CORRESPONDENCE

Anesthesiology
1999; 90:318

The Spinal Inquisition: Heresy of Neurotaxonomy

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.[1] He was spared, fortunately, the duo of paraparesis and incontinence that collectively shape the syndrome's symptom triad.

Although the report's title[1] 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-yr-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.[3,4] 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.[5]

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

1. Gerancher JC: Cauda equina syndrome following a single spinal administration of 5% hyperbaric lidocaine through a 25-gauge Whitacre needle. ANESTHESIOLOGY 1997; 87:687–9
2. Cope RW: The Woolley and Roe Case: Woolley and Roe versus Ministry of Health and others. Anaesthesia 1954; 9:249–70
3. Woolsey RM, Young RR: The clinical diagnosis of disorders of the spinal cord. Neurol Clin 1991; 9:573–83
4. Jaradeh S: Cauda equina syndrome: A neurologist's perspective. Reg Anesth 1993; 18:473–80
5. Horlocker TT, McGregor DG, Matsushige DK, Schroeder DR, Besse JA: A retrospective review of 4767 consecutive spinal anesthetics: Central nervous system complications. Anesth Analg 1997; 84: 578–84

(Accepted for publication March 20, 1998.)

Anesthesiology
1999; 90:318–9

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.[1] As Ferguson and Watkins[2] wrote in their original description of cauda equina syndrome after spinal anesthesia, "This 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).[1] Even Ferguson and Watkins'[2] 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."[3] 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

shine much light through the "cloud" surrounding intrathecal hyperbaric lidocaine. I propose we recognize complications when they occur[1] and formulate reasonable clinical recommendations for the use of spinal lidocaine[4] in the hope of clearing the cloud. I believe that my case report, when combined with the recommendations made by Dr. Drasner in his accompanying editorial,[4] "presents the clinical facts dispassionately" and only help to further this goal.

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References

1. Gerancher JC: Cauda equina syndrome following a single spinal administration of 5% hyperbaric lidocaine through a 25-gauge Whitacre needle. ANESTHESIOLOGY 1997; 87:687–9

2. Ferguson FR, Watkins KH: Paralysis of the bladder and associated neurologic sequelae of spinal anaesthesia (cauda equina syndrome) Br J Surg 1937; 25:735–52

3. De Jong: Last round for a "heavyweight?" Anesth Analg 1994; 78:3–4

4. Drasner K: Lidocaine spinal anesthesia: A vanishing therapeutic index? ANESTHESIOLOGY 1997; 87:469–72

(Accepted for publication March 20, 1998.)

Anesthesiology
1999; 90:319

The Use of Magnetic Resonance Imaging in Patients with Fiberoptic Intracranial Pressure Monitors

To the Editor:—We read with interest the conclusions of the authors of a case report published in the October issue of ANESTHESIOLOGY (Fiberoptic intracranial pressure monitoring during magnetic resonance imaging. 1997; 87:1001–2). We are writing in response to this report because the use of magnetic resonance imaging (MRI) in patients with fiberoptic intracranial pressure monitors in place has become essential at busy trauma centers.

Although we applaud the creative thinking and reasoning involved in hypothesizing the potential for and testing the movement of a Camino fiberoptic intracranial pressure monitor (Camino Laboratories, San Diego, CA) in the magnetic field of an MRI machine, these authors missed the essential point: movement of the catheter inside the head, regardless of the strength of the magnetic field, is impossible when the catheter is inserted properly. This is because the flexible catheter tip extends only 0.5 mm beyond the rigid cranial bolt when applied according to manufacturer's guidelines. Therefore these *ex vivo* testing results are moot when extrapolated to the *in vivo* situation.

Perhaps the more important point alluded to in this case report relates to thermal effects. We investigated this recently with cadaver models and found negligible temperature changes during typical MR sequences (unpublished data, Prall JA, Lillehei KO, Whittaker JB, January, 1998). Others found similar results in other types of intracranial implants.[1,2] The only other potential for radiofrequency absorption causing detrimental effects may lie in epileptigenesis. However, in 10 years of submitting patients with Camino monitors to MRI, we have yet to document a seizure during or immediately after the scan. Prospective confirmation of this observation remains to be performed.

As far as we have been able to determine, there is no documented reason why this type of monitor represents any additional risk to a patient undergoing MRI for traumatic brain and spinal cord injuries. We continue to feel safe in submitting any patient with a fiberoptic intracranial pressure monitor to MRI where indicated.

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References

1. Pohost GM, Blackwell GG, Shellock FG: Safety of patients with medical devices during application of magnetic resonance methods. Ann NY Acad Sci 1992; 649:302–12

2. Chou CK, McDougall JA, Chan KW: Absence of radiofrequency heating from auditory implants during magnetic resonance imaging. Bioelectromagnetics 1995; 18:307–16

(Accepted for publication May 11, 1998.)