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Regional Anesthesia, Intraneural Injection, and Nerve Injury

Beyond the Epineurium

NERVE damage after regional anesthesia is appropriately regarded as a major complication and, when the injury is severe, may take weeks or even months to recover completely.[1,2] There are many possible causes for such injuries.[3,4] These include stretching, compression, ischemia, surgical trauma, and local anesthetic toxicity.[5–7] One causative factor that has been the subject of intense discussion involves the direct intraneural injection of local anesthetics. The deleterious effect of such injections was demonstrated by Selander *et al.*[3] nearly 30 yr ago. Since that time, we have been advised to avoid direct contact between the needle and nerve and to think of the epineurium as a barrier that we should not cross. One consequence of this advice has been a move away from "seeking paresthesias" during the performance of blocks and the use of electrical stimulation and evoked motor responses to estimate proximity to the nerve. However, in this issue of ANESTHESIOLOGY, Dr. Bigeleisen[8] has challenged the idea that intraneural injection is uniformly damaging and is to be avoided at all costs.

In this study, videography and ultrasonography were used to assess local anesthetic distribution when axillary brachial plexus block was performed according to his usual practice, which was seeking paresthesia by needle manipulation. When paresthesia was established, 2–3 ml local anesthetic was administered. If the injection appeared intraneurally, the needle was withdrawn until it appeared outside the nerve, the injection was continued, and the block was completed. The patients were checked 6 months later for the occurrence of neuropathy. The results of the study were surprising: 22 of 26 patients (85%) had nerve puncture of at least one nerve, and 21 of 26 patients (81%) had an intraneural injection of at least one nerve. Assessment 6 months later showed no clinical evidence of nerve damage. Two important new considerations emerge from this investigation: First, intraneural injection of local anesthetic, at least in a small volume, does not seem to result in nerve damage, and second, performance of the paresthesia technique does result in frequent intraneural injection.

The belief that administration of local anesthetic inside the epineurium uniformly results in nerve damage should be reconsidered in view of Bigeleisen's results.[8] The study showed that injection of local anesthetic (2–3 ml) inside the epineurium does not result in severe nerve damage. Some minor, transient neurologic symptoms may have occurred between block performance and neurologic assessment at 6 months and may have been unrecognized, but the occurrence of severe nerve damage would most likely have been brought to the attention of the author or detected by the surgeon.

Ultrasonographic resolution does not allow us to differentiate between an injection into the subepineurium or subperineurium. The perineurium, in contrast to the epineurium, is a tough and resistant tissue withstanding very high pressure.[3] The ability to expand the nerve, as shown in figure 2B in Bigeleisen's article,[8] suggests that the needle lies in a compliant space between the epineurium and perineurium. However, the main issue coming from this investigation is that the barrier that should not be penetrated to avoid severe neural damage is likely the perineurium. The next question, which cannot be answered, is how much volume can be placed in this space until the pressure increases and adversely affects the blood supply. A study will be needed to clarify this question.

A more recent study by Hadzic *et al.*[9] further evaluated the consequences of either subepineurium or subperineurium injections in dogs. In this study, the authors placed the tip of the needle under microscopic control either around the epineurium or intraneurally by piercing the epineurium. In the control group, injection pressures were low (< 4 psi) in all animals. In the intraneural group, the authors were able to distinguish two subgroups: one with a moderately increased injection pressure, and the other with a very high injection pressure (25–45 psi). After the dogs awakened from general an-

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esthesia, motor function returned to normal within 3 h in all animals, except for those with very high injection pressures. In this subgroup, severe and persistent motor deficits were recorded, with varying degrees of damage to the neural architecture. The weakness of this investigation resides in the absence of proof that the needle was effectively placed subperineurally. Similar studies using electronic microscopy and injection of dye should be able to confirm these suppositions.

Another interesting point made by Bigeleisen[8] is the apparent high frequency of subepineurium local anesthetic deposition when using the paresthesia technique for performing peripheral nerve block. These findings give support to those promoting the use of electrical nerve stimulation. However, the volume of the injection that enters subepineurally is unknown in this context, but this observation may explain the greater incidence of minor neurologic symptoms observed by some authors using the paresthesia technique[10] and the observation that the incidence of severe neurologic complication is not greater when using the paresthesia technique compared with electrical nerve stimulation.[10] Therefore, intraneural injection may not cause severe neurologic deficits and might be explained by the relatively good tolerance of low or moderate volume of local anesthetics between the epineurium and perineurium. Another interesting finding reported by Bigeleisen[8] is the heterogeneous description of symptoms observed after eliciting paresthesia—a phenomenon that is poorly explained. It must be emphasized that the possibility to "contact" the nerve without eliciting any paresthesia or dysesthesia may occur. This phenomenon has occasionally been reported in the literature.[11]

This investigation has some limitations. Detractors will criticize the current study because of its relatively small sample size and the lack of any neurologic assessment until 6 months after the injection. It has been demonstrated that most peripheral nerve injuries are transient after regional blocks and resolve within a few weeks after the injury.[1,2] However, this study raises pertinent questions about the importance of penetrating the epineurium and nerve damage during regional anesthesia.

In summary, for neurologic complications from regional anesthesia, the belief that the epineurium as the last barrier should be balanced—local anesthetics should be injected outside of it— but we should recognize that some local anesthetics can be injected without uniformly damaging the nerve. Evidence is growing that the key barrier is the perineurium. The work performed by Bigeleisen[8] contributes to this understanding. However, this new information should not yet change our clinical practice: Nerves should be treated with care, and the basic rule not to inject local anesthetics into the nerve remains.

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