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LETTER TO THE EDITOR

Early involvement of spinal cord in diabetic peripheral neuropathy may influence patient outcome after neuraxial anesthesia

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To the Editor:

I read with interest the clinical report entitled "Subclinical neuropathy in diabetic patients: a risk factor for bilateral lower limb neurological deficit following spinal anesthesia?" by S.D. Angadi and A. Garde, published in the February 2012 issue of the *Journal of Anesthesia*. Although I agree with the authors about the fact that diabetic patients may be at an increased risk of neurological injury after regional anesthesia, I would like to make two comments about this clinical report.

First, in a previous report [1], I included the double crush hypothesis (DCH) as a possible factor (among several) to explain nerve insult after epidural anesthesia in diabetic patients. In 1973, Upton and McComas [2] launched the following hypothesis: patients with carpal tunnel syndrome or ulnar nerve neuropathy present not only compressive lesions at the wrist or elbow but also evidence of damage at the level of the cervical roots of the same nerve. They called this the DCH.

Later, in a retrospective study, Hebl et al. [3] applied the DCH to diabetic peripheral neuropathy (DPN). They reported that patients with pre-existing neural diabetic compromise might be more susceptible to injury when exposed to a secondary insult at another site, such as trauma, ischemia, and toxic compromise. Nevertheless, there is no evidence-based work in the literature to confirm the hypothesis of Hebl et al. [3] regarding DPN. Furthermore, diabetes is a generalized disease, and the vascular and metabolic mechanisms associated with DPN are complex and completely different from nerve entrapment. Despite the fact that the DCH has been accepted in many experimental and clinical reports, studies are needed to confirm this hypothesis. In the literature, modern trends show that there is controversy about the DCH, and recent studies seem to dismiss this interpretation of nerve injury [4]. Furthermore, a focal compression site on a nerve influences other regions on the same nerve according to a linear relationship. Thus, damage could be related to DCH at another site on the same nerve [4]. Accordingly, DCH is less likely to be involved as a damage mechanism after neuraxial anesthesia in patients with DPN. Moreover, secondary neurologic damage after neuraxial anesthesia in diabetic patients could be explained by mechanical trauma, neuronal ischemia, or local anesthetic toxicity. Thus, these risk factors can influence patient outcome, even though the course of spinal anesthesia appears uneventful [1, 4].

Second, DPN is one of the most common forms of neuropathy. There is some evidence that vascular dysfunction (microangiopathy), driven by metabolic changes, is an important cause of DPN [1, 5]. Although these vascularand metabolic mechanisms have been proposed [1, 5], a complete understanding of its pathogenesis remains elusive [5]. All studies have focused mainly on DPN, and central nervous system involvement has been largely overlooked [5]. Selvarajah et al. [5] reported that central nervous system involvement could be seen early in patients with subclinical DPN. Using magnetic resonance imaging, they demonstrated that the cervical spinal cord index was significantly reduced in patients with clinical DPN. Moreover, they demonstrated that this spinal cord involvement is an early process which is present not only in

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subjects with clinically detectable DPN, but also in subjects with modest impairment of nerve function (subclinical DPN). Accordingly, magnetic resonance imaging of the cervical spinal cord and neurophysiologic assessments could be of great value before proceeding to neuraxial anesthesia in diabetic patients [5].

Therefore, in summary, further studies are needed to determine whether diabetic patients are more susceptible to postoperative nerve injury after regional anesthesia.

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