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Perioperative neuropathies, blindness, and catastrophic patient positioning problems

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Perioperative neuropathies, blindness, and catastrophic patient positioning problems

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Perioperative neuropathies, vision loss, and positioning-related problems have received increasing attention from the lay press, plaintiffs’ lawyers, the anesthesiology community, and clinical researchers in recent years. This review will provide an update of current findings and discuss possible mechanisms of injury for these potentially devastating problems.

UPPER EXTREMITY NEUROPATHIES

Any nerve that passes into the upper extremity may sustain an injury or convert from an abnormal but asymptomatic state to a symptomatic state during the perioperative period. Of the major nerve structures of the upper extremity, the ulnar nerve and brachial plexus nerves are the most common to become symptomatic and lead to major disability during the perioperative period (1-3).

Ulnar neuropathy. Improper anesthetic care and patient malpositioning have been implicated as causative factors in the development of ulnar neuropathies since reports by Budinger (4) and Garriques (5) in the 1890s. These factors likely play an etiologic role for this problem in some surgical patients. Other factors, however, may contribute to the development of perioperative ulnar neuropathies. In a series of twelve inpatients with newly acquired ulnar neuropathy, Wadsworth and Williams (6) determined that external compression of an ulnar nerve during surgery was a factor in only two patients. A prospective study at Mayo Clinic found that medical as well as surgical patients develop ulnar neuropathies during inpatient and outpatient care (7). It is clear that both surgical and medical patients may develop ulnar neuropathies during or after an episode of care.

Typically, anesthesia-related ulnar nerve injury is thought to be associated with external nerve compression or stretch caused by malpositioning during the intraoperative period. While this implication may be true for some patients, three findings suggest that other factors may contribute. First, a retrospective study has found male gender, high body mass index (≥ 38) and prolonged bedrest postoperatively to be associated with these ulnar neuropathies (8). Of these, male gender is the factor most commonly associated with perioperative ulnar neuropathy. Various reports suggest that 70-90% of patients who develop this problem are male (1-3,6,8,9). Second, many patients with perioperative ulnar neuropathies have a high frequency of contralateral ulnar nerve conduction dysfunction (9). This finding suggests that many of these patients likely have asymptomatic but abnormal ulnar nerves prior to their anesthetics, and these abnormal nerves may become symptomatic during the perioperative period. Finally, many patients do not notice or complain of ulnar nerve symptoms until more than 48 hours after their surgical procedures (8,9). A prospective study of ulnar neuropathy in 1,502 surgical patients found that none of the patients developed symptoms of the neuropathy during the first two postoperative days (10).

Currently available data suggest that perioperative ulnar neuropathy may be caused by factors other than improper patient positioning and padding of extremities during surgery. Elbow flexion, especially to greater than 100°, can elongate the ulnar nerve and tighten the cubital tunnel retinaculum, directly compressing the ulnar nerve (Figures 1-3) (11-13). The clinical significance of this finding, however, is unclear. Morell et al (14) found that elbow flexion did not inhibit ulnar nerve perception, while direct pressure on the ulnar nerve in the post-condylar groove did.

External compression of the ulnar nerve in the absence of elbow flexion also may damage the nerve. Compression within the bony groove posterior to the medial epicondyle...
may be possible. In a very innovative study Prielipp, et al\(^{(15)}\) have shown that forearm rotation, especially pronation, can increase pressure in the postcondylar groove (Figure 4). Contreras, et al\(^{(16)}\) have noted that the nerve may be more easily compressed by external forces distal to the medial epicondyle where the nerve and its associated artery are quite superficial than in the postcondylar groove (Figure 5).

**Brachial plexus neuropathy.** Brachial plexus neuropathies may masquerade as ulnar neuropathies or be associated with symptoms that suggest injuries to other nerve structures. In general, brachial plexus neuropathies are associated with median sternotomy\(^{(17-19)}\). This neuropathy often involves stretch or compression of the brachial plexus during sternal separa-

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**Figure 1.** The proximal edge of the roof of the cubital tunnel is formed by a retinaculum that originates on the medial epicondyle and inserts on the olecranon. It is distinct from the aponeurosis of the flexor carpi ulnaris (FCU) with which its distal margin blends.

**Figure 2.** In this medial-to-lateral view of the right elbow, the cubital tunnel retinaculum (CTR) is lax in extension (A) as it stretches from the medial epicondyle (ME) to the olecranon (Ol). The retinaculum tightens in flexion (B) and can compress the ulnar nerve (arrow).

**Figure 3.** Intraneural and extraneural pressures for the ulnar nerve within the cubital tunnel increased dramatically with elbow flexion greater than 100°.

**Figure 4.** In supination, the pressure over the ulnar nerve is uniformly low, and most of the data are clustered around the zero line. Prielipp RC, et al: Anesthesiology 1999; 91:345-354.

**Figure 5.** The ulnar nerve and its primary blood supply in the proximal forearm, the posterior ulnar recurrent artery, are very superficial and appear to be susceptible to compression from external pressure as they pass posteriomedially to the tubercle of the coronoid process. The tubercle is larger in men than women, and the adipose layer in this area is thinner in men\(^{(16)}\).
Other potential mechanisms of injury include direct trauma from fractured first ribs. In general, brachial plexus neuropathy does not appear to be related to a patient’s arm position or padding during the sternotomy and related procedures\(^ {20}\).

The brachial plexus also is vulnerable to stretch in a patient who is positioned prone (Figure 6)\(^ {21}\). Stretch of the brachial plexus, especially its lower trunks, is most likely to occur when the head is turned to the contralateral side, the ipsilateral shoulder is abducted, and the ipsilateral elbow is bent. Other potential problems are noted in the legend for figure 6. Although this position is commonly used during surgical procedures and the frequency of perioperative brachial plexus neuropathy is low, it would appear prudent to place the arms at the patient’s side whenever possible to decrease the risk of brachial plexus stretching.

**LOWER EXTREMITY NEUROPATHIES**

Although neuropathies of the lower extremities may occur in a variety of patient postures, most of these occur in patients who are undergoing procedures while placed in a lithotomy position. These neuropathies often have been considered to be preventable and to occur because of poor intraoperative care (e.g.: improper positioning or padding) or judgment (e.g.: excessively prolonged use of lithotomy position)\(^ {22}\). This perception has significant impact on the outcomes of medicolegal cases involving these types of problems\(^ {23}\). Interestingly, the majority of plaintiffs in medicolegal cases involving lower extremity neuropathies name anesthesiologists and surgeons in their complaints. In contrast, plaintiffs in cases involving upper extremity nerves often do not name surgeons.

A number of studies have suggested that there are many factors other than improper intraoperative care that may contribute to the risk of lower extremity nerve injury\(^ {24-26}\). A 1994 retrospective review of patients in lithotomy positions found that the most common lower extremity neuropathies were the common peroneal (81%), sciatic (15%), and femoral (4%)\(^ {27}\). The authors found specific patient characteristics that contributed to the risk of neuropathy. A more recent prospective study found that the longer patients were in lithotomy, the greater their risk of developing a neuropathy\(^ {28}\). The obturator and lateral femoral cutaneous (LFC) nerve were most often involved in this study.

**Obturator and lateral femoral cutaneous neuropathies.** Litwiller, et al\(^ {29}\) subsequently evaluated the strain of the obturator and LFC nerves associated with lithotomy positions in fresh cadavers. They found that neither hip flexion nor abduction increased strain on the LFC nerve. However, abduction to 30 degrees without concomitant hip flexion dramatically increased strain on the obturator nerve.

**Common peroneal neuropathy.** The common peroneal nerve is superficial as it passes down the neck of the fibula. Because it is quite exposed at this level, it may be easily compressed and injured. Although direct compression of the peroneal nerve by leg holders commonly has been considered the primary mechanism of injury in peroneal neuropathy, a recent study suggests that the superficial peroneal nerve may be affected distal to the tuber calcanei\(^ {28}\). The authors speculated that compressive stockings or wraps may be etiologic factors for this finding.

**Sciatic neuropathy.** The same forces that contribute to stretch injuries of the hamstring group muscles (e.g.: biceps femoris muscle) may stretch the sciatic nerve. Simultaneous hyperflexion of the hip and extension of the knee will stretch and possibly injure the sciatic nerve. This set of actions can occur during the establishment and maintenance of a lithotomy position. A patient in a lithotomy position may passively shift towards the caudal end of an operating table when placed in a head-up posture or be actively shifted caudally by a member of the operating team in an attempt to obtain increased exposure of the perineum. This movement may increase the flexion of the hips and extension of the legs if the legs are already fixated within leg holders. It would seem prudent to confirm that the flexor muscles of the knee (e.g.: hamstring group) are not taut after placing a patient’s legs into any lithotomy position.

**Femoral neuropathy.** Unlike most other neuropathies in which the anesthesia provider is often considered to have acted improperly in order for the neuropathy to occur, those involving the femoral nerve and its cutaneous branches often are considered to result from improper placement of abdominal wall retractors and direct compression of the nerve. When related to retractors, the assumption is that retractors place continuous pressure on the iliopsoas muscle and ei-

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**Figure 6.** Sources of potential injury to the brachial plexus and its peripheral components in a pronated patient. Head position stretching plexus against anchors in shoulder (A). Closure of retroclavicular space by chest support with arms at side; neurovascular bundle trapped against first rib (B). Head of humerus thrust into neurovascular bundle if arm and axilla are not relaxed (C). Compression of ulnar nerve in cubital tunnel (D). Area of vulnerability of radial nerve to compression above elbow (E).
ther stretch the nerve or cause it to become ischemic by occluding the external iliac artery or penetrating vessels of the nerve as it passes through the muscle(30).

**PRACTICAL CONSIDERATIONS FOR NEUROPATHIES**

Efforts to prevent perioperative neuropathies are frequently debated, and there often is confusion on how to manage a neuropathy once it has occurred. In general, there are no data to support recommendations on any of these issues. Therefore, the following opinions have been formulated by personal experience, guided by advice from neurologists who primarily care for patients with peripheral neuropathies, and seasoned or supported by speculation derived from anecdotal case reports.

Padding exposed peripheral nerves. Many types of padding materials are advocated to protect exposed peripheral nerves. They often consist of cloth (e.g.: blankets and towels), foam sponges (e.g.: “eggcrate” foam), and gel pads. There are no data to suggest that any of these materials is more effective than any other, or that any is better than no padding at all. A good rule-of-thumb would be to position and pad exposed peripheral nerves to 1) prevent their stretch beyond normally tolerated limits while awake, 2) avoid their direct compression, if possible, and 3) distribute over as large an area as possible any compressive forces that must be placed on them.

*What to do if your patient develops a neuropathy?* Although each situation is unique and requires careful assessment, the following guidelines may suggest a basic course of action that will lead to appropriate care:

- Is the neuropathy sensory or motor? Sensory lesions are more frequently transient than motor lesions. If the symptoms are numbness and/or tingling only, it may be appropriate to inform the patient that many of these neuropathies will resolve during the first 5 days(10). The patient should be instructed to avoid postures that might compress or stretch the involved nerve. Arrangements should be made for frequent contact General Guidelines. There are sufficient, albeit retrospectively-derived, risk factors in cardiac surgical patients to suggest that avoiding prolonged perioperative anemia and hypotension may reduce the risk of AION and/or PION(31). Similar data are not available for patients undergoing spine surgery while positioned prone. However, because many cases of PION in this surgical population occur in patients who receive large intraoperative infusions of crystalloids during long (> 6 hr.) procedures, it may be prudent to use more colloid solutions and avoid prolonged episodes of perioperative anemia and hypotension(40).

*Registry of cases.* The ASA Committee on Professional Liability established a registry for cases of vision loss in 1999. The ASA Postoperative Visual Loss Registry may process a sufficient number of cases in the next several years to allow analyses for risk factors in non-cardiac surgical patients. Dr. Lorri Lee (University of Washington) is the director of the registry. All cases in the registry are anonymous. Please report any known cases to www.asaclosedclaims.org.

**REFERENCES**