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Contemporaneous Evaluation of Intraoperative Ulnar and Median Nerve Somatosensory Evoked Potentials for Patient Positioning: A Review of Four Cases

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ABSTRACT. *Somatosensory evoked potentials (SSEPs) are a valuable tool for assessing changes in peripheral nerve pathways caused by patient positioning during spinal surgeries. These changes, when left undiagnosed, may lead to postoperative neurological sequelae. Why an upper extremity SSEP attenuates due to positioning is not necessarily clear and can be multifactorial, affecting the peripheral nerves or elements of the brachial plexus. A conduction block can occur at any point along the course of the nerve secondary to entrapment, compression, and ischemia. These mechanisms of injury may be caused by extreme body habitus, the length of the procedure, or the patient's metabolic underpinnings. The goal of neuromonitoring for positional injury is to predict and prevent both peripheral nerve and brachial plexus injuries. Using ulnar and median nerve SSEPs contemporaneously may lead to better identification of compromised structures when an SSEP change to one or both of the nerves occurs. The investigators provide four case reports*

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where intraoperative SSEP assessment of contemporaneous ulnar and median nerves prevented postoperative upper extremity neural deficits.

KEY WORDS. *Intraoperative monitoring, median nerve, positional nerve injury, SSEP, ulnar nerve.*

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INTRODUCTION

Somatosensory evoked potentials (SSEPs) are a valuable tool for assessing changes in peripheral nerve pathways caused by patient positioning during spinal surgeries (Chung et al. 2009; Kamel et al. 2006; O'Brien et al. 1994; Schwartz et al. 2000). However, there has been no clear establishment for what nerves to analyze for upper extremity positional injuries, and the mechanism of injury is not well understood (Kamel and Barnette 2014). Current techniques describe the use of ulnar nerve SSEP monitoring as the primary modality when evaluating for upper extremity positional injury during spinal procedures (Chung et al. 2009; La Neve and Zitney, 2014; Schwartz et al. 2006). The use of only one nerve SSEP monitoring for upper extremity analysis may lead to false negative findings and patients waking from surgery neurologically compromised without any detection from the upper extremity SSEP (O'Brien et al. 1994). Evaluation of multiple nerves may increase the specificity of monitoring by isolating the nerve that is at risk. Using more than one nerve due to the broader scope of the nervous system assessed (i.e., brachial plexus injury involving multiple peripheral nerves) enhances the overall sensitivity of the monitoring.

Peripheral nerves in the upper extremities or the brachial plexus can become entrapped (ulnar at cubital tunnel, median at antecubital fossa), compressed, stretched, or ischemic (occlusion of brachial artery) due to patient positioning (Kamel and Barnett, 2014; Swenson et al. 1998). Pressure (arms on arm boards), stretching (taping of shoulders in cervical spine surgeries), dislocation/subluxation of the shoulder, and compression (tucking of arms or blood pressure cuff) are potential contributors to a positionally induced SSEP degradation (Kamel and Barnett, 2014; Swenson et al. 1998). The authors present four separate cases that used contemporaneous ulnar and median nerve SSEPs to assist in the identification of SSEP changes due to patient positioning. With prompt intervention and countermeasures, neural injury was averted based on which nerve was affected.

CASE REPORT 1

A 289-pound, 70-inch, 59-year-old male with a body mass index (BMI) of 41.5 was scheduled for a C4–C7 posterior cervical stabilization with screw and rod instrumentation. Four days prior, the patient underwent an anterior cervical discectomy and fusion from C5–C7. The patient's indication for the first surgery included multiple herniated discs in

the neck causing subsequent radicular pain, weakness, and paresthesia in the bilateral upper extremities. The patient also presented with a preexisting peripheral polyneuropathy. The intraoperative neurophysiological monitoring (IONM) of SSEP, transcranial electric motor-evoked potential (TCeMEP) and spontaneous electromyography (S-EMG) was unremarkable during the first procedure and the patient awoke from anesthesia neurologically intact. The second surgery was for stabilization purposes, and the patient's initial symptoms had diminished to complaints of neck stiffness only.

Methods

Neuromonitoring Methods

Used for this procedure was a multimodal neuromonitoring paradigm with upper and lower extremity SSEPs, upper and lower extremity TCeMEP, and S-EMG. The upper extremity SSEPs assessed the median and ulnar nerves bilaterally. Placed over the median nerve at the wrist were surface stimulating electrodes with the anode positioned over the wrist crease and the cathode positioned 2 cm proximal to the anode between the flexor carpi radialis and palmaris longus tendons. For ulnar nerve analysis, surface electrodes were placed at the wrist inferior to the flexor carpi ulnaris tendon, with the anode positioned over the wrist crease and the cathode positioned 2 cm proximal to the anode. A stimulus intensity of 35 mA was delivered using a pulse width of 200 μsec and averaging 300 trials. Acquisition sites included a subdermal needle electrode placed in the chin at the level of the fifth cervical vertebra (C5s) and corkscrew electrodes placed on the scalp at the CP3 and CP4 positions of the International 10-20 system. The acquisition sites were all referenced to Fz of the International 10-20 system. For lower extremity SSEP assessment, stimulating surface electrodes were placed inferior to the medial malleolus for posterior tibial nerve stimulation bilaterally with the cathode 2 cm proximal to the anode. Placed on the anterior ankle lateral to the extensor hallucis longus tendon for deep peroneal stimulation were another set of surface stimulating electrodes. 50 mA was delivered using a pulse width of 300 μsec and averaging 300 trials. Acquisition sites included a subdermal needle electrode placed at the level of C5s and a corkscrew electrode placed on the scalp at the CPz position of the International 10-20 system referenced to Fz.

For TCeMEP stimulation, corkscrew electrodes were placed on the scalp three fingerbreadths anterior to the CP3 and CP4 locations of the International 10-20 system. Voltage between 270 and 370 was used to elicit distal compound muscle action potentials (CMAPs) from selected muscles bilaterally. The stimulus was elicited using a train of 9 with a pulse width of 75 μsec along with an interstimulus interval of 2 ms (500 Hz). The trapezius, deltoids, triceps, abductor pollicus brevis, extensor digitorum brevis, and abductor hallicus longus muscles bilaterally were the target muscle groups used for TCeMEP acquisition. S-EMG acquisition used the same target

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muscles excluding the extensor digitorum brevis and abductor hallicus longus muscles. SSEPs were deemed interpretable and reproducible based on a sufficient amount of averaging to obtain an optimal signal-to-noise ratio with minimum noise and strong response amplitudes. SSEP intervention used an alarm criterion of 50% amplitude attenuation or greater and/or an increase in peak latency 10% or greater (American Clinical Neurophysiology Society 2009).

Procedure

The patient was administered general anesthesia and intubated while on a stretcher. With the patient in the supine position, application of the IONM electrodes ensued. After the placement of a soft bite block, preposition tracings were acquired from all modalities.

Replicating SSEP recordings from all nerves stimulated were obtained, excluding the bilateral deep peroneal recordings. The inability to obtain baseline deep peroneal recordings was consistent with the patient's first procedure as well. This patient's deep peroneal SSEPs were likely absent secondary to his preexisting peripheral polyneuropathy. TCeMEPs from all four extremities were acquired.

The surgeon secured the patient in a Mayfield head holder and positioned him prone on the operating table. The surgical team tucked the patient's arms to his side and taped his shoulders to provide a more optimal view for X-Ray.

Immediately following the positioning from supine to prone, a significant attenuation from the left median nerve SSEP cortical and subcortical recordings occurred. All other SSEP recordings were unchanged. The neuromonitoring team immediately informed the surgeon and anesthesiologist, and countermeasures were taken to yield a return of the response. With an isolated change to the left median nerve, the neuromonitoring team first checked the antecubital fossa to ensure there was no compression of the left median nerve there. Padding was added to the antecubital fossa as well. A return of the response did not occur with this intervention. Next, the tuck of the arm and the tape on the shoulder were loosened. Again no return of the SSEP occurred. The surgeon then put an arm board on the operating table and rotated the arm forward, so the patient was in a "superman" position with his left arm in front of him. This intervention again did not yield a return of the evoked potential. Due to the inability to obtain a response with multiple repositions, the surgeon decided to abort the procedure. He called for the patient's stretcher and placed the patient's left arm behind the patient's back preparing to reposition supine. With the patient's arm placed behind him (Figure 1), the median nerve SSEP recording returned. When the surgeon allowed the arm to slip out of this position, the median nerve recording attenuated immediately (Figure 2). The surgeon was unable to keep the arm in a position where the median nerve SSEP remained functional for the procedure and decided to abort the surgery. The patient woke up neurologically intact.

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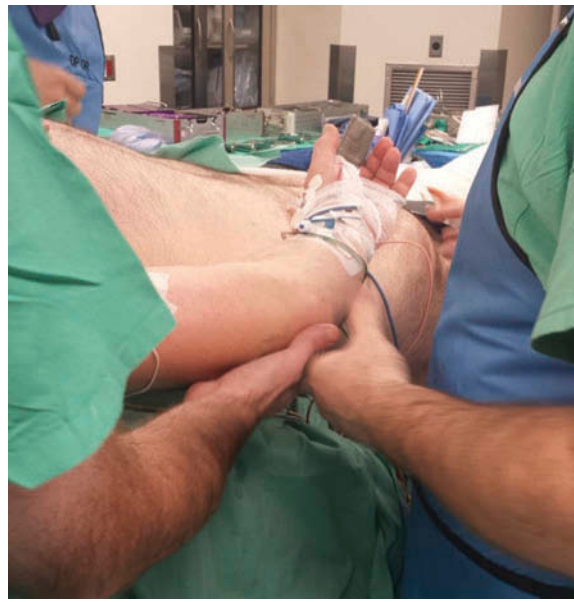

FIG. 1. Patient's arm positioned behind his back allowed for median nerve SSEP recovery.

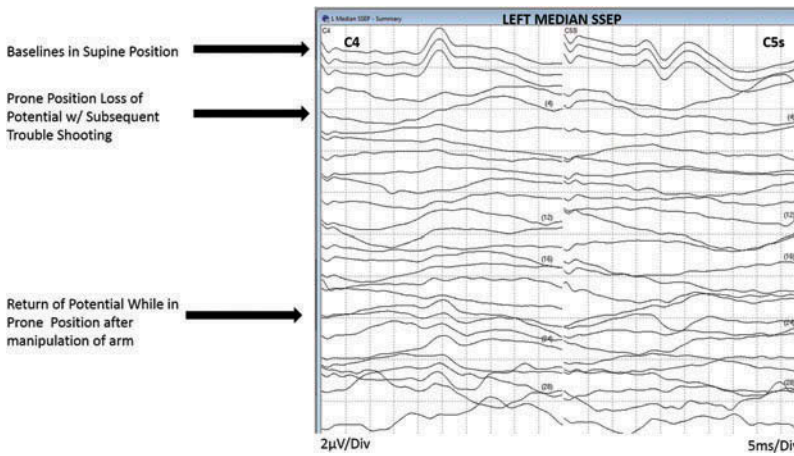

FIG. 2. Left median nerve SSEP waterfall.

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It is important to note that the TCeMEPs to the left thenar muscle in this procedure went unchanged when the loss of the median nerve SSEP occurred. We suspect that the lack of TCeMEP change is due to the sensory fibers being affected first by conduction block, which has been reported in the initial phase of neuropraxia due to acute compression (Kimura 2013; Meancock 1958; Novak and Mackinnon 1997). Colloquially it is similar to when a person experiences paresthesia in a limb when pressure is applied (e.g., arm "falling asleep" while sleeping on the limb). This acute phase of conduction block is thought to induce paresthesia secondary to ischemia or displacement of axoplasm, which manifests itself as a sensory disturbance. However, if the conduction block remains prolonged, motor fibers will become affected, and paralysis will occur (Kimura 2013; Meancock 1958; Novak and Mackinnon 1997).

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CASE REPORT 2

A 270-pound, 75-inch, 33-year-old male with a BMI of 35.0 was scheduled for a left L4–L5 hemilaminectomy and excision of herniated disc. The patient presented antalgic gait on the left, with left lower extremity pain, paresthesia, and burning. The neuromonitoring modalities used for this procedure included upper and lower extremity SSEP, with S-EMG of lower extremity muscles bilaterally. Median and ulnar nerve SSEPs evaluated the upper extremities, with deep peroneal and posterior tibial nerves evaluated for lower extremities. This procedure used the same SSEP stimulation and acquisition methods as described in Case Report 1 with subdermal needles replacing corkscrew electrodes for this procedure.

After the surgical team had positioned the patient prone ("superman" position), the neuromonitoring team obtained replicating SSEP recordings from all nerves stimulated excluding the right median nerve. After it was deduced that the problem was not technical in nature, the surgeon was informed while scrubbing at the scrub sink. He immediately ceased scrubbing and entered the operating room, where he took the patient's right arm off the arm board and placed it by the patient's side. The surgeon found the right shoulder to be dislocated likely during the initial positioning and decided to reduce the shoulder. This maneuver allowed for an immediate recovery of the SSEP (Figures 3, 3A). After repositioning the patient's arm in the "superman" position, no further complications occurred for the remainder of the procedure. The patient awoke neurologically intact.

CASE REPORT 3

A 230-pound, 69-inch, 54-year-old male with a BMI of 34.0 was scheduled for an L3–L4, L4–L5 laminectomy with fusion, instrumentation, and interbody arthrodesis. The patient presented with severe stenosis with neurogenic claudication, which included left lower extremity pain and paresthesia. The neuromonitoring modalities used for this procedure included upper and lower extremity SSEP, with lower extremity S-EMG and triggered-EMG (T-EMG). Median and ulnar nerve SSEPs evaluated the

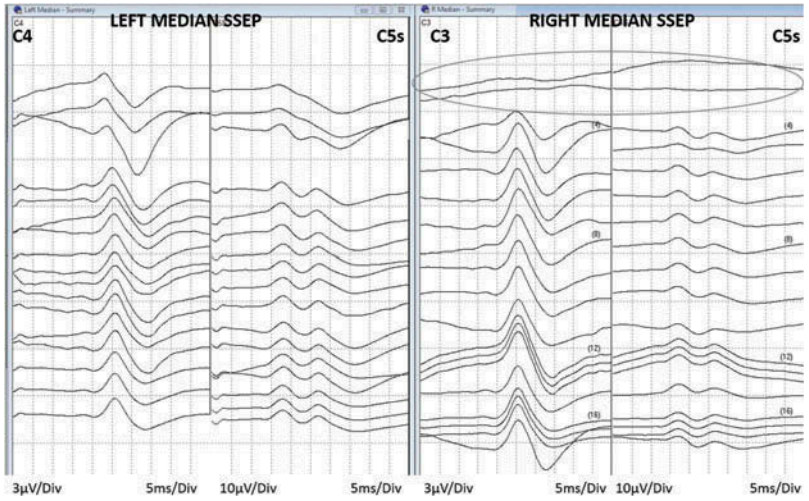

FIG. 3. Bilateral median nerve SSEP waterfall with unobtainable right median nerve SSEP at baseline (circled). Subsequent recovery occurred after surgeon reduced the shoulder.

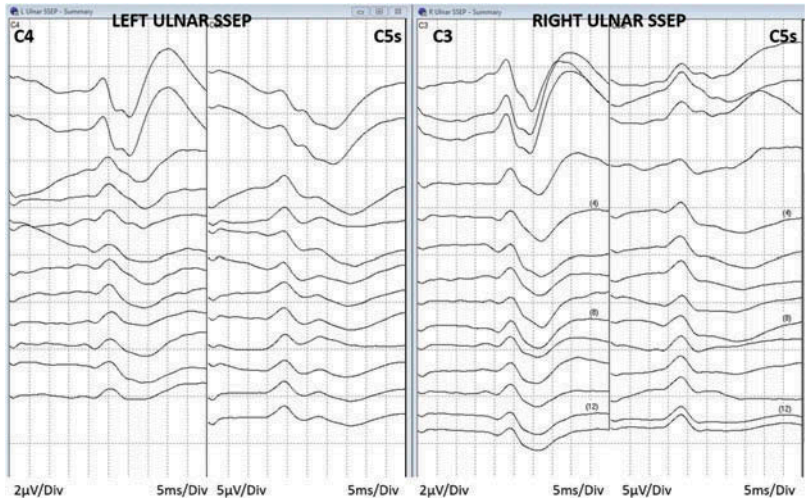

FIG. 3A. Waterfall of bilateral ulnar nerve SSEP showing well-defined replicating data.

upper extremities, with deep peroneal and posterior tibial nerves evaluated for lower extremities. This procedure used the same SSEP stimulation, and acquisition methods as described in Case Report 1 and Case Report 2.

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The surgical team positioned the patient on a Hall-Relton Jackson table in a "superman" position. Next, the neuromonitoring team acquired replicating SSEPs from all nerves stimulated. The first SSEP trial after the surgical exposure revealed a bilateral change in the ulnar nerve SSEP recordings. The left ulnar nerve cortical recording had a prolongation in peak latency (23% increase from baseline), with significant amplitude degradation (80% decrease from baseline) and temporal dispersion of the waveform. The subcortical recording also became temporally dispersed with a diminution of amplitude as well (74% decrease). The right ulnar SSEP recording had a significant attenuation of the cortical and subcortical recordings (91% and 75%, respectively), with both recordings showing a prolongation in peak latency as well (20% and 13%, respectively) (Figure 4). Bilateral median nerve SSEP recordings remained within normal limits (Figure 5). The neuromonitoring team immediately informed the anesthesiologist and surgeon, and countermeasures were taken to prevent a postoperative position-induced neurological injury. The degradation from the bilateral ulnar nerve SSEPs is suspected to be due to both arms significantly pressing into the arm boards just distal to the cubital tunnel likely secondary to the patient's body habitus and subsequently causing compression of the bilateral ulnar nerves. The neuromonitoring and anesthesiology team alleviated the pressure by placing the arms on foam pads just distal to the cubital tunnel bilaterally (Figure 6). This maneuver reversed the left ulnar nerve positional SSEP degradation immediately; however, the degradation in the right ulnar SSEP recording persisted. The anesthesiology team adjusted the right arm three more times, but it was not until the fourth adjustment that the response returned to within baseline limits. Rotating the right arm from a 90-degree angle down to a 45-degree angle was the intervention that worked best for this patient. Forty-three minutes

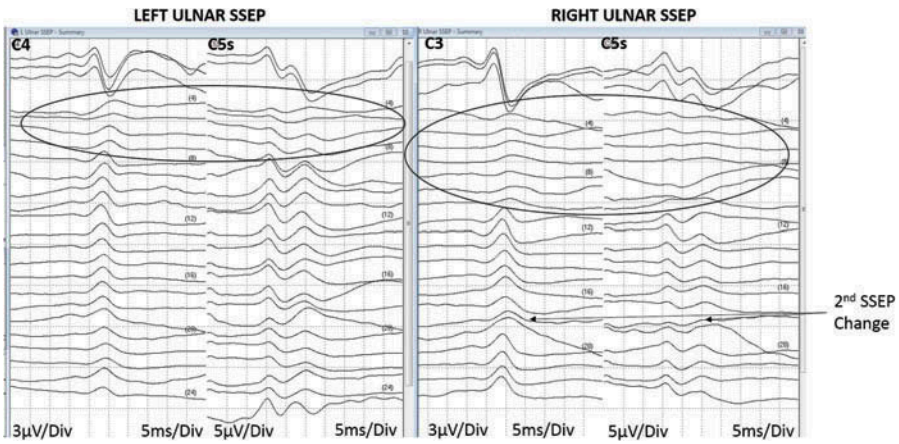

FIG. 4. Bilateral ulnar nerve SSEP waterfall with attenuation of amplitude, prolongation of latency, and temporal dispersion (circled). Arrows indicate the second change to the right ulnar SSEP.

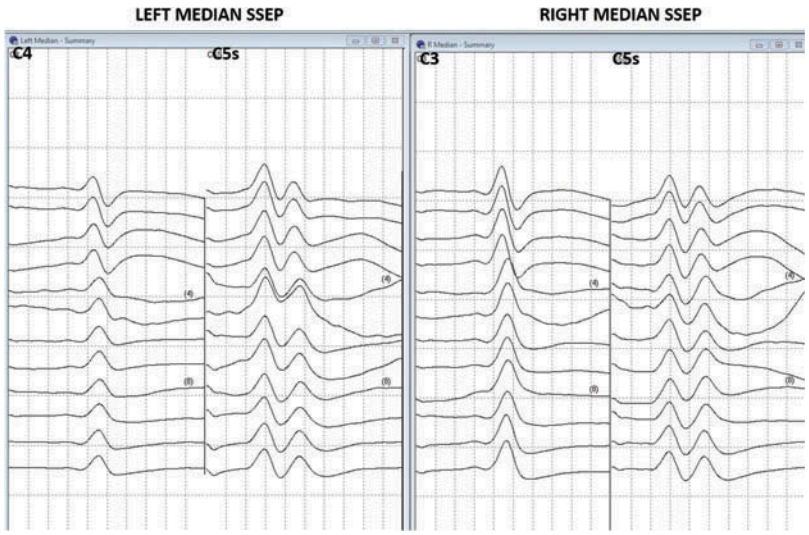

FIG. 5. Waterfall of bilateral median nerve SSEP showing well-defined replicating data.

later, the right ulnar SSEP deteriorated once again, with a drop in subcortical amplitude and significant morphology change with temporal dispersion from the cortical recording (Figure 4). The anesthesiology and neuromonitoring teams placed two pillows under the patient's arm for intervention during the second degradation of the right ulnar nerve SSEP recording, which allowed the SSEP responses to recover. The patient woke up neurologically intact with no new subjective upper extremity complaints.

CASE REPORT 4

A 290-pound, 77-inch, 56-year-old male with a BMI of 34.4 was scheduled for a laminectomy of L1–L5. The patient had a medical history of peripheral polyneuropathy and surgical history for posterior cervical fusion. SSEP evaluation came from stimulating the bilateral ulnar, median, deep peroneal, and posterior tibial nerves. The surgical team positioned the patient in a "superman" position and the neuromonitoring team obtained replicating SSEP baselines from all nerves stimulated. Thirty minutes into the procedure, a contemporaneous attenuation of the left ulnar and left median nerve SSEPs was noted. After ensuring that the deterioration of SSEP recordings was not technical in nature, the surgeon and anesthesiologist were informed, and the anesthesiologist repositioned the left arm at the shoulder. The patient's chest was checked to make sure there was no pressure on the axilla. After multiple attempts to reposition the arm at the shoulder, the SSEPs from both nerves returned to baseline limits (ulnar

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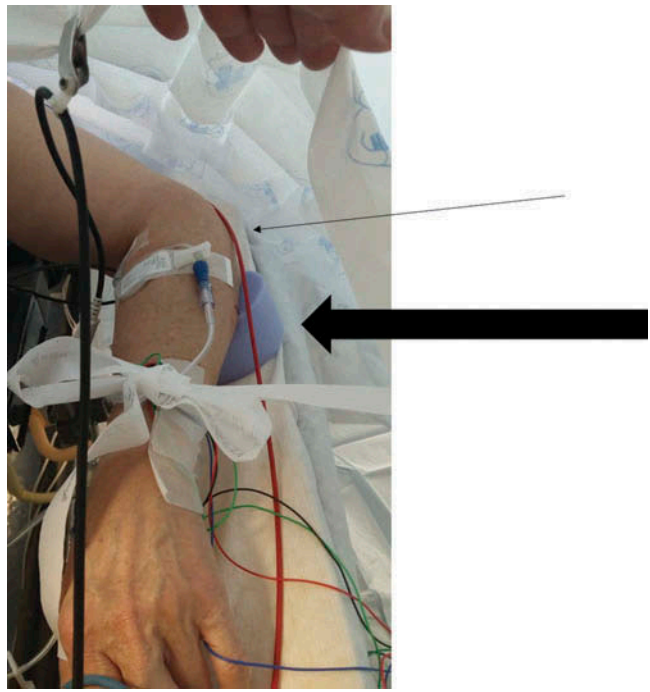

FIG. 6. Foam pad placed distal to cubital tunnel (block arrow). Thin arrow is pointing to cubital tunnel (medial epicondyle of the humerus).

returning before median). However, both nerves deteriorated once more during the procedure and intervention was implemented with the subsequent intraoperative recovery of the SSEP (Figure 7). The patient awoke neurologically intact.

DISCUSSION

The mechanisms of injury for Case Reports 1 and 2 are unknown; however, we suspect that the median nerve became entrapped at the shoulder, possibly due to shoulder dislocation or impingement. Visser et al. (1999) reported a 4% injury rate for the median nerve in anterior shoulder dislocations, whereas, Mesa et al. (1996) reported median nerve injuries occurring with *luxatio erecta* (inferior shoulder dislocation). The degradation in the median nerve SSEPs may also have been due to entrapment of the medial and/or lateral cords of the brachial plexus due to dislocation not affecting ulnar nerve contributions (De Latt et al. 1994; Liveson 1984). Though shoulder dislocations are rare in surgery, the proximity of the median nerve in the axilla when they occur can cause a degradation to the median nerve SSEP intraoperatively (Shriver et al. 2015).

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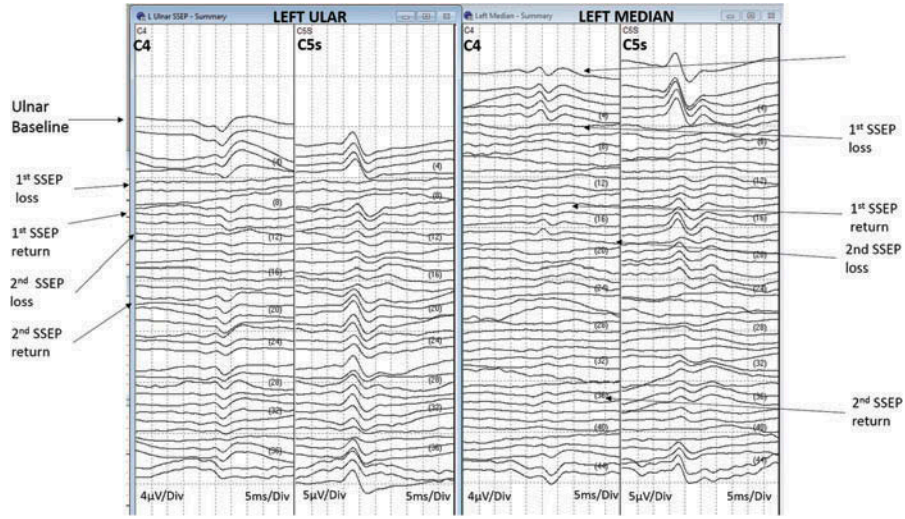

FIG. 7. Contemporaneous loss of the left ulnar and median nerves with subsequent interventional recovery at two different times during the same procedure.

The median nerve originates in the lateral and medial cords of the brachial plexus from the C5–T1 spinal nerve roots. It exits the axilla near the musculocutaneous nerve anteromedial to the humerus. It courses through the upper arm superior to the ulnar nerve until it begins to make a midline approach to the antecubital fossa. The median nerve dives deep into the forearm between the two heads of the pronator teres muscle and travels down the middle of the forearm, where it innervates the majority of forearm flexors. At the wrist, the median nerve courses through the carpal tunnel to give off its motor and sensory innervations to the thenar eminence and digits (Kimura 2013).

The mechanism of injury for Case Report 3 is unknown, however the investigators suspect that compression of the ulnar nerve at or distal to the cubital tunnel bilaterally due to the pressure of the arms on the arm boards occurred. The ulnar nerve arises from the spinal nerve roots of C8 and T1 and projects through the lower trunk and medial cord of the brachial plexus. It travels in a common neurovascular bundle with median nerve and brachial artery between the biceps and triceps muscles in the upper arm. In the mid portion of the upper arm, the ulnar nerve starts to move into a posterior position as it becomes positioned to travel through the cubital tunnel (medial epicondyle of the humerus) at the medial elbow. Once the ulnar nerve traverses the cubital tunnel (its most superficial location), it gives off the innervation to the flexor carpi ulnaris and flexor digitorum profundus III and IV muscles. Next it travels down the medial forearm and passes Guyon's Canal, where it innervates the majority of intrinsic hand muscles and the hypothenar eminence (Kimura 2013).

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The suspected mechanism of injury for Case Report 4 is unknown, but the investigators suspect that the brachial plexus or upper arm became compressed, stretched, or ischemic, leading to a contemporaneous degradation of the median and ulnar nerve SSEPs.

Peripheral nerves have the highest risk for injury due to patient positioning (Bund et al. 2005; Winfree and Kline 2005). Stretching or compression of the nerve is the likely mechanism of injury along with ischemia and pressure (Swenson et al. 1998; Winfree and Kline 2005). Kamel and Barnette (2014) described ulnar neuropathy as the most common perioperative position injury that occurred in their review of anesthesia malpractice closed claim literature at 28%, followed by brachial plexus injury (20%) and median nerve injury (4%). Silverstein et al. (2014) found that ulnar nerve SSEP diminution occurred most often in their cohort of 398 subjects at 8%, with median nerve SSEP deterioration occurring in 1% of their study population. Lee and Espley (2002) describe the incidence of ulnar nerve injury in their case series of 203 subjects at 3% for their study. In a prospective trial to prove ischemia as a mechanism of injury, Swenson et al. (1998), purposely occluded the brachial artery while their patients were under general anesthesia and the results were a rapid degradation of contemporaneous ulnar and median nerve SSEPs. Ohashi et al. (2012) discuss in a case report that a patient who successfully underwent correction of scoliosis awoke from anesthesia with a complete paralysis of the brachial plexus on one side. They further report that the intraoperative neuromonitoring utilized was TCeMEP and SSEP for lower extremity monitoring only. They did not use upper extremity SSEPs and in their conclusion discuss that upper extremity SSEPs should be added to the monitoring paradigm to reduce the risk of an upper extremity positional nerve injury.

It has been well established within the literature that upper extremity SSEPs can be used as a tool for predicting and preventing position-related injuries by monitoring the function of peripheral nerves and brachial plexus during surgical procedures (Balzer et al. 1998; Chung et al. 2009; Fountas et al. 1995; Jones et al. 2004; Kamel et al. 2006; Nuwer, et al. 2012; Schwartz et al. 2000). However, the use of contemporaneous median and ulnar nerve SSEPs is not well documented, nor are appropriate interventional methods. O'Brien et al. (1994), monitored the median and ulnar nerves contemporaneously in their study, which was the first study to describe monitoring for positional injuries. They concluded that the SSEP was valuable in the prevention of brachial plexus injuries but was not reliable to detect isolated postoperative ulnar neuropathies. They describe two of their patients waking up from anesthesia with new ulnar neuropathies without detection from the intraoperative SSEPs. Today it is common practice to stimulate the ulnar nerve at the wrist; however, O'Brien et al. (1994) were stimulating the ulnar nerve at the elbow. This stimulation site was likely at or proximal to the site of the conduction block, which is the suspected reason the injuries went undetected by the ulnar nerve SSEP evaluation in their study.

Using both the median and ulnar nerves via wrist stimulation to evaluate upper extremity SSEPs allows the neurophysiologist to isolate the injury that is occurring and expedite positional recovery by addressing the specific change rather than blindly

adjusting the arm. For example, in Case Reports 1 and 2, if there was not an evaluation of the median nerve SSEP, the patients might have awoken from anesthesia with an isolated median neuropathy or brachial plexopathy undetected by intraoperative ulnar nerve SSEP assessment. Instead, by focusing on the deteriorating median nerve SSEPs and having stable ulnar nerve SSEPs allowed for expeditious countermeasures to be implemented to reduce a non-ulnar nerve postoperative neuropathy from occurring. In Case Report 3, the focal ulnar nerve SSEP degradation with no change to the median nerve SSEPs allowed the neurophysiologist and anesthesiologist to address the ulnar nerve directly and intervene specifically to counteract an ulnar nerve injury. These countermeasures included adding padding around the cubital tunnel and reducing pressure on the elbow and forearm. Case Report 4 illustrates that using both nerves contemporaneously as a means to address a perioperative brachial plexus issue can allow for an adjustment of the arm at the shoulder to attenuate a postoperative brachial plexopathy.

Body habitus, length of procedure, and patient sex can be determinants for a patient developing a positionally induced nerve injury (Kamel and Barnette, 2014; Silverstein et al. 2014). In all four case reports, each patient had a BMI of 34 or greater. Silverstein et al. (2014) found that subjects with a mean body mass index (BMI) of 32 or greater were most at risk for ulnar nerve injuries, whereas patients who had a mean BMI of 41 or greater were more at risk for median nerve injuries. They also found that brachial plexus injuries were more likely to occur when the patient has a mean BMI of 37 or greater (Silverstein et al. 2014). In a retrospective analysis of over one million subjects, Warner et al. (1994) found that men with extreme body habitus were most at risk for postoperative ulnar neuropathy that persisted three months or more.

Position-related degradations of upper extremity SSEPs is not relegated to body habitus only and can be multifactorial. Preexisting conditions, such as peripheral neuropathy, diabetes mellitus, chronic kidney disease, alcoholism, and HIV should be taken into consideration, as patients with these underpinnings are more at risk for compromised nervous systems. Orthopedic joint and nerve surgeries should also be taken into consideration, as scar tissue can cause excess pressure on nerves while the patient is in position for surgery (Kimura, 2013; Prielipp et al. 1999).

There should be an algorithm that the neurophysiologist goes through when addressing a potential position-induced SSEP change. This algorithm is dependent on the change that is occurring. For example, if the patient presents with an isolated degradation of the ulnar nerve SSEP while in the "superman" position, the likely area of conduction block is at or distal to the cubital tunnel. In the lead investigator's experience, placing a piece of foam under or just distal to the cubital tunnel allows for ulnar nerve SSEP recovery. However, if the degradation persists, adjusting the arm by flexing or extending the elbow may also mitigate the issue. In the event of an isolated median nerve SSEP degradation, the antecubital fossa should be assessed to ensure it is free from any pressure (this includes entrapment caused by IVs, compression caused by blood pressure cuff, etc.). If the median SSEP change persists, then the shoulder should be evaluated and repositioned appropriately by the appropriate staff. If

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there is a contemporaneous degradation of the ulnar and median nerve of the same limb, the limb should be evaluated to ensure there is nothing cutting off the blood supply to the upper arm (e.g., blood pressure cuff, C-arm, etc.). If there is no visual violation of the upper arm, the shoulder should be repositioned. If the median or ulnar nerve does not return with the interventions described, we have found it useful (especially when a patient is positioned on a Jackson table) to remove the arm board completely and place the arm on a stool padded with pillows (Figure 8). When using this intervention, the arm is no longer affixed to the operating table, and the anesthesiologist must be aware of this should he or she have to move the table up or down during the procedure. If a median or ulnar nerve SSEP diminution occurs while the patient is in a supine position with the arms tucked, all efforts should be made to loosen the tuck without having the arm fall to the side. Again, the authors have seen this to be very effective in their clinical practice.

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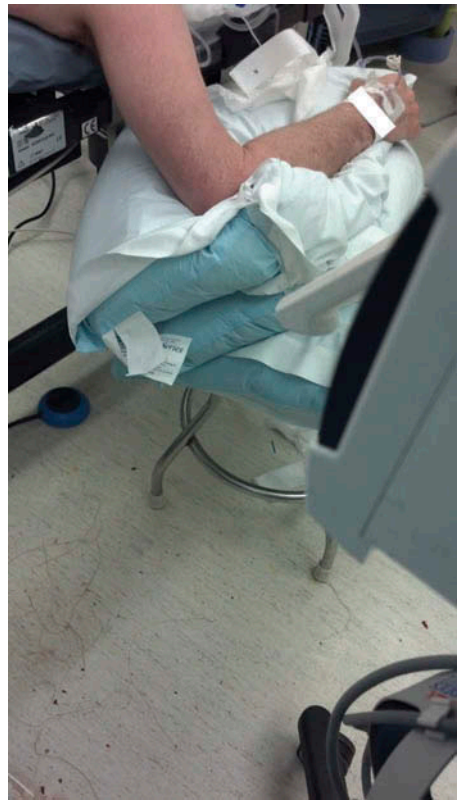

FIG. 8. The stool method used to assist in the recovery of an attenuated upper extremity SSEP when other interventional countermeasures have failed.

CONCLUSION

The goal of neuromonitoring for positional injury is to predict and prevent both peripheral nerve and brachial plexus injuries. Using contemporaneous ulnar and median nerve SSEPs may lead to better identification of the compromised structures when an SSEP change to one or both of the nerves occurs, allowing for specific countermeasures to be taken which promote expeditious intraoperative neural recovery. Although we have shared our thought process as to why we use ulnar and median nerve SSEPs and presented an algorithm for intervention, a larger case series is needed to demonstrate a better definition for the sensitivity, specificity, and efficacy of this neuromonitoring paradigm.

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