Correspondence

Intubating conditions and correct application of cricoid pressure during rapid sequence induction: who should hold the mask?

To the Editor:

In difficult elective intubations, cricoid pressure (CP) is often applied after laryngoscope-assisted head extension and jaw thrust to improve visualization. In contrast, during rapid sequence induction (RSI), CP is applied as the drugs are injected. The head can flex forward as consciousness is lost and make it difficult for the assistant to know where to apply CP. If CP is then applied the resulting skin tension can act like a "bow string" to prevent full extension of the head and forward displacement of the mandible. Difficulty in inserting the laryngoscope into the mouth during RSI for Caesarean section has been attributed to large breasts getting in the way of the laryngoscope handle. Another explanation, "fixed" flexion of the head due to the "bow string" effect deserves consideration.

If the anaesthetist holds the mask and maintains head extension with a jaw thrust as CP is applied, the "bow string" effect is prevented. An intravenous stop cock assembly allows injection with one hand while the other holds the mask and head position. When the anatomy is not obvious, the inferior aspect of the thyroid cartilage can be felt with the fingers of the right hand and the assistant instructed to apply CP from below. Since the jaw thrust prevents the tongue from obstructing the airway, an extra period of apnoeic oxygenation is possible while waiting for the muscle relaxant to take effect.

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Pharmacological properties of the denervated heart

To the Editor:

We read with interest Dr. Haddow's thoughtful review article concerning the management of anaesthesia for patients having undergone lung transplantation.[1] As indicated in his article, one of the considerations for this type of patient is the potential for disruption of the nerves innervating the heart. Dr. Haddow asserts that in the event of such cardiac denervation, heart rate will not change in response to the administration of anticholinesterase drugs. We wish to challenge this opinion which, although widely held, has little evidence to support it. On the contrary, we have demonstrated that neostigmine consistently produces a dose-dependent, atropine-sensitive reduction in heart rate in cardiac transplant patients, and in particular patients transplanted remotely prior to the administration of neostigmine (> six months) may be especially sensitive to its bradycardic effect.[2] We have recently shown that edrophonium also evokes a dose-dependent, atropine-sensitive reduction in heart rate in the cardiac transplant patient, although the magnitude of the bradycardia is smaller and less variable compared to that produced by neostigmine.[3] Animal studies suggest that anticholinesterases may produce a bradycardic response in the denervated heart by their direct stimulation of cholinergic receptors in the peripheral cardiac parasympathetic pathway, or by their anticholinesterase action, whereby acetylcholine tonically released from cardiac parasympathetic postganglionic cells is protected from hydrolysis by acetylcholinesterase.[4] These findings have prompted us to caution that when administering anticholinesterase drugs to heart transplant patients, a muscarinic antagonist should always be co-administered to block the cardiac and other muscarinic side effects.[5] While it remains to be demonstrated that anticholinesterase drugs evoke a bradycardic response in a heart denervated during the course of lung transplantation, such an effect is anticipated in light of the observations in the cardiac transplant patients and animal studies cited above.

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