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 management³ 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 formation⁵ evidence that significant trauma occurs on removal or can it be explained as a delayed presenta-

tion 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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Analgesic effect of intra-articular tramadol compared to morphine after arthroscopic knee surgery

To the Editor:

Intra-articularly administered morphine has been studied extensively and shown to have a definite but mild analgesic effect.¹ Although parenteral tramadol, an analgesic drug with some α_2 -adrenergic and opioid receptor agonist activity, produces analgesia equivalent to parenteral morphine in moderate postoperative pain, the efficacy of intra-articular (IA) tramadol remains unclear.² We compared the analgesic effect of IA morphine with IA tramadol. Seventy-five patients, having elective arthroscopic surgery of the knee were randomized to receive either IA tramadol 50 mg, IA morphine 5 mg or IA normal saline 20 mL. The tourniquet was released ten minutes after analgesic administration. Exclusion criteria were preoperative narcotic use, contraindication to narcotics, seizure disorder, anterior cruciate ligament reconstruction, and traumatic injury to the knee.

We did not observe any differences among the three groups with respect to age, gender, weight, duration of surgery, surgical procedures, laser use during surgery,

duration and severity of preoperative knee pain, time to discharge or side effects. There was also no difference with respect to pain scores or the use of rescue analgesics postoperatively, except for the first pain score when the patients arrived to the postanesthesia care unit and the time to first rescue analgesic (Table).

The dose and volume of morphine used in this study was based on our previous experience.³ We believe duration and severity of the preoperative symptoms and pain can change the effectiveness of the postoperative analgesic. There was a good correlation between the duration of preoperative pain symptoms and time to first analgesic requirement after the operation ($r^2 = 0.731$, $P < 0.0001$). Experimental and clinical evidence shows that analgesic effects of peripheral opioids and the density of opioid receptors on peripheral sensory nerves increase with the duration of painful inflammatory processes.⁴ After examining different time intervals, in the subgroup of patients with more than six months of preoperative pain (14 patients in the morphine and the control groups, ten patients in the tramadol group), the analgesic effects of IA narcotics were more prominent (Table). These patients benefited from the IA administration of tramadol or morphine, having had a chance to increase the expression of peripheral receptors.

We conclude that morphine and tramadol, at the doses used, provide similar analgesia when given intra-articularly. The mechanisms of action of IA opioids are yet to be elucidated.¹ Further studies are needed to compare the adverse effects between IA morphine and tramadol. We believe IA tramadol can be an alterna-

TABLE Patient data

	Control (n = 25)	Morphine (n = 25)	Tramadol (n = 25)
Age (yr)	41 ± 17	34 ± 15	34 ± 17
VRS at rest (0 min)	7 (4-10)	1 (0-9)*	0 (0-9)*
VRS at rest (15 min)	6 (0-10)	5 (0-10)	5 (0-9)
VRS at rest (30 min)	6 (1-10)	5 (1-9)	6 (1-9)
VRS at rest (45 min)	6 (0-10)	4 (1-9)	6 (1-9)
VRS at rest (1st hr)	5 (0-9)	4 (0-9)	5 (0-8)
VRS at rest (2nd hr)	3 (0-8)	3 (0-9)	4 (0-8)
VRS at rest (6th hr)	4 (0-5)	3 (0-8)	2 (0-8)
VRS at rest (24th hr)	2 (0-7)	1 (0-9)	1 (0-7)
Time to first analgesic (min)	25 (15-55)	34 (15-158)*	33 (17-728)*
Time to first analgesic (preoperative pain ≥ 6 months)	25 (15-45)	35 (15-160)*	60 (26-746)*
No analgesics needed	3 (12%)	5 (20%)	5 (20%)
Opioids needed in PACU	18 (72%)	13 (52%)	12 (48%)
Opioids needed in PACU (preoperative pain ≥ 6 months)	12 (86%)	8 (57%)*	3 (30%)*
Number of opioid boluses	1 (0-5)	1 (0-3)	1 (0-3)
Dipyrone needed in 24 hr	4 (16%)	7 (28%)	8 (32%)
Tablets of dipyrone in 24 hr	2 (0-5)	1 (0-3)	1 (0-7)

Pain was assessed by verbal rating score between 0–10 (VRS). Results are given as number of patients (%), mean ± SD or median (95% CI). PACU = postanesthesia care unit; * $P < 0.008$ compared to control, $P > 0.05$ between morphine and tramadol.