THE EMERGENCY TREATMENT AND TRANSPORT OF PATIENTS WITH RESPIRATORY FAILURE

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Acute respiratory failure may occur in a number of conditions and may vary in severity from a minor degree of impairment of respiratory exchange to complete cessation of breathing. The most obvious and serious concomitant of such failure is anoxia, with all of its secondary manifestations on the cardiovascular and central nervous systems. Less often thought of, but yet of as great importance, is the consequent inadequate elimination of CO_2, which may produce effects as disastrous as those due to lack of oxygen.

The conditions most commonly associated with acute respiratory insufficiency are the following:

1. Poliomyelitis involving the muscles of respiration and/or the muscles of the pharynx and larynx.
2. Polyneuritis, e.g. infective and that due to porphyria.
3. Head injuries.
4. Injuries of the upper cervical region of the spinal cord.
5. Poisoning, e.g. from narcotics, insecticides of the anticholinesterase group, shellfish which have ingested poisonous plankton (see below).
6. Respiratory paralysis deliberately induced as a therapeutic measure, e.g. in cases of tetanus.

The occurrence of recent cases[1,2] of fatal paralytic *mussel poisoning* has re-awakened interest in this uncommon condition and has shown that here is another cause of acute respiratory failure which the practitioner may be called upon to treat. Mussels and clams may become poisonous when they have strained the dinoflagellate plankton *Gonyaulax* out of the water; the poison has been shown to be a neuromuscular toxin with effects on both skeletal muscle (curare-like) and peripheral nerves.[3] In addition the poison is said to have a

depressant action on the myocardium and its conducting tissues.[4] As there is no known antidote to this poison the treatment remains purely symptomatic and with this in mind it might be of help to have a clear regime to follow in the cases showing signs of respiratory failure.

It is important from the outset to bear in mind that in patients apparently suffering from respiratory failure there are two equally vital functions to be considered. These are (1) the mechanism responsible for the provision of pulmonary ventilation—i.e. the diaphragm and the intercostal muscles, and (2) the mechanism responsible for the protection of the respiratory tract from foreign material—the pharyngeal and laryngeal mechanisms. Either one or both of these may be affected, according to the nature and extent of the condition responsible, thus giving rise to the three broad categories described by Crampton Smith, Spalding and Russell,[5] each of which requires different management.

I. *Patients with paralysis or weakness of the muscles of ventilation only*

Here there is involvement of neither the larynx nor the pharynx, so that swallowing is intact and the patient can prevent the entry of foreign material into the respiratory tract. Respiratory exchange however, is inadequate, and a varying degree of suboxygenation will be present, depending on the degree of paralysis. Retention of CO_2 is also likely and again its extent depends upon the degree of underventilation. The patient in fact, is suffering from both lack of oxygen and accumulation of carbon dioxide and it is necessary to correct both of these.

If the paralysis is incomplete the administration of an oxygen enriched atmosphere will help to correct the anoxic element, but will do nothing to correct the CO_2 retention; it is important to realize that CO_2 itself, when present to excess, may eventually cause complete respiratory failure through its depressant effect on the respiratory centre.

In cases of more extensive paralysis, even the administration of pure oxygen will not correct the anoxia and it is quite futile to administer oxygen if the patient's lungs are not adequately ventilated with it. Only too often does one see the patient with markedly impaired respiratory ventilation being 'given oxygen', which ineffectively fans the face and does little to correct the oxygen deficiency of the haemoglobin and nothing to correct the CO_2 accumulation which is taxing the pH-regulating mechanism to its utmost. These patients need *active mechanical ventilation* to make up for the inadequacy of their own diaphragms and intercostal muscles and, if such ventilation is adequate, there should be no need even to administer oxygen—air is quite good enough and the CO_2 retention will be corrected. Additional oxygen should only become necessary when pulmonary collapse or pneumonia or interference with diffusion across the alveolar membranes complicates the picture.

II. *Patients with involvement of the protective mechanism only*

Cases of bulbar palsy are frequently treated incorrectly, with disastrous results. The muscles of ventilation are acting adequately, but there is disorganization of the pharyngeal and laryngeal mechanisms, so that saliva or anything else that may be in the pharynx tends to enter the trachea. This may lead to a variety of complications, from atelectasis due to plugs of mucus to severe acute pulmonary oedema due to regurgitated acid gastric juice, but severe pulmonary infection is the most frequent result and often the ultimate cause of death. Because the vocal cords cannot close, an effective cough is not possible and severe respiratory distress rapidly ensues because insufficient alveolar surface is available for normal gas exchange.

Treatment here is aimed firstly at prevention, by early accurate assessment of the condition. Pooling of secretions in the pharynx or trachea is usually accompanied by a characteristic rattling sound on respiration. Movement of the thoracic cage will be good. Phonation may be impaired, the voice being weak or merely a whisper. Attempts to swallow show that movement of the palate and pharyngeal muscles is weak or absent and dribbling of saliva may be evident.

At this stage, if little or no foreign material has entered the respiratory tract, treatment consists of providing protection only. This is adequately secured by posture alone, it being best to place the patient in a head-down position, lying on his side or prone but never on his back, for this will cause pooling of secretions in the posterior pharynx with eventual spill into the trachea. No active ventilatory assistance need be given, as the diaphragm and intercostals will be working well. If, on the other hand, the patient has already inhaled a fair amount of foreign matter when first seen, there will, in addition to the signs mentioned above, be some degree of respiratory distress with clinical signs of pulmonary collapse. It is this stage that is very frequently managed wrongly. What is needed is again immediately to put the patient in a posture which will enable gravity to protect him from further inhalation of foreign matter, and active tracheal and bronchial toilet by suction, either *via* a sterile rubber catheter passed into the trachea or by bronchoscopy. Too often one sees such patients being nursed in the sitting position, which seems to be the traditional position in which to place the dyspnoeic patient. This can only lead to further entry of foreign matter into the trachea and bronchi.

Oxygen administration is often indicated in these cases and is of value if posture and active suction and physiotherapy fail to clear obstructed bronchi or where simple mechanical obstruction has already progressed to bronchopneumonia.

III. *Patients with impairment of both ventilatory and protective components*

In this group, it is obvious that both active ventilation of the lungs and protection of the respiratory tract are necessary —in fact a combination of the treatments outlined above. The means of providing protection, however, may need to be modified to prevent the active ventilation from forcing secretions into the respiratory tract against gravity in spite of posture.

MANAGEMENT

It is the active ventilatory assistance required in groups I and III above that is most frequently neglected in respiratory emergencies, and often no attempt is made to supply this assistance until complete cessation of all respiratory efforts on the part of the patient. By this time myocardial failure is imminent and anoxia may have been present for a sufficient length of time to cause permanent damage to the central nervous system.

In the main, patients in category I require active ventilation, and this should be started too early rather than too late. A rapid clinical assessment of ventilatory adequacy can be made quite easily, without the use of spirometric apparatus,

by simply asking the patient to take as deep a breath as possible and count out loud for as long as possible. The average normal adult can count up to 40 or 50 with a single full breath. If this ability to count is found to be reduced to 20 or 30, active ventilatory assistance will most probably be necessary if a further degree of respiratory failure due simply

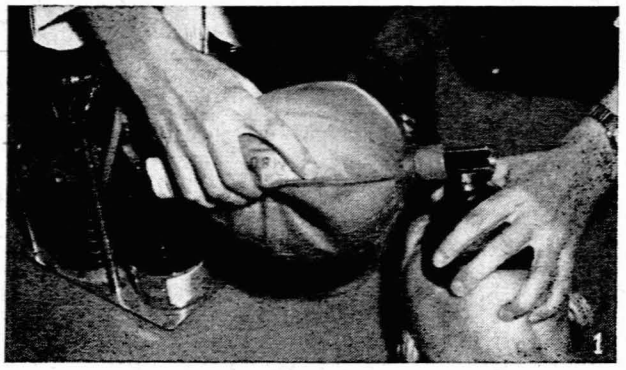

Fig. 1. The self-inflating Ruben resuscitator in use.

to retention of CO_2 is to be avoided. A count of below 20 indicates that assistance is already overdue and is therefore most urgently needed. Adequate ventilation should be maintained by whatever method is available and artificial respiration by Schafer's method or Eve's rocking method may be necessary in the first instance if paralysis is complete. Such methods however, soon become tiring, and a hand respirator or even an anaesthetic machine should be used if available or if paralysis is incomplete. Various pieces of equipment are obtainable for providing manual artificial

Fig. 2. The Oxford inflating bellows (left) and Kreiselmann resuscitator.

respiration, this being far more effective than the first-aid methods. Good examples of these are the Ruben resuscitator, the Kreiselmann resuscitator and the Oxford inflating bellows (Figs. 1 and 2). It does not matter what method is used, so long as it is efficient and sustained.

Patients in category II require no more than correct posture and tracheo-bronchial toilet as has been mentioned.

Patients in category III present a combined problem in that they require both protection and ventilation. They should be placed in a head-down position at once and maintained thus with the application of first-aid methods of artificial respiration until more adequate measures can be taken. As soon as possible, a cuffed endotracheal tube should be passed and the cuff inflated. This can very often be done without any anaesthesia or with topical anaesthesia alone. Once in position with the cuff inflated, such a tube at once isolates the trachea and bronchi from the mouth and pharynx. It provides excellent protection against the entry of foreign matter into the lungs and will enable a suction catheter to be passed. Active ventilation can be carried out *via* this tube by any method available, preferably an efficient manual inflator such as the Oxford bellows or Ruben resuscitator. Thus

Fig. 3. Foot-operated portable suction machine in use.

protected and ventilated the patient can be safely transported to a centre where prolonged respiratory therapy can be carried out. If a cuffed endotracheal tube is used, it is advisable to deflate the cuff for 1 minute at intervals of 4-6 hours. This is in order to prevent ischaemic damage to the tracheal mucosa from pressure. Before releasing the cuff it is important to tip the patient head down and aspirate all saliva etc. from the pharynx (Fig. 3). Every effort should be made to reach a properly equipped centre for prolonged respiratory treatment within a period of 8 hours so that a cuffed tracheotomy tube may be substituted. If the journey is to be one of less than 100 miles then it may be made by road, but for greater distances it is far more practical to travel by air. During the journey ventilation still has to be maintained and the services of a trained anaesthetist should be obtained for this task if possible.

In the above, 3 clear-cut categories have been dealt with. It must be realized however that varying degrees of overlap will occur and that a patient in say, category I may rapidly or insidiously change to category III. Continued vigilance is therefore necessary on this score.

SUMMARY

1. The causes of respiratory failure, with or without, paralysis of the protective mechanisms of the pharynx and larynx, are discussed; together with the recognition of these conditions.

2. Stress is laid on the fact that accumulation of CO_2 is as dangerous as lack of oxygen, and that, without adequate ventilation of the lungs, neither can be properly corrected.

3. The emergency treatment of patients with these conditions, and their transport to a centre fully equipped for the necessary long-term management is outlined.

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