Discussion

Dollfus, P. (France), asked Dr. Agerholm whether, amongst the 15 deaths, she had a case of acute respiratory exhaustion during sleep. He remembered a polio patient in Copenhagen with a complete paralysis of the upper limbs, who went home and led a nearly completely normal life and suddenly one night she died. She obviously had not represented any symptoms of hypoventilation or respiratory distress, and actually to his knowledge this happened in Copenhagen twice.

Agerholm, M., said she had not seen this at all, and of course this series did not apply to people who were weaned completely—but she had not done so in other circumstances. Two of the people were found dead in the morning while their curasses were working. Both were elderly patients, and she thought the limit of life for these people was probably somewhere around the 50 mark. In neither of them did a P.M. show any reason for the death, apart from the fact that one wondered why they were alive for other reasons.

Frankel, H. L. (England), would like to agree with Dr. Agerholm on the need to hyperventilate patients on respirators. They had constantly exceeded the norms in the published books by about 100 per cent. over the last few years and the patients seemed to do very well on it. He would agree that while on a respirator the patient was always right but he wondered whether Dr. Agerholm had anything to say about whether the patient was also always right when he or she had been weaned from the respirator.

Agerholm, M., replied that, yes, she did still feel the patient was always right except in the sense that he should have rather more aid than perhaps he was trying to manage to do without. She once got herself into a very difficult situation by criticizing at a physiotherapy meeting the way they weaned a patient on 'Emergency Ward 10' in which patients were encouraged and forced to breathe rather longer than they wanted to! But she did very much believe that the patient was the best judge, given the guidance, and that one must never press people to do without apparatus unless one's slant was that one should insist on their using apparatus when they did not want to. This she thought one occasionally had to do because patients got a bit bloody-minded when hypoventilated.

RESPIRATORY AND METABOLIC MANAGEMENT IN ACUTE TETRAPLEGIA

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THE management of the respiratory problems of acute traumatic tetraplegia has been discussed in previous papers. This paper presents a modification and extension of the same subject and, in addition, attempts to correlate the respiratory and metabolic facets of management into a pattern of treatment which will maintain adequate alveolar ventilation and nutrition and a normal fluid and electrolyte balance.

The maintenance of adequate alveolar ventilation may be threatened by:

1. Increasing paralysis of the muscles of respiration.
2. The supervention of either pulmonary consolidation or collapse. These complications are related to the aspiration of vomitus, the inability to deliver sputum to the mouth, and the inspissation of sputum.
3. Pulmonary oedema, due to overhydration of the patient.

Arising from a knowledge of these potential complications, a respiratory regime is presented, which includes the timely use of tracheostomy and intermittent position pressure respiration. In the management of a patient with a tracheostomy, the importance of the humidification of the inspired air, and frequent and adequate bronchial suction is stressed.

The effect of body temperature on the oxygen requirements of the tetraplegic patient is also discussed, and the suggestion is made that there may be advantages in nursing such a patient at 34° to 35°C.

The patient with acute traumatic tetraplegia may show one or more of a number of disturbances of function, each of which may stress homeostatic mechanisms for fluid and electrolyte. In particular, shock, paralytic ileus, vomiting and acute renal failure are common and gastric aspiration and intravenous infusion are almost always required. Among the more common disturbances of fluid, electrolyte and acid-base balance which must be prevented are:

1. Respiratory acidosis.
2. Alkalosis associated with loss of gastric hydrochloric acid.
3. Acidosis associated with loss of small intestinal secretions. This loss may be external or into dilated gut.
4. Acidosis associated with starvation—this may be made worse by co-existing hypoventilation or acute renal failure.
5. Hypokalaemia.

The final pattern which may require correction by intravenous therapy could be the resultant of any combination or all of these disturbances.

An important point in the management is the intravenous therapy. The fluid volumes have been designed to keep the patient on a fluid balance with a urinary output of 2 to 3 litres per day. If, for any reason, the urinary volumes are significantly more or less than this, the volume of intravenous fluid is adjusted accordingly.

The electrolyte composition of this regime is directed to covering the continuing gastro-intestinal losses, to impose a minimal electrolyte load on the kidney and to achieve and maintain normal extracellular and intracellular patterns and normal buffer concentrations. A relatively high potassium is required, as the recovery of smooth muscle function will be delayed unless these requirements are met. In many patients, extra bicarbonate ion is necessary to correct the buffer system, and it is our practice to meet these requirements by adding 20 or 40 ml. 8.7 per cent. (saturated) sodium bicarbonate to a litre of intravenous fluid.

Calories are provided in this regime in four forms: fructose, alcohol, sorbitol, and synthetic amino acids. Fructose is preferred to glucose because it is much less likely, in solutions more concentrated than 5 per cent., to cause thrombophlebitis. It has the further advantage of entering the Embden-Meyerhof glycolytic pathway more readily than glucose and of having a metabolism at least partly independent of insulin.

Alcohol is used as a further caloric source because it acts as a very mild sedative and also because it has a peripheral local venodilator effect, thereby further minimising any tendency to thrombophlebitis with concentrated hexose solution.

Sorbitol is used in the synthetic amino-acid solutions because it is relatively non-reactive *in vitro* and therefore does not react with the amino acids in solution as would occur with a more reactive sugar such as glucose or fructose.

These three substances are all employed to meet the body's energy requirements and to spare body protein.

Insulin is used to facilitate carbohydrate metabolism but more importantly to promote potassium and amino-acid uptake by cells. It is quite probable that in many cases endogenous insulin would be adequate to facilitate potassium and amino-acid uptake.

Discussion

Benassy, J. (France), said that there was a great difference between tetraplegia at C5 and C6 levels; on the higher lesions, patients could neither cough nor laugh nor whistle, and for them tracheostomy, though very often necessary, was dangerous because they could not cough by themselves. Another problem was that in tetraplegia above C7, the sweating centres were destroyed. The patients could not sweat and therefore were subject to hyperthermia. One could cure it now by refrigerating them, but before this treatment was introduced hyperthermia was very dangerous and ten years ago nearly all patients died. The danger was greatest in the first stage of the tetraplegia, when the muscles of the abdomen were completely flaccid, and during the first stage the vital capacity was often not more than $\frac{1}{2}$ to 1 litre. When the muscles of the abdomen, which were the normal expiratory muscles, recovered some spasticity, the power of expiration was much higher and could go up to $3\frac{1}{2}$ litres. With the difficulty of expiration came the terrible risk of sudden death caused by embolism. Last year they had had three threatened with sudden death who could fortunately be cured by mouth-to-mouth breathing and massage of the heart. A fourth case occurred unfortunately during the night and the patient died. The respiratory problem was the most dangerous and nearly the only one for high cervical lesions and occurred especially when there was an additional thoracic trauma which unfortunately was rather often.

Rossier, A. (Switzerland), asked Dr. Cheshire whether he had seen in his quadriplegics also cases who did not have hyperthermia, as was suggested by Dr. Benassy, but hypothermia. He had had some cases who were running 35° instead of hyperthermia.

Cheshire, D. J. E., said that this question of body temperature was one that interested him a great deal at the moment and it was one that he had thought might be a red herring to drag in at the moment. Following the hyperthermic death that occurred during a Melbourne heat-wave he had pressed to get his acute ward air-conditioned, and when the ward was maintained at a temperature of 70°F . (21°C .) they found that the patient with a complete C5 lesion nursed without blankets stabilised at a body temperature of 34° – 35°C ., and the thing he was paying attention to at the moment was what was the effect of reducing the oxygen consumption of a tetraplegic by approximately 20 per cent. by permitting him to cool from a body temperature of 37°C . to a body temperature of 34°C .

Meinecke, F. W. (Germany), said they had seen last year a patient with a high lesion of C5. A few days afterwards he had become extensively hyperthermic and they cooled him, and when they stopped treatment he had a bulbar disturbance. Fortunately this status improved in the last months, but the rest was still in *status quo*. Had Dr. Cheshire cases like this, because they would like to know whether the hyperthermia or the hypothermia was the cause of this bulbar paralysis?

Cheshire, D., said he had never seen such a case but he had seen now partly by accident and partly by intention probably 25 acute complete tetraplegics who had been maintained at body temperature of 34° to 35°C . without any such complications. He would think probably on general terms, that hyperthermia was much more possibly the cause.

Holmes, G. (England), thought the decision to do a tracheostomy was a formidable one and should not be embarked upon lightly. However, indications were rather well and truly defined but no one seemed to know when to take a tracheostomy tube out. Physical presence of the tube in the trachea caused a certain amount of swelling of the trachea, and

a relatively small decrease in the diameter of the trachea due to oedema, mucosal swelling and so forth caused relatively enormous decrease in the surface area of the trachea. One was frequently seeing respiratory obstruction caused by the physical presence of the tracheostomy tube and the respiratory obstruction cured by taking the tracheostomy tube out. He would like to ask Dr. Cheshire when he took the tube out of his patients?

Cheshire, D., replied that this was a question he found difficult to lay down criteria for. He thought the important ones were that the chest should be clear, that there should be no respiratory infection, that any collapse should have been re-expanded, and that the patient should have a vital capacity which was adequate to maintain him without assisted respiration, and that one felt that the need for both suction and ventilation had passed. This was, he thought, probably the only way one could define it.

Walsh, J. J. (England), agreed with what Dr. Cheshire said about the criterion of having a clear chest and so on before removing a tracheostomy tube. With regard to suction, he had noticed that the continuous presence of a tube made it necessary to carry on suction—in other words, the tube was the cause of the need for a suction. The other point was that he thought he would go further when Dr. Cheshire said 'adequate respiration'. They had a very rough rule at Stoke Mandeville—something over one litre of vital capacity.

Cheshire, D., thought he should clarify what he had been saying. When he was talking about suction he referred particularly to endo-bronchial suction and not to endo-tracheal suction.

Guttmann, L., said the question of adequate respiration was an important point, and one must clearly understand what it meant. First of all, as Dr. Walsh had said, they must test the vital capacity quite systematically, and if it reached about 800 to 1000 and remained at that level then was the time to think of taking out the tube. There was, however, another point which one had to consider. He had said that one had to be satisfied that the vital capacity remained at that level. From this point of view one had to investigate the function of the auxiliary respiratory muscles. Two of the most important ones in the very high cervical lesions were, apart from the diaphragm, the trapezius and the sternocleidomastoid, and this was a point to which they had drawn their attention for many years. One must not underestimate the power of the sternomastoid. It has a very important function—it could lift the chest upwards and by so doing it increased the anterior-posterior diameter, and thus increased the vital capacity. The other point was the question how far the paralysed intercostal muscles could take part in the restoration of the vital capacity and adequate respiration of the tetraplegic once the spinal shock was over and the isolated cord had developed its reflex activity. This was a problem to which he had paid attention for some years and he had carried out with Dr. Silver electromyographic studies on these muscles. There was no doubt that in the stage of spinal shock the intercostals showed hardly any response to even a deep breath. But, once the spinal shock disappeared and automatic function of the isolated cord had started, then the rhythmic movement of the lungs produced by the function of the diaphragm elicited a definite reflexogenic function of the intercostals. The afferent impulses set up by the rhythmic movement of the lungs went into the isolated cord and elicited an appropriate effector response by contraction of these paralysed muscles. In the later stages, the reflex function of the intercostal muscles and abdominal muscles helped to promote better respiration in the tetraplegic, and cases with lesions at C5 develop a good vital capacity which enabled them to take part in the usual daily activities. Therefore, one must be aware of the tremendous compensatory mechanisms one had in the neuromuscular system which could be developed by proper treatment.