Correspondence

Vision loss after spine surgery: a new hypothesis

To the Editor:
As new reports concerning vision loss after spine surgery continue to be published,[1,2] it is crucial to explore possible mechanisms. The incidence of vision loss after noncardiac surgery is 0.0008%,[3] while it is 0.2% following spine surgery,[4] with the causes usually taken to be anemia or hypotension.[5] We hypothesize a possible additional mechanism: that the use of large amounts of crystalloids in such cases can lead to an accumulation of edema in the eye socket with the production of an "eye compartment syndrome". Presumably, increased eye socket pressure in this setting can lead to ischemia of the optic nerve, especially in the posterior segment having a single blood supply derived from the ophthalmic artery.[6,7]

Our hypothesis is supported by the frequent observation of facial edema and chemosis in long spine cases carried out in the prone position, as well as by occasional cases of blindness after bilateral neck dissection where the internal jugular veins have been sacrificed.[8] Here, the loss of vision is attributed to back pressure from the distended ophthalmic veins, leading to ischemic neuropathy.

Experience suggests that prone positioning for spine surgery may play a particularly important role. We recommend that, where possible, prone patients should be positioned so that their heads are higher than the heart, and that any position that might impair venous drainage of the head be avoided. In addition, anything that might lead to direct pressure on the orbit must be carefully prevented.

We believe that eye compartment syndrome as a cause of blindness should be considered in any prone spine case managed with extensive use of crystalloids and where anemia and hypotension have been excluded. We also speculate that the use of colloids for spine cases in the prone position may offer the potential to reduce eye socket edema and subsequent vision loss.

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Accepted for publication November 26, 2005.

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Objective techniques for identification of the epidural space in infants and children

To the Editor:
I read with interest the results of a survey of Canadian pediatric anesthesiologists regarding identification of the epidural space in infants and children.[1] However, I was somewhat disappointed to learn that the *iv* micro-drip infusion set technique for identification of the space[2] is not being practiced in Canada. This technique enables the operator to hold the epidural needle with two hands, thus providing more precise control of the needle, and identify the epidural space by an objective sign, either free flow of fluid from the drip

chamber, or by movement of a tiny air bubble at the hub of the needle towards the epidural space.[3,4] We have used this technique in more than 2,000 infants and children at our institution.[4] In 1,385 infants and children, lumbar epidural puncture was successful on the first attempt (90.2%) and on the second attempt in an additional 8.0%.

The survey[1] also failed to include another method to identify the epidural space as reported by Suwa *et al.*[5] In place of a drip infusion set, these authors connected the epidural needle via an *iv* extension tube to a pressure-transducer. They identified the epidural space in 25/25 (100%) children without dural puncture by observing a sudden decrease in pressure when the epidural space was accessed. As a control, they used the loss-of-resistance to saline, and their success rate was 84% (21/25), with a dural puncture incidence of 4% (1/25). This pressure-guided method is also objective, but may be too expensive for some clinical settings.

I hope that by drawing the readers' attention to these methods of identifying the epidural space, more clinicians will consider replacing the loss-of-resistance techniques to saline and air, with these alternative objective techniques.

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Accepted for publication November 30, 2005.

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Factors which may influence mean arterial pressure measurement

To the Editor:

The letter by Ahn and Lim[1] in the November 2005 issue of the *Canadian Journal of Anesthesia* presents direct arterial blood pressure data in a patient under anesthesia, showing that variations in mean arterial pressure (MAP) may result from differences in pressure waveforms even in the presence of stable systolic and diastolic pressure readings. Quoting a previous letter from our team on the empirical equations for MAP estimation,[2] the authors suggest that researchers "sometimes forget the limitations in using these equations".[1] We provide the following comments.

A critical evaluation of several empirical equations for estimating MAP has been reported recently.[3] As in our previous letter,[2] it was stressed that results pertain strictly to the conditions under study, namely patients investigated at rest, under stable hemodynamic conditions, with their pressure being recorded at the aortic root level by using a high-fidelity pressure catheter.[3] As previously discussed,[2,3] our conclusions do *not* apply to peripheral pressure recordings, nor to patients with unstable or rapidly varying hemodynamic conditions. We have obtained data in 139 patients, and the mean bias between MAP estimates and true MAP was < 0.5 mmHg with a precision (standard deviation of the bias) < 3 mmHg.[3] While there are no guidelines for such comparisons, it must be noted that the Association for the Advancement of Medical Instrumentation recommends that bias < 5 mmHg and precision < 8 mmHg are required for validating the accuracy of a new pressure device when compared to a reference standard.[4]

Although MAP is essentially similar in the aorta and large peripheral arteries, the pressure waveform obtained at the aortic root differs significantly with that recorded at the peripheral level. The basic hemodynamic principles explaining such differences have been reviewed.[5] We agree with Ahn and Lim[1] that acute changes in blood volume, inotropic state, heart rate, vascular tone and arterial stiffness may lead to discrepancies between the actual MAP value and the MAP empirically estimated at the peripheral level. However, we feel that our viewpoint has been misquoted, and we hope that the present comments help to clarify other factors which may influence mean arterial pressure estimation, including the recording site (central *vs* peripheral), the characteristics of the recording system (high-fidelity *vs* conventional) and the nature of hemodynamic conditions (stable *vs* unstable).