Ulnar nerve injury and perioperative arm positioning

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Primum non nocere. Hippocrates included this admonition in Epidemics, Book I, Second Constitution, “to do good or to do no harm.” However, even the most conscientious health care provider will encounter unexpected and serious adverse medical events. This discussion focuses on one relatively common, often perplexing, and usually unexplained perioperative complication: ulnar neuropathy. Perioperative ulnar neuropathy has received increased scientific attention because it accounts for one third of all nerve injury claims in the American Society of Anesthesiologists (ASA) Closed Claims Study database [1–3]. In addition, these injuries may result in chronic pain or paresthesia, employment disability, catastrophic economic damages, and malpractice litigation [2–4]. We will explore the current understanding of perioperative ulnar nerve dysfunction by summarizing the relevant scientific literature and information within the ASA closed-claims database, describing the epidemiologic features of perioperative nerve injuries, discussing relevant clinical investigations and recommendations for safe arm positioning during anesthesia, and reviewing the medico-legal issues inevitably intertwined with this topic, particularly the doctrine of res ipsa loquitur.

ASA Closed Claims database

The Committee on Professional Liability of the ASA initiated the Closed Claims study in 1985 [1]. The closed claims database includes the experiences of 35 medical insurers (providing coverage for 50% of anesthesiologists in the United States) and contains nearly 5000 cases. Even though the closed claims project examines only a small fraction of total events that occur, attempts are
made to glean the maximal amount of information from each incident [1].

Because the data involve a large group of patients NOT severely compromised medically, this database offers important insights into how the process of care contributes to adverse events in typical patients. Types of adverse outcomes and demographics show tight clustering (Table 1), and only three categories account for two thirds of all adverse outcomes: death, nerve injury, and brain damage. Surprising to some, nerve injuries now represent the second largest class (16%) of adverse outcomes in the ASA Closed Claims Study database. Indeed, if anything, the relative frequency of nerve injury claims has increased over the last decade as claims for hypoxemia and inadequate ventilation have declined. Just three sites—the ulnar nerve, the brachial plexus, and the lumbosacral roots—account for most nerve injury claims [2]. Among these, the ulnar nerve has been, and continues to be, the mostly commonly cited nerve injury associated with surgery.

**Mechanism of perioperative nerve injuries**

Theoretically, perioperative neuropathies may result from a number of mechanisms including excessive pressure (compression), stretch, ischemia, metabolic derangement, direct trauma, laceration of a nerve, and probably other yet unknown factors. Nerve compression may occur through 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 condylar groove at the elbow. Such uninterrupted pressure can ultimately produce nerve ischemia and injury [4–6]. Fig. 1 is an illustration of such a situation. In addition, some evidence highlights the increased susceptibility of the ulnar nerve to ischemia compared with either the radial or the median nerves [6–8]. Furthermore, the ulnar nerve and its blood supply may be compromised by internal compression by the coronoid tubercle of the ulna (Fig. 2). This bony prominence is at least 50% larger in males, consistent with their greater susceptibility to perioperative ulnar nerve damage. As the ulna moves through

**Table 1**

<table>
<thead>
<tr>
<th>Adverse outcome</th>
<th>All Closed Claims (%)</th>
<th>Demographic variables</th>
<th>All Closed Claims (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>32</td>
<td>Age &gt; 18 y</td>
<td>90</td>
</tr>
<tr>
<td>Nerve injury</td>
<td>16</td>
<td>Nonemergency</td>
<td>73</td>
</tr>
<tr>
<td>Brain damage</td>
<td>12</td>
<td>ASA status I and II</td>
<td>70</td>
</tr>
<tr>
<td>Airway trauma</td>
<td>6</td>
<td>General anesthesia</td>
<td>70</td>
</tr>
<tr>
<td>Pulmonary aspiration</td>
<td>2</td>
<td>Male</td>
<td>41</td>
</tr>
</tbody>
</table>

Summarized from references [1–3].
Fig. 1. MRI in the axial plane of the ulnar nerve (N) as it courses through the superficial condylar groove at the elbow in a young male volunteer. A and V identify the ulnar collateral artery and vein. For illustration, an artificial marker (X) is highlighted to simulate a possible external object exerting pressure on the ulnar nerve. These conditions may create tissue pressure that could induce direct compression of the nerve against the bony prominence of the medial epicondyle or could interrupt nutrient blood flow to the nerve by compression of the nearby ulnar collateral artery and vein. (From Prielipp RC, Morell RC, Walker FO, et al. Ulnar nerve pressure: influence of arm position and relationship to somatosensory evoked potentials. Anesthesiology 1999;91:345–54; with permission.)

Fig. 2. The ulnar nerve and its blood supply traverse a superficial course in the proximal forearm, where it is also susceptible to compression from the tubercle of the coronoid process. Of special note, the tubercle of the coronoid process of the ulna is significantly larger in men, perhaps further contributing to the male predisposition to perioperative ulnar nerve injury. (From Contreras MG, Warner MA, Charboneau WJ, et al. Anatomy of the ulnar nerve at the elbow: potential relationship of acute ulnar neuropathy to gender differences. Clin Anat 1998;11:372–8; with permission. Copyright (c) 1998 John Wiley & Sons, Inc.; This material is used by permission of Wiley-Liss, Inc. a subsidiary of John Wiley & Sons, Inc.)
its range of motion, it may exert internal pressure against the ulnar nerve and its accompanying artery and vein [9].

Other mechanisms of perioperative nerve injury should be considered. The ulnar nerve, like most peripheral nerves, is intolerant of stretch beyond 10% of its normal length. Fig. 3 illustrates the cubital tunnel retinaculum, which is lax while the forearm is extended but becomes taut as the elbow is flexed [10]. Thus, persistent elbow flexion creates two mechanisms of potential nerve injury: direct internal compression and internal fixation within the cubital tunnel, which renders the remainder of the nerve more vulnerable to stretch along its course. One other factor often relevant is the phenomenon of the double-crush syndrome, a general term describing the coexistence of dual compressive lesions along the course of a nerve. Well-described in animal experiments, one lesion occurring along a nerve renders the nerve less tolerant of compression at the same or a second locus [11,12]. Therefore, nerves with some preexisting injury or compression are at

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**Fig. 3.** CTR is identified in laxity during forearm extension (top frame, highlighted in dashed box) but becomes taut in flexion (bottom frame). The retinaculum may cause compression of the ulnar nerve, indicated by the solid arrow. This likely represents one mechanism of ulnar nerve compression in supine, hospitalized males resting with their elbows flexed. (*From* O’Driscoll SW, Horii E, Carmichael SW, et al. The Cubital tunnel and ulnar neuropathy. J Bone Joint Surg Br 1991;73:613–7; with permission.)
much greater risk for a second, possibly subclinical, insult producing a permanent nerve injury [11,12]. Lastly, certain medical diseases (see box 1) or concomitant drug therapy (see box 2) may render peripheral nerves more vulnerable to injury in the perioperative period. The low incidence of nearly all such preexisting

**Box 1. Diseases and conditions that may predispose patients to neuropathies**

- Acromegaly
- Amyloidosis
- Carcinoma
- Cryoglobulinemia
- Chronic obstructive lung disease
- Diabetes mellitus
- Hereditary predisposition to pressure palsy
- Hypoglycemia
- Hypothyroidism
- Liver disease
- Lymphoma
- Macroglobulinemia
- Malabsorption syndromes and vitamin deficiencies
- Monoclonal gammopathy
- Multiple myeloma
- Polycythemia vera
- Porphyrias
- Uremia

**Box 2. Drugs and chemicals that may predispose patients to neuropathies**

| Acrylamide | Amiodarone |
| Arsenic | Aurothioglucose |
| Buckthorn (toxic berry) | Carbon disulfide |
| cis-Platinum | Dapsone |
| Diketone hexacarbons | Dimethylamino propionitrile |
| Diphtheria | Disulfiram |
| Hydralazine | Isoniazid |
| Lead | Metronidazole |
| Misonidazole | Organophosphates |
| Perhexiline | Phenytoin |
| Pyridoxin | Thalidomide |
| Thallium | Vincristine |
diseases or conditions may be one reason that perioperative ulnar nerve dysfunction is rarely, if ever, observed in young children.

Ulnar neuropathy: anatomy and epidemiology

The ulnar nerve originates from ventral nerve roots of C8 and T1 (motor fibers) and from the C8 dorsal root ganglion (sensory fibers), forming the lower trunk of the brachial plexus. After dividing into anterior and posterior divisions, the bulk of the fibers of the medial cord continue on as the ulnar nerve. This nerve travels with the medial head of the triceps muscle to the posterior aspect of the medial epicondyle, where several anatomic variations can result in nerve entrapment during the extensive range of motion of the elbow. For instance, there are noteworthy variants of the cubital tunnel retinaculum (CTR), which functions to hold the ulnar nerve in position [10]. The CTR is the roof of the cubital tunnel formed by a 0.4-mm fibrous band, extending from the medial epicondyle to the olecranon, perpendicular to the flexor carpi ulnaris aponeurosis. Certain variants of the CTR may potentiate the likelihood of either static or dynamic compression of the ulnar nerve during flexion or extension of the elbow [10] (Fig. 3). Indeed, a defined subgroup of ulnar neuropathies arises at the elbow known collectively as the cubital tunnel syndrome [13]. A number of other variations at the elbow of ulnar nerve structures also exist, such as accessory epitrochleoanconeus muscles or other dense fibrous bands directly bridging the medial epicondyle to the olecranon, and have been implicated in ulnar neuropathy. These variants are reviewed separately [14].

Among patients with perioperative ulnar nerve injury, males predominate by a ratio of at least 3:1. This may be explained, in part, by anatomic differences of the elbow between men and women [3,9,15]. Although there are no gross anatomic differences of the ulnar nerve between genders, females exhibit a strikingly greater (2–19 times) fat content on the medial aspect of the elbow, presumably providing a greater degree of subcutaneous padding for the superficial ulnar nerve along its course past the elbow [9]. As described earlier, the tubercle of the coronoid process of the ulna is markedly larger in males and may compromise nutrient blood flow to the nerve [9] (Fig. 2).

Retrospective studies suggest perioperative ulnar nerve (and brachial plexus) injuries occur frequently after cardiac surgery, with an incidence estimated between 1.5% to 24% of patients [16–18]. Fortunately, the incidence is lower in noncardiac surgery. Warner [15] reviewed more than 1 million records of patients undergoing noncardiac surgery at the Mayo Clinic over a 35-year period and found an incidence of 1:2729 patients (0.04%) of persistent ulnar neuropathy; of these, 9% were bilateral. In patients surviving at least 1 year after surgery, approximately half recovered complete motor and sensory function and were asymptomatic [15]. More recent prospective data from the Mayo Clinic suggests a higher incidence of 1:215 (≈ 0.5%) in adult patients undergoing noncardiac surgery [19]. This frequency closely approximates that of Alvine et al [20], who prospectively identified postoperative ulnar neuropathy in 0.26% of 6538 surgery patients.
Numerous factors have been associated with perioperative ulnar nerve injuries [5]. These include induced or prolonged hypotension, use of automated blood pressure cuffs [21], subclinical diabetes or other unrecognized medical illness [22], local anesthetic toxicity, manipulations of the brachial plexus during surgery, and, of course, stretch or compression [23] during surgical positioning [4,5,24]. However, the most consistent risk factors appear to be male gender, prolonged hospitalization, and extremes of body habitus [15,19,20,25]. We also speculate that people who have nocturnal or positional ulnar nerve paresthesias or dysesthesias could be at increased risk for perioperative neuropathy [19,20].

Thus, prolonged supine positioning of males, often with concomitant administration of sedative or narcotic medication (which obtunds the sensorium) may increase the risk for aggravating a preexisting, subclinical ulnar nerve disorder. This is supported by the consistent observation that patients with perioperative ulnar neuropathy do not report symptoms until 2 to 7 days after surgery [19,20,25]—that is, often after several days of supine bedrest.

Ulnar neuropathy appears to be most often caused by unknown factors rather than by the extent of positioning or padding of extremities during surgery and anesthesia. This conclusion is supported by the observation that ulnar nerve injury occurs with an equal frequency in medical and surgical patients [25] hospitalized for more than 2 days (Table 2).

Thus, the real etiology of ulnar neuropathy may be predisposing anatomic and other risk factors in males (due to anatomic gender variations of the cubital tunnel) associated with prolonged periods of bedrest in the supine position (whether during or after surgery or for medical conditions) [19,25]. Supine patients tend to flex their elbows naturally to a resting angle of 90° to 110°. This position produces the anatomic condition of the CTR illustrated in Fig. 3, emphasizing how the retinaculum can directly stress the ulnar nerve at the elbow flexed to this degree. We have ongoing clinical investigations that corroborate these anatomic findings. We recently reported significant changes in ulnar nerve sensory thresholds associated with elbow flexion of 110° as compared with 0°. In the flexed position, nearly all volunteers reported ulnar nerve paresthesias, and nearly all demonstrated significant increases in C fiber (second pain) sensory threshold without influencing either Aα or Aδ fiber function [26].

<table>
<thead>
<tr>
<th>Primary diagnosis of hospitalized patients</th>
<th>Prospective incidence of ulnar neuropathy (after 48–72 h)</th>
<th>Percentage</th>
<th>Confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical</td>
<td>2/986</td>
<td>0.2</td>
<td>0.02–0.73</td>
</tr>
<tr>
<td>Surgical</td>
<td>7/1502</td>
<td>0.47</td>
<td>0.2–1.0</td>
</tr>
<tr>
<td>Arithmetic sum</td>
<td>9/2488</td>
<td>0.36</td>
<td>—</td>
</tr>
</tbody>
</table>

Summarized from information in references [19,25].
Clinical investigations and anesthetic implications

Anesthesiologists and surgeons may make erroneous assumptions about ulnar neuropathy, enabling incorrect conclusions regarding etiology [27,28]. Our goal has been to develop quantitative and physiologic models of ulnar nerve injury, such as determining which arm position minimizes pressure on the ulnar nerve in patients about to undergo anesthesia and surgery. Previous admonitions were in conflict because some authors recommended abduction of the arm to more than 60° and supination of the hand and wrist [29], whereas others stressed the need to avoid pronation of the forearm [30]. We examined the role of arm position on pressure exerted over the ulnar nerve at the elbow [31], using a pressure-sensing pad, illustrated in Fig. 4 [32]. This surface map detected pressure to 1 cm² resolution and allowed us to determine surface pressure distribution over the ulnar nerve (Fig. 5).

Forearm supination minimized direct pressure exerted over the ulnar nerve, even while accounting for the fact that this position produced the least contact

Fig. 4. Photograph of the 46-cm × 46-cm flexible, pressure sensor pad that contains 1296 embedded microsensors, each 0.64-mm thick. Pressure mapping software samples each cell at 5 Hz and determines pressure between 2 and 220 mm Hg [9,33]. The subject’s arm shown here is resting passively with the forearm in pronation, part of the protocol described in the text. (From Prielipp RC, Morell RC, Walker FO, et al. Ulnar nerve pressure: influence of arm position and relationship to somatosensory evoked potentials. Anesthesiology 1999;91:345–54; with permission.)
area between the ulnar nerve and the weight-bearing surface [31] (see Table 3).

Note that pressure localized over the ulnar nerve was greatest with the forearm pronated (see Table 3). Indeed, with the forearm in supination, only 6 of 50 subjects manifest any pressure over the ulnar nerve. Furthermore, supination minimized direct pressure to the ulnar nerve, whereas pronation of the forearm produced the largest pressure regardless of arm abduction between 30° and 90°. With the forearm in neutral orientation, pressure over the ulnar nerve decreased as the arm was abducted from 30° to 90° (Fig. 6).

In a related study [31], we compared the onset of clinical paresthesia to the onset and severity of somatosensory-evoked potential (SSEP) electrophysiologic changes during intentional ulnar nerve compression. The ulnar nerve of 16 male subjects was monitored with standard SSEP techniques. We then intentionally

Table 3
Pressure recorded over the ulnar nerve of 50 volunteers with the forearm in three positions

<table>
<thead>
<tr>
<th>Arm position</th>
<th>Ulnar nerve pressure (mm Hg)</th>
<th>Ulnar nerve contact area (cm²)</th>
<th>No. subjects with no pressure on ulnar nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean Median (Range)</td>
<td>Mean Median (Range)</td>
<td></td>
</tr>
<tr>
<td>Supination</td>
<td>2 0 (0–23)</td>
<td>2.2 1 (0–9)</td>
<td>44</td>
</tr>
<tr>
<td>Neutral</td>
<td>69 22* (0–220)</td>
<td>5.5 5* (0–13)</td>
<td>14</td>
</tr>
<tr>
<td>Pronation</td>
<td>95 91*† (0–220)</td>
<td>5.8 6* (0–12)</td>
<td>7</td>
</tr>
</tbody>
</table>

(Modified from [31]; with permission.)

* $P \leq 0.0001$ by Mann-Whitney $U$ test (supine compared with pronated and neutral).

† $P \leq 0.05$ by Mann-Whitney $U$ test (pronated compared with neutral).
placed a wooden dowel snugly in the ulnar groove, allowing the full weight of the arm to rest directly on the wooden block while simultaneously monitoring pressure and function of the ulnar nerve. Subjects were specifically instructed to report the first symptoms of tingling, numbness, paresthesia, weakness, or altered temperature sensation distal to the elbow in their test extremity, for a maximum of 60 minutes. Maximal decreases in SSEP waveforms were recorded, especially the N9-N9' wave amplitude, sometimes called the Erb point, which is superficial to the brachial plexus [33,34].

Eight subjects reported progressive hand paresthesia 37 minutes after placement of the wooden block in the ulnar groove, and all 8 of these subjects also manifested significant SSEP changes with a mean decrease in the N9-N9' amplitude of −44% (range, −20 to −71%). In contrast, 8 volunteers reported no ulnar paresthesia during 60 minutes of similar pressure from the wooden block in the ulnar groove. Nevertheless, these 8 subjects demonstrated a mean SSEP decrease in the N9-N9' waveform amplitude of −44% (range, −19 to −72%) (Table 4). These results suggest that up to 50% of male patients who experience pressure on peripheral nerves sufficient to impair electrophysiologic function may be at risk because they do not perceive a concurrent paresthesia of that ulnar nerve. Thus, significant ulnar nerve compression and dysfunction can occur in unsedated males in the absence of perceived symptoms. Recognizing that post-surgical patients are often sedated or receive opioids, we speculate that male surgical patients could be at even greater
Medicolegal implications

Nerve injuries represent the second largest class of adverse outcome in the ASA Closed Claims Study database [1,2]. In adults, these injuries often result in chronic pain and permanent disability, thereby initiating tort litigation and claims for damages [2–4]. Until recently, approximately two thirds of cases for ulnar nerve injury settled in favor of the plaintiff, despite the fact that the alleged mechanism of nerve injury could be identified in only 1 of 20 and that independent reviewers concluded that the standard of care had been met in most closed claims. Indeed, 27% of cases involving ulnar nerve injury occurred despite the documentation of specific protective padding at the elbow.

Many such tort cases were based on the assumption that nerve injury does not occur without negligent external pressure or stretch against a nerve [4,8,35]; therefore, anesthesia personnel must have deviated from standards of care in positioning the patient (plaintiff). Indeed, nerve injury represented a classic damaging event in which the legal doctrine of res ipsa loquitur (the thing speaks for itself) was evoked. When res ipsa loquitur is accepted by the judge or jury, the burden of proof is shifted from the plaintiff to the anesthesia provider-defendant to prove that he or she did not cause the injury through negligence (a challenging legal hurdle). Because anesthesia providers render patients unconscious and unable to protect themselves, this doctrine is invoked more frequently in cases against anesthesiologists than any other specialty.

Thus, while some experts continue to opine that “but for negligence” (ie, improper positioning of the affected arm in an anesthetized or sedated patient) ulnar neuropathies should not occur, we know that peripheral neuropathies can arise in the absence of identifiable predisposing conditions, depression of

Table 4
Data from 16 male subjects with SSEP monitoring during intentional application of pressure to ulnar nerve

<table>
<thead>
<tr>
<th>Paresthesia</th>
<th>No. subjects</th>
<th>Parameters</th>
<th>Time to SSX (min)</th>
<th>SSEP change (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>8</td>
<td>Mean</td>
<td>37</td>
<td>−44</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Median</td>
<td>33</td>
<td>−45</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range</td>
<td>20–59</td>
<td>20 to −71</td>
</tr>
<tr>
<td>No</td>
<td>8</td>
<td>Mean</td>
<td>60</td>
<td>−44</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Median</td>
<td>60</td>
<td>−45</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range</td>
<td>—</td>
<td>19 to −72</td>
</tr>
</tbody>
</table>

P value: Mann-Whiney U test comparing subjects who reported ulnar nerve paresthesia (n = 8), with those who denied symptoms of ulnar nerve paresthesia (n = 8).
consciousness, or trauma [36]. As previously noted, ulnar nerve injury occurs even when there is documentation of specific protective padding at the elbow. We also now know that ulnar neuropathy occurs in 0.2% of hospitalized patients who have not undergone surgery or anesthesia. And last, our data [31] confirm that changes in ulnar nerve function can occur in the absence of clinical paresthesia even in awake, unsedated males.

**Recommendations**

Anesthesiologists should familiarize themselves with the ASA Practice Advisory for the Prevention of Perioperative Neuropathies [37]. This document summarizes the findings of a 10-member task force focused on issues of perioperative positioning of adult patients during anesthesia and surgery. Some of the important consensus highlights regarding the ulnar nerve include:

- Arm abduction should be limited to \( \leq 90^\circ \) in the supine position.
- Arms should be positioned to decrease pressure on the postcondylar groove of the humerus (ulnar groove). When arms are tucked at the sides, a neutral forearm position is recommended.
- Padded arm boards should be used.
- Padding at the elbow *may* decrease the risk for upper extremity neuropathy, but data are lacking or are in conflict.

It is particularly helpful to do a final check and make a notation about the position of the arms and hand as part of every anesthetic record, such as: “arms 85° bilaterally, palms up, elbows padded, ulnar grooves clear.” Indeed, for particularly long surgeries, it may be prudent to recheck and again document the

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**Fig. 7.** Surgical treatment for cubital ulnar nerve compression syndrome includes medial epicondylectomy, simple decompression, or anterior transposition of the ulnar nerve into subcutaneous, intramuscular, or submuscular locations. For patients with severe (McGowan grade 3) ulnar nerve symptoms, or those in whom nerve subluxation is confirmed intraoperatively, anterior intramuscular transposition (as illustrated here) may have the best long-term outcome.
safe position of the arms and elbows, if the surgical procedure and position allow for such observation. Last, some institutions are routinely asking patients about symptoms and documenting function of the ulnar nerve just before post-anesthesia care unit discharge. Such notations help establish a time line when and where nerve symptoms and nerve dysfunction first occur. To the above recommendations, we would add that for most patients, supination of the forearm minimizes pressure over the ulnar nerve during positioning of the supine, adult patient. In the neutral (thumb up) position, ulnar nerve pressure decreases as the arm is abducted between $30^\circ$ and $90^\circ$. Because many male patients fail to perceive clinical symptoms of ulnar nerve compression, attention should include the postoperative convalescence of bedridden patients. Increased vigilance on the part of nurses and other health care personnel could reduce the time during which patients rest their flexed elbows on a firm mattress.

**What to do if your patient has symptoms of perioperative nerve injury**

Many patients have musculoskeletal symptoms consistent with perioperative nerve injury. It is always reasonable to perform and record a careful neurologic examination on such patients. If preexisting nerve injury is suspected, it may be useful to request electrodiagnostic testing in the first few days after surgery. (If muscle fibrillations are identified, chronic muscular denervation must have been present.) However, for most patients it is reasonable to delay electrodiagnostic testing for at least a few days because most patients will improve rapidly. In any case, there is no known effective treatment. We recommend that you contact your risk management specialist or insurance carrier if your patient has not recovered within a few days. You may want to consult with a neurologist familiar with recent developments in this field, especially if evidence for motor weakness is present. It is not helpful to have consultation notes that include such jargon as positioning injury recorded on official medical records. Conversations with the patient should urge a tone of optimism tempered with the realistic possibility that the patient may be left with some degree of nerve dysfunction. Orthopedic or neurosurgical consultation may even recommend surgical treatment [38,39] (Fig. 7).

**References**


