who had a mean PCV of 49.4% with a control group who had a mean PCV of 42.1%. The mean TEI in the first group was 31.25 Ω, which was significantly greater than that in the second group—27.5 Ω (P < 0.05) [5].

These observations may help to explain the “surprisingly increased response in TEI” noted by the authors. The influence of PCV clearly assumes greater importance with larger variations in PCV, seen, for example, after cardiac surgery.

Failure to understand the limitations of TEI in the estimation of lung water may explain why a technique that was well described more than 20 years ago has yet to find its way into routine clinical use.

Sir,—Thank you for the opportunity to respond to the comments of Drs Thomas, Vohra and Pollard. We do agree that the change in intravascular volume as demonstrated by haemodilution of different magnitude in the two groups of treatment complicates the interpretation of transthoracic electrical impedance (TEI). However, as blood losses were similar in the two groups, the smaller PCV in the 3% Dextran group indicates that intravascular volume is larger after Dextran treatment than after Ringer’s acetate. The decrease in TEI after Ringer’s acetate, therefore, seems mainly to represent an increase in extravascular lung water. The decrease in resistivity of blood caused by decreased PCV as pointed out by Drs Thomas, Vohra and Pollard, would have affected Dextran-treated patients more, thereby decreasing TEI more in this group. However, only a moderate change was seen, in contrast with the more pronounced decrease in TEI after Ringer’s acetate.

In summary, both the smaller PCV per se and the larger intravascular volume would have augmented a decrease in TEI in the Dextran-treated patients as a result of an increase in extravascular lung water. A more direct method for investigation of extravascular lung water would thus probably have resulted in even more pronounced differences between the two kinds of volume loading before extradural Caesarean section.

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REFERENCES

Sir,—Although the recent paper by Kalli [1] on the effect of surface electrode position on evoked EMG response appears to be well designed, experience from our department is at variance with some of his results. Kalli reported a peak-to-peak amplitude of 8.5 mV when the evoked compound action potential (ECAP) was measured using the adductor pollicis muscle and the index finger (TD2) as the recording electrode pair. He found that the response of the first dorsal interosseous was significantly greater (12.5 mV) and hence preferred the latter muscle for clinical monitoring.

Using a study design and instrumentation that appeared to be similar to that of Kalli, we found that TD2 electrode placement produced a peak-to-peak amplitude of 13.1 (SEM 0.7) mV or 50% greater than the evoked response that Kalli recorded. However, a modest resting tension was applied to the thumb in all of our patients. In Kalli’s investigation the arm to be studied was secured in a splint, but there was no mention of any preload applied to the thumb. As there is some evidence that preload can effect the ECAP [2], it would be helpful if Kalli could be a little more specific regarding the exact method used to secure the experimental hand and arm. If no resting tension on the thumb was used, Dr Kalli’s conclusions on the usefulness of the adductor pollicis compared with the first dorsal interosseous may need to be re-examined.

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REFERENCES

Sir,—I read with interest Dr Kopman’s comments on evoked compound muscle action potential (ECAP) monitoring of muscle relaxation.

The aim of the recent study [1] was to compare the ECAP responses of different muscles of the hand. The hand was fixed in a neutral position on a splint; no pre-tension was applied to the thumb. Increased hand muscle mass may have an impact on ECAP. Female patients with similar characteristics were selected to minimize interindividual variability. Depending on the methodology used in various studies, the results may vary.