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New procedure

Neural blockade and nerve damage – changing concept

ALAIN BORGEAT

Department of Anesthesiology
Balgrist University Hospital
Forchstrasse 340
CH-8008 Zurich / Switzerland
E-mail: alain.borgeat@balgrist.ch

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Nerve injuries are a well-recognized complication of anesthesia *(1)*. Postoperative brachial plexopathy has been documented in the literature for more than 100 years. Postoperative brachial plexopathy is generally believed to be a result of traction injury of the nerves with compression a contributing factor. Both stretching and compression of the nerve ultimately lead to ischemia of the vasa nervorum and subsequent injury to the nerve. In addition, there may be rupture of intraneural capillaries and hematoma formation with further compression. Several factors have been associated with intraoperative brachial plexus injury, including concomitant patient disease, anatomical variations, positioning of the patient, surgical factors and physiological factors.

The frequency of peripheral neuropathies reported after regional anesthetic blockade is generally low and varies from zero to more than 5% *(1)*. Lesions to the brachial plexus seem to be reported most frequently *(2, 3, 4),* but injuries of the sciatic and phrenic nerves have also been reported. The high incidence of postanesthesia neuropathies in the upper extremity probably reflects the relatively high rate of hand and arm lesions and the concomitantly frequent use of brachial plexus blocks for surgery.

The symptoms of a nerve lesion can appear within a day or two but sometimes may not become apparent until 2 or 3 weeks after the injury *(5)*. This variation in time to debut is likely to depend on the origin of the nerve lesion and on confounding factors such as »normal« postoperative pain and effects of surgery, position, plaster casts, and other bandaging. The intensity and duration of symptoms vary with the severity of the injury, from light, intermittent tingling and numbness lasting of few weeks, to persistent, painful paresthesia, neuropathic pain, sensory loss, and motor weakness lasting several months or years, sometimes developing into distressing complex regional syndrome type I or II *(5, 6, 7)*.

Neurologic sequelae after a local anesthetic blockade basically depend on three classes of insult: trauma, toxicity, and ischemia. In most cases, the lesion is probably caused by the combined action of two, or all three, of these factors *(8)*.

Trauma to the nerves can be caused by the injection needle, intraneural injection, and compression or stretching due to other external factors such as patient position and effect of retractors. The search for paresthesias can increase the risk of postblock neuropathies was found by Plevak *et al. (9)* in prospective clinical studies. This report revealed a trend for a paresthesia-nerve lesion relation, and metaanalysis of different studies demonstrated that the frequency of post-anesthesia neuropathy was significantly higher ($P<0.05$) when paresthesia techniques were utilized, compared with "nonparesthesia" techniques *(10)*.

Painful paresthesias on injection indicate intraneural needle position. Such injections are initially painful, but as the local anesthetic very quickly blocks the nerve, the pain fades and information regarding the nerve injury disappears until the block wears off. Thus, it is important that the anesthesiologist maintain adequate contact with the patient and react to such information by immediately stopping the injection to reposition the needle away from the nerve.

The destructive effects of intrafascicular injections are probably due to a combination of three factors. First, direct needle trauma leads to perforation of the perineurium and other nerve sheaths, cutting and disrupting the nerve fibers and compromising the microvasculature, with a risk of intraneural or intrafascicular bleeding and hematoma *(11)*.

All local anesthetics are potentially neurotoxic, and the neural blockade can be seen as a reversible expression of this. Commercial local anesthetics are supplied in concentration that under normal conditions do not cause nerve damage; however, the use of higher concentrations and intraneural injections, especially when epinephrine has been added, can result in severe damage with persistent dysfunction and pain *(6, 13)*.

Different factors may be involved in neurological complications after peripheral nerve blocks

– the tourniquet

– the surgeon. Surgery is implicated in neural damage secondary to traction and/or compression. Experimental studies have shown that stretch and compression act synergically for nerve damage.

– the anesthetist. The following rules should be applied to avoid anesthetist induced neural damage

- do not touch the perineurium
- do not go into the peri-endoneurium space
- do not inject local anesthetics into the nerve *(15)*

The occurrence of paresthesias is a warning signal that any of the above may occur. Moreover, the use of the lowest concentration of the friendliest local anesthetic is recommended. Therefore paresthesias may be considered as a risk factor for neural damage. It means in the majority of cases the needle is too close to the neural structure. A block can be safely performed without taking this unnecessary risk.

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