Cricoid pressure

To the Editor:

I feel that Jean-Francois Hardy, in his recent review "Large volume gastro-esophageal reflux: a rationale for risk reduction in the perioperative period," described some of the literature on cricoid pressure but did not subject it to critical analysis. I am concerned that advising readers that "cricoid pressure is simple" encourages the wrong attitude and approach. Moreover can its 100 per cent effectiveness be taken for granted? Complacency about such subjects surely needs to be avoided.

For a time I followed Selwyn Crawford's technique of applying cricoid pressure prior to the induction of anaesthesia but I gave up this approach when a patient hiccoughed, as the thiopentone circulated, and thereby filled up her pharynx with fluid from the stomach with resulting aspiration and subsequent death a few hours later. More recently, in a case where cricoid pressure was applied, an easy and simple intubation was converted into an impossible one; laryngoscopy displayed only the epiglottis with its base resting on the posterior wall of the pharynx. Last week I was told of a colleague who was sure that cricoid pressure was properly applied but upon inserting the laryngoscope found the pharynx full of fluid.

Such examples bring home the pitfalls facing anaesthetists with each and every laryngoscopy, intubation and application of cricoid pressure and the degree of concentration and dedication required if perfection is to be achieved in their performance every time. Moreover, although such incidents occur few of them seem to get as far as being published which can give us all a false impression.

Thank you for your interest.

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REPLY

Sellick's manoeuvre is a simple concept, sometimes difficult to implement in everyday practice. Despite the sophisticated equipment used in contemporary anaesthesia, proper application of cricoid pressure remains based on sound clinical judgment and experience. It is, in effect, a form of medical art. Though not always easy to achieve, a smooth induction of anaesthesia and perfect timing of cricoid compression are of the utmost importance to avoid either provoking active vomiting or passive regurgitation.

I believe Sellick's recommendations still hold true. Judiciously applied, these will effectively prevent gastrooesophageal reflux in a majority of patients at risk of regurgitating on induction of anaesthesia. However close to perfection, I do not believe any one single measure will ever prevent gastrooesophageal reflux in 100 per cent of cases. But, is this not inherent to the practice of medicine?

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Interpleural or paravertebral block at thoracotomy?

To the Editor:

I am writing in response to the Editorial "Interpleural block" published in the Journal in March 1989.

Dr. Lewis refers to the "open chest" technique used by Kambam JR *et al.*[1] whereby an epidural catheter is inserted into the chest at thoracotomy to provide access to the pleural cavity for postoperative infusion of bupivacaine. He suggests that this technique offers an alternative to more established postthoracotomy analgesic methods such as epidural, parenteral narcotics or intercostal nerve blocks. He does not mention paravertebral block, which we have recently described as an alternative "open chest" technique[2] placing the epidural catheter in the paravertebral space under direct vision via an extrapleural route. We believe that paravertebral block attained thereby is the method of choice for a number of reasons.

The anaesthetic is delivered to the most logical space through which runs the intercostal nerve as it exits the intervertebral foramen, its dorsal primary ramus (supplying posterior spinal muscles and costovertebral ligaments strained at thoracotomy), its collateral branch (supplying the parietal pleura), and the sympathetic afferents on that side (supplying the visceral pleura). Inhibition of the endocrine-metabolic stress response to operative trauma requires effective sympathetic and somatic block.[3]

We have demonstrated radiologically that the infusion using this technique is confined to the paravertebral space by the fifth day of infusion.[4] Paravertebral infusion of local anaesthetic agents does not suffer the disadvantages of interpleural infusion after thoracotomy, namely loss via the chest tube, dilution by irrigation fluid or blood, binding to blood proteins in a haemothorax, or rapid absorption by damaged lung quoted as possible explanations for unsatisfactory analgesia.[1] Paravertebral infusion is not contraindicated in pneumonectomy patients who are unsuitable for interpleural block. It is unlikely to affect the phrenic nerve, and should only produce a unilateral sympathetic block. Paravertebral block should provide adequate analgesia for anterior thoracotomies where interpleural block may not be effective.[1]

In a randomised controlled trial, the "open chest"

paravertebral block has been shown to reduce significantly postoperative decline in lung function at 24 hours, improve its subsequent restoration, and reduce pain scores and opiate requirements.[5] In 160 patients who have had this form of pain relief after thoracotomy, we have seen no instances of clinical toxicity to bupivacaine, infusing 0.5 per cent plain bupivacaine at $0.5\ mg \cdot kg^{-1} \cdot h^{-1}$ for four days.

There appears to be no advantage in leaving a catheter in the pleural cavity at thoracotomy when it can so easily be placed in the paravertebral space.

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REPLY

I would like to thank Mr. Sabanathan for his interesting comments. I applaud their ingenuity in the use of yet another catheter technique to provide postoperative anaesthesia on patients having undergone thoracotomy. Mr. Sabanathan makes no comment as to whether or not this technique can be used for analgesia for abdominal incisions but one would intuitively think so.

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Should vecuronium be used in renal failure?

To the Editor:

We read with interest the paper by Starsnic *et al.* (Can J Anaesth, 1989; 36: 35–9) on the use of vecuronium in renal transplant patients. Their conclusion is that vecuronium may accumulate in the renal transplant patient. Perhaps it is timely for us to stand back and examine the use of vecuronium in patients with impaired renal function. There are a number of papers which have shown little difference between the pharmacokinetics of vecuronium in patients with and without renal failure. These papers have, however, examined single bolus doses or very short infusions (a few minutes only).[1,2] In situations where repeated doses have been administered or a continuous infusion has been used, various extents of prolonged paralysis, up to 90 hours in one report,[3] have been noted.[3–6] The weight of evidence seems now to be such that there is little doubt about the tendency for vecuronium to accumulate in patients with renal failure. It must surely therefore be appropriate now to make the recommendations that caution be exercised in the use of vecuronium in patients with impaired renal function and that when it is used, neuromuscular function should be carefully monitored.

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