Clinical Case of the Month

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

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This case will review what is a common problem in patients with a high level spinal cord injury, the transitional neurological level between the presence and absence of diaphragm function. The case submitted to the discussants is as follows:

The case

This is a 24-year-old man in good health prior to sustaining a fracture dislocation at cervical 3 – 4 in a skiing accident. At the scene of the accident, he was immediately tetraplegic, but was able to breathe. He was immobilized and evacuated to hospital where X-rays confirmed that there was a fracture dislocation of cervical 3 on 4 and complete tetraplegia with a sensory level last pin sensation preserved at the top of the shoulder at the acromio clavicular joint, complete distal ASIA Impairment Scale A. Note: function revealed intact upper trapezius muscle bilaterally but no deltoid or biceps function bilaterally. He was able to breathe on his own, and initial respiratory measurement was a vital capacity of 1000 cc's. After completing the evaluation and initial medical stabilization treatment, he was taken to the operating room at which time a posterior reduction and internal fixation with wire and iliac bone graft was done at the cervical 3 – 4 level. No unusual complications or findings were identified during the operative procedure. In the post-operative recovery room, he did not meet the parameters necessary to extubate and was maintained on oral endotracheal intubation and was placed on ventilator support for the first night post-injury.

The following morning, his vital capacity is 700 cc's and NIF measures – 20 cmH2O. Chest X-ray reveals right lower lobe atelectasis. The questions for consultants to focus on was how would you go about assessing this individual’s potential for weaning from the ventilator, and how would you go about managing this individual so that he would come off the ventilator?

Discussant Dr D Brown
(With the assistance of Dr Geoff Gutteridge)

This man’s initial neurological examination suggests that he is tetraplegic complete below C4. We assume he is a male of average weight, ie 70 kg. Given the vital capacity of 1000 cc, we know that there is partial loss of diaphragmatic innervation. He is described as being in good health prior to the accident. There is no specific indication of smoking status or the presence of asthma. We will assume that he is a nonsmoker and is not an asthmatic.

On admission, his vital capacity was border line but satisfactory. We would observe him at this stage in the absence of other complicating factors. He would be likely to need mechanical ventilation within 24 – 48 h, whether or not he had surgery, as a result of diaphragmatic failure due to the increased work-load associated with the flaccid paralysis of the intercostal muscles. It would be our practice to admit him to the Intensive Care Unit in order to observe him closely and treat him prophylactically for respiratory decline and to be in a position for supported ventilation if required. He would have chest physiotherapy as required.

This patient went to theater shortly after admission. It is well recognized that there can be some neurological deterioration with surgery, although it is often of a relatively temporary nature. Following theater we would return him to the Intensive Care Unit on mechanical ventilation and would not consider extubation within the first 8 – 12 h post surgery. We would then assess his neurological status and respiratory function at a time when all the anesthetic drugs would have worn off. We would not consider extubation unless he had a vital capacity of 15 ml/kg or more, was cooperative and had no respiratory complications, ie no atelectasis.
The following morning his vital capacity has decreased to 700 cc and his chest X-ray revealed right lower lobe atelectasis. These would be contraindications to extubation. We do not regularly measure negative inspiratory force. There are no scientific tests which determine a readiness for weaning, ie there is no single value or set of values which are accepted as predicting ability to wean or to extubate. Our general principles for weaning are as follows:

1. Resolution of pulmonary complications and any obvious extra pulmonary restrictive factors, eg paralytic ileus.
2. The patient should ideally be afebrile as fever increases respiratory requirements.
3. The vital capacity should be 8–10 mg/kg to begin weaning.

The weaning process is one of trial. One cannot complete weaning until the vital capacity is 15 ml/kg. At that level, it is likely that the patient will be able to remain off the ventilator. At the point of weaning, we would require acceptable blood gases, eg arterial PO2 of 80–100 on 40–50% oxygen, want acceptable minute ventilation of 12 L/m and acceptable lung mechanics, ie compliance and resistance.

If the patient was not ready to wean early, and was in established respiratory failure, we would arrange for an early tracheotomy to be performed. We would do this surgically rather than percutaneously in order to minimize trauma to the fracture site which may be associated with the percutaneous technique. The reasons for a tracheotomy are that it decreases the imposed work of breathing, it is more comfortable for patient, it allows laryngeal oedema from endotracheal tube (ETT) to resolve and prevent long term complications of the EET.

The patient’s right lower lobe atelectasis would be treated initially with postural change (left lateral position) and hyperventilation. If the lung is not clear within 12 h, we would undertake a flexible bronchoscopy. Antibiotics would be used for a clinical diagnosis of pneumonia guided by sputum culture.

Once the patient is weaned, we would see that he is stable off mechanical ventilation for 5–10 days or more, that sputum is much reduced and clear, and that the patient is able to clear his chest with assisted coughing and swallows his saliva. Prior to removing the tracheotomy tube, we would plug ‘cork’ the tracheotomy tube for periods and would have the patient on a program of assisted coughing and chest management with positioning 5–6 times per day, of which three would be by the ward physiotherapist and the rest by the nursing staff. Parameters monitored would be vital capacity, oxygen saturation with a pulse oximeter, auscultation, and if necessary, blood gases, chest X-ray, amount and character of the sputum, strength of cough, ability to assist cough and reduction in percentage of inspired oxygen towards normal room air. If the individual continued to borderline in pulmonary toilet – the tracheostomy tube could be replaced by a mini tracheostomy tube to assist with sputum clearance. BiPAP may be used for shorter intervals over night for respiratory muscle fatigue, eg 1 h on and 4 h off. It may also be used if the vital capacity is falling and the patient is having difficulty with a cough and if the PCO2 is high.

Discussant Dr John Watt

The VC measurement of only 1000 cc at this stage is already lower than would be expected simply by the loss of the intercostal contribution to breathing in a previously fit young man. It could be due to right diaphragmatic paralysis and early right lower lobe atelectasis, or to temporarily good accessory muscle compensation masking profound weakness of one or both diaphragms. This essential information could be verified clinically but would normally be confirmed by imaging, and in few centers by diaphragmatic EMG studies.

I would be worried by a decision to operate so early, especially considering the tendency of the neurological level to ascend in the next couple of days, and the recently reviewed evidence points towards early surgery as slightly increasing this risk while conferring no advantage in terms of better neurological outcome. The relative contributions of tracheal intubation, patient positioning, intraoperative cardiovascular instability and surgery itself cannot easily be quantified but each has the potential to cause neurological decline. The intraoperative use of anti-cholinergic agents for bradycardia will increase the tendency for viscous mucus to plug off branches of the bronchial tree which may have happened in this case.

Weaning assessment

Having made a decision to undertake surgery, I would not contemplate immediate extubation even without the atelectasis and even if the patient had maintained the following weaning parameters for 2 h: PaO2 >80 mmHg at FiO2 <0.4; VC >10 ml/kg; max. insp. pressure >20 cmH2O; and respiratory rate <35 bpm.

These criteria are more applicable for non-neurological cases since there is a greater incidence of weaning failure after extubation in neurological cases associated with limited muscle reserve in whom fatigue supervenes later. Elective ventilation after surgery would help minimize the chance of further atelectasis causing suboptimal blood gases which in turn would exacerbate cord edema formation and fatigue itself might lead to the need for an emergency reintubation. In this patient’s case tracheal intubation will also facilitate access to the airway for the purposes of re-expanding the area of collapse and for dealing with lower respiratory infection which is also a possible complication.
Weaning technique

It is my experience that such a patient would wean but the average weaning time in our records of 74 patients with neurological levels from C1 incomplete to L1 was 36 days and I would advise early tracheostomy. Not only does this facilitate weaning partly due to a reduction in dead space and associated reduction in upper airways resistance, but it also allows for earlier discontinuation of sedation and earlier enteral feeding. Formal studies of the success of weaning modalities suggest that SIMV is not particularly successful in comparison with graded time breathing spontaneously on a T-piece which is probably on account of a combination of the added work of breathing and unrecognized, unrelieved muscle fatigue.3,4 While pressure support ventilation has been more successful, this mode of ventilation also depends upon the patient’s effort and encourages a pattern of shallow breathing with basal atelectasis. It does not allow for periods of muscle rest which is crucial in the rebuilding of muscle strength. For this reason, intermittent T-piecing remains as good a method of weaning as any and has the additional advantages that the patient’s morale is boosted by active participation in the weaning process, the vital capacity is closely monitored during the spontaneous breathing and finally, even patients with profoundly weak inspiration e/C128ort can start to train in a weaning program while breathing supplementary oxygen. The present patient’s VC was 700 ml at the end of surgery and I suspect both his right lower lobe atelectasis and the inability to breath himself or herself from using IPPV by taking fewer and fewer assisted breaths as necessary.

Discussant Dr John Bach

The primary focus of my respiratory program is non invasive ventilation therapy.5 The program focuses upon educating individuals with marginal respiratory muscle function whenever possible in the use of respiratory muscle aids before the introduction of the aids. This may not be practical in the acute care setting but can be performed in the non acute setting or in the acute setting if the program specializes in respiratory rehabilitation. The program depends on training the individual in receiving intermittent positive pressure ventilation (IPPV) via mouth pieces and/or nasal interfaces and in the use of manually and mechanically assisted coughing (mechanically insufflation-exsufflation). With preparatory education and training, following the surgical procedure, the patient is introduced to oximetry and instructed that he or she must maintain an oxyhemoglobin saturation (Sao2) greater than 95% by using, as necessary, assisted ventilation and assisted coughing.

Following surgical procedures, when the patient is fully alert, any supplemental oxygen that the patient may have been receiving is discontinued. The Sao2 usually decreases precipitously at this point but it increases just as quickly once the patient is aggressively exsufflated (using the In-exsuflator, JH Emerson Co, Cambridge, MA) via the translaryngeal tube. If the resulting expulsion of airway secretions results in normalizing the Sao2 that patient is considered a candidate for extubation despite the appearance of right lower lobe atelectasis and the inability to breath autonomously. Often, however, there continues to be a decrease of the Sao2 baseline below 95% which requires ongoing insufflation-exsufflation every 10 to 15 min while the patient is awake to assist in eliminating airway secretions until the Sao2 remains above 94%. If the Sao2 levels do not return to normal after 2 days of aggressive therapy, the individual is considered for bronchoscopy. Once the Sao2 remains normal on room air and the patient no longer requires narcotics or sedatives, which is usually by the fourth or fifth post-op day, the patient is switched to using a portable volume ventilator and extubated despite radiographic signs of atelectasis and the need for ongoing ventilatory support.

Immediately upon extubation, the patient is placed on mouth piece or nasal IPPV, as he or she prefers, and is instructed to use noninvasive IPPV as necessary to maintain Sao2 greater than 94% and to avoid shortness of breath. Simple mouth piece or nasal IPPV is used for daytime aid and nasal or lipseal IPPV is used for sleep. Any dips in Sao2 below 95% while using noninvasive IPPV are treated by mechanical insufflation-exsufflation until Sao2 returns to normal. The patient weans himself or herself from using IPPV by taking fewer and fewer assisted breaths as necessary.

Editor Dr Robert R Menter

In evaluating an individual with a high level spinal cord injury, there are many different perimeters of significance. First is the neurological level.6,7,8 In my clinical experience, the presence of intact sensation to pin prick to the level of the acromio clavicular joint implies that the diaphragm muscle is innervated and will be functional at some point in the future, even if it is not functioning at full strength at the time of the examination. If the neurological level of intact pin prick is more proximal than the acromio clavicular joint, one has to suspect that the diaphragm muscle is not innervated at the time of examination and it is unclear when the innervation will return.

An important component of the neurological level is its asymmetry. It is very common that the injury level is not always symmetrical and there may be the presence of hemidiaphragm paralysis. To understand whether the low vital capacity is due to asymmetric function or bilateral symmetrical paresis, fluoroscopy of the diaphragm off the ventilator should be done and the exact movement of the diaphragm, under voluntary control can be measured. If a symmetric diaphragm function is present, different mechanics exist within the lung and different vulnerability to
abdominal distention and ileus. In the presence of hemidiaphragm paralysis, there must be added a major expansive force to re-expand and maintain expansion of the passive basilar segments of the lung above paralyzed diaphragm. The expansion force initially may involve a ventilator but later may be reduced to intermittent expansion therapy during the 24 h period.

In the initial stages of tetraplegia, the chest wall is flaccid and diaphragm measurements may actually produce paradoxical movement of the chest wall, resulting in lower movements of VC than actually represented by diaphragm function. After emerging from the spinal cord injury shock (flaccid) stage, tone returns to the chest wall creating some stability which enhances diaphragm function. Even in the presence of bilateral diaphragm function, there is a range from very weak symmetrical function up to full strength symmetrical function. Bilateral normal diaphragm function implies at least a vital capacity of 1500 cc’s or more. Whenever the vital capacity is less than 1500 cc’s, one has to suspect that there is some degree of paresis or paralysis, either asymmetrical or symmetrical, contributing to the decrease in the value.

As pointed out by the discussants, there is, in many cases, an ascending neurological loss of one segment and in a smaller percentage of patients, two segments, which may recover with time. The length of time for paralyzed diaphragms to recover varies from hours to months and during that time there must be a strong respiratory expansion program to maintain the anatomy and integrity of the lung until the paralyzed segment returns to function.

Perhaps the most important nonrespiratory issue in managing an individual with this injury is the presence of abdominal pathology. Various conditions such as ileus, with associated abdominal distention, will interfere with the movement of the diaphragm and will accelerate respiratory decline and prevent resolution of respiratory problems. The abdomen must be followed in close association with the respiratory system. Any abdominal pathology must be managed aggressively to decrease the abdominal contents resistance to diaphragm motion.

Another significant issue is the presence of obesity. The presence of obesity increases the resistance to the movement of the diaphragm when it is in a state of weakness and fatigue. This will accelerate respiratory decline and prevent respiratory improvement. Unfortunately, there is no simple treatment for the problem of obesity which will mitigate the respiratory problems. Accordingly, the clearing of respiratory problems in the presence of significant obesity, requires great patience and perseverance.

Lastly, the issue raised by Dr Brown of premorbid respiratory history such as smoking, and/or asthma, are major problems encountered in respiratory management. Smoking creates significant secretion problems which accelerate atelectasis and prevent re-expansion of lungs once atelectasis has occurred. Asthma with bronchospasm seems to occur with increasing frequency in industrial societies and will require specific medications beyond the general pulmonary toilet initiated with the spinal injured individual.

As the discussants have presented, there are several ways of approaching ventilation of the spinal injured individual, invasive ventilation versus noninvasive. As Dr Bach points out in his discussion which focuses on noninvasive ventilation, it may be difficult to initiate the education process and patients’ participation when they are under the influence of significant drugs necessary for pain in the acute injury phase. There may also be the problem of decreased mental responsiveness, secondary to head injury and other injuries which may impair a person’s ability to participate in the educational process and the actual participation in the program once it is initiated.

By far, the most common problem in spinal cord injury management is the lack of anticipation of these events and the tendency to react only after there has been respiratory deterioration. Hopefully this discussion has given some insight on how to anticipate, prevent and minimize the respiratory problems in critical spinal injury care.

Listed below are bibliographies of some suggested reading which covers these topics. My thanks to the discussants and their participation.

General References

Specific References