maximum tenderness in failed carpal tunnel surgery. Incidentally, superficial to this lesion is the dreadful palmar cutaneous branch of the median nerve, which has not only turned hand surgeons into "scaredy cats," but also has led Mackinnon and Dellon[4] to the erroneous conclusion that the cause of this tenderness is scarring of the palmar cutaneous branch; which explains why we have yet to see a successful outcome of surgery for "scarring of the palmar cutaneous branch."

According to our protocol, this patient will require repeat stellate blocks, and when the pain, stiffness, and swelling are resolved, then and there we will surgically repair the residual carpal tunnel syndrome with complete and methodic flexor tenosynovectomy and neurolysis of the median nerve.

The inextricable link between reflex sympathetic dystrophy and patient dissatisfaction—the long searched-for cause of the so-called "individual predisposition"—calls for competent and compassionate management of these patients to regain their trust.

I conclude with the prediction that, at the end of the day, we will all be convinced that pain is a simple sensory messenger from the damaged tissue crying out loud, "Please fix me."

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In Reply:—We appreciate the opportunity to respond to Drs. Khan and Erjavec. Their comments show several of the issues and concerns we discussed in our article.

It is difficult to understand the rationale of Dr. Khan's position with regard to the usefulness of psychologic treatments for pain. His confusing references to the empirical basis for psychologic treatments and his global dismissal of such interventions as "neither successful nor economic and, therefore unprudent [sic]" reveals a fundamental misunderstanding of the contemporary pain literature that defies further comment. His perspective should not be dismissed lightly as the ill-informed opinion of one isolated practitioner. Unfortunately, it represents an attitude that pervades the world of biomedicine.

Dr. Khan's concluding remarks eloquently portray the biomedical myth that lies at the root of the problem of intractable chronic pain. His prediction that, "at the end of the day, we will all be convinced that pain is a simple sensory messenger from the damaged tissue crying out loud 'Please fix me'" is quaint, but very disturbing. Although, unlike Dr. Khan, numerous practitioners do not grossly oversimplify this complex clinical problem, many well-meaning physicians practice as if this is the case. Contemporary medical education emphasizes nociception while ignoring the psychologic and social aspects of chronic pain. One of the major points of our article is that as long as the practitioner remains limited to a biomedical model those patients who are the most overwhelmed by pain will remain enigmatic.[1] It is our belief that to help those patients who show "minimal pathology with maximum dysfunction"[2] the anesthesiologist needs to learn a whole new set of conceptual and clinical skills.

In his letter, Dr. Erjavec shows an awareness of the importance of psychologic and social factors of chronic pain. He acknowledges that the proceduralist must "first and foremost be a communicator and a behavioralist and well-rehearsed in the biopsychosocial skills of pain medicine before being given the privilege of performing procedures." It appears that, as we suggest in our article, he restricts nerve blocks to those patients who have high levels of psychosocial functioning and clear organic etiologies indisputably amenable to nerve block therapy.[1] Therefore, according to Dr. Erjavec's stated position, we disagree mainly about practical matters rather than about conceptual issues. We applaud his efforts to screen patients for psychosocial problems that mitigate the decision to perform a procedure. We suspect, however, that most anesthesiologists lack the skills to perform such evaluations. Our article outlines the changes in training that we believe are necessary.[1]

Unfortunately, in the real world, many "needle-jockeys" function more as technicians than as physicians. The incentives and pressures of modern medicine leave little time to practice the "art" of medicine. The major purpose of our article was to describe new opportunities for anesthesiologists to learn old medical skills that are devalued by biomedicine. It is important to understand that overwhelming chronic pain is not a biologic event, but rather an all-consuming personal experience.[3] We advocate a biopsychosocial approach that is tolerant of incomplete medical knowledge and that accommodates medicine's limitations. When complete understanding is abandoned as a goal, the traditional tasks of the physician—listening, witnessing, and relieving suffering—are not relegated to a small corner of medicine, the so-called art of medicine, but are returned to the core of medical practice and medical education.[4]

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Anesthesia Safety, Outcomes, and Prospective Study Design

To the Editor:—We congratulate Auroy *et al.*[1] for the new and valuable information resulting from their survey of regional anesthesia in France. The study provides data that confirm the safety of modern anesthesia practice. Clearly, it is rare for regional anesthetic techniques to produce serious complications. Although some excellent information is provided, we are concerned that the study results have been overinterpreted.

First, this was not truly a prospective study. Prospective data collection is achieved when "the relevant prognostic and outcome variables are collected from patients as they are treated."[2] In this study, the questionnaire used to assess total number of anesthetics and number of serious complications was "sent to all participating anesthesiologists 15 days before the end of the 5-month period."[1] Although not technically prospective, this portion of the data collection could be considered prospective only if the questionnaire eliminated, rather than added, new data fields to those on the original log data sheet. We accept that it is unlikely that anesthesiologists would forget to report serious complications that occurred in the previous 5 months and that these data are likely to be accurate.

In contrast, data regarding potential risk factors for serious complications clearly were not collected prospectively. Data collection forms for potential risk factors (e.g., local anesthetic choice, sensation of paresthesia during needle insertion, use of continuous microcatheters) were designed and mailed to investigators 1 month after the study was completed. Furthermore, because accuracy of these data were not verified by retrospective review of patient records, the possibility of incomplete and biased data reporting cannot be excluded.

Indeed, the data suggest a recall bias in reporting for patients with neurologic deficits. Of the 14 patients in whom a neurologic deficit developed after bupivacaine spinal anesthesia, 11 anesthesiologists remembered the occurrence of a paresthesia during placement of the spinal needle (table 1). In contrast, of the 10 patients in whom neurologic injury developed after lidocaine spinal anesthesia, only one anesthesiologist remembered the occurrence of a paresthesia during placement of the spinal needle. Because these data were not collected prospectively, it would seem prudent, at least, to retrospectively review anesthetic records to verify the accuracy of these recollections.

Even if it is assumed that the recollections are accurate, one must ask, are these data sufficient to support speculation regarding risk for nerve injury? We think the answer is no, for several reasons. First, the denominator was not measured. By design, detailed data were only collected for patients experiencing a complication. For example, anesthesiologists were asked to recall choice of local anesthetic for only 97 of 103,730 regional anesthetic procedures included in this study. As a result, it is impossible to calculate the true incidence of injury associated with any specific factor, including choice of local anesthetic. It is also impossible to determine whether the incidence of nerve injury differs based on choice of local anesthetic.

Second, even if incidences were identified accurately, univariate data analysis is not sufficiently robust to support speculation regarding potential cause–effect relationships. For example, it is certainly possible that lidocaine was chosen more frequently in a patient population with an inherently high risk for nerve injury (e.g., patients placed in the lithotomy position). In this scenario, lidocaine would be used more frequently in patients in whom nerve deficits developed, and univariate statistical analysis may incorrectly identify lidocaine as a risk factor for nerve injury. Because data regarding other potential risk factors were not collected, it is impossible to conclude whether any single factor, such as lidocaine use, is associated with an increased risk for serious complications.

Finally, assumptions made in the *post hoc* data analysis clearly bias interpretation of the results. For example, it was assumed that a transient paresthesia occurring during insertion of the spinal needle was the cause of subsequent neurologic deficit. However, transient paresthesias frequently are elicited during placement of spinal needles, and there are no data to prove this causes postoperative neurologic deficit. The decision to exclude patients with transient paresthesias during needle insertion skews the analysis (11 of 12 patients excluded because of paresthesia received bupivacaine). When all cases of neurologic deficit are included in the analysis (table 1), 10 patients (42%)

Table 1. Characteristics of Patients with Neurologic Deficit after Spinal Anesthesia

	Lidocaine 5%	Bupivacaine 0.5%	Total
Paresthesias or pain	1	11	12
No paresthesias or pain	9	3	12
Total	10	14	24