Spinal Cord (1998) 36, 446–447

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Letters

A review of the respiratory management of a patient with high level tetraplegia

This clinical case is of great interest since respiratory failure is the major acute cause of death in traumatic tetraplegic patients, and the study of such patients has added to our knowledge of the physiology and the mechanics of breathing.

We are not given enough information about this 24 year old man:

1. Did he have a normal chest X-ray on admission?
2. What were his blood oxygen and CO2 levels on admission?
3. What was his respiratory rate?

Immediately after injury, patients with cervical injuries can compensate for a reduced vital capacity by breathing very rapidly with shallow breaths.

The vital capacity must be measured in all positions that the patient is nursed in, since if one leaf of the diaphragm is paralysed, whilst it may support ventilation when the patient is on their back and be adequate when the paralysed leaf is underneath, when they are turned on the side with the paralysed leaf uppermost, this can lead to respiratory embarrassment and death.[1]

Recording these simple parameters can enable the patient to be effectively monitored without recourse to some of the detailed studies alluded to.

I was concerned with these problems when I first started working in spinal injuries in 1956 and found an acute mortality of 38% in patients with a complete tetraplegia. I initiated various studies to investigate the respiratory problems.[2]

EMG studies have demonstrated in the stage of spinal shock, flaccid paralysis of the intercostal muscles.[3] At that stage it may be possible, though extremely difficult, to monitor by cutaneous electrodes, the function of the diaphragm. Accurate localisation can be obtained by needle studies or wires but there is a risk of producing a pneumothorax which would be fatal in a tetraplegic patient at this stage.

A systemic study of diaphragmatic function by means of an oesophageal electrode has been performed in the later stages[4,5] but the electrode needs anchoring with a balloon and, again, in the presence of ileus and a sick patient, is not really justified. They did reveal that when the diaphragm was partially innervated, re-innervation could be accompanied by spasm of the diaphragm. I have made one study in the stage of spinal shock in collaboration with Mike Goldman and Jeremiah Mead with an oesophageal electrode but this cannot be recommended for routine use.

With regard to the increased workload associated with flaccid paralysis of the intercostal muscles referred to by Dr Brown, study of the oxygen consumption of respiratory muscles in established tetraplegic patients when compensatory and hypertrophy of the accessory muscles has occurred, has shown that the oxygen consumption is raised.[6] I know of no studies in the acute stage.

Under normal circumstances, the respiratory muscles work at optimum efficiency to drive the respiratory system. In these conditions, energy demands will comfortably be exceeded by supply. If the system is inefficient, the energy demands of respiratory muscles will increase, and if they outstrip supply, fatigue will ensue until the body can reduce the load or make adaptive responses. The energy demands of the respiratory muscles increase considerably when the respiratory rate or tidal volume increases above the optimum efficiency. The work of breathing will also be increased if the lungs are stiff and hard to inflate, or the rib cage compliance is decreased.

The rapid breathing alluded to in the acute stage to try and compensate to the reduced vital capacity can lead to exhaustion over a period of, maybe, 2 or 3 days, coupled with the development of oedema in the lungs or pneumonia so the patient becomes exhausted and dies. It is advisable, when the vital capacity is reduced, to support the ventilation by means of a ventilator.

Ledsome and Sharp[7] made a systematic study of five patients with lesions at C4 and found results very similar to this patient. They found the average vital capacity in the first week was 1.28 L, which was 24.4% of predicted (range, 11 to 33%). All five patients required ventilator assistance, begun at 1 to 5 days after injury because of severe hypoxaemia or hypercapnia. Two patients with higher FVC (28 and 33%) were not ventilated initially, but their FVC did decrease before ventilator support was begun. After the initial stage, 2 to 3 weeks, all patients were eventually able to maintain spontaneous ventilation. After 3 months the average FVC was 2.24 L (44% of predicted; range, 18 to 62%).

These patients did not have paralysed diaphragms.

In the initial stages of tetraplegia, the chest wall is flaccid and diaphragm contractions may produce paradoxical movement of the chest wall, resulting in lower recording of VC than actually represented by diaphragm function.

Flaccid paralysis has been demonstrated electromyographically in the acute stage.[3] Movement studies were carried out in the later stages which showed that the diaphragm can produce a paradoxical movement.[8] This is due to the fact that the abdominal muscles are paralysed and instead of the contraction of the diaphragm being resisted by intact abdominal muscles, thus causing expansion of the lower rib cage, paradoxical inward sucking of the chest is produced. Naturally the greater the force, the greater the vital capacity; the greater the paradox.

There is variability in the findings and I know of only one study in the acute stage which was followed up over time.[9] This showed paradox initially. Movements became positive with time. This work needs amplifying.

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In reply to Dr J Silver

The basic information describing the 24-year-old man was deliberately limited to allow the consultants freedom in explaining what they felt were the important parameters in assessing a newly injured SCI individual. Dr Silver presents an interesting discussion and in a comprehensive lengthy review article, these many pieces of information would need to be brought out. However, that was not the intention of this article.

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