Perioperative Nerve Injury: A Silent Scream?

WELCH et al. add 112 well-documented adverse events from 380,680 patients to the third most commonly recognized cause of anesthesia litigation: Perioperative nerve injuries.1 This retrospective analysis from the University of Michigan collates three concurrent data sets (quality assurance, an internal closed-claims collection, and billing codes) to summarize the clinical experience with nerve injuries at one large tertiary university hospital from 1997 to 2007. The casual reader might be reasurred by the relatively low global frequency of nerve injuries at 0.03% (1:3,400), but there were likely other patients who developed these problems.

There are a number of potential reasons that the current study may have underestimated the incidence of nerve injuries. First, although the authors were diligent, using three separate departmental and institutional databases to identify patients with potential perioperative neuropathies, they did not report the crossover identification rates between the three databases. That is, how many patients with new peripheral nerve injuries were identified in more than one database? As reported, it appears that each database contributed new cases that were not found in the other databases. This lack of cross-identification suggests a high likelihood that other cases were missed. Second, in an effort to specifically seek new nerve injuries that were likely the result of intraoperative or immediate postoperative care, the authors sought information on peripheral neuropathies that were identified only during the first 48 h postoperatively. Previous prospective studies have shown that a number of perioperative neuropathies are first identified more than 48 h after surgery. Third, retrospective studies of perioperative nerve injuries document only injuries that are either sufficiently symptomatic to be noted in records or missed entirely in postoperative surveillance, a problem especially true for retrospective studies performed at tertiary medical centers where patients do not necessarily have long-term care. Thus, the true incidence of perioperative neuropathies is still unclear, but likely exceeds 0.03%, and will probably require a major prospective study. Nonetheless, the authors should be congratulated for the extensive efforts they used to compensate for the problems posed by the retrospective methodology.

Regardless of methodology, there was approximately one anesthetized patient each month in the University of Michigan Medical Center who had additional unexpected pain, distress, and perhaps a disability unrelated to his or her primary operation. As a profession, have we been deaf to a silent “Scream” (with apologies to Edvard Munch, The Scream, 1893), of a nerve within an appropriately padded and positioned limb in the operating room, which is often assumed (“res ipsa loquitur”) to be the cause of unexpected perioperative nerve injury?

What Characterized the Nerve Injuries Seen in This Study?

In their remarkable effort, Welch et al. collated the anesthetic outcomes from a single institution of over 380,000 anesthetics during a 10-yr epoch, ending in 2007. They found that perioperative nerve injuries were associated with medical conditions afflicting the health and function of small blood vessels, which are critical to the sustenance of major peripheral nerves. These conditions included hypertension, diabetes, and smoking (hazard ratios of 2.2, 2.1, and 2.4, respectively). Interestingly, general and epidural anesthesia were significantly associated with nerve injury, whereas monitored anesthesia care, spinal anesthesia, and even peripheral nerve block were not. Upper extremity injuries predominated by a 60:40 margin, and patients undergoing cardiac, general, orthopedic, and neurosurgery had higher risk. Two-thirds of the recorded injuries were sensory only, followed by combined motor and sensory (27%) or isolated motor injuries (just 16%). Interestingly, patients with few identified comorbid illnesses (American Society of Anesthesiologists Classes 1 and 2) were equally vulnerable to developing a perioperative neuropathy, as compared with those of American Society of Anesthesiologists Classes 3, 4, and 5. Moreover, patients from age 13 to 86 yr of age were identified with new neuropathies, although demographics of weight or body surface area were not included.

Mechanisms of Perioperative Nerve Injuries

Theoretically, perioperative neuropathies may result from excessive pressure (compression), stretch, isch-
emia, metabolic derangement, direct trauma, laceration of a nerve, and other, yet unknown, factors. Nerve compression may occur via external or internal mechanisms. Inappropriate placement of noncompliant external objects or improper arm positioning (such as allowing the elbow to rest on the steel frame of a surgical table) may create external pressure on the ulnar nerve, trapping it as it courses within the rigid bony canal of the superficial postcondylar groove at the elbow. Such uninterrupted pressure can ultimately produce nerve ischemia and injury. In addition, most peripheral nerves are intolerant of prolonged stretch beyond 5% of normal resting length. Extremes of extension or flexion should generally be avoided to minimize this risk.

Double Crush Syndrome and Anesthetic Implications

An additional factor which may contribute to perioperative nerve dysfunction is the phenomenon of the double crush syndrome. This syndrome describes the coexistence of two (or more) clinical or subclinical insults along the course of a nerve. Double crush was first described in 1973 by Upton and McComas, and reflects the phenomenon whereby one compressive lesion occurring along a nerve renders the nerve less tolerant of compression at the same or a second locus. Therefore, nerves with a preexisting injury or compression are at much greater risk of a second, possibly subclinical, insult. Together, these may result in a permanent nerve injury. This phenomenon is schematically illustrated in figure 1. While the exact mechanism is not definitively understood, it likely involves disturbances in axonal flow of nutrients and/or disruption of the architecture of neurofilaments.

Clinically, proximal upper extremity nerve root pathology has been shown to lessen the median nerve compression necessary to produce symptoms of carpal tunnel syndrome and to worsen outcome after carpal tunnel decompression. In addition, some evidence highlights the increased susceptibility of the ulnar nerve to ischemia, as compared with either the radial or median nerves.

Finally, certain medical diseases and/or concomitant drug therapy may have physiologic and/or toxic effects on peripheral nerves, rendering them more vulnerable to injury in the perioperative period. Smoking, hypertension, and diabetes may all contribute to microvascular changes as identified here by Welch et al., which may contribute to the development of peripheral neuropathies and may well predispose peripheral nerves to be more vulnerable to other, relatively minor insults. Indeed, many diseases and conditions as well as medications and toxins may predispose patients to neuropathic injury.

General Impressions

Evidence is rapidly mounting that indicates that mechanisms of perioperative nerve injury are complex and multifactorial. In 27% of cases reviewed by Welch et al., an ulnar nerve injury occurred despite the specific documentation of protective padding at the elbow. In a separate study, Alvine and Shurrer detected bilateral nerve conduction abnormalities in 12 of 14 patients who presented with unilateral symptoms of a perioperative ulnar neuropathy. Curiously, the present study found that patients undergoing monitored anesthesia care did not develop peripheral nerve injuries. The authors postulated that these patients were sufficiently alert to move uncomfortable extremities and prevent the stresses that may have caused clinical symptoms of nerve injury. However, previously described somatosensory evoked potential data demonstrated that only 50% of awake, unmedicated volunteers were able to perceive sensory alterations or paresthesias resulting from direct pressure applied to the ulnar nerve, despite significant alterations in somatosensory evoked potential latencies.

This finding suggests that awake patients may not perceive sensory changes of adversely affected limb nerves during surgery, and may not alter their limb position automatically to a “safer” position. Indeed, a number of reported upper extremity perioperative nerve injuries have occurred and resulted in medicolegal claims even when the patient was awake and conscious during lower extremity surgery under regional anesthesia. Thus, clinicians must remain alert to patient position at all times and modes of anesthesia care.
Fortunately, etiologies for some perioperative neuropathies are reasonably clear, and clinicians should always endeavor to optimize limb positioning. Brachial plexus injuries may occur by hyperabduction of the arm to >90 degrees, perhaps excessively stretching major nerve axons beyond their resting length. The use of shoulder braces coupled with steep head-down positioning has been implicated in the development of compression and brachial plexus injuries. Prolonged pressure on the radial nerve as it courses in the spiral groove of the humerus may result in an ischemic/pressure perioperative neuropathy. For example, direct pressure in this area may occur with a mismatched armboard, causing a step-off from the operating room table. Retractor holders, ether screens, and operating room equipment may also produce direct pressure in vulnerable areas. Hyperextension of the elbow may stretch the median nerve and contribute to postoperative disability. We also have observed and are aware of a number of cases of transient median and ulnar nerve dysfunction that mimic carpal tunnel syndrome and vice versa. In the latter instance, we theorize that transient perioperative edema results in excess pressure under the transcarpal ligament, and other carpal tunnel connective tissue may cause short-term symptoms that mimic ulnar or median neuropathies.

Ulnar neuropathy may occur despite the specific use of extensive padding of extremities during surgery and proper positioning of both arms. This observation is further amplified by the finding that ulnar nerve injury occurs with nearly equal frequency in medical and surgical patients hospitalized for more than 2 days.7,8 In addition, predisposing anatomic and other risk factors in men (because of anatomical gender variations of the cubital tunnel), combined with prolonged periods of bedrest in the supine position (whether during or after surgery, or for hospitalization as a result of strictly medical conditions), may contribute to the development of perioperative ulnar nerve injury.

Summary

The data from Welch et al. denote the (minimal) frequency, scope, and demographics of perioperative neuropathies that impact clinicians in daily practice.1 The etiology of perioperative peripheral nerve injuries remains complex, multifactorial, and incompletely understood. Despite avoidance of traction, stretch, and excessive or prolonged pressure during surgery, perioperative peripheral nerve injuries can and do occur. Prospective studies suggest that many nerve injuries initially become symptomatic more than 2 days after surgery and anesthesia. Thus, it appears evident that perioperative neuropathies occur despite appropriate positioning and padding of limbs during surgery; that awake patients vary in their ability to detect peripheral nerves at risk, and individuals may or may not reposition afflicted limbs on their own; and that some adverse events classified as perioperative neuropathies have their origins and onset beyond the operating room or postoperative care unit.

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References