterior diameter of the thorax. The elevation of the ribs is effected by the external intercostal muscles. The fibers pass obliquely downwards and forwards from the lower border of one rib to the upper border of the rib below. When the muscle contracts it exerts a pull upon these attachments which tends to depress the upper rib of the pair and to raise the lower. The first rib, however, acts through the contraction of the scalene muscles, as a

fixed point above, so that contraction of the external intercostals can only result in an elevation of the ribs. Owing also to the obliquity of the fibers which are attached below to the anterior end of the long arm of a lever and above to the posterior end of the long arm, a distinct mechanical advantage is given to the upward movement. The internal intercostal muscles are, in the cat at any rate, as shown by Bronk and Ferguson (11), expiratory in function. The internal and external intercostals receive impulses alternately along the intercostal nerves.

The strongly bowed mid-portion of the body of each rib from the second to the sixth also becomes elevated, in relation to its two ends, rotating around an oblique anteroposterior axis. This movement which is compared to the raising of a bucket-handle to a more horizontal position increases the transverse thoracic diameter.

The lower costal series and the diaphragm

The ribs from the seventh to the tenth also swing outwards and upwards (bucket-handle movement) during inspiration, rotating around an oblique anteroposterior axis which passes through the midline in front and the rib necks behind. The subcostal angle is widened by this movement and the transverse diameter of the lower part

of the thorax increased; the anteroposterior diameter is slightly reduced.

The diaphragm is the chief muscle of respiration, its movements being responsible in deep breathing for 60% of the total amount of air breathed. It consists of a musculo-tendinous sheet arched toward the thoracic cavity. The tendinous portion is centrally placed and attached to the pericardium. The muscular tissue is placed circumferentially. The diaphragm consists of two parts which differ from one another in their actions.

(1) The costosternal part arises from the back of the ziphoid and the cartilages and adjacent portions of the six lower pairs of ribs. It is attached to the anterior edge of the central tendon.

(2) The lumbar or crural part arises from the fibrous arches over the quadratus lumborum and psoas muscles and by two fleshy bundles (the crura of the diaphragm) from the bodies of the upper lumbar vertebrae. The fibers so originating are inserted into the posterior margin of the central tendon.

Action of the diaphragm

The diaphragm descends during inspiration and ascends during expiration. In full expiration its upper limit

lies at a level situated between the costal cartilages of the fourth and fifth ribs. In quiet breathing the range of its movement is about 1.2 cm. and in forced breathing about 3.0 cm. The total diaphragmatic surface is about 270 sq. cm. A descent of 1.0 cm. therefore (assuming that all regions descend practically to the same extent) will increase the thoracic capacity by 270 cu. cm. and cause a corresponding volume of air to enter the lungs.

As the diaphragm descends its domed shape alters very little; it may be seen by the flouroscope to move up and down like a piston. At the end of expiration a considerable proportion of the diaphragmatic surface is in contact with the chest wall as high as the sixth or seventh rib, but during inspiration it is "peeled off" the thoracic boundary, and the base of the lung expands coincidently to fill the space (pleural sinus). The costosternal portion of the diaphragm, using the lower ribs, which through the action of the external intercostals serve as fixed points, moves downward and forward pushing the abdominal viscera before it. Thus, the capacity of the lower part of the thorax is increased. The abdominal wall distends but when, as a result of the resistance offered by the abdominal

muscles, the downward movement of the viscera becomes arrested these act as a fixed point for the continuing contraction of this part of the diaphragm. Its force is now spent in raising the lower ribs to which it is attached; through this action the sternum is thrust forward and upward. The spinal or crural part in its descent acts solely in increasing the vertical diameter of the thorax.

The excursions of the diaphragm and consequently its mid-position are influenced by (1) the upward pull of the subatmospheric intrathoracic pressure, (2) the abdominal viscera. In the standing position the weight of the latter exerts a downward pull and so aids the descent of the diaphragm but hinders its ascent; the mean or mid-position of the diaphragm is therefore taken up at a lower level than in recumbency when the viscera exert an upward pressure. (3) The abdominal muscles; these, when lax and the body in the standing position, allow the viscera to subside to a lower level and so increase the downward pull upon the diaphragm. In persons with extremely weak abdominal muscles, such as subjects with visceroptosis, the downward pull upon the diaphragm greatly interferes with its movements. The breathing is largely costal.

The floating ribs (11th and 12th) and the abdominal muscles.

Functionally the floating ribs must be considered with the abdominal muscles which are the antagonists of the diaphragm. The recti and oblique muscles relax as the diaphragm descends; they contract with its ascent.

Expiration

Expiration is to a large extent a passive movement; in quiet breathing it is probably entirely so. That is, the contraction of the inspiratory muscles ceases; the thoracic framework tends through its own weight to resume its former position, the elastic lungs recoil and the relaxed diaphragm is drawn upwards toward the thoracic cavity by the "negative" intrathoracic pressure which is greatest at the end of inspiration. There is, however, also a definitely active element in expiration when this is forced. As mentioned above, the abdominal muscles contract and by pressing upon the viscera aid the ascent of the diaphragm, i.e., the latter is pushed up by the increased intrabdominal pressure resulting from the contraction of the abdominal muscles, as well as "sucked" up by the subatmospheric pressure within the thorax.

During forced expiration the internal intercostals whose

fibers like those of the external intercostals course obliquely but in the opposite direction (downwards and backwards) contract and so aid in the depression of the ribs. The restoration of the thorax to its previous diameters is, of course, accompanied by a corresponding reduction in the capacity of the lungs and the expulsion of air from its air spaces.

The act of coughing

There are three mechanisms for the expulsion of foreign material. First, the action of the cilia; second, the peristaltic motion in the bronchioles; and third, the cough reflex.

The ciliated cells are found in the nasal part of the pharynx, in the lower part of the vestibule of the larynx and in the trachea. The cilia beat with a motion which propels material toward the mouth. They become even more abundant when the large bronchioles are reached, but are largely replaced by cuboidal or flattened cells in the respiratory bronchioles. These cells are highly efficient when performing under normal conditions. However, a multitude of things effect their motility. They are not influenced by nerve impluses, but are very susceptible to chemical changes in the

blood and to substances applied locally. Certain general anesthetics depress their activity and many sedatives exert the same effect. Ciliary action is depressed by cold and increased when the temperature is slightly above normal. Their effectiveness may also be varied by changing the properties of the material as well as by increase or decrease in the rate or force of beating.

The peristaltic motion of the bronchioles

From the studies of Ellis and Nicholson (23), the rhythmical bronchial movements appear to be purely passive in nature and not integrated through reflex mechanisms with inflation and deflation of the lungs. The bronchioles also exhibit a peristaltic movement which can be detected by X-ray photographs of the bronchioles after the injection of radio opaque materials. It is not thought that the peristaltic movement plays any part in the movement of air, but it appears to assist in the movement of foreign material towards the large air tubes.

The cough reflex

The cough reflex is most commonly initiated by the stimulation of afferent nerve endings in the region of the tracheal bifurcation, the most sensitive area, on

the laryngeal mucosa. It may also be initiated from the excitation of vagal afferents in the lungs, or from nerve endings in the pleura. Ear disease, through the stimulation of terminals of the auricular branch of the vagus (Arnolds nerve), may also cause coughing. The act itself consists of a short inspiration followed immediately by the closure of the glottis and a forcible expiratory effort. A considerable degree of pressure is there by developed within the lung. The glottis then opens suddenly and offending material is moved a variable distance along the air passage. If it reaches an insensitive area the coughing ceases. During the subsequent inspiration the irritating particle, if not large enough to seriously obstruct the air passage, remains in its new position, from which it is carried forward again during succeeding expulsive efforts until it is swept away from sensitive areas.

Influence of respiration on blood pressure in man

The effect of respiration on the blood pressure is important in the body economy.

According to Battro et al (4), the negative pressure within the pleura is greater during inspiration than during expiration. During ordinary inspiration, a larger quantity of venous blood goes to the heart, because of the greater negative pressure within the thorax. At the same time, with the descent of the diaphragm, the abdominal pressure is increased. Both factors contribute to augment the inflow of blood to the heart at the end of inspiration. The result is an increase in the cardiac output in this phase.

During expiration, the phenomena are reversed, and there is a consequent diminution in the blood pressure.

Considering other factors, the intervention of which can alter the results stated above, the anatomicophysiologic condition of the pulmonary bed, the variations in the diameter of the pulmonary blood vessels and the resistance which these vessels offer to the blood must be mentioned. The intrapericardial pressure is also significant.

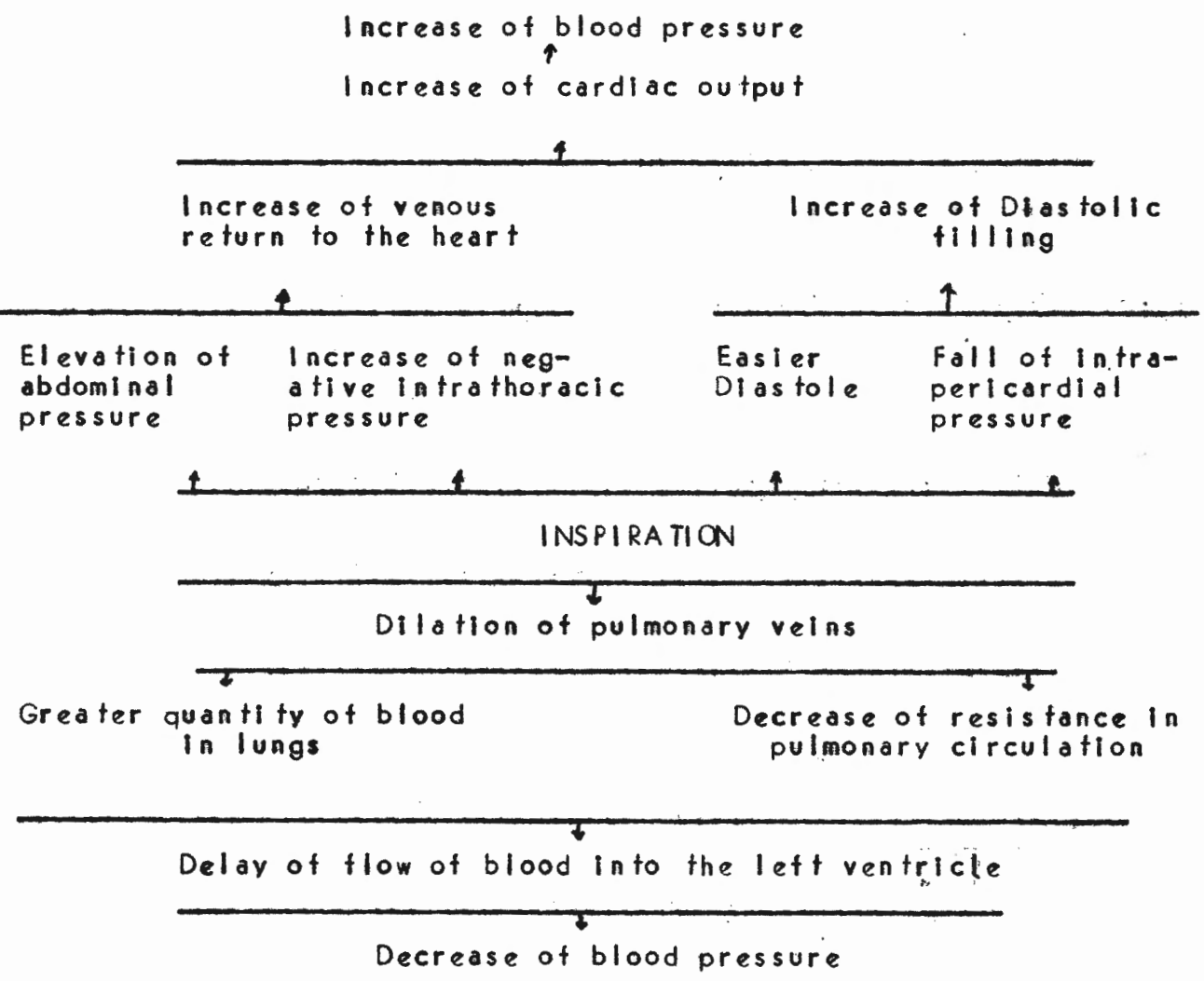
According to the findings of Heinbecker and Trimby and

Nicholson (60) the pulmonary bed admits a greater quantity of blood through expansion of the lung during inspiration, especially at the beginning. Consequently, the resistance to the blood in its pulmonary course and the inflow of blood to the left ventricle are lessened and the blood pressure tends to decrease.

In expiration, in spite of greater resistance of the pulmonary vessels, the blood flows more easily into the left auricle. The consequence of this is an increase in the systemic blood pressure. In addition, Lewis (40) states that during inspiration there is a decrease in the intrapericardial pressure with an increase in the diastolic inflow and the cardiac output.

The relationship between all these factors may explain the fluctuations of blood pressure in the course of normal breathing. In the following table are considered the different factors which act on the blood pressure.

Factors Which Contribute to Variations of Blood Pressure During Inspiration



(adapted from Battro et al (4)

In ordinary breathing there is generally an inspiratory fall and an expiratory elevation of the blood pressure. The vascular capacity of the lung probably predominates over the thoracicabdominal factor in these cases.

During deep, slow thoracic breathing, the variations of the blood pressure are clearer than during ordinary breathing. As has been previously stated, there is generally an increase in pulse rate and a fall in blood pressure during inspiration and a rise during expiration and the respiratory pause. The behavior of the blood pressure is neither constant nor uniform as cases have been found in which it has risen during inspiration. These variations are independent of the heart rate. Expiratory slowing has been occasionally found which commenced sometimes at the beginning and sometimes at the end of expiration and persisted during the respiratory pause.

A decrease in blood pressure during inspiration is probably due to predominance of thefactor of negative intrathoracic and positive abdominal pressure. This predominance favors the venous return to the heart and hence the systolic discharge of the ventricle. An increase in blood pressure during expiration leads one to suppose the predominance of the pulmonary factor, with

a consequent increase in the venous return to the heart.

During deep, fast thoracic breathing an increase of the blood pressure during expiration has been observed, and with deep, slow abdominal breathing there was shown a rise of blood pressure during inspiration.

Bbod pressure during inspiratory apnea

The record of the arterial pressure curve during apnea following a deep inspiration shows that both the systolic and the diastolic pressure fall gradually in the first three to five seconds, and the dicrotism becomes more evident. The cardiac rate increases 6 or 7 beats per minute. The initial fall is followed by a gradual rise of the arterial curve and the cardiac rate until the end of the apnea. As soon as breathing is recommenced, the arterial curve regains its original state and the blood pressure returns to its original level, though during a few seconds it may be higher. These variations may even be recorded in arteries far from the thorax, e.g., in the posterior tibial artery.

The changes in the first seconds of the period of apnea may be explained on the basis of mechanical interference, chiefly blocking of the venous return to the heart,

which tends to reduce the amount of blood flowing to the heart and produces diminution of the cardiac discharge.

The changes of the second phase may be explained by the intervention of reflex phenomena due to stimulation of vasomotor centers by the hypercapnemia or hypoxemia associated with any more or less prolonged respiratory pause.

Arterial pressure during expiratory apnea

In the course of the interval of apnea following an ordinary expiration, it is observed that after a slight decrease in blood pressure during the first seconds there is a progessive increase in the maximal, the medial and the minimal pressure until the end of the test, which ggenerally lasts from fourteen to twenty seconds. The pulse pressure also increases toward the end. Holding the breath for a longer period causes a series of muscular and diaphragmatic contractions with a terminal elevation of the arterial pressure and a slowing of the pulse.

The initial descent could be interpreted as the result of stopping the respiratory movements. The consequent elimination of the thoracicdiaphragmatic factor reduces the venous flow to the heart and diminishes the cardiac

output. The subsequent rise could be explained by the excitation of the vasomotor centers caused by oxygen hyposaturation of the arterial blood (hypoxemia) or by accumulation of carbon dioxide (hypercapnemia). The cardiac rate increases slightly as a result of the sympathetic stimulation. If the test is prolonged muscular contraction produces abrupt changes in intrathoracic pressure.

Effect of cough on arterial pressure

The intra-arterial pressure was studied during the exertion of coughing and it was found that it rose sometimes to very high levels. When the cough coincides with the top of the pulse curve, the systolic pressure may duplicate its normal values.

The influence which this phenomenon has on the cerebral arteries has been studied by Hamilton, Woodbury and Harper (28). The simultaneous registration of intra-arterial, intrathoracic and intraspinal pressure has shown that an abrupt rise of intra-arterial pressure caused by coughing is accompanied by an increase in intraspinal pressure. Therefore, a rise in the intra-arterial pressure in the brain is accompanied by a correlative increase in the pressure of the cerebrospinal

fluid, and possible rupture of an artery is avoided. They have proved that identical protecting mechanisms against abrupt rises in blood pressure exist in the thorax and in the abdomen.

THE PHYSIOLOGY OF RESPIRATION

Respiratory Center

The respiratory center is located in the reticular formation and extends from slightly below the pons to the level of the calamus scriptorius. Transection of the brain stem below the latter level stops respiration. According to Pitts et al, 1939, 1940; (49)(50) Beaton and Magoun, 1941 (5), when the interior of the medulla is explored in cats or monkeys with a needle electrode, through which stimulation is applied to one point after another in the reticular formation, either inspiration or expiration can be produced depending on the location of the point stimulated. The points in the reticular formation which cause expiration lie dorsal to those that cause inspiration. By this method it has been possible to determine the extent and outline of the respiratory center and to show that it includes a dorsal expiratory division, corresponding to the dorsal part of the reticular formation, and a ventral inspiratory division, corresponding to the ventral part of the reticular formation.

It was formerly supposed that the respiratory center possessed an inherent rhythmicity of its own; but whether or not this is true, its activity is controlled

by the condition of the blood in its capillaries and by impulses reaching it from various sources especially from the lungs by way of the vagi.

Pitts et al (49) discovered that electrical stimulation within the expiratory center causes expiration; if stimulated during inspiration or during apneusis these movements are inhibited. Regular respirations -- inspiration alternating with expiration -- are induced by rhythmical stimulation of the inspiratory center; expiration then occurs passively. Rhythmical stimulation of the expiratory center also produces regular respiration, spontaneous inspirations then alternating with the expiratory movements. Intimate connections exist apparently within the centers and between the two. Reciprocal inhibition of one center upon stimulation of the other can be demonstrated. The similarity in the effects of stimulation of the expiratory center and of the central end of the vagus, namely, inhibition of inspiration and of apneusis, has led Pitts and his colleagues (50) to the conclusion that the vagal effects are mediated through the expiratory center.

The chemical control of respiration under ordinary physiological conditions is carried out through the action of carbon dioxide on the respiratory center.

Haldane and Priestley (26) found that in man a rise in the carbon dioxide of the alveolar air of only 0.2 per cent (equivalent to an increase of 1.5 mm. Hg. pressure) was sufficient to double the pulmonary ventilation. Changes in the oxygen tension and hydrogen ion concentration are less effective.

The respiratory center is connected with the neurones of the phrenic and intercostals nerves in the cervical (C. 3, 4 and 5) and upper thoracic segments of the cord (T. 2-6) by descending tracts which run in the anterior columns and in the ventral part of the lateral columns of the spinal cord.

Voluntary control - Emotional influences (7)

That the respirations are under voluntary control for short periods of time is common knowledge. This control, though we are scarcely conscious of it in most instances, is being exerted in numerous ways in the ordinary affairs of daily life, such as in speaking, swallowing, laughing, blowing, coughing, sucking, etc. But the power of the will to inhibit respiration is strictly limited. The breath can be held for only a brief time (about 45 seconds) before automatic or involuntary control asserts itself; the inhibitory influence is overridden and the muscles of respiration contract

despite all one's efforts to "hold the breath". The nerve elements giving origin to the voluntary impulses are probably in the motor area of the cerebral cortex. Stimulation of area 6a in monkeys accelerates the respirations; area 6b is inhibitory.

The respirations may be affected profoundly by impulses arising in higher cerebral centers as a result of various emotional or other mental states, e.g., fear, grief, surprise, interest, amusement, etc.

Reflexes from other parts of the body

According to Best and Taylor (7), stimulation of almost any afferent nerve may bring about a reflex change in respiration. Stimulation of pain fibers is especially potent in this regard and the respiratory effects of the excitation of the cutaneous nerves by extremes of heat or cold are well known. The great increase in pulmonary ventilation occurring in muscular exercise is to a large extent dependent upon reflexes originating in the active skeletal muscles. Proprioceptive impulses from the diaphragm and other respiratory muscles during one respiratory phase exert an important influence upon the succeeding movement. Stimulation of the abdominal viscera, either during surgical operations or as a result of disease, may

cause profound changes in breathing. The glossopharyngeal nerve contains afferent fibers which inhibit respiration during the second stage of the act of swallowing. Abrupt inhibition of respiration is also caused by the inhalation of an irritant gas through stimulation of nasal branches of the 5th nerve. In other instances irritation of these endings may cause sneezing -- a modified respiratory act. Cough, though it can be brought about by a voluntary effort, is most commonly reflex in character, initiated by the stimulation of afferent nerve endings in the trachea and larynx.

Carotid and aortic bodies

Heymans and Heymans (36) made the surprising discovery in 1927 that respiratory reflexes could be elicited from the aortic area. This discovery and the researches of Heymans and his associates on the corresponding role played by the structures in the region of the carotid bifurcation opened a new field in the physiology of respiration. These men discovered that the carotid and aortic areas each contain two types of receptors; one type, pressoreceptors, responds to mechanical, the other, chemoreceptors, respond to chemical stimulation. The pressoreceptors situated among the collagenous fibers in the wall of the carotid sinus and in the wall of the aortic arch, are stimulated by a stretching force,

as by a rise in arterial blood pressure. The chemoreceptors are contained in small glandular structures, the carotid and aortic bodies. The respiratory reflexes initiated from these two types of receptors are contrary in their effects. Stimulation on the pressoreceptors inhibits respiration, and abrupt rise in blood pressure such as follows the injection of adrenaline causing respiratory arrest (apnea). Excitation of the chemoreceptors increases the rate and depth of breathing. Impulses reach the respiratory center from the carotid body andccarotid sinus by way of the glossopharyngeal nerve and those from the aortic body and aortic arch by way of the vagus nerve.

Though of the utmost importance in the control of circulation, the pressoreceptors do not appear, in mammals at least, to serve any respiratory function under physiological conditions according to Best and Taylor (7). They state further that there is no circumstance of a physiological nature under which the inhibition of respiration is caused by a rise in blood pressure or by the stimulation of these receptors in any other way. Nor is it apparent that such a reaction would serve any useful purpose. That they give a respiratory

response when very strongly stimulated appears to be accidental and without physiological significance.

According to Barach et al (3), who made observations on a group of subjects, there must be a rather severe lowering of arterial oxygen saturation of the blood to produce a marked change in respiration. In those of the group who retained good functional activity the oxygen saturation was 79.3 per cent and the expiratory exchange increased from 9.7 to 10.98 liters per minute. In those who were confused the oxygen saturation was 77.8 per cent and the respiratory exchange only 8.09 to 9.36. Increases of 13 and 16 per cent of the control value indicate that the hyperpneic response was certainly not marked. Data such as this indicate that, in stable, normal subjects, the hyperpnea of anoxemia is not marked at all until the oxygen saturation is around 77 per cent. If the saturation becomes lower the chemoreceptors respond to stimulation of the respiratory center.

The chemoreceptors are less sensitive still to carbon dioxide. Schmidt, Dumke and Dripps (54) illustrate this by isolating the carotid body, in the dog, from the general blood stream but its nerve supply is retained, and then perfused with a solution containing

carbon dioxide. The smallest change in carbon dioxide tension which causes an appreciable change in respiration is around 10 mm. of Hg. When, on the other hand, the reflex is abolished by denervation of the carotid area, a change in carbon dioxide tension of only 3 mm. of Hg. in the blood supplying the respiratory center is sufficient to induce hyperpnea.

These experiments indicate clearly that the chemoreceptors are relatively insensitive to a reduction in oxygen or to a rise in carbon dioxide tension and therefore play a very minor role, if any, in the control of respiration under ordinary physiological conditions. However, in more exacting emergencies the reflex response of the chemoreceptors, especially to anoxia, is of highest importance.

The findings of Davenport et al (18) show that anoxia, in its direct effects on the respiratory center, is both stimulant and depressant, the implication being that the stimulation is slower on onset and reversal than is the depression. The chemoreflex mechanism, on the other hand according to Comroe and Schmidt (15), is highly resistant to anoxia, retaining its viability and continuing to exert its influence upon the center

which other wise would be unresponsive in the body's emergency. These observers look upon the chemoreceptor mechanism as a more primitive type of respiratory control which serves as a last line of defense against respiratory failure -- the ultimum moriens of the respiratory control system.

Hering-Breuer reflexes

The importance of afferent impulses from the lungs in the control of respiration was first pointed out in 1868 by Hering and Breuer (35), who showed that inflation of the lungs arrested inspiration, expiration then ensuing, while deflation inhibited expiration and brought on inspiration. These reflex effects are mediated through the afferent fibers of the pulmonary vagi, for they are abolished after these nerves have been divided. The receptors are sensitive to a stretching force and are thought to be situated in the walls of the alveolar ducts, the most distensible part of the lungs.

In ordinary breathing the inflation reflex alone comes into play, thus checking, at the end of inspiration of the usual duration further distension of the lungs. According to Adrian and Partridge (1), the corresponding deflation reflex does not, apparently, play a part under ordinary circumstances. In order to elicit it,

extreme deflation of the lungs is required, as can be produced in the laboratory by forcible compression of the chest, by colapse of the lungs through the production of a pneumothorax, or by sucking air from the trachea.

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 therespiratory muscles.

(3) Integrity of the motor nerves of these muscles, including the vagosympathetic 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 (35) reflex (in which expansion of the alveoli cause arrest of inspiration and starts expiration, and colapse of the alveoli causes arrest of expiration and starts inspiration.)

(8) Integrity of the carotid and aortic bodies (chemoceptors recording oxygen and carbon dioxide).

The asphyxiated patient, regardless of the cause of asphyxia, will exhibit a variety of symptoms. There are many investigators who have grouped these symptoms into classifications. It is the opinion of the writer that the method used by Flagg (25) in arranging the physical signs and symptoms of asphyxia into three stages is inclusive and readily understood.

Stage of depression

The patient is in a stuporus or semiconscious state. He may be aroused by severe stimulation, slapping, etc. but usually relapses. 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 is completely unconscious and cannot be aroused. 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; extermities relaxed; circulation sluggish or with a bounding pulse of anoxemia; pharyngeal reflexes active.

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.

A clinical discussion of anoxia

The effectiveness of artificial respiration is governed by many different factors as well as the actual manuver performed. If these conditions produce anoxic anoxia, histotoxic anoxia, anemic anoxia or stagnant anoxia and are not recognized and corrected at once, any attempt to revive the patient is futile. These factors and method of corrective treatment are considered in the following discussion.

Anoxic anoxia

Obstruction by foreign matter

Solid or fluid matter in the mouth, pharynx or any part of the respiratory tree due to any of the obvious causes should be aspirated as soon as possible or the solid material be reguritated or removed, surgically if necessary.

Obstruction by glottic spasm

The glottic reflex is a profound reflex controlled by relatively powerful muscles and is excited by contact with fluid or solid material therefore, normally, food and drink will not pass into the trachea. If material should pass, coughing and severe spasm results. If the glottic reflex is impaired because of general anesthesia, barbiturates, local anesthesia, central paralysis, etc., the physician should protect the patient from aspiration of any foreign material by suction. It is much better, of course, to protect the mechanism so it will function if possible.

Obstruction due to loss of muscle tone

The patency of the airways depends upon the tone of the muscles of the tongue, faucial pillars and muscles of the soft palate. If this muscle tone is lost by cerebral hemorrhage, or injury, certain diseases, drugs, anesthesia (local or general) or by asphyxia from its many causes patency of the airways is lost. In such cases the indicated treatment is to correct the position of all unconscious patients, regardless of the cause of unconsciousness, whether surgical or medical, and to provide free ventilation.

Obstruction by paralysis of muscles of respiration

Certain diseases or injuries of central origin cause an easily recognized paralysis. 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 envolvement, which limits the normal respiratory excursions, not infrequently results in obstructive complications from the accumulation of 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 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 inspiration may be readily observed in any neck operation. This regular intermittent relief of the right heart pressure, when suddenly eliminated by a stoppage

of the respiration, throws a heavy burden upon the right heart. So called controlled respiration in anesthesia, shallow respiration or excessive carbon dioxide absorbtion, 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-carbon dioxide tension will gradually become atelectatic. If the nitrogen is displaced by an anesthetic gas, this atelectasis is very rapid and may proceed to complete collapse within a few minutes. The indication here is to 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 is commonly seen in high altitude flying or on mountain tops. The air in high altitudes is still in the correct proportion but is reduced in amount per unit

volume. Also at or below sea level gas dilution may occur because the atmospheric oxygen is replaced by another gas, e.g., smoke from fires or from gases in a mine. In such situations recognize the oxygen deficiency.

Histotoxic, Anemic and Stagnant Anoxia

A short discussion of tissue fluids is necessary at this time to clarify the next portion of anoxia. Tissue cells and the fluids with which they are bathed constitute the internal enviroment. The normal condition of this enviroment 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. Viewed physiologically, the purpose of dehydration of the tissues is to maintain plasma volume. When urine volume drops to one pint in 24 hours, uremia, delirium, coma, blood viscidity, low blood pressure and anoxia result. Tissue fluid balance is maintained when the urine output is between two and three pints a day.

Dehydration is characterized by sunken facies, thirst, weakness, dry skin, and low urine volume. It occurs in all delayed care of accidents where there is tissue

destruction, such as burns and crushing inuries. In these cases there is little blood lost but plasma and serum escape. Dehydration induces stagnant anoxia which sets in a vicious circle, i.e., hemo concentration, oxygen, carbon dioxide, tissue tension imbalance, capillary stagnation, right-heart embarassment, myocardial weakness and anoxia. This situation is to be avoided at all costs.

The first and best method of administering water to these patients is by the mouth. Often by allowing the patient to sip the water from a glass through a tube is better than to allow him to drink it naturally. The next method of choice by some is per rectum, using half strength normal saline. Lastly, the intravenous method may be the one of choice using 500 cc. of normal slaine alternating with 500 cc. of five per cent glucose not exceeding one pint every two hours. If given too rapidly the resulting hemodilution will cause pulmonary edema. Along with the administration of water, oxygen is indicated with rebreathing of the expired air. The elimination of anoxia will raise the blood pressure and help reestablish oxygen-carbon dioxide tissue balance.

When there is no tissue dehydration and moderate hemorrhage is complicated by psychic shock and pain, the condition

is referred to as primary shock. This condition is characterized by a small, rapid-running pulse, low blood pressure, cold perspiration and loss of muscle tone. When the primary shock lasts for longer than an hour with a blood pressure of 100 or less, the condition is referred to as secondary shock. The passage from primary to secondary shock is usually credited to hemorrhage. It may be precipitated however, by progressive anoxia. Where such primary shock is further complicatedby histotoxic anoxia from gassing, from anemic anoxia, or from red cell loss, the indication for oxygen therapy is urgent.

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. It can be added that by increasing the oxygen, hemoglobin and plasma oxygen tension, oxygen therapy may tide over a temporary large loss of red blood cells. This is only temporary. The oxygen-carrying capacity of the

blood must be returned to normal as soon as possible.

In summary, before beginning the discussion of the methods of administering effectively oxygen and carbon dioxide, the indicated treatments for anoxic, histotixic, anemic and stagnant anoxia are mentioned as follows:

(1) Remove foreign matter from airway.

(2) Eliminate or overcome the effects of 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) Mantain 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.

METHODS OF ADMINISTERING OXYGEN AND CARBON DIOXIDE

Oxygen Therapy

During the 18th century oxygen began to be used for every kind of disease known to man, resulting in its being discredited as a therapeutic agent.

Oxygen remained a questionable therapeutic agent until World War I when Haldane, Barcroft, Hunt, Dutton (126) and others used it successfully in the treatment of soldiers suffering from exposure to poisonous gases. Since then much scientific investigation has been put forth and it has gained considerably in its use as a valuable therapeutic agent.

Whenever the oxygen pressure falls below normal it is important that oxygen be administered immediately. Its early employment will often lessen the length of time necessary for it to be administered, especially in cases where the throat or any part of it may have been damaged. At the same time, it is essential that the percentage of oxygen administered should be very high or even pure -- depending on the case.

Cohen (14) states, "Oxygen to be efficacious must be used freely, frequently, fearlessly, and almost constantly, nor must its use be postponed until the patient is moribund for it will not revive the dead."

Since many unsatisfactory results have been due to errors in administration, many are becoming interested in improving the efficiency and technique of oxygen therapy.

It would be incongruous to the purpose of this paper to discuss, in detail, the physiological and chemical aspects of carbon dioxide and oxygen. It is only the aim of the writer to dwell on the methods and effectiveness of their practical administration.

Cameron (12) administered oxygen by mouth in the form of a souffle, made by bubbling oxygen through water to which foam extract has been added. It has also been used intravenously. However, the general method of administration has been by inhalation. One of the first methods being that of an inverted funnel to which was attached a tube and a bubble bottle containing water. This method was not successful because it was too wasteful of oxygen, unpleasant to wear, and it increased the concentration of oxygen in the inspired air by about two per cent.

The method of using an intranasal catheter was utilized during the first World War. Its chief advantage was that several patients could be treated at once from

one tank of oxygen. Since then, the catheter method has been widely employed.

The nasal catheter contains multiple perforations at the tip, and may be lubricated with a little vaseline or by the patient's saliva. It should be passed along the floor of one nostril to a depth equal to an inch less than the distance between the alae of the nose and the tragus 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, for if it is inserted before the gas is turned on, the sudden flow of gas may cause gagging. A flow of 2-3 liters per minute should be used to begin the administration; this may then be stepped up to 4-5 liters per minute. If no 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 chief objection to the administration of oxygen by this method has been the occurrence of drying of the mucous membrane of the nasopharynx as a result of insufficient moisture in the oxygen. This causes the patient discomfort and results in infection of the naso-

and oro-pharynx. In order to meet this objection, a type of apparatus has been used, in which conventional types of reducing valves and gauges to indicate tank pressure and oxygen flow are utilized. Attached to this set of valves, by means of a metal tube, is a closed cylinder, the wall of which is an alloy, which, due to its porous texture, is p rmeable to gases. This cylinder is immersed in water contained in an ordinary jar of quart size. The tightly fitting lid contains an outlet through which oxygen is delivered to the patient. The essential feature being the porous wall through which water must pass. Thus, the oxygen is finely devided exposing more surface to the water for absorbtion as the bubbles rise to the surface. As a result oxygen, carrying enough water, is delivered to the patient.

It is felt that this apparatus has many advantages. It is simple in construction and operation. There is adequate humidification of the oxygen. The parts of the apparatus that may be broken are easily and quickly replaced.

Inhalation from oro-pharynx by oral tube

According to Flagg (25), Carl Connell of Roosevelt Hospital introduced the Connell tube. This tube,

moulded to the contour of the average oro-pharynx, provides relief to the obstruction so commonly caused by the tongue falling back into the pharynx. The relief so obtained completely eliminates the emergency use of mouth props and tongue forceps. Today, these tubes are made of hard rubber and are quite efficient if properly used. Care must be exercised by the user lest he damage the mucus membrane in the throat.

This pharyngeal tube cannot be employed unless the patient is sufficiently relaxed to permit separation of the teeth by the operator's fingers, and unless the pharyngeal reflex is sufficiently in abeyance to permit retention of thetube. If difficulty is met, the nasal catheter should be used.

Transpharyngeal Insufflation

To quote Dr. Joseph O'Dwer (47): "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 (42) of the Rockefeller Institute devised his resuscitation apparatus, he recognized the principles pointed out by O'Dwyer (47). 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.

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."

If the principles pointed out by O'Dwyer (47) and accepted by Meltzer (42) 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.

In Flagg's (25) 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, 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 according to medical literature.

The outstanding objection to the method of endotracheal insufflation is the lack of 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.

The technique must definitely 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. Butwhen 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 (47) for intubation in diphtheria; and intubation under direct vision by the use of the laryngoscope, in accordance with the technique popularized by Chevalier Jackson (38).

Mouth to mouth insufflation

According to Corylios (17) this form of resuscitation is very efficient and rapidly effective. Physiologically this is a sound procedure because it allows the immediate administration of the correct mixture of oxygen and carbon dioxide, under the correct conditions of temperature, moisture, pressure, at proper intervals and lastly, it is always with the administrator. Moreover, it takes advantage of the powerful Hering-Breuer reflex. The most serious drawback, common to all resuscitation procedures, being that the airways are not always patent. The administrator or the patient may become infected with a contagious disease, such as, tuberculosis, influenza, etc.

The oxygen tent

An oxygen tent of standard make, operated under proper supervision furnishes an excellent and economical means of administering oxygen. However, there are certain fundamental principles which should be recognized.

The motor should be mechanically efficient and readily controllable. There should be sufficient space so that the patient will not feel restricted. Means should be provided for variation of the rate of circulation and height of temperature in the tent. Adequate means for controlling humidity and for controlling the concentration of carbon dioxide should be provided. In those tents providing ice for cooling, and soda lime for absorption of carbon dioxide should be insulated containers for these agents. Soda lime does not absorb efficiently unless it is warm. Lastly, and most important, the tent must be thoroughly checked during use to make sure that adequate concentrations of oxygen are being administered. Trained personel should operate oxygen tents.

Carbon Dioxide Therapy

The administration of carbon dioxide has been used favorably in some cases but has fallen into disrepute in others. After the information has been sifted, both experimental and clinical, there is every indication that carbon dioxide has a definite place in the resuscitation of the individual in certan cases.

In considering the experimental work, Shaw, Behnke and

Messer (56) have shown that in the anesthetized dog which rebreathes in a closed system without provision for the absorption of carbon dioxide but with provision for the maintenance of normal oxygen pressure, the following facts may be set forth: (1) That percentages of carbon dioxide as high as twenty may stimulate temporarily the intact respiratory center in the dog, (2) that percentages of carbon dioxide above 30 are associated with gradual depression and ending in respiratory failure, and (3) the initiation of respiration following failure, provided that the carbon dioxide pressure is allowed to fall rapidly, occurs only with little reflex stimulation associated with two or three manual compressions of the thorax. Later tests show that a concentration of carbon dioxide in the alveolar air, maintained at 10 percent, stimulated respiration for a period of one and one half to 3 hours without depressing the blood pressure.

Murphy and Drinker (44) have pointed out the beneficial effects of 10 per cent carbon dioxide in contrast with 5 per cent carbon dioxide in the treatment of carbon monoxide poisoning. Behnke (6) demonstrates that carbon dioxide is an effective physiological agent in bringing about not only increased pulmonary ventilation

but also augmentation of cerebral blood flow. However, Eastman, Dunn and Kreiselman (22) demonstrated that resusciation utilizing tank oxygen promoted a better recovery of blood pressure and respiration than the employment of a mixture of 90 per cent oxygen and 10 per cent carbon dioxide. This is due to the initial stimulation of respiration brought about by the oxygen lack producing, essentially, an acid effect on the chemoreceptors of the carotid body according to Von Euler (62).

In conclusion, carbon dioxide is an effective physiological agent in bringing aboutnnot only increased pulmonary ventilation but also augmentation of cerebral blood flow.

MECHANICAL AND MANUAL ARTIFICIAL RESPIRATION

Mechanical methods of artificial respiration

In 1924 Professor Thunberg (21) invented a mechanical appliance for artificial respiration known as the barospirator. In the description by Drinker (21) the barospirator functions as a cylinder completely to enclose the patient. Ventilation is accomplished by pressure changes of 55 millimeters of mercury at the rate of 25 strokes per minute. These pressure pulsations serve to move airrin and out of the lungs without the necessity for volume changes in the pulmonary air spaces.

If the barospirator is now modified so as to envelope the patient's body with the head protruding through a rubber collar to the atmosphere, then the principle underlying the operation of the Drinker and the Emerson respirators can be illustrated. For the maintenance of artificial respiration cyclic negative pressure of 10 to 15 millimeters of mercury around the chest permits normal atmospheric pressure to expand the lungs without muscular effort. Expiration occurs passively when during the cycle the pressure is allowed to return to normal in the cylinder.

The E and J apparatus (17) for mechanical resuscitation

This apparatus consists of two parts, one of which

acts as a simple inhalator and the other as a resuscitator. The latter is a mechanical devise composed of a number of valves which are activated by the pressure of the usual oxygen or carbon dioxide in oxygen mixture delivered in tanks under a pressure of two thousand pounds. When a positive pressure of 14 millimeters of mercury is built up in the mask of this apparatus the valves are automatically reversed and a suction of -9 millimeters of mercury is produced which is again followed by a positive pressure and so it continues. The principle objectionsbeing: (1) The intrapulmonary and intrapleural pressures produced by this apparatus during inspiration and expiration are positive during inspiration and negative during expriation. That is opposite to those of normal respiration. (2) According to Wilson (67) negative pressure when applied to lungs, which are essentially capillary beds filled with blood, may lead to congestion and to an increased tendency to edema and hemorrhage into the alvoeli. Barach (3) has shown that there is a definite advantage to the positive pressure in the prevention and treatment of pulmonary edema. Actual proof of these problems remains for further experimenters to determine.

Drinker method

In 1929 Drinker and Shaw (21) developed a new apparatus for artificial respiration in which the patient is completely enclosed in a cylindrical, sheet-metal tank sealed at one end. At the other end is a flat lid, to which is attached a rubber collar. The patient's head and part of his neck protrude through the collar, the head lying upon an adjustable support outside the tank. Since the patient is thus enclosed in an air-tight chamber, the pressure around the body may be alternately raised and lowered while the head always remains at atmospheric pressure. When the pressure in the tank is lowered, the chest expands and air rushes into the lungs. When the pressure is raised, the chest is compressed and air is forced out of the lungs. By this method movement of the chest is induced in such a manner as to simulate the natural respiratory movements. To determine the correct pressure for optimum comfort and to induce breathing and the like in the patient, there is a set of readily adjustable valves which can adjust the pressure to the required level. There is no certain way of adjusting the level to suit any patient except by trial and error.

A thermostat for controlling the temperature and humidity

of the air in the tank is present. This is because the body heat of the patient, especially when the pumps are not running, raises the temperature within to an uncomfortable degree. This is overcome by running the air through a can of ice which serves as a dehumidifier and as a cooling agent. Exhaust air is blown into the room and fresh air enters the tank all of the time.

The jacket model of Sahlin and the Bragg-Paul pulsator

Two other types of apparatus should be mentioned; (1) the jacket model of Sahlin (7) which operates like the Drinker machine but is applied only to the chest, and (2) the Bragg-Paul (25) pulsator which consists of a hollow elastic bandage placed around the chest. The bandage, when inflated by an electrically diven bellows, compresses the chest, which returns to the mid-position by virtue of its own elasticity during the intervals between the compressions.

Manual methods of artificial respiration

In comparatively recent years, manual artificial respiration has made new advances. These improvements tend toward greater efficiency with more consideration for the patient. It is to be noted, however, that the progression of new ideas came about only as rapidly as the concepts of physiology became established.

Prone pressure Schafer method

The Schafer (53) method is the most widely used in English speaking countries today. A decription of this standard technique is as follows:

(1) Lay the patient on his belly, one arm extended directly overhead, the other arm bent at the elbow and with the face turned outward and resting on the 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 a position which is comfortable and allows freedom of movement. 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 rib, with the thumb and fingers in a natural position and the tips of the fingers just out of sight.

(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. Elbows are to be kept straight. This operation should take about two seconds.

(4) Now immediately swing backward so as to remove the pressure completely.

(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.

In this method all efforts are directed towards producing expiration. Inspiration, the life saving phase, occurs automatically or not at all. Therefore, before beginning the application of the treatment, the patient is put in the best inspiratory position, with both arms extended so that there is increased tension on the chest muscles. The tempo should be slow enough, that is, the intervals between compression should be long enough to allow for an expansive recoil of the chest.

Supine position (Viswanathan procedure)

While experimenting on warm cadavers and on patients under deep anesthesia, Viswanathan (61) found that by pulling on the lower costal margins in an upward and outward direction with hooked fingers of both hands under the costal margins of the ribs, the chest can be made to assume the raised position of inspiration. Release of the pull, coupled with gentle pressure on the lower ribs with the palms of the hands, is quite sufficient to bring about expiration.

If the patient is on a bed or on an operating table lying on his back, the operator stands at the head of the table and places the palms of the hands on the lower part of the chest, the middle fingers being on the anterior axillary lines. The fingers are hooked round the lower costal margins on both sides. The pull is made in an upward and outward direction, thereby raising the anterior portions of the ribs as well as widening the lower costal arches. After a steady pull for three seconds the tension is released and the chest is gently pressed downward and inward, while the extended fingers exercise the same pressure on the abdomen. Doing this about twelve or fifteen times a minute brings about a respiratory exchange of over "7000" c.cm. of air. In addition, the expansion of the chest and outward stretching of the diaphragm, owing to the widening of the lower costal arches, helps the venous return of the blood. If the patient is in the face down position, the operator stands as before, but his hands are brought up underneath the chest and the lower costal arches are pulled upwards and outwards.

The Nielsen procedure (46)

This procedure has withstood much criticism and is considered to be of great value in Belgium where it

is used extensively. The patient is placed in the prone position with his head resting on his folded arms. The operator kneels at the patient's head and pushes firmly down and towards the patient's feet. He gradually increases the weight applied on his outspread hands by slowly applying his body weight to his stiffly extended arms. Next, the operator relieves the pressure, hooks his hands under the patient's folded arm near the axilla and lifts outward and upward, thus, stretching the pectorals and increasing the expansion of the chest.

This method depends for its efficacy, first on the force exerted by the operator in forcing air out of the lungs, and second on the elastic recoil of the body in drawing in air. Expiration is thus a positive event, but inspiration depends on the degree of elasticity possessed by the patient. Field experience shows that it is quite efficient but it cannot be denied that it is not too efficacious in the worst degrees of asphyxia.

Drinker and Shaw (21) suggest a change to the method which could easily be adopted. This consists merely of the operator using Schafer's (53) prone pressure technique and then when an other person arrives on

the scene to advise him to use the arm lift in addition. There need be no change in rhythm and no disturbance of the regular rules for giving prone pressure artificial respiration. This promotes the flow of air into the lungs and so increases ventilation. Inhalation can be instituted as easily as with the prone method alone.

Eve's method

Upon examining the different methods of artificial respiration and noticing the various changes that have taken place up to the latest approved methods, it is notable that artificial respiration advances only as rapidly as the physician understands; (1) the basic physiology of respiration, (2) the needs of the attendant for apparatus, (3) protecting the patient from over zealous administration of artificial respiration and a quick, efficient method. Dr. Frank Eve (24) of Hull, England recognized the necessity of reviving the suffocated and water-logged patient without causing injury and discomfort to the patient. He devised the rocking chair method which is standard for the British Navy.

Surgeon Commander Gibbons (24) of the Navy realized that the Schafer (53) technique was inadequate even

in skilled hands. The reason being, no doubt, that the chest had lost it's customary tone, therefore being flaccid, and this, in turn, leading to further asphyxia.. Because of the loss of elasticity no additional areation of the lungs occurs resulting in the nerve cells suffering further embarrassment. The loss of tone caused the diaphragm to rise into complete expiration being pulled up by the elastic lungs. Schafer's method would then be completely useless because it depends upon muscle elasticity and tone. If Silvestor's (57) method is used, greater areation is obtained but fluids or solids will still obstruct the pathway in the face-upward position and harm may be done. Aspiration of the materials could lead to lung abcess, pneumonia, etc. Therefore, both methods have great disadvantages. Breathing machines have many disadvantages which make them undesirable for the administration of artificial respiration. First and foremost disadvantage is that the machine is not present at the time of the injury and another method must be used immediately.

Because of the disadvantages which have been briefly discussed above, Dr. Eve devised his method. Schafer's method is promptly used till the rocking method (Eve's)

can be set up. This arrangement is accomplished by having a stretcher, plank or door or some such flat object that can be easily handled, placed on a rack or with two men holding each side, at the middle of the plank, till more reliable support is obtained. In so doing, the respiration can be promptly carried out. The patient, artificial respiration being maintained, is placed on the plank and tilted to an angle of 45 degrees to allow the fluids and foreign material to run out. After a few seconds the table is tilted 30 degrees each way at the rate of ten times a minute. This will be enough to ventilate the lungs about 450 cc. with each tilt.

According to Dr. Eve, resuscitation is actually a trinity of ventilation, circulation and warmth directed to supplying warm blood, oxygenated by moving lungs, to the microscopic nerve cells which maintain respiration and tone. These cells are easily paralyzed by cold and quickly revived by warmth. The ventilation is accomplished by the weight of the abdominal contents pushing and pulling the diaphragm up and down like a piston.

Restoration of the circulation by other methods is not so effective. The Silvester (57) method produces a

pressure change of 26 cm. of water in the stopped heart. The Schafer (53) method only 4 cm. and is increased to 22 by the Nielsen (46) procedure. These pressure changes could possibly help to restart the heart. The problem still remains of supply an adequate amount of blood to the lungs for oxygenation. In Eve's rocking chair method by tilting the patient whose heart is stopped, or nearly so, into the head-down position, the pressure of about four feet of blood will slam shut the aortic valve and the blood will have no option but to travel through the oxygen starved heart muscle. This should be invaluable aid in starting a stopped heart or reviving a feeble one. Similarly, the nerve cells of the brain and breathing center will receive blood rhythmically at a hydrostatic pressure which should be about normal. Theveins of the extended arms will acquire a reservoir of blood ready to fill the heart again when the legs are tilted down. Every drowning person is in some stage of shock, and in shock the venous side of the heart is starved of blood. In this case, the head-down tilt will fill it and encourage it to beat and pump. Hence, in rocking, gravity propels blood alternately in arteries and veins. Reflux is prevented by the valves in the veins

and heart. Experiments show that blood flow to the brain can be kept by alternating head-up and head-down positions.

Due to exposure and often being wet, the patient will lose much of the body warmth in a short time. To attempt to restore this warmth, it is advisable to place hot water bags over the shoulders to revive the chilled nerve cells, especially those controlling the respiration. As a further stimulation to the respiratory center carbon dioxide, mixed with 5 per cent oxygen, is a good stimulant.

CONCLUSION

There are certain obvious criteia necessary for an adequate means of performing artificial respiration. These are: (1) The method must give sufficient ventilation for a normal subject at rest. (2) The method must stimulate the heart and circulation so that the blood may transport oxygen to the tissues and yet be harmless in itself. (3) The method must be carried out with ease and rapidity in attaining results.

The mechanical methods do accomplish the above requirements. However, there are definite disadvantages which are difficult for mechanical therapy to overcome. These are: (1) the machine is not always present at the time of need. (2) It requires skilled operators and adjustments. (3) It is apt to impede the attempts of the patient to breathe by going into a series of reversals. (4) There is danger of over ventilation. And, (5) inspiration produced by negative pressure may cause lung injury.

Mechanical methods should not be too harshly discredited, however, because if properly controlled and employed they are a great boon to mankind in cases of partial paralysis of the respiratory center, for example, poliomyelitis. The Drinker respirator is

very successful in maintaing the patient's ventilation and often complete recovery of the patient results with its use.

The manual methods have disadvantages that are of a less severe nature. These are: (1) injury may occur from over enthusiasm and rough handling by the operator resulting in broken ribs, ruptured spleen, etc. (2) ignorance of the method or of a method to be used can result in little or no air exchange. (3) it may become arduous to the operator.

For immediate, safe artificial respiration, using the Schafer (53) method until arrangements for the use of Eve's (24) method are accomplished, is the method of choice where it is adaptable.

Eve's method produces a pulmonary ventilation of 580 cc. of air for each tilt of 45 degrees. It promotes recovery of the heart with the resulting improvement of pulmonary circulation; it increases the circulation to the central nervous system which prevents anoxial injury to the nerve cells in the severely asphyxiated individual; it is not fatiguing for the operator if the conditions require long, continued application; it is harmless to the patient; it is foolproof; the patient can be made comfortable and, additional treatment can easily be instituted.

In conclusion, the thoughts of Henderson (32) are worthy of consideration: ..."There are many modes of dying that bring men to their ends; but only one final common cause. Whether the brain is destroyed, or the lungs blocked, or the heart stopped, death finally occurs in but one way only. When breathing and the heart come to a standstill, the oxygen to the body ceases. Unless a man is burned alive, the tissues of his body always die of asphyxia."

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