High doses of local anaesthetic during spinal anaesthesia may increase the risk of life-threatening vagal reactions

Editor—Dr Wahl and his colleagues provided an excellent service to the anaesthesia community by sharing their findings from two healthy young women who experienced multi-vessel coronary spasm during spinal anaesthesia.1 The authors provided a concise explanation of how excessive vagal tone led to coronary spasm and myocardial infarction in two patients despite normal coronary arteries. It appears that myocardial ischaemia may not be a rare event in healthy patients during spinal anaesthesia. Palmer and colleagues2 studied the electrocardiographic changes in 93 patients during Caesarean delivery with T4 levels of spinal anaesthesia and observed changes that were consistent with ischaemia in more than one-third of patients. Fifteen of the patients with ECG changes also had symptoms of chest pain, pressure or dyspnoea. This suggests that, even with healthy patients, anaesthetists should be vigilant for signs of ischaemia with T4 levels of spinal anaesthesia.

These cases raise another issue that has not been given sufficient emphasis. As the extent of sympathetic block and the resulting vasodilation are related to the extent of the block, the dose of local anaesthetic should be limited to provide a level of anaesthesia that is appropriate for the surgery. While many factors can affect the ultimate spread of the block, the total dose of local anaesthetic has a profound influence on the extent and duration of the block. For pelvic and hip surgery, it is difficult to justify the 20 mg dose of plain bupivacaine that was selected for the two patients in this case report.1 Such a large dose increases the risk of vagal predominance and should not be necessary when an epidural...
catheter was available to augment the block, if necessary, later in the procedure. Similarly large doses of local anaesthetic are often reported to have been used in the cases where there is rapid onset of vagal symptoms during spinal anaesthesia. Lovstad et al. reported a series of four patients who experienced cardiac arrest following intrathecal injection of lidocaine 85–150 mg. A fifth patient arrested 12 min after administration of hyperbaric bupivacaine 16 mg. The vagal-enhancing effects of such large doses can be long-lasting. Ponhold and Vicenzi et al. observed that in a series of 60 patients who received 20 mg of spinal bupivacaine, there were 26 episodes of severe bradycardia (heart rate <50 beats min⁻¹) during the recovery period. These severe bradycardias occurred up to 5 h after arrival in the postoperative care unit. These episodes occurred with low sensory levels of spinal anaesthesia, demonstrating that both the extent and duration of the sympathetic block is far greater than the sensory block. Although determining the appropriate dose for a specific clinical situation is part of the art of anaesthesia, one may use objective information to help select the optimal dose for an individual patient. Reviewing textbooks or the package insert and tempering this with what is done in actual practice at other institutions can be useful. Most texts recommend a maximum of bupivacaine 15 mg or less for lower body surgical sites. For hyperbaric bupivacaine, the manufacturer recommends 9 mg for lower and 12 mg for upper body surgery. A review of over 2000 bupivacaine spinal anaesthetics in one setting revealed a mean dose of 14 mg was utilized. The 20 mg doses of isobaric bupivacaine that were given in the cases reported were higher than what is recommended, and more than two standard deviations above the mean dose used at the Mayo Clinic. Admittedly, even with standard dosing, a subset of patients will continue to experience both the more common and the more severe vagal related side-effects of spinal anaesthesia. Fortunately, with moderate dosing of our spinal anaesthetics and prompt use of i.v. atropine 0.4–0.6 mg at the onset of common vagal symptoms such as nausea or bradycardia, the incidence and permanent sequelae of the more severe side-effects can be minimized.

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Editor—We agree with Dr Pollard that high doses of intrathecal local anaesthetics speed the onset of anaesthesia and will therefore increase the risk of rapidly occurring vagal predominance, and that the dose of the spinal anaesthetic should be adapted to the field of surgery. Vagal predominance occurring with high sensory levels is known to be an important risk factor for cardiac arrest in the context of spinal anaesthesia, as is overdose of local anaesthetic drug. Coronary vasospasm with wide complex tachycardia and myocardial infarction is a less common and less well-known effect of vagal predominance under spinal anaesthesia, irrespective of the cause. We therefore focused the discussion of our two cases on this issue, and on the fact that the frequently occurring vagal symptoms—if not treated appropriately with atropine—may sometimes lead to potentially life-threatening complications. Thus, we did not comment on the potential role of the dose of bupivacaine.

The 20 mg dose in the second patient scheduled for hip arthroscopy was clearly too high. The first patient, however, was scheduled for pelvic osteotomy, where the surgeon gains access to the acetabulum from inside the pelvis, possibly causing compression of the lower peritoneum. At sensory levels of block of T10 or lower, patients may feel uncomfortable, so we recommend a level of T8 for this operation, which was not exceeded in this patient. Dose selection in spinal anaesthesia is affected by the unpredictability of the achieved level. The most important predictor of the extent of a spinal block is the volume of lumbar cerebrospinal fluid, which cannot be derived from any clinically available patient characteristic. Pargger and colleagues injected plain bupivacaine 18 mg intrathecally and observed a mean upper level of T6 with a large variation (SD 3.5 segments). In an earlier study, the mean sensory level after 25 and 30 mg of plain bupivacaine intrathecally was T3 to T4 with a similar SD. In a recent survey of 3315 patients, the most important risk factors for hypotension detected in a multiple logistic regression model were chronic alcohol consumption, history of hypertension, increased body mass index, block level >T6, and urgent surgery. There was no relevant difference between the mean doses of bupivacaine injected in the patients with and without hypotension (17.5 ± 17 mg; P = 0.01). As we aim at reaching the requested sensory level for surgery with the initial intrathecal dose of bupivacaine, we occasionally administer 20 mg of plain bupivacaine even in planned combined spinal–epidural anaesthesia, but it is the maximal dose injected intrathecally in our department. The unpredictable level of spinal anaesthesia achieved in an individual patient suggests that severe vagal side-effects could occur even at moderate or lower doses of spinal anaesthetics.

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