

## CORRESPONDENCE

1996; 85:  
Anesthesiology  
1996; 85:429-30  
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## Spinal Cord Infarction after Surgery in a Patient in the Hyperlordotic Position

*To the Editor:*—Amoiridis *et al.* are to be congratulated on redirecting attention to the potentially catastrophic dangers of an exaggerated lordotic posture in patients receiving epidural blockade.<sup>1</sup> Surgeons and anesthesiologists have been unwilling to acknowledge the harmful effects of prolonged hyperextension of the spine, though the evidence has been available for nearly half a century,<sup>2,3</sup> and cited repeatedly for more than 20 yr.<sup>4-6</sup> The cardiovascular mechanism of spinal venous infarction from hyperlordosis is quite clear. Hyperlordosis causes rotation of the liver and obstruction of the inferior vena cava in its intrahepatic course. Pressure increases within the alternative venous pathway (the azygos system) and is transmitted via the intercosto-vertebral veins and epidural venous plexuses to the intraspinal veins.<sup>2,3</sup>

In their opening paragraph, Amoiridis *et al.* list a total of four published reports of spinal cord infarction, including their own case, attributable to an imposed hyperlordotic posture, and one of these cases holds the key to appreciating the underlying pathophysiology. It is not appreciated generally that two of those cases (references 4 and 8 in Amoiridis' reference list) were operated by the same surgeon, in the same year (1969), in the same surgical posture of imposed hyperlordosis, and under the same general anesthetic technique, except for the method of controlled arterial hypotension. In one case, high epidural blockade was induced with 36 ml 1.5% lidocaine and 1/200,000 epinephrine, and the intraoperative blood pressure was maintained at 80-90 mmHg; this case was published in the journal *Anaesthesia* under the title "Paraplegia following epidural analgesia."<sup>7</sup> In the second case, arterial pressure was reduced with intravenous pentolinium, and no epidural was used. This second case is critical to resolving the etiologic issue of arterial *versus* venous infarction of the cord, because it is the only one to receive thorough macroscopic and microscopic examination of the spinal contents and to show all the signs of venous infarction. Both cases revealed a patent anterior spinal artery. After being rejected for publication by a widely read anesthesia journal in 1969, the second case was eventually published 5 yr later, by the *New Zealand Medical Journal*, a journal not widely read by Western anesthesiologists.<sup>8</sup>

The educational influence of these two cases and their publication history is reflected in subsequent listings in *Science Citation Index*, with the epidural case receiving 24 citations, most imputing causality to the epidural block. The second has received only four citations, two of which were by this writer, inveighing against the dangers of spinal hyperextension and the folly of ascribing them to a coincidental epidural blockade.

One more case must be added to the list of parapareses after spinal hyperextension.<sup>9</sup> In this recent report, hyperlordosis was maintained for 2 h 45 min, but nowhere in the discussion was this considered as a possible etiologic factor, because epidural analgesia was used

as an adjunct to general anesthesia, and accidental subarachnoid cannulation was suspected. Once again, the publication process failed to acknowledge the unphysiologic posture of hyperlordosis as a possible cause of injury.

Finally, epidural analgesia will continue to attract obloquy unless appropriate monitoring standards are maintained to avoid associated but unrelated complications. Any analgesic medication of the spinal cord is a neurologic intervention that requires informed and appropriate neurologic surveillance on the part of the anesthesiologist. Any hope of rescuing a jeopardized spinal cord depends on prompt diagnosis and treatment within 8 h of injury.<sup>10</sup> In my opinion, a prudent course of management for postoperative epidural analgesia should assume that rare, unrelated neurologic dangers are always lurking, and that steps must be taken to provide early warning of their possible intrusion and their timely containment. This implies that early and regular neurologic examination of the lower limbs must take precedence over continuous intense epidural analgesia, and epidural infusions should be interrupted periodically while alternative pain relief is administered, to allow neurologic examination and confirmation of uncompromised neural function.<sup>11</sup>

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(Accepted for publication April 29, 1996.)

Anesthesiology

1996; 85:430

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*In Reply:*—We thank Prof. Bromage for his comments on our recent publication.<sup>1</sup> His suggestion that rotation of the liver, as caused by hyperlordosis, can lead to inferior vena cava (IVC) obstruction is a possibility, with the most probable site being just before entry into or in the lower part of the hepatic IVC sulcus. We agree with his view that epidural analgesia should be interrupted periodically to allow assessment of spinal cord function.

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(Accepted for publication April 29, 1996.)

Anesthesiology

1996; 85:430-1

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## Nonanesthetic Haloalkanes and Nicotinic Acetylcholine Receptor Desensitization Kinetics

*To the Editor:*—Raines reports the effect of volatile anesthetics (enflurane and isoflurane) and 'nonanesthetics' (2,3-dichlorooctafluorobutane and 1,2-dichlorohexafluorocyclobutane) on the desensitization kinetics of the Torpedo nicotinic acetylcholine receptor (nAChR).<sup>1</sup> The less pronounced effect of nonanesthetics on the desensitization kinetics of this membrane protein is interpreted as evidence that the system is a valid model of the volatile anesthetic site of action, based on a somewhat contentious criterion suggested by Eger and colleagues.<sup>2-4</sup> Indeed, these nonanesthetics were described recently as inducing at least one component of the anesthetic state.<sup>5</sup>

The nonanesthetics studied were chosen, in part, because they lack a bromine atom that may quench fluorescence. However, halogenated molecules that contain chlorine atoms also may quench fluorescence,<sup>6</sup> in some cases with an efficiency (the probability that

an encounter between quencher and fluorophore will result in energy transfer) that exceeds that of brominated compounds. Therefore, using quenching of indole fluorescence in methanol (as a model for tryptophan residues in proteins), 1,2-dichlorohexafluorocyclobutane is found to be a superior quencher compared with halothane, whereas 2,3-dichlorooctafluorobutane is 84% as efficient as halothane (fig. 1). The effective fluorescence quenching exhibited by these bichlorinated compounds is presumably due to the electron withdrawing influence of the neighboring fluorines, which results in favorable conditions for electron transfer to the chlorine atoms. The lack of a bromine atom, therefore, is not a valid reason to exclude direct fluorescence quenching by the test compound.

An alternative interpretation of the results reported by Raines<sup>1</sup> is that quenching of a portion of the native nAChR tryptophan fluores-