MANAGEMENT OF THE APNOEIC PATIENT WITH SPECIAL REFERENCE TO BULBAR POLIOMYELITIS AND TETANUS

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Apnoea is compatible with life only so long as artificial respiration can be maintained. By modern methods, artificial respiration can be continued for an unlimited time, but its prolonged application may result in serious biochemical and circulatory disturbances if its form and volume are incorrect.

Apnoea may result from many causes. It may be due to obstruction in the airway, to changes in the brain produced by injury, disease or drugs, or to impairment of the bellows action of the chest. In some conditions—such as combined bulbar and spinal poliomyelitis—all three types may co-exist.

Apnoea in the patient undergoing abdominal surgery is a common condition. It does not normally outlast the operation, and its management is simple, for it requires no more than the correct application of the apparatus already available to the anaesthetist.

This apparatus, however, has a defect which has long been recognized, and which is shared by most types of mechanical respirator. It brings about an inversion of the normal pressure relationships within the thorax, so that introthoracic pressure increases during inspiration and decreases during expiration; and it raises the mean intrathoracic pressure. This impedes venous return and puts the cardiovascular system at a disadvantage.

In fit patients subjected to this form of artificial respiration for only a few hours the harmful effect cannot easily be demonstrated; but Maloney and others (1953) have shown that in patients whose cardiovascular systems are already impaired, elevation of the mean intrathoracic pressure may result in serious falls in blood pressure and in cardiac output. There is no doubt that a system of imposed respiration which alternates positive with negative pressure is to be preferred to one which alternates positive with zero pressure; and machines which will produce the preferred form of respiration are being designed.

Methods of Artificial Respiration

Many of the methods of artificial respiration—whether manual or mechanical—and much of our understanding of it, derive from techniques evolved in connection with the use of muscle relaxants during surgery under general anaesthesia. It is recognized that, even for short periods, the volume of ventilation which is imposed on the surgical patient should not be very different from what he requires. If it is too great, recovery of tone in the respiratory muscles as the relaxant wears off may be masked by apnoea due to hyperventilation; if it is too small, carbon dioxide may build up to a toxic concentration (Pask, 1955) which can itself result in apnoea. When artificial respiration has to be carried on for longer periods, inequality between the volume of ventilation and the patient's needs will tend to alter the reaction of the blood, and throw a serious strain on the body's compensating power. It is for medical rather than surgical patients that prolonged artificial respiration is likely to be required, and of these the sufferer from poliomyelitis is the most important. If the virus is confined to the anterior horn cells below the medulla the bellows action of the chest may be gravely impaired, but the larynx will still be able to perform its function as the watchdog of the lungs; and the management of the case will consist mainly of the provision of an adequate minute volume of ventilation. This can be achieved by a cabinet-type respirator, and patients have been kept alive and in good biochemical balance for years by the use of these machines. Cabinet respirators, however, have several disadvantages. They are cumbersome and expensive, they impede close observation, they make nursing and physiotherapy difficult, and they are incompatible with a tracheotomy. An improved type of cabinet respirator (Macrae, 1954) has a perspex dome which can enclose the patient's head. A change-over valve causes the pump to apply positive pressure to the dome instead of negative pressure to the cabinet, which can then

be opened without interrupting the patient's ventilation, and access to the body can be provided. In addition, this cabinet respirator allows the lateral or the prone position to be adopted.

Another means of maintaining ventilation without denying access to the body of the patient is provided by the cuirass type of respirator. This, in its simplest form, does no more than apply rhythmic compression to the patient's chest, as with Schäfer's method for the apparently drowned, leaving the elastic recoil of the chest wall to provide for inspiration. Cuirass respirators alone cannot usually ventilate a completely paralyzed patient sufficiently to prevent carbon dioxide accumulation, and they have to be augmented by some other method (Engström, 1954).

The achievement of active contraction of the diaphragm by repeated stimulation of the phrenic nerve is a good idea, and the electrophrenic respirator, which allows unrestricted access to the patient, has been developed by Sarnoff (1950). It produces a form of respiration in which the natural intrathoracic pressure relationships are preserved. But though respiration can be successfully maintained in some cases by this means, the spread of the stimulus to the muscles of the neck and shoulder, and the difficulty of placing and maintaining the stimulating electrode in the correct position, limit its general application.

Protection of the Lungs

When respiratory insufficiency is due only to spinal poliomyelitis the main problem is simply the provision of adequate ventilation, and any type of respirator that will do this can be used, but when the pathological process involves the brain stem as well as the spinal cord, the picture becomes much more complicated. Bulbar poliomyelitis, tetanus and certain other diseases of the nervous system may lead to disorganization of the vagal nuclei. The larynx can then no longer act as 'the watchdog of the lungs,' and the respiratory tract is in grave danger of invasion. Most of the patients who die of bulbar poliomyelitis or of tetanus succumb to respiratory invasion. As soon as it is recognized, therefore, that the vagus nerve—which, through its receptors, nuclei and effectors, marshals and coordinates the vital defences of the lungs—is out of action, prompt steps must be taken. These steps are directed towards one end: the isolation of the respiratory tract from contamination by foreign matter or bacteria. Let it be said at once that tracheotomy is the most important of these steps, and it should be undertaken, without delay, whenever vagal control of the larynx and the pharynx becomes ineffective. Properly managed, a tracheotomy performs two vital functions. It provides a pathway and a

barrier: a free pathway for respiratory gases, and an impassable barrier to invaders. It also provides a ready means for the removal, by suction, of the bronchial secretions which, with the normal expulsive mechanism out of action, can rapidly accumulate.

Having substituted a pathway other than the useless glottis for the respiratory gases, we must provide a force other than the paralyzed respiratory muscles to make them flow. A cabinet respirator can provide this force, but it is a dangerous instrument in a case of bulbar poliomyelitis. It is incompatible with a tracheotomy because of the proximity of the stoma to the neck seal. Its undeviating pattern of forced inspiration will cause it to suck into the lungs the accumulating secretions which the paralyzed pharynx cannot swallow and which the paralyzed larynx cannot shut out. Moreover, the supine position which conventional models impose on the patient is the very one which favours the accumulation of these secretions, and the immobility and incarceration which they involve prevents effective treatment of the infected atelectasis which inevitably follows. The flow of respiratory gases can be as efficiently, and much more safely, secured by intermittent positive pressure. There are many devices achieving this type of artificial respiration. In some, the energy is supplied by hand power, rhythmic compression of a rubber bag, in some by a pump worked by an electric motor, and in some from the kinetic energy of compressed air or other gases. A description of positive pressure respirators is beyond the scope of this paper, but the reader is referred to Mushin and Rendell-Baker's review article (1954).

By use of a machine for providing intermittent positive pressure, coupled to a tracheotomy tube which is made to fit closely into the trachea, efficient ventilation can be restored to the paralyzed patient, and at the same time the protection of his lungs can be assured. The development of this technique, and the appreciation of its importance, due largely to the work of Lassen (1954) and of Ibsen (1954) in Denmark, have greatly increased the chances of survival of patients suffering from bulbar poliomyelitis and from tetanus.

Bulbar Poliomyelitis

In bulbar poliomyelitis with rapid onset the patient may die from respiratory insufficiency before he reaches hospital. Some of these patients could be saved by early recognition and prompt action by the medical attendant. He should suspect bulbar involvement if the patient is obviously ill and febrile, with pain and stiffness in the neck; if there is weakness of the facial muscles, difficulty

in speaking and swallowing, or a history of regurgitation of fluids through the nose (Russell, 1955). If—as is so often the case—the patient is supine, inspiration of secretions pooling in the pharynx can rapidly result in massive atelectasis or death from suffocation. If acid stomach contents are inhaled, necrosis of the lung is a likely complication. These dangers can be obviated by a simple manoeuvre: turning the patient on his face and raising the foot of the bed. In this attitude he should be transported to hospital. If the virus has affected the spinal cord as well as the bulbar centres, artificial respiration during the journey may be necessary. This can be simply applied through a face mask by one of the manual methods, such as the Oxford bellows (Macintosh, 1953), or by the Blease inflator.

Though the principles to be observed in the treatment of bulbar poliomyelitis are clear enough, the management of a case presents many practical difficulties. It is generally agreed that one of the first things to do is a tracheotomy, but there is some difference of opinion on the best type of anaesthesia for this operation. Local anaesthesia is sometimes used, but general anaesthesia by inhalation is to be preferred. The coughing which usually occurs when the trachea is opened and the tube is being inserted is most distressing to a patient with poliomyelitis, and so is the bronchial suction which should follow it. Moreover, inhalation anaesthesia facilitates the respiratory support which may be needed. The presence of a tracheotomy tube for days or weeks may give rise to the fear that tracheal stenosis may follow its removal. This must be accepted, however, as the lesser risk, and intubation has been maintained for long periods without any complication. Some people advocate the insertion of a cut-down cuffed Magill tube into the trachea through the tracheotomy opening. This is certainly the most effective means of providing a barrier against secretions or ingested material penetrating into the bronchi; but if the cuff remains inflated for more than a few hours it may damage the tracheal mucosa. It should therefore be deflated at intervals.

An alternative to tracheotomy is the insertion of a cuffed endotracheal tube through the glottis under direct vision. This should be carried out under general anaesthesia, and at the same time the pharynx, trachea and main bronchi may be cleared of secretions by suction and a feeding tube can be passed into the stomach. The period immediately after intubation—whether transglottic or transtracheal—is often an anxious one. The rapid removal of accumulated carbon dioxide reduces the respiratory drive, and bronchial spasm, tenacious secretions and commencing pulmonary oedema may complicate the picture. The

suction catheter and oxygen may have to be passed into the respiratory passages in rapid alternation. Later, the tracheal tube may become partly blocked by inspissated secretions. It should be cleaned frequently and replaced every two or three days. The tube should be connected to the positive pressure respirator by an adaptor of the Cobb type, so that the suction catheter can be readily introduced into it without disconnecting it from the respirator.

So long as the pharynx and larynx remain disorganized, it is dangerous to give the patient anything by mouth. Nourishment and hydration must be maintained intravenously or by stomach tube. The level of plasma non protein nitrogen and the specific gravity of the urine are useful guides to this (Crampton Smith *et al.*, 1954). In the early stages the patient may require hourly feeding through the stomach tube.

When ventilation must be maintained by mechanical means, the sensitive control normally exerted on it by the respiratory centre is lost, and over-ventilation or under-ventilation can all too easily be produced. It is not so much the supply of oxygen which is thereby upset as the removal of carbon dioxide. Under-oxygenation is unlikely if oxygen enriched mixtures are being used, and will in any case be revealed by cyanosis; and over-oxygenation, at the pressures normally used, does no harm. But deviation from the normal tension of carbon dioxide in the blood can easily be produced if the degree of ventilation is not properly suited to the patient's needs, and can be dangerous. Under-ventilation results in respiratory acidosis. This in turn throws a strain on the renal compensatory mechanism, and if it is prolonged pathological changes in the kidney, and later in the vascular system, may result. Except when gas exchange is impeded, by areas of collapsed lung, however, over-ventilation is the commoner fault. The resulting respiratory alkalosis tends to produce a rise in plasma pH. This displaces the haemoglobin dissociation curve, and impedes oxygen uptake in the tissues (Astrup, 1954). It is easy to tell that a patient under artificial respiration is doing badly, but it is not always easy to say whether he is being under- or over-ventilated. It is useful to remember that over-ventilation often produces tetany. The urinary pH is also a useful guide (Crampton Smith *et al.*, 1954) and should be regularly measured. Direct measurements of plasma pH, and of total and combined CO_2 , are also very valuable (Astrup, 1954). Measurement of alveolar CO_2 gives an even more direct indication; and some of the gas analyzers now becoming available (Woolmer, 1953) enable this to be done relatively easily.

The respiratory gas may be air or air enriched

with oxygen, and it should be humidified (Marshall and Spalding, 1953). Dry gases result in viscid, tenacious secretions which predispose to atelectasis. For the same reason, atropine should be avoided.

Frequent bronchial aspiration should be performed. This is best done by introducing a rubber suction catheter through the tracheotomy tube, and passing it into each main bronchus in turn. Additional protection to the lungs should be given by determined physiotherapy, with percussion of the chest and frequent changes in position (though with the head-down tilt maintained), and by prophylactic penicillin from the outset of the disease.

Tetanus

It used to be believed that if the first signs of tetanus occurred within a week of the primary infection, or if the disease progressed rapidly from premonitory signs to convulsions, no treatment would be of any avail. That was before the muscle relaxants had become generally available. The establishment of a new line of treatment, based on the use of relaxants and on the principles already set forth for the treatment of bulbar poliomyelitis, brings new hope (*Lancet*, 1954).

Patients who die of tetanus, like those who die of bulbar poliomyelitis, succumb from respiratory insufficiency, though toxæmia and exhaustion also play a part. The tetanus toxin acts on the spinal cord to abolish synaptic inhibition (Brooks *et al.*, 1955) and on the brain stem and cranial nerve nuclei to impair coordination (Baker, 1942). The spastic respiratory muscles can no longer bring about rhythmic respiration, and, as with bulbar poliomyelitis, the uncoordinated larynx can no longer protect the lungs against invasion. The flaccid respiratory muscles of the poliomyelitic patient make effective artificial respiration easy: the spastic muscles of the tetanic patient make it impossible. If rigidity could be replaced by flaccidity, it should be as easy to ventilate the tetanic as the poliomyelitic patient. The muscle relaxants enable this to be done. They convert the tetanic patient into one closely akin to the patient with bulbar poliomyelitis; and such a patient has nowadays as good a chance of living as, a few years ago, he had of dying. It should be said, however, that this hopeful view is not shared by those who believe that tetanus toxin fixed in the body will not relax its grip on the nervous system, and cannot be opposed by circulatory antitoxin, in however high a titre.

Though the idea of the rational treatment of severe tetanus is now as clear as is that of bulbar poliomyelitis, its practical application is a great deal more complicated. To the difficulties of the

treatment of poliomyelitis, already formidable enough, are added those of maintaining the correct concentration of relaxant at the synapses or motor end-plates, and of combating toxæmia. These difficulties have been overcome in a number of recorded cases, and in one (Bjørneboe *et al.*, 1954) treatment with continuous infusion of relaxants and artificial respiration was kept up for no less than seventeen days, with a successful result.

There is not yet complete agreement about practical details in the treatment of tetanus. Some authorities (Belfrage, 1947; Torrens *et al.*, 1948; Godman and Adriani, 1949) favour the use of mephenesin as the relaxant, arguing that its supposed site of action (the synapses in the spinal cord) make it more appropriate, as an antagonist to tetanus, than drugs which act more peripherally. Mephenesin, however, cannot give the degree of relaxation which is required if the patient's respiratory function is to be completely taken over, and it is therefore suitable only for mild cases. Also, its capacity for causing haemolysis and kidney damage (Hewer and Woolmer, 1947; Pugh and Enderby, 1947) should not be forgotten. Of those who favour the peripherally acting drugs, many (Evans and Whiting, 1948; Bjørneboe *et al.*, 1954) prefer the long acting ones, holding that a condition lasting days or weeks requires a drug whose action lasts for hours or days. They use curare (sometimes in a vehicle intended to delay absorption and prolong its action), or gallamine (Vander Bergen and Buckely, 1952). Others (Woolmer and Cates, 1954; Shackleton, 1954) prefer a short acting drug, and they use succinylcholine by continuous infusion. They believe that in tetanus the degree of muscle relaxation required to oppose spasm and prevent convulsions varies from minute to minute. Muscle tone (which is an expression of the balance between acetylcholine and cholinesterase at the muscle end-plates) depends on many factors. Among these are sensory stimuli and tetanus toxin, which increase it, and sedatives and antitoxin, which decrease it. Since these factors vary widely from time to time the hypertonus cannot be expected to remain constant, and a varying amount of relaxant will be required to oppose it. A slow acting drug like curare cannot follow these fluctuations: a quick acting one like succinylcholine can. If arrangements are made for the rate of infusion to be accurately controlled over a wide range—and this is best done by an electrically driven syringe to be described elsewhere—fluctuations in tone can be closely followed. This facilitates the avoidance of spasms on the one hand and of unnecessary degrees of paralysis on the other, and at the same time enables the progress of the disease to be closely followed.

The old treatment of tetanus consisted simply

in the giving of antitoxin and in the use of drugs which had a depressant effect on the central nervous system. No other means of controlling spasms or convulsions was known. Since the central nervous system is already under attack by the toxin, it would be unwise to add to its difficulties, by adding depressants to the toxin, if there were any other way of reducing muscle spasm. Now that there is another way, the use of depressants in tetanus is dying out, albeit slowly. Some workers (Shackleton, 1954), nevertheless, advocate that light general anaesthesia with nitrous oxide and oxygen should be maintained throughout the period when relaxants are needed, and have had successful results. They argue that light nitrous oxide does not act as a depressant to the vital medullary centres in the same way as morphine, for instance; and that the maintenance of light general anaesthesia reduces, to a fixed level, the otherwise varying amount of sensory and psychic stimulation which makes control so difficult.

The maintenance of general anaesthesia, however—even if it does render control less critical—adds another burden to the already very exacting task of correctly managing a case of severe tetanus with spasticity, convulsions, vagal incoordination, respiratory insufficiency and toxæmia.

Patients who recover from tetanus and from purely bulbar poliomyelitis usually recover abso-

lutely, so that though the treatment of these conditions is exacting and fraught with difficulty, it can be very rewarding.

BIBLIOGRAPHY

- ANNOTATION (1954), *Lancet*, 2, 175.
 ASTRUP, P., et al. (1954), *Brit. med. J.*, 1, 780.
 BAKER, A. B. (1942), *J. Neuropath. & Exp. Neurol.*, 1, 394.
 BELFRAGE, D. (1947), *Lancet*, 2, 889.
 BJORNEBOE, M., et al. (1954), *Danish med. bull.*, 1, 129.
 BROOKS, V. B., et al. (1955), *Nature*, 175, 120.
 CRAMPTON SMITH, A., et al., (1954), *Lancet*, 1, 939.
 ENGSTRÖM, C.-G. (1954), *Brit. med. J.*, 2, 666.
 EVANS, P. R. C., and WHITING, R. J. (1948), *Brit. med. J.*, 2, 1022.
 GODMAN, H. E., and ADRIANI, J. (1949), *J. Amer. med. Assn.*, 141, 11.
 HEWER, T. F., and WOOLMER, R. F. (1947), *Lancet*, 2, 909.
 IBSEN, B. (1954), *Proc. R. Soc. Med.*, 47, 72.
 LASSEN, H. C. A. (1953), *Lancet*, 1, 37.
 MACINTOSH, R. R. (1953), *Brit. med. J.*, 2, 202.
 MACRAE, J., et al. (1954), *Lancet*, 1, 704.
 MALONEY, J. V., et al. (1953), *J. Amer. med. Assn.*, 152, 212.
 MARSHALL, J., and SPALDING, J. M. K. (1953), *Lancet*, 2, 1022.
 MUSHIN, W. W., and RENDELL-BAKER, L. (1954), *Brit. J. Anaesth.*, 26, 131.
 PASK, A. E. (1955), *Anaesthesia*, 10, 4.
 PUGH, J. I., and ENDERBY, G. E. H. (1947), *Lancet*, 2, 387.
 RUSSELL, W. RITCHIE (1955), *Brit. med. J.*, 1, 98.
 SARNOFF, S. J., et al. (1950), *J. Amer. med. Assn.*, 143, 1383.
 SHACKLETON, P. (1954), *Lancet*, 2, 155.
 TORRENS, J. A., et al. (1948), *Lancet*, 2, 807.
 VAN BERGEN, F. H., and BUCKLEY, J. J. (1952), *Anesthesiology*, 6, 599.
 WOOLMER, R. F. (1953), *Brit. J. Anaesth.*, 25, 315.
 WOOLMER, R. F., and CATES, J. E. (1952), *Lancet*, 2, 808.

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- HARRIS, A. S., BISTENI, A., RUSSELL, R. A., BRIGHAM, J. C., and FIRESTONE, J. E. (1954), *Science*, 119, 200.
 JOPPICH, G., and SCHAFER, H. (1955), *Dtsch. med. Wschr.*, 80, 73.
 JUNG, R., DOUPE, J., and CARMICHAEL, E. A. (1937), *Brain*, 60, 28.
 KHALIL, H. H., and MACKEITH, R. C. (1954), *Brit. med. J.*, 11, 734.
 LABORIT, H., and HUGENARD, P. (1951), *J. Chir., Paris*, 67, 73.
 LASSNER, J. (1954), *Cah. d'Anesthesiol.*, 2, 269.
 LEWIS, F. J., and TAUFIC, M. (1953), *Surgery*, 33, 52.

- MELROSE, D. G. (1953), *Brit. med. J.*, 11, 57.
 MILLER, J. A., and MILLER, F. S., (1954), *Surgery*, 36, 916.
 ROSS, D. N. (1954a), *Lancet*, 1, 1108.
 ROSS, D. N. (1954b), *Guy's Hosp. Rep.*, 103, 97, 116.
 SAYERS, G., and SAYERS, M. A. (1949), *Ann. N.Y. Acad. Sci.*, 50, 522.
 SIMPSON, D. (1954), Personal communication.
 STOKES, T. L., and GIBBON, J. H., JUN. (1950), *Surg. Gynec. Obstet.*, 91, 138.
 SWAN, H., ZEAVIN, I., HOLMES, J. H., and MONTGOMERY, V. (1953), *Ann. Surg.*, 138, 360.
 WYNN, V. (1954), *Lancet*, 11, 575.