⌂ Letters to the Editor

Unilateral Tremor of the Upper and Lower Limb After an Axillary Brachial Plexus Block

To the Editor:

We read with interest the case of Moorthy and Dill[1] who reported a tremor of the forearm during performance of axillary brachial plexus block. We would like to describe a case that developed unilateral tremors that were not limited to upper limb but extended to the lower limb after an axillary plexus block using Xylocaine (AstraZeneca AB, Södertälge, Sweden)-bupivacaine mixture.

A right-handed, American Society of Anesthesiologists 1, 29-year-old man, weighing 70 kg, presented for right-hand arthrotomy. He had no significant history of any disease. Hemoglobin, electrolytes, and plasma glucose values were normal. Standard monitoring was applied and with the help of nerve stimulator 50-mm needle (22-G; Stimuplex, B/Braun, Melsungen AG, Germany), an axillary block was performed by using 35 mL 0.5% bupivacaine/2% Xylocaine (isobaric mixture) without epinephrine. A blood aspiration test was negative. Blood pressure and heart rate were respectively 130/85 mm Hg and 65 beats/min. A few minutes later, the patient developed a unilateral tremor on his right arm and right leg. The patient was unable to control and/or cease these involuntary movements. The patient was fully cooperative, and these movements were not associated with tinnitus, metallic taste, convulsion, or visual disturbance. This tremor lasted about 3 minutes and ceased immediately after administration of 3 mg midazolam.

Moorthy and Dill[1] attributed the tremor either to peripheral effect or to central mechanism. The occurrence of tremor in both the upper and lower limb in our patient, as well as the immediate relief after the administration of midazolam, supports a central mechanism or possibly a psychogenic one. It is possible that tremor may be a sign of central nervous system reaction after local anesthesia administration and early administration of midazolam may be recommended to abolish tremor.

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Accepted for publication January 12, 2005.
doi:10.1016/j.rapm.2005.01.001

Re: Combination of Intraneural Injection and High-Injection Pressure Leads to Fasicular Injury and Neurologic Deficits in Dogs

To the Editor:

With great interest, I read the experimental study by Hadzic et al.[1] on pressure recordings and other effects of intraneural injections in dogs. To some extent, the study is a repetition of an earlier study by Selander and Sjöstrand,[2] and the results of the two studies basically seem to agree. However, the terminology and methodology used in Hadzic et al.'s study raise some questions.

The authors measured the injection pressures in PSI (pounds per square inch), which is outside the SI system. Why not use mm Hg, which still is the international standard unit for human physiological pressures (e.g., arterial blood pressure)? The use of PSI makes the results rather difficult to interpret (e.g., when considering the effects of intraneural pressure on neural blood circulation).

Unfortunately, the authors use somewhat confusing descriptions of the intraneural needle positions. Their terminology deviates from the standard nomenclature for the peripheral nerve anatomy, which was introduced by Key and Retzius in 1876,[3] and which, for the sake of clarity, is described in the next paragraph.

A peripheral nerve consists of a number of fascicles held together by an external sheath called the epineurium (which includes the tissue between the fascicles). Each fascicle is surrounded by a strong and protective sheath called the perineurium, which has some specific qualities, (e.g., it constitutes the outer part of the blood-nerve barrier [the inner part is the endothelium of the intrafascicular blood vessels]). Each fascicle contains a varying number of nerve fibers embedded in an inner supportive and well-perfused sheath called the endoneurium. Thus, an intraneural needle tip can be positioned epineurially (i.e., in the epineurium) or endoneurially (i.e., in the endoneurium; = intrafascicularly). The word perineural usually indicates a needle position close to/just outside the nerve, whereas perineurially means a localization within the perineurium. However, in Hadzic et al.'s text "perineurally" means a position "within the epineurium, but outside the perineurium," and "intraneurally" means "within the perineurium." It would be more consistent and easier to understand the needle-tip position if the anatomically adequate standard nomenclature had been used.

To illustrate the "pathohistologic data" (= histopathological), the authors took "a series of tissue slices approximately 1.5 cm (not inches!) proximal and distal to the injection site" (transverse or longitudinal slices?). The article includes only one "microscopic image of a fascicle after an intraneural injection under high pressure." This

microphoto is said to show "degeneration of axons and marked cellular infiltration" but it is not very clear, and there is no detailed description of what it shows, although there seems to be no traces of the normal nerve structures. It would have been good with a control image of a normal nerve and perhaps also a closer discussion on the possible pathogenesis of the detected nerve lesions. There may also be some other questions, but to me these seemed most important.

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2. Selander D, Sjöstrand J. Longitudinal spread of intraneurally injected local anaesthetics. *Acta Anaesth Scand* 1978;22: 622-634.
3. Key A, Retzius G. *Studien in der Anatomie des Nervensystemes und des Bindegewebes* (*2. Hälfte*). Stockholm: Samson & Wallin; 1876.

Accepted for publication January 19, 2005.
doi:10.1016/j.rapm.2005.01.003

Reply to Dr. Selander

To the Editor:

We thank Dr. Selander for his comments on our article[1] and for bringing up some important concepts. However, we must emphasize that our study was not a repetition of their earlier study.[2] As the title of their article indicates ("Longitudinal Spread of Intraneurally Injected Local Anesthetics") Drs. Selander and Sjostrand in their work sought to explain development of total spinal anesthesia after interscalene block and not to study neurologic outcome after intraneural injections. Their methods comprised a small-animal model, microinjections (10 to 200 μmL), miniature needles, clinically irrelevant injection rates (100 to 300 μmL/min) and did not include neurologic evaluation. For these reasons, their work has limited clinical relevance with regard to the dynamics and outcome of intraneural injection pressures. In contrast, our large-animal model was specifically designed to study injection pressures, neurologic and histologic consequences of intraneural injections by use of clinically more relevant needles, rates of injection, and volumes of injectate.[3] In addition, Dr. Selander's data suggested that intraneural injection results in sustained intraneural pressure that exceeds neuronal capillary perfusion pressure (50 mm Hg), neuronal ischemia, and, consequently, nerve injury. In contrast to this generally accepted concept,[4] we did not find sustained intraneural pressures. In our studies, once the intraneural injection was initiated and the perineurium ruptured, the anesthetic solution freely escaped into the subepineurial space and resulted in rapid return of the pressures to baseline (Fig 1). In contrast to the focus on the ischemic nature of nerve injury in Dr. Selander's study, our study suggested that high injection pressures (>20 psi) predicted intraneural injections and mechanical, rather than ischemic, nerve injury. The likely reason for this disparity between our studies and the study of Selander and Sjostrand is our use of a more clinically relevant intraneural injection model.

Although Dr. Selander's criticism of our use of psi (pounds per square inch) instead of mm Hg (which is

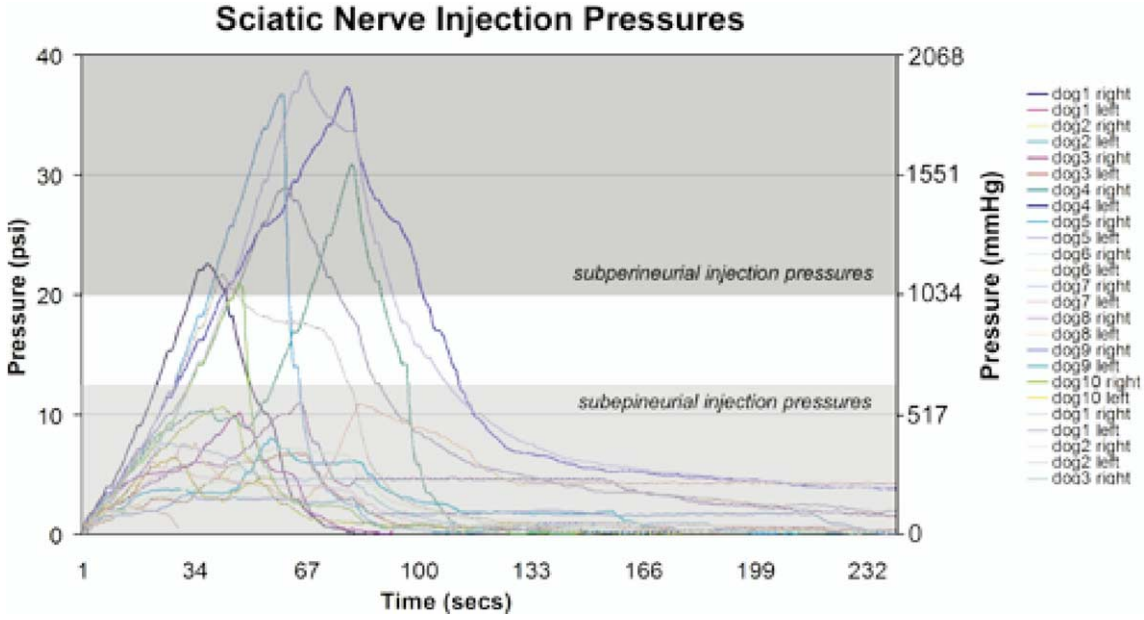

Fig 1. Sciatic nerve injection pressures.