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A Call for Caution Regarding Cervical and Ulnar Nerve Injuries and General Anesthesia

To the Editor:

I read with interest the recent editorial by Lanier and Warner of the Mayo Clinic on new perioperative cervical injury.[1] The authors state in a somewhat cavalier manner that "Spinal cord injury associated with airway instrumentation was uncommon, accounting for a mere 11% of patients.[1]" They seem to be suggesting that anesthesia providers do not need to be greatly concerned with these injuries, and they compare them with postoperative ulnar nerve injuries. They cite a study from their institution that they claim dismisses the culpability of anesthesia providers as the cause of these postoperative ulnar nerve injuries because ". . . ulnar injuries were never present at the completion of surgery, and most did not appear until 1 or 2 days after surgery.[2]" However, Miller and Camp have indicated that ulnar injuries were noted in five patients immediately upon awakening from general anesthesia and were attributed to preventable errors.[3]

Lanier and Warner state "We wonder whether future research will also lessen the culpability and legal risk of anesthesia providers regarding new onset cervical injuries.[1]" I suggest that anesthesiologists maintain a careful and cautious approach in an attempt to prevent both neck and ulnar nerve injuries by using every means at their disposal to lessen the incidence of these serious and persistent problems.

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In Reply:

We wish to thank Dr. Sosis for his letter commenting on our editorial. His communication, along with the original article of Hindman *et al.*,[1] and our editorial[2] all agree that new-onset perioperative neurologic deficits, whether minor or severe, are always of concern to clinicians and patients. Every effort should be made to lessen patient risk; however, we reaffirm that these efforts must be based on scientific foundations, not speculation and innuendo.

A central purpose of the Hindman *et al.* article, previous research from Mayo Clinic (cited in our editorial[2]), and our editorial was to offer insights into the scope of new-onset neurologic deficits after anesthesia and surgery, factors contributing to those deficits, and remediable limitations in the delivery of health care affecting outcomes. Before relatively recent research, patients, clinicians, plaintiffs' lawyers, and expert witnesses representing plaintiffs largely assumed that if a patient experienced a perioperative neurologic deficit, some individual or individuals on the healthcare team must

be at fault, with improper positioning, management of physiology, or related factors resulting in negative outcomes. The important concept introduced by the Mayo Clinic research on ulnar neuropathy and the Hindman *et al.* report on new-onset cervical injuries is that, whether these injuries are studied prospectively or retrospectively, it is difficult to identify any wrongdoing on the part of healthcare providers in the vast majority of cases. Instead, the more common finding is preexisting patient characteristics that likely placed the patient at enhanced risk for new-onset injury, whether or not there were shortcomings in patient care.

Such a concept is comparable with what we know about new-onset perioperative tissue insult and injury in other organ systems. For example, in patients who experience myocardial ischemia and infarction during the perioperative period, it is most commonly baseline patient characteristics (*e.g.*, coronary artery arteriosclerosis, diabetes mellitus, inflammatory syndromes) that account for adverse outcomes, not shortcomings in the delivery of health care in otherwise healthy patients.

It is informative that Dr. Sosis selected the Miller and Camp article on perioperative ulnar neuropathy[3] to criticize the interpretations within our editorial. The article was published in 1979, an era in which perioperative patient monitoring and management were quite different from today and—by Miller and Camp's own admission—new-onset ulnar neuropathy was not widely recognized as a postoperative problem. Miller and Camp studied their patients retrospectively, and there was a selection bias in their 8-patient case series, compared with data from the larger Swedish series of 30,000 patients that they cited.[4] Miller and Camp studied patients who were referred for persistent, severe ulnar neuropathy, and the authors focused their report on nerve conduction studies in the ipsilateral limb. There is no mention of studies in other nerve groups, which would have been critical for assessing baseline patient predisposition to injury (as later reported in the Mayo Clinic research we cited[2]). Thus, all we can conclude from the Miller and Camp article is that their eight patients had nerve conduction anomalies consistent with nerve injury in the cubital tunnel. There was no reporting on the anesthetic management or the fine details of patient positioning. However, based on the types of surgery performed (see their table 1), the specialty backgrounds of the two authors, and the methods of patient identification, we can conclude that the authors had no firsthand knowledge of intraoperative risk factors in the overwhelming majority of these patients. Despite this, in their discussion of the data, the authors stated that "position of the arm seems to be critically important in the development of cubital tunnel compression" and advised that the arm should be protected by a "padded shield or sleeve." They further opined that "the injury to the ulnar nerve is probably delivered by the metal edge of the operating table or by the steel rail used to attach appliances to the table. The arm is pressed against this noncompliant surface with considerable force (the weight of the patient or his surgeon)." Unfortunately, these speculations on "preventable errors" (as characterized by Dr. Sosis) have no support whatsoever from Miller and Camp's own data. Despite this, speculation of the type offered by Miller and Camp helped pave the path for a generation of erroneous communications with patients and unfounded legal actions against physicians.

As we mentioned in our editorial, the original research from Mayo Clinic, which addressed patient care in the 1990s, demonstrated in a series of hundreds of patients that none of the cases of postoperative ulnar neuropathy could be traced to substandard care or wrongdoing on the part of clinicians. Instead, the deficits in all patients were first observed one or more days after surgery had ended, and similar deficits were observed in hospitalized patients who had not undergone anesthesia and surgery. The Mayo Clinic investigators discovered that the most common factors *associated* with postoperative ulnar neuropathy were baseline aberrations of patient anatomy and physiology, which likely predisposed the patients to injury. Similarly, the Hindman *et al.* research discovered that, in patients who experienced new-onset cervical injuries, baseline patient characteristics were far more commonly *associated* with injury than were any identifiable weaknesses in delivery of health care.

Ironically, Miller and Camp, neither of them anesthesiologists, had the opportunity to reach these same types of conclusions in their report of 1979. Of the eight patients in their report, two had underlying diabetic polyneuropathy and another had baseline idiopathic axonal polyneuropathy; in three of the patients, the first evidence of neuropathy was noted at 24–48 h after surgery. Despite this, the authors focused their speculation on shortcomings of patient positioning, mentioning the increased risk of polyneuropathy only in the context of patient positioning (*e.g.*, "patients with underlying polyneuropathy should receive particular attention in avoiding ulnar nerve compression").

The concept that endogenous patient factors can produce postoperative peripheral neuropathies, independent of limitations in the delivery of health care, recently has been revisited in the 2010 publication of Staff *et al.* from Mayo Clinic.[5] These authors retrospectively reported on 33 patients who experienced new-onset postoperative peripheral neuropathies that were clinically indistinguishable from compression injuries, with the exceptions that (1) there was no evidence of trauma and (2) the neuropathies were temporally and spatially segregated from the surgery.[5] In all cases, inflammation was suspected as the underlying pathomechanism. In the 21 cases of biopsy-confirmed inflammatory neuropathy, none of the patients had a history of autoimmune disorder. All patients reported acute or subacute onset of symptoms, beginning a median of 2 days after surgery (range 0–30 days). Nerve biopsies in all 21 demonstrated epineuronal perivas-

cular lymphocytic inflammation, with 15 diagnostic or suggestive of microvasculitis. Seventeen patients who underwent biopsy were treated with immunotherapy (typically corticosteroids) and, of the 13 who were followed longitudinally, there was a significant resolution of neurologic impairment ($P = 0.001$). Based on these data, the authors concluded that "it is important for physicians to recognize that not all neuropathies that occur in the postsurgical setting are due to compression, transection, or stretch." Inasmuch as the inflammatory response may be altered dramatically during the postoperative period, and inflammatory microvasculitis neuropathy is a previously unrecognized or underrecognized cause of peripheral neuropathy after surgery, large epidemiologic studies will be required to determine the role of this disease entity in the origin of new-onset perioperative neuropathy.

In his criticism of our editorial, Dr. Sosis is mistaken that we were "cavalier" in our assessments. Quite the contrary, we are extremely interested in ongoing research to identify the causes of new-onset perioperative neurologic deficits and remediate any shortcomings in the contemporary delivery of health care. However, we view it as irresponsible, both to the patients and the physicians who care for them, to fabricate origins of patient injury, mislead patients with those fabrications, and downstream place clinicians at inappropriate legal vulnerability. This is not to discount that shortcomings in the delivery of health care—independent of other risk factors—can contribute to adverse patient outcomes. However, attempts to criticize clinicians' shortcomings absent convincing evidence that a shortcoming has occurred serve no one except those who benefit financially from misdirected legal actions or who otherwise advance their professional careers based on unsubstantiated claims.

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Is It Dangerous to Quit Smoking Shortly before Surgery?

To the Editor:

We read with interest the work of Turan *et al.*,[1] who used the American College of Surgeons National Surgical Quality Improvement Program Database and found that cigarette smoking increased risk for perioperative mortality and major morbidity in patients having noncardiac surgery. The accompanying editorial by Katznelson and Beattie[2] provides a valuable perspective on their work, and we applaud their call for anesthesiologists to take a leadership role in efforts to help surgical patients quit smoking. In addition to potential beneficial effects on the acute perioperative risk nicely documented by Turan *et al.*, surgery also represents a teachable moment for smoking cessation (*e.g.*, undergoing a surgical procedure increases the chances that smokers will successfully quit),[3] and the benefits of smoking cessation to long-term health are unquestioned. The issue of the optimal timing of preoperative smoking cessation is of practical importance, and the duration of preoperative abstinence necessary for maximum benefit is not defined (and may differ among various smoking-related complications). For example, recent data suggest that even prolonging postoperative abstinence in smokers who had smoked up to the time of their surgery may benefit patients who have undergone orthopedic surgery.[4]

Unfortunately, in their excellent commentary Katznelson and Beattie perpetuate a concept that hinders perioperative tobacco control efforts: the fear that brief preoperative abstinence from smoking may actually have deleterious effects. They raise the concern that abstinence from smoking may exacerbate preoperative stress, citing a paper from our group that showed that although smokers report more stress than nonsmokers, stress was not increased by perioperative abstinence, and cravings for cigarettes were surprisingly low.[5] This finding actually favors attempts at smoking cessation during the immediate perioperative period, especially when considering the forced abstinence created by smoke-free healthcare facilities. They also state that several studies suggest that patients who experience sudden withdrawal from tobacco may be at increased risk for pulmonary complications, referencing two observational studies to support this assertion.[6,7] However, the study of Bluman *et al.* did not analyze patients who quit smoking shortly before surgery, but rather those who "cut down" the number of cigarettes smoked by a relatively modest amount.[6] The study of Nakagawa *et al.* did not find a significant difference in pulmonary complications between those patients who quit from 2–4 weeks before surgery and those who had smoked within 2 weeks before surgery.[7]

It is beyond the scope of this letter to fully review this topic, but a recent meta-analysis of available studies, which as Katznelson and Beattie note are primarily observational and have sig-

This letter was sent to the author of the above-mentioned article. The author felt that a reply was not necessary.—James C. Eisenach, M.D., Editor-in-Chief