Bupivacaine chondrotoxicity

Editor—We commend Webb and Ghosh1 for their editorial reviewing the growing evidence for the chondrotoxicity of intra-articular bupivacaine. We would like to add three further points for your readers’ consideration. First, neither magnesium nor bupivacaine is licensed for intra-articular use, although bupivacaine has a good safety record given by this route. Nevertheless, no patient harm has been raised as a potential concern, it should be incumbent on anaesthetists to consider alternative methods of providing analgesia after arthroscopy, until such time as research is performed to confirm or refute chondrotoxicity.

Secondly, as stated in the editorial, ropivacaine 0.5% appears to be less chondrotoxic than bupivacaine 0.5%.2 Although not yet reported, the relative analgesic potencies of ropivacaine and bupivacaine suggest that 0.375% (or even 0.2%) intra-articular ropivacaine may provide equivalent postoperative analgesia, but with reduced chondrotoxicity.

Finally, and although unlicensed, magnesium (1 g in 20 ml saline) might provide a viable alternative or dose-sparing adjuvant to intra-articular bupivacaine.3 4 Crucially, recent in vitro research suggests that supraphysiological doses of magnesium appear to be chondroproliferative.5 We would advocate the use of magnesium sulphate 1 g as the primary intra-articular analgesic for knee arthroscopy, augmented (if at all) by ropivacaine 0.2%.

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Editor—We would like to thank the authors for highlighting the increasing awareness of the possible toxicity of intra-articular local anaesthetic, particularly bupivacaine.1 They concentrate on postarthroscopic glenohumeral chondrolysis (PAGCL), as this has been found to occur after the use of intra-articular infusion of local anaesthetics.6 Gottschalk7 in his letter suggests that the use of these techniques in the subacromial bursa, outside the glenohumeral joint, would avoid the risk of PAGCL. We would contend that this may be the case in surgery where the anatomical barriers to spread of anaesthetic remain intact; but where they are disrupted, as may occur with rotator cuff repair, local anaesthetic may leak into the glenohumeral space and thus may still cause PAGCL.

Bearing these points in mind, we would like to question the need to ever use intra-articular local anaesthetic. The published data comparing intra-articular injection of local anaesthetic with interscalene block clearly indicate that interscalene block provides better analgesia.8–10 In the face of this evidence, it should be questioned why intra-articular local anaesthetic techniques are still being used for shoulder surgery where there is an alternate, more effective method of analgesia which involves no risk of PAGCL.

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Editor—We wish to thank Drs White and Turner and Drs McNaught and McCartney for their letters in response to our article. We cannot support the use of intra-articular ropivacaine 0.2% and intra-articular magnesium as advocated by Drs White and Turner. Although the risk of chondrotoxicity appears to be less with ropivacaine 0.5% compared with bupivacaine 0.5%, the absence of chondrotoxicity associated with ropivacaine 0.375% or 0.2% has not been demonstrated.2 Intra-articular magnesium sulphate may provide effective postoperative analgesia, but its safety in clinical practice has yet to be established.5

We agree with Drs McNaught and McCartney’s suggestion that interscalene brachial plexus block should be considered as an alternative strategy to the intra-articular administration of local anaesthetics for postoperative analgesia after shoulder surgery.

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3 Elsharnouby NM, Hala EE, Abou Elezz NF, Moharram AN. Intraarticular injection of magnesium sulphate and/or bupivacaine for postoperative analgesia after arthroscopic knee surgery. Anesth Analg 2008; 106: 1548–52
7 Gottschalk A. Avoid intra-articular catheters for continuous infusion of local anaesthetics. Br J Anaesth 2009
Bispectral index sensor as a possible cause of postoperative visual loss after frontal craniotomy

Editor—Postoperative visual loss (PVL) after non-ocular surgery is rare, but a devastating complication.1–3 We report a patient with unexpected PVL after brain tumour removal by frontal craniotomy. The development of PVL was attributed to the bispectral index (BIS) sensor (BIS standard, Aspect Medical Systems) fixed on the forehead.

A 38-yr-old female patient was undergoing right frontal brain tumour removal. She had complained of headache and nausea suggesting increased intracranial pressure, but otherwise she was quite healthy. After placing a BIS sensor on the left forehead, general anaesthesia was induced with a target control infusion of propofol and continuous infusion of remifentanil 0.5 μg kg\(^{-1}\) min\(^{-1}\). Her trachea was intubated after vecuronium 6 mg. Anaesthesia was maintained with target control infusion of propofol and continuous infusion of remifentanil 0.15–0.25 μg kg\(^{-1}\) min\(^{-1}\). Skin incision was performed bilaterally along the hairline on the forehead, and the myocutaneous flap was retracted anteriorly and inferiorly near the orbit. Right frontal craniotomy was performed, during which the connector between the BIS sensor and cable was turned down to the left side and also fixed on the left forehead. The surgical procedure was uneventful, but lasted >8 h. Systolic arterial pressure was controlled between 120 and 140 mm Hg. BIS value stayed between 40 and 50. Total amount of fluid infused was 5200 ml, blood loss was 290 ml, and urine output was 2680 ml. She emerged from anaesthesia uneventfully after surgery. Her first complaint of left visual loss and several small blisters above her left eyelid were noted after admission to the intensive care unit. An ophthalmologist’s examination revealed the following: her left eye was completely blind, the left direct light reflex disappeared although the consensual light reflex was normal, the left ocular muscle function was slightly impaired, and the fundal appearances presented normal optic disk and retina. Dexamethasone was administered immediately. The fundal appearances continued to be normal without the appearance of cherry red spot. Imaging studies revealed neither cerebral infarction nor surgery-related injury to the orbit or optic nerve. Electroretinogram showed slight b-wave reduction suggesting left retinal damage, whereas fluorescein angiography demonstrated normal retinal circulation. The absence of visual evoked potential suggested injury between the optic nerve and the optic tract. The left direct light reflex began to appear after 1 month, but her vision is still only to hand movement. Left optic disk became pallid after 2 months, suggesting the development of optic nerve atrophy.

Bilateral orbital infarction attributed to orbital compression by retraction of a myocutaneous flap over the eyes during frontal craniotomy has been reported,\(^4\) and occlusion of the ophthalmic artery and its branches led to ischaemia of the whole orbit because of increased intraorbital pressure. In our case, the bulky BIS sensor and connector fixed on the left forehead may have caused orbital compression when the myocutaneous flap was retracted near the orbit, increasing left intraorbital pressure. Several small blisters above the left eyelid suggested firm retraction. Increased intraorbital pressure caused vascular insufficiency to the optic nerve, leading to the development of PVL. Left retinal damage and ocular muscle dysfunction also developed as the result of ischaemia caused by increased intraorbital pressure. On the other hand, impaired orbital venous drainage caused by increased cerebrospinal fluid pressure associated with brain tumour might contribute to vascular insufficiency.\(^5\) We should be cautious when placing a BIS sensor on the forehead during frontal craniotomy.

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