cauda equina syndrome have been reported associated with the use of small-bore catheters, but only one case since 1984 associated with a larger catheter. However, do we have any information on the frequency with which continuous spinal anesthesia has been used since the advent of smaller catheters, compared with the previous era when the idea of making an 18-G or larger dural puncture made continuous spinal anesthesia a less attractive anesthetic alternative? Do we know the relative frequency of use of small-bore *versus* larger-bore catheters since microcatheters became freely available? Might not the surge in awareness of the risk of cauda equina syndrome with the technique since 1991 have produced an increase in reporting of a complication that may often have escaped the attention of anesthesiologists in the past?

I am concerned that the anesthesia community might be distracted by the focus on microcatheters and their withdrawal by the FDA, from the wider and perhaps more relevant question of safe *versus* unsafe ways to administer a continuous spinal anesthetic through a catheter of any size. The current response of regulatory bodies both in the United States and in Australia has done little to prevent the administration of hyperbaric local anesthetic solutions through larger-bore catheters (such as the 20-G epidural catheters that have been used intrathecally for years) by practitioners who are not well informed about the important issues that Rigler *et al.* and Rigler and Drasner have raised.[1,2]

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*In Reply:*—We appreciate Peyton's comments. We share his concern that the current focus on microcatheters and their withdrawal by the Food and Drug Administration not distract us from considerations more fundamental to the safe intrathecal administration of local anesthetic.

As his letter suggests, recent regulatory decisions have not (nor could they have) eliminated the risk of injury—to avoid injury requires understanding the factors that contribute to neurotoxicity and informed clinical decisions. A small diameter may, in fact, adversely affect distribution; however, maldistribution can occur with catheters of any size. As Peyton notes, in our model, administration of anesthetic through any of the sacrally directed catheters resulted in high "subarachnoid" concentrations; clinically, maldistribution has been documented with catheters as large as 3.5 French.[1] Even if the relative incidence of neurotoxic injury were to differ according to catheter size, risk remains—indeed, one of the 12 cases occurred with a large-bore catheter. And, as Peyton points out, recent regulatory decisions do not prevent the administration of hyperbaric local anesthetic through large-bore catheters.

Moreover, maldistribution occurs not only with catheters of any size, but also with any spinal technique. Maldistribution is perhaps the most common cause for a failed single-injection spinal anesthetic; because repeat injection to overcome failure has the potential to produce the same restricted distribution, if maldistribution is the etiology for failure, there is risk of neurotoxicity.[2]

The events of the last 3 yr also have identified other issues relevant to the safe practice of spinal anesthesia. For example, there is a substantial body of evidence to suggest that neurotoxicity is concentration-dependent.[3,4] Consequently, we have suggested that anesthetics be administered at their lowest effective concentration.[5] Thus, the use of 5% lidocaine for either continuous spinal anesthesia or single-injection spinal anesthesia should be reconsidered since this concentration far exceeds what is needed for adequate blockade. Similarly, consideration should be given to administering anesthetics at the lowest effective tonicity, since tonicity may be an important factor in neurotoxicity.[6,7]

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# Regulation of Skeletal Muscle Acetylcholine Receptors

*To the Editor:*—In a recent review it is stated that if an upper motor neuron (UMN) dysfunction is bilateral and the patient is tetraparetic, "The upper limb muscles relative to the lower appear more sensitive to nondepolarizing muscle relaxants (NDMR)."[1]

To my knowledge, the only report that might confirm this statement refers to patients with syringomyelia in whom the motor deficit of the upper limbs is caused by a *lower* motor neuron (LMN) dysfunction.[2]

I wish to stress this point because, according to the authors of the review, any evidence of muscle denervation should be accompanied by hyposensitivity to NDMR, and any hypersensitivity exhibited by denervated or injured muscles should be regarded as a "contradictory finding."

I think that any evidence of axonal sprouting (even that occurring after muscle injury[3]) should be regarded as a predisposing factor to the subsequent development of hypersensitivity to NDMR.

As mentioned by the authors,[1] a definite hyposensitivity to the action of d-tubocurarine has been observed in the gastrocnemius muscle of the rat 14 days after a 75–80% transection of the sciatic nerve.[4] It also should be remembered, however, that a definite hypersensitivity to the action of the same drug has been demonstrated in the anterior tibial muscle of the rabbit 42 days after the peroneal nerve has been crushed and then allowed to regenerate.[5]

A distinction, therefore, should be made between the early effects produced by denervation and the late effects produced by reinnervation:[6] by the reduced amounts of acetylcholine (ACh) released by intact terminals,[7] and by the low synaptic efficacy of neuromuscular junctions in multiply innervated muscle fibers.[8]

So far as hyperkalemic accidents are concerned, I think that the distinction to be made in patients with UMN dysfunction is not between congenital and acquired lesions, but between lesions that cause muscle *decentralization*[9] and lesions that cause decentralized muscles to be transiently *denervated*.[10]

Diaschisis phenomena, which are a frequent complication of acute UMN lesions, may result in transient states of functional denervation that may cause an extrajunctional proliferation of ACh receptors (AChRs)[11] similar to that induced by sepsis or by chronic treatment with NDMR.

Such an *UMN* dysfunction, which may be unrecognized in comatose patients,[12] should be considered an important factor to which ascribe hyperkalemic accidents caused by succinylcholine in patients recovering from acute UMN lesions.

If it is considered, on the other hand, how frequently decentralized muscles of hemiparetic or tetraparetic patients have been challenged with an intubating dose of succinylcholine in the past three decades, the likelihood of any correlation between UMN dysfunction and hyperkalemia appears to be very poor[9,13] (in the absence of other predisposing factors such as diaschisis, sepsis, and chronic treatment with NDMR).

These observations indicate that extrajunctional proliferation of AChRs is not a normal consequence of UMN dysfunction. They also suggest that the altered sensitivity that decentralized muscles exhibit toward the action of cholinergic agonists,[14–16] to the action of anti-AChR-antibodies,[17,18] and to that of NDMR should be ascribed to junctional or prejunctional factors.

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