Postoperative ulnar neuropathy typically presents 2 to 7 days after surgery. Symptoms can be purely sensory or may include motor deficits as well. The ulnar nerve provides cutaneous sensation to the fourth and fifth digits and innervates the majority of the intrinsic muscles of the hand. Therefore, patients may report decreased sensation in the ulnar digits and decreased grip strength and weakness with finger abduction. Prolonged motor deficits can result in clawing of the ulnar digits, as the maintained strength of the unaffected extrinsic muscles overpowers the weaker intrinsics. The available literature suggests that roughly half of patients with ulnar nerve PPNI recover in the short term (weeks to months), whereas the other half continue to exhibit persistent deficits at 1 to 2 years postoperatively.[12,13]

The ulnar nerve is most sensitive to stretch and compression in and around the cubital tunnel, and this area should always be well padded. Biomechanical cadaveric studies have shown that the cross-sectional areas of both the cubital tunnel and ulnar nerve significantly decrease with increasing elbow flexion.[14,15] The ulnar nerve has also been shown to flatten and press against the medial epicondyle with increasing elbow flexion.[15] In one study of intraneural pressures, ulnar nerve pressures were found to increase significantly with an elbow flexion of 90 degrees or more.[14] Another study demonstrated that the proximal ulnar nerve lengthened significantly with progressive elbow flexion, resulting in 18% nerve lengthening at 135 degrees of flexion.[16] Taken together, these findings suggest that an elbow flexion of 90 degrees or more places the ulnar nerve at risk. Positioning of the forearm also plays a role in the development of ulnar neuropathy. A biomechanical study of awake volunteers demonstrated that ulnar nerve compression was lowest with forearm supination, with a significant increase in pressure with neutral forearm rotation, and the highest pressures noted in forearm pronation.[17]

Brachial Plexopathy

PPNI of the brachial plexus is the second most common form of peripheral neuropathy in the upper extremity. These injuries represent 20% of claims related to PPNI in the anesthesia closed claims database.[2] The brachial plexus is a confluence of all of the cervical and thoracic nerve roots supplying the entire upper extremity; disparate presentations of brachial plexus injury may therefore be seen. Most PPNIs stemming from the brachial plexus affect the upper nerve roots, resulting in an Erb's palsy-like constellation of symptoms (ie, motor dysfunction of the proximal shoulder girdle and intact hand function).[18] The majority of brachial plexus injuries related to spine surgery tend to be positioning related. There are a number of positioning factors that predispose patients to brachial plexopathy. In the prone position, risk factors include shoulder abduction over 90 degrees, posterior pressure on the shoulder, and external rotation of the arm. In the lateral position, hyperabduction of the elevated arm with the neck tilted downward has been identified as a risk factor. Finally, in the supine position, traction on the shoulder with contralateral neck tilt has been associated with risk of brachial plexopathy. The majority of PPNIs of the brachial plexus resolve over time but may require months of physical therapy to preserve motion.[18-20]

Median Neuropathy

PPNI of the median nerve is rare, particularly when compared with ulnar nerve and brachial plexus injury. Median nerve injuries represent only 4% of claims related to PPNI in the anesthesia closed claims database.[2] The median nerve provides sensation to the radial 3 digits of the hand and innervates most of the thenar muscles. Therefore, injury to the median nerve usually presents with decreased sensation in the thumb, index, and long finger and with decreased thumb opposition and pinch strength. It is unlikely for operative patient positioning to result in supraphysiologic stretching of the median nerve unless the patient has a significant preexisting elbow flexion contracture. Extension of the elbow in such patients may result in stretch injury to a chronically shortened median nerve, as it crosses the elbow. Direct compression resulting in median nerve injury has been observed. One case series described 6 cases of postoperative median neuropathy due to brachialis syndrome (ie, compressive necrosis and swelling of the brachialis, as it crosses the trochlea resulting in median neuropathy) requiring decompression.[21] Of note, none of these cases followed a spinal procedure.

Radial Neuropathy

PPNI of the radial nerve is even less common than median neuropathy, representing only 3% of claims related to PPNI in the anesthesia closed claims database.[2] The radial nerve provides sensation to a small area on the dorsoradial aspect of the wrist and innervates the major wrist extensors and extrinsic finger extensors. Therefore, radial neuropathy would be expected to present postoperatively as a wrist drop. The radial nerve is vulnerable to direct pressure about the spiral groove of the humerus, which might occur in the lateral position with the elevated arm pressing against a support for a prolonged period.[22]

PPNI OF THE LOWER EXTREMITY

PPNI of the lower extremity due to positioning in spine surgery is observed less frequently than that of the upper extremity, as the lower extremities typically rest in near-anatomic fashion, thereby limiting pressure and strain on the peripheral nerves. The 2 lower extremity peripheral nerves that course near bony prominences and may therefore be at risk are the lateral femoral cutaneous nerve (LFCN) and the peroneal nerve.

Peroneal nerve palsy may occur with any surgery in the lateral decubitus position due to the proximity of the common peroneal nerve to the bony prominence of the fibular head. Ensuring there is adequate padding about the knee is typically sufficient for prevention. The rarity of this adverse effect is highlighted by the fact that the authors were unable to identify a report of peroneal nerve palsy after lateral position spine surgery in the literature.

LFCN palsy, also known as meralgia paresthetica, is more common after spine surgery. It is typically due to pressure

against the anterior superior iliac spine and the inguinal ligament in a prone-positioned patient, resulting in compression of the LFCN, as it passes over the pelvic brim. The LFCN is a pure sensory nerve that provides cutaneous innervation to the anterolateral aspect of the thigh. LFCN palsy therefore typically presents as pain, numbness, or dysesthesia in this distribution. The incidence of LFCN palsy has been estimated at between 12% and 23.8% in prone-position spine surgery, with higher rates noted in obese patients and with prolonged surgical time.[23,24] However, LFCN palsies are often mild compared with other PPNI, and they typically resolve with observation. Complete resolution of symptoms is expected in 92% to 100% of cases.[23,24]

IONM FOR DETECTING AND PREVENTING PPNI

The use of IONM in spine surgery has become increasingly prevalent during the last 50 years. Although initially developed to allow for detection and prevention of spinal cord injury during major deformity procedures, IONM is now used by some surgeons for smaller procedures indicated for degenerative conditions.[25] IONM typically involves the use of 3 modalities: somatosensory-evoked potentials (SSEPs), motor-evoked potentials (MEPs), and spontaneous electromyography (EMG). SSEPs monitor the amplitude and latency of retrograde transmission of sensory signals from a distal stimulus on the upper or lower extremity to cranial recording electrodes. MEPs monitor the antegrade transmission of muscle action potentials in response to transcranial stimulation of the motor cortex. EMG measures skeletal muscle action potentials in response to direct nerve or spinal cord stimulation, which may occur during surgery. SSEPs, MEPs, and spontaneous EMGs are used in tandem in most cases of IONM because they monitor pathways in distinct areas of the spinal cord and measure different types of nerve function.

Because the neural pathways used for IONM generally include several different peripheral nerves of the upper and lower extremity, changes in monitoring signals can indicate pathology of both the central and peripheral nervous systems. Intermediary leads are typically placed at the popliteal fossa in the lower extremity and at Erb's point in the upper extremity to identify potential peripheral causes of signal changes, such as limb ischemia and/or peripheral nerve injury.

Given the increasing use of IONM in spine surgery and the fact that it can be used to detect changes associated with peripheral nerve pathology, it stands to reason that neuromonitoring may be a useful modality to detect and potentially prevent PPNI. Indeed, there is some literature to support the efficacy of IONM in detecting positioning-related signal changes. For example, Kamel and colleagues[26] reviewed 1000 consecutive cases for the presence of upper extremity positioning-related SSEP changes. They determined that 92% of the observed SSEP changes were positioning related, as signals returned to baseline after patient positioning was adjusted. There were no postoperative upper extremity deficits noted in any of the patients with reversible SSEP changes, leading the authors to conclude that SSEPs were effective in identifying and preventing position-related PPNIs.[26] Other retrospective studies,[27] prospective studies,[28] and case reports[29,30] have shown similar results using the same methodology. These studies have demonstrated that, for spine surgery patients, IONM is effective at identifying reversible positioning-related signal changes, and once the positioning is corrected and signals improve, patients do not tend to wake up with neurologic deficits. O'Brien and colleagues[31] performed a retrospective study in which they reported a sensitivity of 78% and a specificity of 98.5% for SSEPs in predicting upper extremity PPNI. However, it is important to notv e that this evidence is not sufficient to prove that IONM is effective in *preventing* PPNI—without control groups, it is impossible to know whether reversible signal changes would have resulted in postoperative deficits in the absence of IONM. Studies directly comparing PPNI rates in patient cohorts with and without IONM are necessary to demonstrate that it is neuroprotective, but no such studies have been performed to date.

SUMMARY

PPNI in spine surgery is a rare and preventable, but a potentially debilitating adverse effect. It is most commonly caused by peripheral nerve ischemia due to abnormal nerve lengthening or pressure and can be exacerbated by systemic hypotension. Peripheral nerve injury is more common in the upper extremities due to the frequent employment of nonanatomic positions during spine surgery. Among upper extremity PPNI, ulnar nerve injury is the most common, resulting from some combination of direct pressure and stretch. PPNI of the lower extremities is less common, except for meralgia paresthetica, which is generally characterized by mild symptoms and tends to resolve spontaneously. IONM has emerged as a potential adjunct to help detect and prevent PPNI due to positioning. Although the existing evidence supports the ability of IONM to detect reversible signal changes, controlled studies are needed to establish neuroprotection.

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