To the Editor.—Syndrome: "A group of symptoms that collectively characterize a disease..." (American Heritage Dictionary, 1996). After visions of a modern-day Woolley and Roe catastrophe ("Cauda Equina Syndrome following single [dose] spinal... Hyperbaric Lidocaine...") it was a relief to learn that the patient fared no worse than having residual perineal hypesthesia, constipation, and difficulty voiding. He was spared, fortunately, the duo of paraparesis and incontinence that collectively shape the syndrome's symptom triad.

Although the report's title1 trumpets yet another catastrophe linked to intrathecal hyperbaric lidocaine, the actual case description paints an altogether different (albeit no less unfortunate) picture of persistent bilateral midsacral dorsal radiculopathy. The case made for neurologic sphincter muscle incompetence is tenuous; difficulty voiding more probably is caused by inability to sense bladder fullness (or to injury of preganglionic sacral parasympathetic axons) than by incontinence from bladder sphincter paralysis. Attributing laxative-responsive constipation in a 74-year-old patient—with a clearly functional anal sphincter— to neurologic dysfunction smacks of denying Mother Nature.

We need to be explicit: *cauda equina syndrome* proper is the triad of bilateral paraparesis or paraplegia of leg and buttock muscles, saddle anesthesia plus sensory deficits below the groin, and incompetence of bladder and rectal sphincters causing incontinence of urine and feces. Scattered below that ultimate asymptote of the drug-exposure/toxicodynamic cumulative probability curve lies a continuum of *cauda equinopathies* that range from transient radicular irritation or radiculopathy through lumbosacral sensory deficits, monoparesis, and sphincter incompetence, culminating in full-blown chronic cauda equina syndrome.3

The impact of hot-button trigger words on public and press all too easily could cripple spinal anesthesia in North America. Intrathecal hyperbaric lidocaine already is under a cloud. Let us present the clinical facts dispassionately, and so, offer spinal lidocaine an impartial hearing.

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References

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In Reply.—Like Dr. de Jong, I am an advocate of regional anesthesia and therefore welcome his critical appraisal of the clinical aspects of this case report. As Ferguson and Watkins2 wrote in their original description of cauda equina syndrome, "The report is published not with the intention of disparaging a very valuable, if not indispensable, form of anaesthesia, but in the hope that the result of these investigations may help to obviate such unfortunate incidents in the future..."

I am unable to agree with Dr. de Jong when he implies that the current case report 'paints an altogether different picture' than other reported cases of cauda equina syndrome. As outlined in the case report (and by Dr. de Jong) *cauda equina syndrome* consists of a triad of symptoms. As also outlined in the case report, the patients in other modern cases after spinal and continuous spinal anesthesia usually have not presented with this entire triad (table 1). Even Ferguson and Watkins2 detailed classic descriptions of 14 patients lacked lower extremity paralysis as a prominent symptom. Similar to most of these other patient's labeled with cauda equina syndrome, our patient walked out of the hospital, but with a urinary catheter in place. Also similar to some of these other patients, he gradually regained control of micturition, but only after the passage of a very long year for all parties involved.

I agree that spinal lidocaine deserves an impartial hearing, especially at a time when some authors are suggesting that "the hyperbaric lidocaine formulation as dispensed presently carries a substantial risk of neurotoxicity."5 Using terminology such as *cauda equinopathy*, injury of preganglionic sacral parasympathetic axons, or monoparesis and sphincter incompetence to describe these patients' symptoms may very well be the most technically correct thing to do. Unfortunately taking this approach seems unlikely to

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