ARTIFICIAL RESPIRATION AND RESPIRATORS¹

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IT WAS REALIZED in the eighteenth century that persons who had apparently drowned might be resuscitated by the "Elijah" method of mouth-to-mouth insufflation, but it was not until the nineteenth century that artificial respiration was thought to have any important part in the treatment of these people. It is perhaps rather more surprising that until about ten years ago the administration of oxygen was considered to be much more important than the achievement of ventilatory adequacy in a wide variety of diseases which cause partial respiratory failure.

Advances in respiratory physiology and pathology have now brought our knowledge of artificial respiration and the indications for its use to a reasonably sophisticated level, and technical improvements in apparatus have matched these advances. In fact, workers in the technical field have been outstanding in their ingenuity in devising machinery for artificial respiration, and the choice would appear to be difficult. Only a consideration of the basic principles will enable us to make a choice. Many of the intermittent positive pressure machines available work very well within their limited field of assistance to respiration during surgical anaesthesia, but in no way could most of them be considered suitable for the prolonged treatment of respiratory failure.

The indications for artificial respiration can be broadly divided into two categories: (a) where disease or injury, or drugs, prevent adequate work from being done on ventilation (for example, myasthenia gravis, head injury, and barbiturate poisoning), (b) where, for some reason, the work required to produce adequate ventilation is beyond the capacity of the individual (for example, in crush injury of the chest, massive infiltration of lung tissue, or postoperative mechanical impairment of respiration).

The respirator chosen has to be adaptable to the conditions present in a very wide variety of conditions.

It is accepted that, from the physiological viewpoint, there is no difference between the external suck applied to the chest wall by a cabinet type of respirator and the force exerted on the airway by an intermittent positive pressure respirator. They both achieve ventilation by raising the intrathoracic pressure. Because of their greater convenience in nursing and general management and greater versatility, intermittent positive pressure machines are now more commonly used. Cuirass respirators, rocking beds, and electrophrenic respirators are still used, but they are not within the scope of this paper.

When positive pressure is applied throughout the respiratory cycle to the airway of normal persons, the intrathoracic pressure rises, the venous return to the heart is impaired and the cardiac output and blood pressure fall. Within a few heart beats vasoconstriction occurs, the venous pressure rises to restore right

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atrial filling, and the cardiac output and blood pressure return to normal. A normal person can so compensate for sustained mean pressures of about 20 cm. water. Cournand *et al.* (1) showed that the significant factor in the reduction of cardiac output during artificial respiration was the mean tracheal pressure and that any rise in this would produce these effects.

In the face of a mean tracheal pressure above atmospheric pressure, any factor which prevents an effective increase in venous pressure will prevent compensation so that cardiac output and blood pressure will fall. In the application of artificial respiration, most commonly we meet with the effects of prolonged hypoxia and carbon-dioxide retention, surgical shock with hypovolaemia and with the central failure of vasomotion, as for example in bulbar poliomyelitis. The first principle is, therefore, that the mean tracheal pressure should be kept to a reasonable minimum so that little vasoconstrictor response is demanded by the therapy itself.

When a positive pressure is applied to the airway the stretching force applied to the alveoli depends upon several factors. If the pressure is applied for sufficient time, the alveolar pressure will become equal to the airway pressure and gas flow into the lungs will cease. The effect of this applied pressure will depend upon the state of the thorax at the time. If, for example, the subject has full muscle power he could prevent any distension of his alveoli, even with very high applied pressures, by opposing the force, but if he assisted the applied pressure by inspiring synchronously with it, his alveoli could be grossly overdistended by the same pressure. If no active respiration is present, the distension of the alveoli will depend upon the over-all resistance to airflow into the lungs, elastic and non-elastic, and upon the applied pressure and its duration of application. This would be the case where the alveolar ventilation is uniform throughout the lung. In bronchospasm it is unreasonable to assume that all parts of the lung are equally affected by the spasm so that, if the applied pressure is raised to maintain ventilation in the face of bronchospasm, some alveoli served by the least constricted bronchi may be grossly overdistended while others are poorly ventilated. In reduced compliance of the lung, increasing the airway pressure to maintain ventilation is theoretically only safe if the compliance is reduced uniformly throughout the lung. It is better to increase the time allowed for gas flow into the lungs than to increase the pressure. In order to maintain the mean tracheal pressure then within reasonable limits, this increased duration of pressure application can be compensated for by the application of negative pressure during some part of the expiratory phase. Negative pressure, and by this is meant a reduction of pressure below atmospheric pressure, applied to the airway, however, by making the diameter of the bronchioles smaller, increases the resistance to the inflow of gas and it may prevent efficient ventilation of some parts of the lungs because some of the bronchioles may reach their critical closing pressure. It is then theoretically better to apply a small negative pressure for a longer duration than to apply a high negative pressure for a short time.

In normal persons breathing spontaneously, regular respiration is frequently interrupted by a deep inspiration or sigh. Physiologically this appears to be due to the need to open up areas of lung which have tended to collapse and become

underventilated during resting quiet respiration. If artificial respiration is adjusted so that the respiratory rate is normal for the individual and provides the calculated correct alveolar minute volume, then this mechanism is not simulated. Every anaesthetist is aware of the good clinical practice of fully inflating the lungs from time to time during the course of anaesthesia. During artificial respiration it is, from the viewpoint of alveolar collapse, better to slow the rate of respiration and to increase the volume for a given alveolar ventilation so that more of the lung is ventilated. The respiratory rate and tidal volume in health or in disease represents the combination of these in which the individual does least work on respiration. In artificial respiration this necessity can be ignored because the machine is doing the work. For example, if a patient required an alveolar ventilation of 5 L./min. and with a tracheostomy had a dead space of 50 cc., then he would be ventilated adequately by 20 respirations of 300 cc./min., but it would be much better from the point of view of alveolar collapse to ventilate him with 10 respirations of 550 cc./min. The machine would be doing more work but this would be of no importance, nor does it make any subjective difference.

Both pressure-cycled and volume-cycled respirators are available so that first of all we have to decide which type is most desirable. The volume-cycled machines all have a pressure blow-off valve which limits the maximum pressure to be applied to the lungs and, when used properly this has to be set to blow off at the pressure just above that which will push in the pre-set volume. They are in practice used as pressure-cycled machines. Unless the maximum blow-off pressure can be controlled in this way, this type of machine can be dangerous. The purely pressure-cycled machines are much more controllable and are to be preferred. The argument generally raised in favour of the volume-cycled type is that one can set the minute volume and that this is a comfort to the physician in control. The estimated alveolar ventilation calculated from one of the nomograms evolved for this purpose presupposes that both ventilation and perfusion of the lung are normal and in patients requiring artificial ventilation this may be very far from being the case. In artificial respiration it is better to judge the ventilation by its results than by any form of prescience. The greater versatility of pressure pattern possible with pressure-cycled machines is a decided advantage in the varying circumstances likely to be met with in practice.

In a pressure-cycled machine there are several parameters which require precise control for efficient artificial respiration in all circumstances.

- (1) The rate of inflow of gas from the respirator should be controllable and variable between 5 L. and at least 60 L./min. This allows between 80 cc and 1,000 cc. of gas to be pushed into the lungs in one second.

- (2) The respirator should cycle at any pre-set pressure up to 40 cm. water pressure and should be infinitely variable within this range. The tidal volume and the duration of inspiration are, therefore, controlled by setting the flow rate and the cycling pressure at any given over-all thoracic compliance.

- (3) The expiratory pause should be controllable in duration from 0.5 sec. to infinity.

- (4) The transition from inspiration to expiration should be very abrupt and should occur as soon as the peak cycling pressure is reached.

(5) In the expiratory phase the airway should be open to the atmosphere through a low resistance pathway so that the tracheal pressure rapidly returns to atmospheric pressure. This is most important in the achievement of low mean intratracheal pressures.

(6) A negative pressure phase is desirable and it should be infinitely variable between 0 and 20 cm. water pressure. It must however have sufficient flow capacity to augment the expiratory gas flow. During normal expiration, gas flows from the lungs at rates of from 10 L./min. upwards depending upon the tidal volume and expiratory period. A respirator must, therefore, either function by allowing a free flow of gas out of the lungs and then applying a moderate negative pressure or be capable of sucking air out of the lungs at varying rates of flow up to 30 L./min. Of the two possible designs, the machine which first allows a free flow of gas out of the lungs and then applies a moderate negative pressure for the remainder of the expiratory period is to be preferred because it ensures that normal expiratory flow rates can always be allowed passively without having recourse to a negative pressure phase to achieve them.

(7) The machine should be capable of being triggered by the patient so that a small inspiratory effort will produce a full respiratory cycle. This feature should be adjustable so that minimal effort is required by the patient and yet not trigger on movement of the tubing. Good criteria are that it should trigger on the exertion of 1.0 cm. water pressure by the patient with a volume shift of about 10 cc. of gas. This feature will allow the machine to be used as an assister to respiration where some respiratory activity is still present.

(8) The machine should have efficient humidification of the gases going to the patient.

These considerations should allow us to choose a good machine from among the many available. The general design should also be taken into account because they have to be rugged and dependable. Finally, it should be possible to adapt the machine to anaesthetic use so that everyone in the anaesthetic department will become familiar with its use.

The necessity for artificial respiration is generally obvious, but in many patients with low respiratory reserve it can be difficult to decide. Where arterial blood $p\text{CO}_2$, pH, and oxygen saturation estimations are available, the decision cannot be in doubt for long, but one cannot obtain these in all centres. In general, the patients are dyspnoeic and cyanosed. Consciousness is disturbed and, depending upon their vasomotor state, there may be obvious signs of carbon dioxide retention such as vasodilatation, sweating and rise of pulse rate and blood pressure. The presence of bronchial secretions and the inability to cough them up is universal. It is a reasonable procedure when in doubt to intubate the patient under local anaesthesia and to impose artificial ventilation and, within a reasonable time to be sure that you are or are not improving the situation.

REFERENCES

1. Cournand, A., Motley, H. L., Werko, L., & Richards, D. W. Physiological Effects of Intermittent Positive Pressure Breathing on the Cardiac Output of Man. *Am J Physiol* 152, 162 (1948).