

through use of a screening questionnaire. *Can J Anaesth* 1998; 45: 87-92.

Potentially incorrect terminology-lumbosacral plexopathy

To the Editor:

Crosby *et al.* present a case of permanent neurological dysfunction following abdominal aneurysctomy and attribute it to a lumbosacral plexopathy caused by an iliopsoas haematoma.¹ I agree with the authors that there is sufficient reason to believe that the iliopsoas haematomas are at least partially responsible for the deficits seen in the femoral and lateral femoral cutaneous nerves (i.e. lumbar plexus). Their conclusion is supported by the extensive literature review they present and by a review of the relevant anatomy. However, I believe that they should refer to the observed neurological dysfunction as a lumbar plexopathy. There is little reason to believe that the iliopsoas haematoma is responsible for deficits seen in the distribution of the "entire" sciatic nerve. While the lumbosacral "trunk" (technically a part of the lumbar plexus at the level of the pelvic brim) may, in theory, be compressed by a large psoas haematoma, it is extremely unlikely that the rest of the sacral plexus would be affected in like manner. The deficits in the sacral divisions of the sciatic nerve (sacral plexus) likely have an alternative explanation as suggested by the authors (i.e. vascular compression or vessel rupture). Therefore, I believe that the latter should be referred to as a sacral plexopathy to avoid confusion and that the combined terminology "lumbar and sacral plexopathy" be used to emphasize the disparate etiologies for the two pathological entities - especially as the aetiology of the sacral plexopathy is unclear!

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REFERENCE

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Haemodynamic effects of adding ephedrine to propofol and alfentanil

To the Editor:

One important problem during induction of anaesthesia with propofol is hypotension.¹ Preinduction

vagolytic agents have been used in an attempt to overcome it.^{2,3} Recently, the haemodynamic outcome of adding ephedrine to propofol during induction was studied⁴ but the effect of intubation was not evaluated.

In a randomised double-blind study in 54 healthy ASA Class I patients, we investigated the haemodynamic effects of adding graded doses of ephedrine (20 mg or 30 mg *iv* vs placebo) to 20 ml propofol 1%. The anaesthetic induction sequence was standardised using 2 mg·kg⁻¹ propofol and 20 µg·kg⁻¹ alfentanil. Vecuronium (0.15 mg·kg⁻¹) was used to facilitate tracheal intubation and anaesthesia was maintained with infusion of propofol and alfentanil mixture (50 µg·ml⁻¹) at 1 ml·kg⁻¹·hr⁻¹.

Decreases in heart rate and systolic blood pressure were observed in the placebo group, but not in either treatment group, following induction of anaesthesia. However, modest increases in systolic blood pressure and heart rate were observed in response to tracheal intubation in both treatment groups. Ephedrine 20 mg when added to 20 ml propofol 1% provided the better haemodynamic profile. (Figure)

Although the addition of ephedrine to propofol and alfentanil may prevent the decrease in blood pressure after induction of anaesthesia, it caused an increase in heart rate and systolic blood pressure after intubation. Whether this results in increased cardiac morbidity in patients with coronary artery disease is unknown, but caution should be taken when administering ephedrine with propofol.

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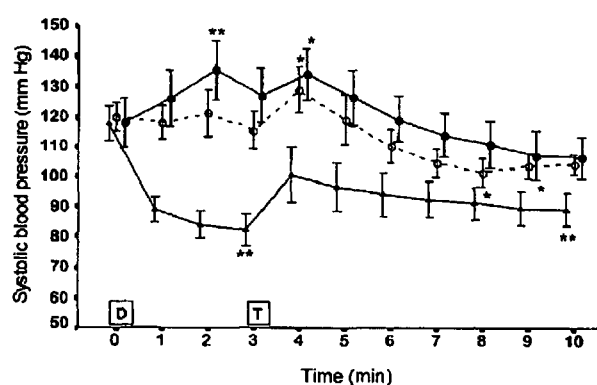


FIGURE Median (range) of the haemodynamic variables at baseline (0), after induction (D) and intubation (T) using propofol 1% (○), 20 mg ephedrine in 20 ml propofol 1% (□) or 30 mg ephedrine in 20 ml propofol 1% (●).

* $P < 0.05$ vs baseline value.

† $P < 0.05$ vs baseline and the other two Groups.

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Endobronchial intubation

To the Editor:

We were very interested to read the letter of John Oyston entitled "Detecting endobronchial intubation during breast surgery" (*Can J Anaesth* 1997; 44: 1129). The use of a "bagged" stethoscope is ingenious and logical. We would have doubted that breath sounds could be heard but obviously there was enough solid contact for satisfactory sound transmission, and we thank Dr. Oyston for the lesson in auscultation.

Endobronchial intubation is the commonest reason for desaturation as was shown in the Australian Incident Monitoring study.¹ It is also of interest that, for Dr. Oyston, the $P_{ET}CO_2$ was unchanged and this is consistent with our findings where capnography was found to be unhelpful in 88% of incidents.

Obviously prevention is the best course. For this reason, in our analysis, we advocated the use of a tracheal marking 3 cm above the upper edge of the cuff. If this mark which identifies the 3, 4, and 5 cm points (Figure) is clearly visible above the vocal cords then the cuff is just below the cricoid ring and the risk of endobronchial intubation is avoided. It has been used in Australia on Curity (Kendall) tubes (7 mm - 9 mm) since mid 1997. The marks are a guide for both oral and nasal tubes as they are referenced to the vocal cords.

A number of authors have advocated single marks on the tracheal tube but only this mark defines the critical 3 to 5 cm above the cuff to ensure the highest

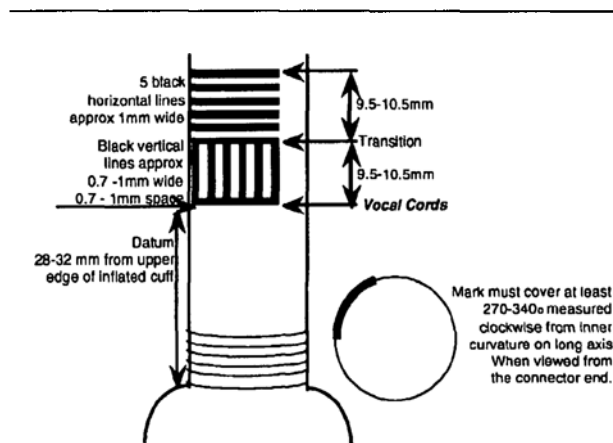


FIGURE Illustration of tracheal tube marking to assist in positioning the tube in the trachea. Normally, in adults, the vocal cords should be positioned as shown. If any upward movement is anticipated during anaesthesia, a deeper insertion to 4 or 5 cm may be appropriate. It should rarely be necessary to insert the tube deeper. These marks are appropriate for both oral and nasal intubation.

possible position of the tube in the anaesthetized patient. The general suitability of the marks is why we are recommending it to the International Standards Organization Technical Committee 121 which deals with standards for tracheal tubes.

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