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.

Gerard Bruin MD FRCPC, Norm Buckley MD FRCPC
Hamilton Health Sciences Corporation
Department of Anaesthesia/McMaster Campus
1200 Min Street West, Room 2U3
Hamilton, Ontario, L8N 3Z5

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.

S.B. Backman MDCM PhD FRCPC
G.S. Fox MD FRCPC
F.E. Ralley MB CHB
Department of Anaesthesia
Royal Victoria Hospital McGill University
Montreal, Quebec

REFERENCES

1 *Haddow GR.* Anaesthesia for patients after lung transplantation. Can J Anaesth 1997; 44: 182–97.
2 *Backman SB, Fox GS, Stein RD, Ralley FE.* Neostigmine decreases heart rate in heart transplant patients. Can J Anaesth 1996; 43: 373–8.

3 *Backman SB, Stein RD, Fox GS, Polosa C.* Heart rate changes in cardiac transplant patients and in the denervated cat heart after edrophonium. Can J Anaesth 1997; 44: 247–54.
4 *Backman SB, Stein RD, Blank DW, Collier B, Polosa C.* Different properties of the bradycardia produced by neostigmine and edrophonium in the cat. Can J Anaesth 1996; 43: 731–40.
5 *Backman SB, Ralley FE, Fox GS.* Anaesthesia for cardiac transplant patients (Letter). Can J Anaesth 1994; 41: 655–6.

REPLY

I would like to thank Drs. Backman, Fox and Ralley for their interest and comments on my review article "Anaesthesia for patients after lung transplantation." As they correctly point out the majority of texts and the majority opinion has been that no change in heart rate occurs with the administration of anticholinesterases to patients with a denervated heart. Their recent research contradicts this view and may indeed change the previous previously held notions.[2,3] Their publications also may lead to new insight into the physiology of the transplanted heart and into the possibility of whether re-innervation occurs. I agree with them that anticholinesterases should not be administered without a muscarinic antagonist to block the muscarinic side effects (indeed this may be why the bradycardic effect has been previously missed).

G.R. Haddow MD
Department of Anesthesia
Stanford University Medical School
Stanford, California

REFERENCES

1 *Haddow GR.* Anaesthesia for patients after lung transplantation. Can J Anaesth 1997; 44: 182–97.
2 *Backman SB, Fox GS, Stein RD, Ralley FE.* Neostigmine decreases heart rate in heart transplant patients. Can J Anaesth 1996; 43: 373–8.
3 *Backman SB, Fox GS, Stein RD, Polosa C.* Heart rate changes in cardiac transplant patients and in the denervated cat heart after edrophonium. Can J Anaesth 1997; 44: 247–54.

Sample size estimation for nominal data

To the Editor:

In the review *Study Design in Clinical Research*,[1] sample size estimation for nominal data involved transforming the data.[1,2] However, clinicians may either be hesitant to or not have the means to transform their data. To estimate the sample size for nominal data without a transformation, readers should be aware of two equations that use the observed proportions:

Equation 1:[3]

$$n = \frac{(p_1 q_1) + (p_2 q_2)}{(p_2 - p_1)^2} \times f(alpha, power)$$

where p_1 and p_2 are the observed proportions, q_1 and q_2 are $(1-p_1)$ and $(1-p_2)$ respectively, and f is a constant that is determined by the alpha (α) and power (1-ß). For $\alpha_2 = 0.05$ and a power of 80% or 90%, the values for f are 7.9 and 10.5 respectively.

Equation 2 takes another approach to estimation of the sample size for nominal data:[4]

$$n = k \times \frac{[\bar{p}(1-\bar{p})]}{(p_2 - p_1)^2}$$

where, k = 15.7 for $\alpha_2 = 0.05$ and a power of 80%; and 21.02 for $\alpha_2 = 0.05$ and a power of 90%; and

$$\bar{p} = \frac{(p_1 + p_2)}{2}$$

Both equations yield similar estimates of the sample size for nominal data and similar estimates to those that use the transformed data, for the entire range of proportions.[1,2,5] However, the reader should take note that at the extremes of proportions (i.e., values <0.05 or >0.95), sample size estimates using the above equations may exceed those determined using equations based on transformed data by approximately 10%.[5]

Jerrold Lerman BASC MD FRCPC
Toronto, Ontario

REFERENCES

1 *Lerman J.* Study design in clinical research: sample size estimation and power analysis. Can J Anaesth 1995: 43; 1–8.
2 *Lerman J.* Erratum. Can J Anaesth 1996: 43; 880.
3 *Wassertheil-Smoller S.* Biostatistics and Epidemiology. A Primer for Health Professionals. New York: Springer-Verlag, 1990: 99–101.
4 *Kramer MS.* Clinical Epidemiology and Biostatistics. A Primer for Clinical Investigators and Decision-Makers. Berlin: SpringerVerlag, 1988: 179–81.
5 *Cohen J.* Statistical Power Analysis for the Behavioral Sciences, 2nd ed. Hillsdale, New Jersey: Lawrence Erlbaum Associates, 1988: 179–206.