the tourniquet for both groups (T3).

The specific activity of citrate synthase (CS), enzymes of the mitochondrial electron transfer chain[1] and manganese-superoxide dismutase (Mn-SOD)[2] were evaluated in the mitochondrial fraction; the specific activity of copper, zinc-superoxide dismutase (Cu,Zn-SOD)[2] was determined in the crude extract (Table).

We observed no significant biochemical differences between patients submitted to GA and those submitted to SA. A significant decrease in CS activity, perhaps due to mitochondrial dysfunction and swelling,[3] was observed during ischemia (T2) from the T1 value, whereas an increase observed in CS activity at T3 suggests recovery due to reoxygenation.

An unexpected significant increase in the activity of the electron transfer chain enzymes was also observed during ischemia (T2) from the T1 values in both groups of patients. The significant reversal in this increase at T3 suggests the activation of a short-term regulatory mechanism during ischemia.

There were no significant changes in SOD enzyme activities during ischemia and reperfusion. This may be because, at T3, the muscle had not had enough time to produce enough free oxygen radicals to stimulate SOD activity.

The reversal at T3 of the increasing or decreasing tendencies at T2 of CS and electron transfer chain enzyme activities suggests the almost immediate onset of recovery from the combined effects of tourniquet induced ischemia and anesthesia. This should be of interest to orthopedic surgeons and to anesthesiologists.

The few data collected do not permit a firm conclusion on the effects of SA or GA. Since no difference was found between the two anesthetics in their effects on skeletal muscle oxidative metabolism, it may be that the main metabolic effects observed were due to ischemia rather than to anesthesia.

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Spinal anesthesia in an obese patient with osteogenesis imperfecta

To the Editor:
A 33-yr-old gentleman (body mass index of 44) with a history of osteogenesis imperfecta (OI) type IV was scheduled for dynamic screw fixation of a right femoral fracture sustained during an epileptic fit. He had significant co-morbidities. These included non-insulin dependant diabetes mellitus, hypopitutarism, obesity, and learning difficulties. He had limited mobility but clinically had no significant symptoms or signs relevant to his cardiorespiratory system. His medications included thyroxine, hydrocortisone, benzodiazepine, sodium valproate, triazine, carbamazepine, testosterone, and sulphonylurea. A preoperative full blood count, platelets, and coagulation screen were normal. He had consistently low serum sodium (124 $mmol \cdot L^{-1}$), chloride (90 $mmol \cdot L^{-1}$), and calcium (2.09 $mmol \cdot L^{-1}$). Such chronic electrolyte disturbances were due to the associated endocrine dysfunction.

As endotracheal intubation was expected to be difficult and hazardous, spinal block was chosen. Despite the associated kyphoscoliosis no difficulty was experienced finding the subarachnoid space using a midline approach with a 25-gauge spinal needle. Three millilitres of L-bupivacaine and 10 μg of fentanyl produced anesthesia to the level of T8 bilaterally. Temperature was monitored in addition to routine ASA monitoring. The operation lasted for three hours and the patient made an uneventful recovery.

Skeletal abnormalities of the lumbar spine, as in OI, are considered relative contraindications to spinal anesthesia. Associated cardiac lesions and a coagulopathy may also present problems. Management of the difficult airway due to abnormal cervical spine mobility, fragile teeth and the risk of mandibular and facial fractures are the risks associated with general anesthesia. The use of anticholinergics may exacerbate the risk of hyperthermia. Although general and epidural anesthesia have been reported in patients with OI,[1,2] the use of spinal block is infrequent. This is possibly due

to technical difficulties, but more importantly due to the difficulty in predicting the spread of local anesthetics. Administration of anesthesia to a patient with OI is a rare and challenging occasion for an anesthesiologist. The problems directly and indirectly associated with the disease affect the anesthetic management. With careful attention to the associated problems, spinal anesthesia appears to be a safe technique and was associated with a favourable outcome in our patient.

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Spinal epidural hematoma and epidural analgesia

To the Editor:
The controversy surrounding perioperative thromboprophylaxis and neuraxial analgesia continues and will likely accelerate with the introduction of newer and more powerful thromboprophylactic agents.[1,2] To complicate matters further, we do not have a good appreciation of the pathogenesis of spinal epidural hematomas or their ideal management[3] and there are several other possible explanations for conclusions drawn in the literature and published as guidelines.

It has been assumed that venous bleeding is the principle source of blood in an epidural hematoma. Why then, is there such a great discrepancy between the frequency of occurrence of blood via needle and/or catheter and the occurrence of a symptomatic epidural hematoma?[1,4]

Traumatic insertion of an epidural needle is an independent risk factor for epidural hematoma formation.[5] However, whether "trauma" increases risk via a relatively common phenomenon (e.g., venous bleeding) or some other, unknown, risk factor (e.g., arterial injury) is not known. Is the correlation between epidural catheter removal and epidural hematoma formation[5] evidence that significant trauma occurs on removal or can it be explained as a delayed presentation of a hematoma formed earlier?[3]

Anticoagulation is associated with increased frequency of epidural hematoma formation.[5] However, this correlation may exist regardless of whether bleeding is principally venous or arterial in origin. The practice of performing neuraxial techniques in patients who are subsequently fully anticoagulated continues and appears to be safer with each case done.[6]

Understanding the pathogenesis of spinal epidural hematoma formation and its role in the subsequent clinical syndrome that may ensue is vitally important. For example, if we learn that we must avoid contact with an arterialized vessel in the path of an epidural needle, we may be able to introduce measures that specifically address that risk (e.g., the use of Doppler ultrasound).[7] Without that knowledge, however, we are reduced to fumbling in the dark, multiple opinions, and great variation in the accepted standard of care.

All interested individuals are invited to attend the Regional Anesthesia Section Breakfast at the 2003 Canadian Anesthesiologists Society meeting in Ottawa where a panel of experts will be discussing this topic of growing concern to anesthesiologists.

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