

Correspondence

Is a decrease in capillary density dangerous?

Editor—I would agree with the authors' interpretation of the interesting observations¹ that not only propofol by itself, but associated hyperoxia and increases in sympathetic discharge, may affect the microcirculation. What are the clinical implications of the observations? Could it be potentially dangerous deterioration of microcirculation induced by propofol, or could an observed decrease in capillary density simply reflect a decrease in oxygen demand within the tissues (due to slight decrease in temperature, hyperoxia, and propofol-induced decrease in metabolic requirements) and metabolically mediated auto-regulation of nutritive flow? In other words, could tissues require less oxygen and decrease capillary density in order to meet this decreased requirement? The authors correctly speculate that as reported in the literature no change in capillary filtration coefficient during propofol anaesthesia probably reflects the absence of increase in microvascular permeability and, one can speculate further, may reflect also an absence of oxygen deprivation. Thus, the question is whether these observations reflect any real homeostatic disturbance or an adjustment to a decrease in metabolic/oxygen requirements?

S. Gelman

Boston, USA

E-mail: sgelman@partners.org

Editor—We agree with Dr Gelman that it is not possible to determine whether the propofol-induced decrease in small vessel density we observed¹ was a detrimental effect of the propofol or an adaptative response, as we did not measure local metabolism or local indices of tissue hypoxia. However, a decrease in capillary density is not a typical finding in the microvascular adjustment to decreased local metabolism, which is characterized by a decrease in microvascular flow velocity.^{2,3} In addition, using near-infrared spectroscopy, De Blasi and colleagues⁴ recently reported that propofol anaesthesia did not alter muscle oxygen consumption (VO₂). In agreement with our findings, these authors also reported that the post-ischaemic haemoglobin re-oxygenation slope was decreased after propofol anaesthesia, which indirectly suggests that microvascular recruitment was altered. Altogether, these data suggest that propofol decreases microvascular perfusion by a non-homeostatic mechanism. This may have detrimental consequences in a diseased microcirculation.

D. De Backer

M. Koch

J.-L. Vincent*

Brussels, Belgium

*E-mail: jlvincen@ulb.ac.be

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Real-time visualization of ultrasound-guided retrobulbar blockade: an imaging study

Editor—We read with interest the article by Luyet and colleagues.¹ The authors' elegantly designed study using CAT scan imaging to localize ultrasound-guided contrast dye injected into cadaveric specimens confirms the utility of this tool. However, having already introduced the use of real-time bedside ophthalmic ultrasound into our clinical practice, and published² and lectured on the subject internationally, we must emphasize important theoretical caveats. The bioeffects of ultrasound on the eye must be considered before encouraging translational application and clinical use of sonography for ophthalmic anaesthesia. Ultrasonic oscillating waves are essentially pulsed energy. They create both thermal and mechanical perturbations in tissue and can induce tissue heating and acoustic pressure. Two indices, thermal index (TI) and mechanical index (MI), are denotive of heat and mechanical agitation that may be generated by a particular ultrasonic transducer.³

Recognizing the special nature of orbital tissue, the FDA⁴ and Health Canada⁵ permit an MI of ≤ 1.9 for all target tissues, with the exception of the eye (≤ 0.23). Additionally, they recommend an equipment that has a maximal achievable TI of 6.0; however, the limit for the eye is 1.0. The British Medical Ultrasound Society⁶ concurs that TI be no more than 1.0. Most commercially

marketed ultrasound transducers do not comply with these recommendations.

Bedside ultrasound may indeed have application in improving the safety profile of ophthalmic regional anaesthetic blocks, principally perforation, or penetration complications associated with needle-based techniques. Clinicians should be cognizant of differences between probes and ensure that they use appropriate orbital-rated transducers.

S. Gayer^{1*}

H. Palte¹

C. Kumar²

¹Miami, USA

²Middlesbrough, UK

*E-mail: sgayer@miami.edu

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Editor—We thank Drs Gayer and Palte and Prof. Kumar for this important note regarding the technical side of using ultrasound for regional anaesthesia in eye surgery. We are aware of the potential risk of ultrasound waves, especially for sensitive structures like neuroretinal tissue. Our cadaver study was intended to prove the concept of a new technique without danger to patients. We investigated the ultrasound-guided correct placement of the needle and the proper spread of the applied drug. The bioeffects of ultrasound were not in the scope of our investigation.

The ultrasound device used for our study on cadavers did not meet the quoted FDA limits,⁴ especially not the limits for the MI which was 0.4 and therefore higher than 0.23. In this case, the energy applied to the eyes did not make any difference as the subjects were cadavers. We agree that the use of ultrasound for eye block anaesthesia should improve the safety rather than represent a potential risk to the eye tissues. Therefore, only orbital-rated transducers for *in vivo* sonography meeting FDA recommendations should be used for clinical studies and daily routine practice. The lower output energy of these transducers does not impair the detection of intraorbital structures or needles used for the eye block—and the method described in our study is also valuable with other small curved array transducers. Companies providing ultrasound equipment will produce suitable transducers in the near future which will be in accordance with the regulations.

C. Luyet

U. Eichenberger*

C. Tappeiner

R. Greif

Bern, Switzerland

*E-mail: urs.eichenberger@insel.ch

I Luyet C, Eichenberger U, Moriggl B, Remonda L, Greif R. Real-time visualization of ultrasound-guided retrobulbar blockade: an imaging study. *Br J Anaesth* 2008; **101**: 855–9

Convulsions associated with ropivacaine 300 mg for brachial plexus block

Editor—We read with interest the case report by Satsumae and colleagues,¹ describing convulsions associated with ropivacaine 300 mg for brachial plexus block. The article raises several questions about the authors' selection of regional anaesthetic technique and the dose of local anaesthetic used.

First, regarding the choice of regional anaesthetic technique for navicular (scaphoid) surgery, adequate intra-operative anaesthesia should be provided by axillary brachial plexus block alone. A blind technique without a peripheral nerve stimulator or ultrasound will inevitably result in a higher incidence of musculocutaneous and radial nerve failure, but this is hardly an indication for additional interscalene block. Occasionally, additional interscalene brachial plexus block or a specific low-volume block of C5/6 with ultrasound or peripheral nerve stimulator can be useful to provide analgesia of the shoulder when arm positioning is problematic, but this approach seems excessive if used for supplementation or tourniquet analgesia alone. Using a peripheral nerve stimulator or ultrasound will improve the success rate² and safety profile of the axillary technique by allowing accurate injection of local anaesthetic and more importantly facilitate reduction in the dose of local anaesthetic administered.

Secondly, regarding the dose of local anaesthetic used, although the authors acknowledge that the dose of local anaesthetic used was excessive, we wish to highlight that the use of a 'safer agent' should not be an excuse to use larger doses of local anaesthetic but to improve the margin of safety for a 'conventional' dose. In our practice, the majority of brachial plexus blocks can be performed with <170 mg of ropivacaine, despite the maximum dose limit of 250–300 mg recommended by the manufacturer and