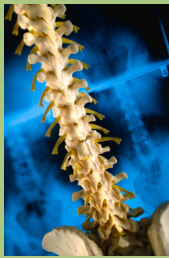


# AnesthesiaDotCalm Newsletter

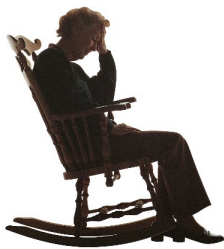


News You Can Use

Mar 31, 2007

## Spinal Anesthesia for the Geriatric Patient

Age and change are inseparable. They are like politicians sharing the same dais and orating at the same time. The cacophony of their intermingling is a conundrum.



Philosophers want to find meaning in the interaction and scientists seek to separate the two. Of the myriad of changes that accompany aging the most obvious in-

volves one's physicality. I can personally attest to that, for now at the age of 61, as I look into the mirror I see my father staring back at me. This is not a bad thing. My father, even in his waning years, is (in my opinion) a rather good looking man. My brother is not so fortunate. He looks more like my dog. But I digress.

As we age, we tend to move slower. We are less flexible and our body movements are more rigid. Scientists have been able to peek behind the grand drape of this phenomenon called aging and have given us some insight into this process. They have found, for example, that action potentials decrease with age in both the peripheral nervous system and the central nervous system. This they contribute to the poorer vascular perfusion of the cell body as well as damage to the myelin sheath. The number of spi-

nal cord motor neurons and functioning motor units also declines with age, by as much as 50% after age 60 years.(1) And unfortunately, this age-associated denervation is thought to be continuous and irreversible,(2) causing the muscle fibers to atrophy and motor neurons to eventually die.(3)

Knowing these things makes me want to stay in bed. It also gives me some degree of consternation especially when it comes to the decision to providing spinal or epidural anesthesia to members of this aging population. After all, these changes can contribute to altered nerve block characteristics following epidural and subarachnoid administration of local anesthetics. And let's just face it, local anesthetics in the laboratory setting have been proven to be quite toxic.

Drug toxicity to the spinal cord or nerve roots can manifest itself in basically three ways: histologic, physiologic, or behavioral/clinical derangements. Histologic refers to neural injury, gliosis, and/or damage to the myelin sheath; it also describes inflammatory changes and involvement of the arachnoid cell layers. Physiologic neurotoxicity of spinal drugs includes changes in spinal cord blood flow,

disruption of the blood-brain barrier, and changes in the electrophysiology of impulse conduction. And histologic and physiologic changes can manifest themselves in behavioral and clinical signs such as pain, motor and sensory deficits, and bowel and bladder dysfunction.

But anesthesia providers have not always been concerned about the toxic reaction of these drugs. Consider that the evolution of spinal local anesthetic use in humans typically involved self-experimentation, followed by widespread application with little or no controlled testing for neurotoxicity. In 1898, Bier and Hildebrandt (4) were the first to perform spinal anesthesia and this they did using cocaine which they administered to each other. Essentially all of the earliest local anesthetics for spinal anesthesia were introduced in this fashion without toxicity studies. But all this changed by the middle of the twentieth century.

In 1985, Ready et al. (5) evaluated the neurotoxic effects of single injections of local anesthetics in rabbits. They reported that spinal cord histopathology remained normal and that persistent neurologic deficits were not seen with clinically used concentrations of tetracaine, lidocaine, bupivacaine, or chlorprocaine. However, histopathologic changes and neurologic deficits did occur with higher concentrations of tetracaine (1%) and lidocaine (8%). More recently, studies have used desheathed peripheral nerve models, designed to mimic unprotected nerve roots in the cauda equina, to further assess electrophysiologic neurotoxicity of clinically relevant concentrations of local anesthetics (4,5,6). These models demonstrated that clinically used concentrations of 5% lidocaine and 0.5% tetracaine caused irreversible conduction block, whereas 1.5% lidocaine, 0.75% bupivacaine, and 0.06% tetracaine did not. It was further elucidated that the electrophysiologic toxicity of lidocaine in these models is both concentration-dependent and time-dependent(6)

Effects of local anesthetics on spinal cord blood have been found to be equivocal. Spinal administration of, mepivacaine, and tetracaine causes vasodilation and increase spinal cord blood flow (7,8,9), whereas bupivacaine, lidocaine and ropivacaine may or may not cause vasoconstriction and reduction in spinal cord blood flow (10). For example, using a radioactive microsphere technique, Kozody and colleagues found that when 100 mg of lidocaine with and without epinephrine were given intrathecally to 12 mongrel dogs spinal cord hyperemia occurred in the plain lidocaine group whereas as epinephrine mitigated these findings(11). Myers(12), on the other hand, using solutions of isotonic sodium chloride solution, 1% and 2% lidocaine with and without epinephrine, and epinephrine alone applied to isolated rat sciatic nerves found that blood flow was significantly depressed for all solutions except isotonic sodium chloride solution. Epinephrine by itself significantly reduced nerve blood flow, and, when added to local anesthetic solutions, it reduced blood flow to a greater extent than the reduction caused by local anesthetics alone.

Bupivacaine, too, has been implicated in reducing spinal cord blood flow. Kozody and his group(13) again using a radioactive microsphere technique in mongrel dogs found that subarachnoid bupivacaine with epinephrine produced a significant decrease in thoracic and lumbosacral spinal cord blood flow; however, cervical cord blood flow remained unchanged. Thoracic and lumbosacral dural blood flows were significantly decreased in both groups following subarachnoid injection.

Although experimental studies in animals have provided ample evidence that some local anesthetics in clinically relevant concentrations can injure nerve tissue, the exact mechanisms of injury are unclear. Recent work on neuronal cell lines has attempted to determine the mechanism of local anesthetic neurotoxicity. Johnson and Uhl (14) have shown that direct application of 2.5%–5.0% lidocaine caused a greater than 3-fold increase in intracellular calcium

and up to a 20% incidence of cell death during 60 min of exposure in the neuronal cell line. They postulated that the mechanism of neurotoxicity was not likely from sodium channel blockade, because such a block would not lead to an increase in cytoplasmic calcium. Subsequent work in this model determined that 0.5% and 1.0% lidocaine, as well as 0.625% bupivacaine, lead to transient, moderate increases in calcium, probably from the endoplasmic reticulum, without cell death (15). Thus, several different laboratory models have proven that all local anesthetics can be neurotoxic but that lidocaine and tetracaine are potentially more neurotoxic than bupivacaine

Despite the knowledge that all local anesthetics can be neurotoxic in the laboratory model, large-scale surveys of the complications of spinal anesthesia attest to the relative safety of spinal local anesthetics in humans. Retrospective (16), prospective (17) and historical studies (18,19,20) report 0%–0.7% incidence of postoperative neurologic injury in patients undergoing spinal anesthesia. Moreover, information from closed-claims databases corroborate these findings (21,22). Thus, the neurotoxic potential of spinally administered local anesthetics has not manifested itself in large-scale studies. Ultimately, what this means to the individual anesthesia practitioner boils down to personal experience. As for me, I'm going to get a Scotch.

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## Spinal anaesthesia with 0.5% hyperbaric bupivacaine in elderly patients: effect of site of injection on spread of analgesia

**Veering BT, Burm AGL, Stienstra R, and van Kleef JW. *British Journal of Anaesthesia*, Vol 77, Issue 3: 343-346,**



In this randomized, observer-blind study, the authors examined, in elderly patients, the effect of site of injection on analgesia levels after spinal injection of 0.5% hyperbaric bupivacaine

solution. Thirty male patients, aged 68-87 yr, undergoing minor urological surgery during spinal anaesthesia received 3 ml of a 0.5% hyperbaric bupivacaine solution at either the L3-4 (n = 15) or L4-5 (n = 15) interspace. The solution was injected with patients in the sitting position; they remained sitting for 2 min and were then placed in the supine horizontal position. Analgesia levels were assessed bilaterally using pin-prick. The highest analgesia levels did not differ between groups (medians were approximately T7). There were no significant differences in the time to maximum cephalad spread of analgesia, maximum degree of motor block or hemodynamic changes. The authors concluded that injection at the L4-5 interspace has no advantage compared with injection at the L3-4 interspace. Moreover, recovery characteristics did not differ between the groups.