Anterior Cervical Spine Surgery

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John F. Bebawy, Antoun Koht,
and Srdjan Mirkovic

Key Learning Points

- The safety of ACDF for patients with cervical radiculopathy, with or without neurophysiologic monitoring, is extremely high, with very low rates of temporary or permanent neurologic sequelae.
- The risk of neurologic injury during an ACDF for those patients with myelopathic symptoms and requiring a cervical corpectomy, laminectomy, or foraminotomy, while unknown, is thought to be higher than that for patients with a radiculopathy alone. For these cases, multimodality neurophysiologic monitoring (SSEP, MEP, EMG) may play a significant role in detecting, and hopefully averting, impending neurologic injury.
- While EMG monitoring is beneficial in detecting mechanical insults to the nerve roots or spinal cord, it lacks the ability to detect changes related to ischemia.

Introduction

An anterior cervical discectomy and fusion (ACDF) is a routinely performed surgery to relieve spinal stenosis, remove intervertebral disk and bony matter that may be impinging upon neural elements, and also to mechanically stabilize the cervical spine after such material is removed. Herniated intervertebral disk material or osteophytes in the spinal canal or intervertebral foramina may cause compression of the spinal cord or nerve roots, respectively. Such compression may lead to radiculopathy, myelopathy, or both, and patients can present with significant symptoms such as pain, numbness, paresthesias, weakness, or paralysis.

The ACDF procedure can be performed at one or multiple levels with varying amounts of complexity depending on the extent of neural tissue compression. An anterior approach in the cervical spine is often preferred to a posterior approach for discectomy due to anatomic favorability. However, in more extensive anterior surgeries, a

J.F. Bebawy, M.D. (✉)
Department of Anesthesiology and Neurological Surgery, Northwestern University, 251 East Huron Street, Suite F5-704, Chicago, IL 60611, USA
e-mail: j-bebawy@northwestern.edu

A. Koht, M.D.
Departments of Anesthesiology, Neurological Surgery and Neurology, Northwestern Memorial Hospital, Northwestern University Feinberg School of Medicine, 251 East Huron Street, F-5-704, Chicago, IL 60611, USA
e-mail: a-koht@northwestern.edu

S. Mirkovic, M.D.
Department of Orthopedic Surgery, Northwestern University Feinberg School of Medicine, Chicago, IL, USA
e-mail: chicagospinedoc@gmail.com

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posterior cervical fusion may also be warranted to stabilize the cervical spine (*see* Chap. 32, "Posterior Cervical Spine Surgery"). The safety of an ACDF for patients with cervical radiculopathy, with or without neurophysiologic monitoring, is extremely high, with very low rates of temporary or permanent neurologic sequelae [1]. One exception to this is the occurrence of a C5 palsy, which may have an incidence of up to 5.9 %. While it is unclear why the C5 nerve root is at higher iatrogenic risk than other nerve roots, this complication seems to be associated with more profound spinal cord compression and possibly ischemic axonal injury secondary to microvascular trauma [2]. Other rare forms of injury include hypoglossal nerve injury, C6 injury causing a Horner's syndrome due to sympathetic chain injury, and basilar artery ischemia due to excessive cervical extension.

In contrast, the risk of a neurologic injury during an ACDF for those patients with myelopathic symptoms requiring cervical corpectomy, laminectomy, or foraminotomy, while unknown, is thought to be higher than that for patients with radiculopathy alone [3]. Likewise, upper cervical spine surgery is often considered more risky than surgery at lower cervical levels [4]. For any of these cases, and particularly for those requiring complex reconstruction, multimodality neurophysiologic monitoring may play a significant role in detecting, and hopefully averting, impending neurologic injury [5–7]. The most frequently used modalities of neurophysiologic monitoring in these cases are somatosensory-evoked potentials (SSEPs), spontaneous electromyography (EMG), and transcranial motor-evoked potentials (MEPs) [8, 9], with dermatomal sensory-evoked potentials and direct epidural (D wave) MEP recordings being used to a lesser degree. Transcutaneous, mid-thoracic D-wave recording has been advocated due to the false-positives that may arise from SSEP recording alone and in circumstances in which transcranial MEPs may be difficult or impossible [10].

Neurophysiologic monitoring has generally been accepted during the surgical management of scoliosis, but has mixed acceptance during cervical operations such as ACDF [11, 12]. The value of SSEPs alone has been questioned in these cases; however, the combined usage of SSEP, EMG, and MEP monitoring is gaining support [13, 14]. When SSEPs are used for ACDF, median nerve responses are generally thought to be more helpful when higher cervical levels (C3–C6) are operated on, while ulnar nerve responses are more frequently favored when lower cervical levels (C6–T1) are the operative target [15].

Because SSEPs function specifically as a monitor of the posterior elements of the spinal cord (i.e., dorsal columns), and nonspecifically as a monitor of the entire spinal cord, they are prone to false-negatives in the detection of injuries to the anterior spinal cord (e.g., corticospinal tracts) [3, 12] and/or nerve roots. Hence, spontaneous EMG monitoring has been advocated in these surgeries, in combination with SSEPs and MEPs, as a way to specifically monitor the motor component of the nerve roots [4]. Identification of muscle-specific spontaneous EMG discharges would be the most efficient indicator of mechanical irritation of a nerve root, preceding the more concerning changes in SSEPs and MEPs, which might be related to ischemia.

While EMG monitoring is beneficial in detecting mechanical insults to the nerve roots or spinal cord, it lacks the ability to detect changes related to ischemia [16]. Therefore, many authors have advocated the routine use of MEPs in those patients thought to be at high risk for intraoperative ischemia due to compression (e.g., severe myelopathy due to critical spinal canal stenosis, severe spondylolisthesis) [13, 17]. SSEPs may be less sensitive than MEPs in this regard, as anterior (motor) spinal elements tend to be at higher risk during anterior spinal surgery. In all reported case series, MEPs were employed in conjunction with SSEPs in order to improve both sensitivity and specificity of the neurophysiologic monitoring being performed. In most published reports, SSEPs have a low but finite incidence of false-positives (Taunt et al. [12] reported 1.8 %), and an even lower incidence of false-negatives [1]. MEPs and EMG may serve a confirmatory role in these cases.

A study by Cole et al. [5] revealed that for single-level spine surgery, neuromonitoring was

only helpful in lowering neurologic complication rates for lumbar laminectomies, with no added benefit for lumbar discectomies, lumbar fusions, or ACDFs. Furthermore, a large prospective study by Helseth et al. [6] found that outpatient microsurgical cervical decompression was feasible without neuromonitoring and with a very low overall complication rate. Others, however, advocate for multimodality neuromonitoring, even in single-level ACDFs. Epstein argues that because quadriparesis/quadriplegia is one of the more common reasons for malpractice suits after single-level ACDFs, neuromonitoring (and specifically MEP monitoring) should be employed in these cases.

Another consideration related to nervous system injury during ACDF, and also a concern during thyroid and parathyroid surgery, is injury to the recurrent laryngeal nerve (RLN). This is the most common type of neurologic injury related to ACDFs and can be caused by direct surgical trauma to the nerve, nerve compression between a retractor and the shaft of the endotracheal tube, high endotracheal tube cuff pressure, or a combination of these elements [18]. RLN injury usually occurs on the side of the surgical approach. The incidence of injury has been shown to increase with the number of cervical spine levels operated on, when the lowest level instrumented is T1, when the surgical approach is from the patient's left side, when the Cloward retractor is opened greater than 3 cm to expose the spine, and with previous surgery at that location [19, 20]. Although the incidence of postoperative vocal cord dysfunction is 2–5 %, most vocal cord injuries resolve within several months.

The RLN is often monitored using EMG, specifically by employing an endotracheal tube adapter or commercially available endotracheal tube with surface electrodes that contact the true vocal cords. Direct visualization of the vocal cords and the electrodes on the endotracheal tube is necessary to ensure proper positioning of these devices (depth and tube rotation). A new method for monitoring RLN function is the use of corticobulbar track motor-evoked potentials with recording from the vocal cords [21]. Many practitioners use succinylcholine, rather than a longer acting muscle relaxant, to facilitate intubation in these cases so that muscle function will recover before the time that monitoring is necessary (although this practice is not always required, since the vocal cords are relatively resistant to the effects of intermediate acting muscle relaxants when given in judicious doses) [22]. An alternative method for intubation if succinylcholine is contraindicated is to use ephedrine, 15 mg; remifentanil, 4 μg/kg; and propofol, 2 mg/kg. Dimopoulos et al. [23] have gone further to describe a method by which they objectively quantify the amount of RLN irritation in ACDFs by the amount of EMG activity, and determined that longer surgeries, multilevel surgeries, previous surgical intervention, and the use of self-retaining retractors are all associated with more RLN irritation. Also, it is important to be cognizant of the use of topical lidocaine on or near the vocal cords, as might be performed during an awake intubation, as this might preclude adequate monitoring of the RLN [24]. Despite this, RLN monitoring is used more commonly in thyroid and parathyroid surgery, being used less frequently for ACDF surgery where other neuromonitoring modalities predominate. Another and long used way to minimize laryngeal nerve is to deflate the cuff of the endotracheal tube. However, there is concern about aspiration in addition to circuit leak.

Case 1

A 68-year-old male, 85 kg, ASA PS 3, with a past medical history of poorly controlled DM and HTN, presents for an ACDF of C4–C7 (right-sided approach) for severe myelopathic and radiculopathic symptoms. The patient has been complaining of bilateral upper extremity weakness, numbness, and bilateral lower extremity paresthesias. A cervical MRI examination reveals critical spinal canal stenosis at C5 and C6, and extensive osteophytic lesions throughout.

The patient was monitored with standard ASA monitors, and multimodality neurophysiologic monitoring was employed, including SSEPs, MEPs, and EMG. Because of the patient's

myelopathy, an awake fiberoptic intubation was planned so as to clinically examine the patient after intubation. Intubation proceeded smoothly, a neurologic examination was performed with no change from preintubation status, and anesthesia was then induced. Induction consisted of propofol, remifentanil, and rocuronium. Rocuronium was chosen to facilitate positioning because prepositioning responses were not deemed necessary and its relatively rapid metabolism would allow MEP and EMG monitoring as soon as possible. If monitoring of the patient's neck position had been required (such as with an unstable cervical spine injury), the avoidance of muscle relaxants could have been planned. A radial arterial line was then placed to facilitate close monitoring of the patient's hemodynamic status. Maintenance of anesthesia consisted of propofol, 50–150 μg/kg/min; remifentanil, 0.05–0.5 μg/kg/min; and desflurane end-tidal 3.3 % (0.5 MAC) in oxygen and air (FiO_2 0.5); no muscle relaxant was used after intubation. Stimulating and recording electrodes were placed for the planned neuromonitoring, and the patient's arms were padded, tucked at his sides, and wrapped firmly with sheets. Baseline SSEPs and MEPs were obtained after a "steady state" of anesthesia, with slightly diminished amplitudes in the upper extremity SSEPs, more diminished amplitudes and increased latencies in the lower extremity SSEPs, and slightly diminished MEPs in all four extremities.

What are the possible causes for the diminished baseline SSEP and MEP signals in this patient?

The diminished responses, involving both SSEPs and MEPs, observed at baseline (i.e., before the start of surgery), were not caused by surgical maneuvers, since surgery had not commenced. Moreover, because a global diminishment in signals was seen without complete loss of the signals, a positional or technical cause was unlikely. Physiologic factors such as hypothermia and hypotension may produce such changes; however, these too are unlikely reasons for the changes because both parameters were within normal limits. In some patients, blood pressure that is borderline-low may lead to a global decrease in signals. Because hypotension is a common cause of SSEP changes in these patients [25], the anesthesiologist will often raise the blood pressure by 20 % while continuing to troubleshoot for a cause. In this case, raising the blood pressure did not significantly correct the observed low signal parameters.

At this point, we are left with whether anesthetic effects, underlying pathology, or a combination of the two is responsible for the decreased signals. Certainly, the diminished MEP signals could be partially related to the residual effects of the muscle relaxant used during intubation. A train-of-four (TOF) nerve stimulation test would help identify such a cause. This was done in this case and showed 80 % TOF recovery. Increasing the MEP stimulation intensity by 50 V produced a better global MEP signal. Other anesthetic drugs may also be contributory, especially in the presence of a volatile anesthetic. Sedatives/hypnotics (e.g., propofol) and opioids tend to have minimal effects on SSEPs and MEPs unless they are given in large doses. Volatile anesthetics, however, may cause more depression of evoked potentials; nonetheless, when given at 0.5 MAC or less as part of a balanced anesthetic technique, the volatile anesthetics are usually compatible with adequate evoked potential tracings unless the patient has significant neurologic dysfunction (e.g., myelopathy). To test the possibility that the volatile agent was responsible for the decreased SSEP and MEP signals in this case, the desflurane was turned off and the propofol infusion was increased to maintain anesthetic depth. No sizable improvement was seen in either the SSEP or MEP amplitudes after sufficient time to remove the volatile agent. During the following 20 min, SSEP signals remained stable, whereas MEP signals improved slightly; consistent with near-complete recovery from the muscle relaxant.

Most likely in this patient, severe cervical canal stenosis coupled with severe peripheral neuropathy secondary to poorly controlled diabetes are the major contributing factors to the globally diminished evoked potentials seen at baseline.

The surgery was begun and proceeded uneventfully throughout exposure. During the course of deeper dissection, however, a slight decrease in

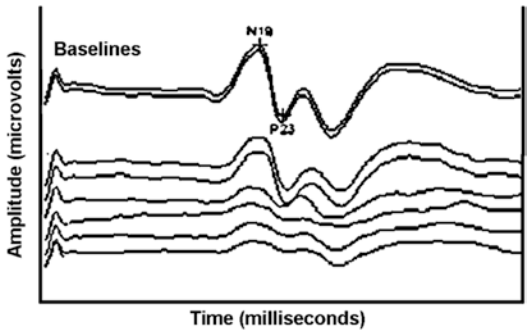

Fig. 31.1 Representative of cortical SSEP changes caused by unilateral carotid occlusion due to retractor malposition

the amplitude of the SSEP of the left arm was noticed. Repeated testing confirmed further SSEP deterioration by more than 50 % in amplitude in both the left arm and left leg although the left Erb's point waveform was not changed (Fig. 31.1). No changes were seen on the right side. EMG activity was also negative. The surgeon was notified and MEPs were tested, which revealed a complete loss of signals of the left hand and left leg with normal right-sided responses.

What are the possible causes for the diminished left-sided SSEP and MEP signals at this point in time?

Given that this change is focal (not global) in nature, anesthetic and physiologic causes for the diminished signals are less likely. Hence, surgical, technical, or positional causes should be sought to explain this evoked potential change. In this case, the anesthesiologist raised the blood pressure by 20 % above its current level while troubleshooting other causes. The position of the arms was checked while the neuromonitoring technologist was assessing the technical fidelity of the signals. There were no apparent technical or positional problems, leaving us only with a potential surgical cause for these changes. The surgical causes for signal changes may be related to mechanical stress, thermal injury, surgical injury, or ischemia. Mechanical stresses are usually associated with EMG discharges and are related to either nerve root irritations or dural insults. Neither instrumentation nor thermal devices were being used on any of the neural structures at this time. Ischemia to the left arm could explain MEP changes but it would not explain the SSEP changes in the lower extremity nor those in the upper extremity (since the response from Erb's point was normal). In fact, occlusion of the right-sided cerebral blood supply is a more likely cause of the changes that were seen. An ischemic or hemorrhagic stroke to the right hemisphere, caused by manipulation of an atherosclerotic right carotid artery, is a possibility. Even more likely is cerebral ischemia caused by obstruction of the right carotid artery, which is in close proximity to the surgical field and may be distracted by the surgical retractor.

Absent blood flow within the right carotid artery was confirmed by the anesthesiologist by palpation of the right superficial temporal artery (transcranial Doppler could also have been used for this indication). The surgeon was informed and repositioned the retractors, which resulted in an immediate restoration of the right superficial temporal pulse as well as both the MEP and SSEP waveforms.

Case 2

A 36-year-old, ASA PS 1, woman without significant past medical history is scheduled for a C5–C7 ACDF for disk herniation and removal of an osteophyte. Anesthesia is performed with standard ASA monitoring and neurophysiologic monitoring consisting of EMG recorded from the deltoid, biceps, and triceps, SSEPs, and MEPs for the upper and lower extremities. Normal baselines of all monitoring parameters were obtained prior to surgery. Discectomies at the C4–5, C5–6, and C6–7 interspaces were performed and significant bony overgrowth along the pedicles of the vertebral canal was removed. At one point, during shaving along the pedicle of C5, there was a burst of spontaneous EMG activity recorded from the biceps (Fig. 31.2).

What could be the cause of this EMG change?

EMGs are used during such operations to continuously monitor for mechanical irritations to the spinal cord or nerve roots induced by the different surgical instrumentation used. EMG discharges are related to mechanical insults that cause depolarization, and are not related to ischemia. The premise of using EMG monitoring is

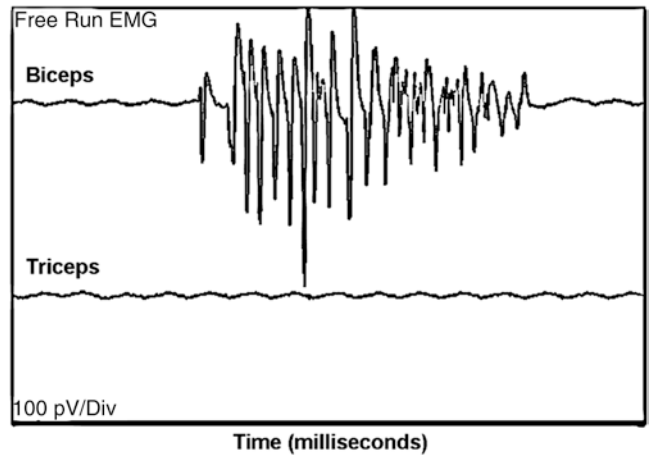

Fig. 31.2 Spontaneous EMG firing at the biceps, with minimal noise in other muscles

to alert the surgeon to changes related to such mechanical irritation before a greater insult occurs that can lead to more permanent neuronal damage. Notably, "light" anesthesia can be a cause for abnormal EMG discharges that are not surgically related (however, light anesthesia usually produces activity in multiple muscles rather than the one muscle noticed here). The use of other electrical devices, such as cautery, may also produce "false" EMG discharges. EMG discharges have been graded in intensity according to a four-level system [16]. In this case, the discharges were mild, the surgeon was notified, and the discharges disappeared immediately thereafter. The surgeon continued to work but a few minutes later severe discharges reappeared (which might indicate the potential for a larger mechanical and/or ischemic insult). The surgeon was again notified and paused surgical activity while MEPs were obtained. During this time, SSEPs were also being acquired. These modalities were used as confirmatory tests in the presence of the EMG changes, testing for any potential spinal cord injury that might be caused by ischemia as a result of mechanical distortion.

MEPs were acquired and revealed no changes. SSEP responses were also stable. How should we proceed?

Because the only change seen in the neurophysiologic monitoring was an increase in EMG activity at C5 or C6, there is most likely a surgical reason for the observed change. In this case, the most likely scenario is that the C5 or C6 nerve root emerging from the intervertebral foramen has been mechanically irritated during attempted decompression at the foramen. EMG provides a real-time alert for impending neurologic deficits related to a mechanical insult. The SSEP and MEP waveforms were most probably not affected because they tend to transmit along major peripheral nerves, which originate from many individual nerve roots, thus masking irritation or impending injury to a single nerve root.

A disadvantage of EMG monitoring compared to MEP monitoring is that it can be "contaminated" by artifact from various sources, including patient movement, Bovie interference, etc., while this is not the case with the relatively high amount of stimulation needed to generate MEPs. A potential advantage of EMG monitoring compared with MEP monitoring, as was seen in this case, is the continuous nature of EMG signal acquisition, which might detect compression/injury to a nerve with greater sensitivity than an evoked MEP, whose acquisition is intermittent and would require deliberate acquisition at or after the time of the insult to the nerve to detect it. Most importantly, as illustrated in this case, EMG also has the advantage of being able to detect irritation to a single nerve root, which is less likely with either SSEP or MEP monitoring, because these modalities monitor major mixed sensory/motor

nerves and muscles that have overlapping nerve root innervation.

The surgeon stopped working in the C5–C6 nerve root area and the EMG recording returned to a silent state. The surgeon proceeded to complete the surgery without any further changes in the neuromonitoring signals. The patient was awakened, extubated, and examined neurologically, with no change in the examination as compared with her preoperative status.

Case 3

A 47-year-old woman, 140 kg, ASA PS 2 with a past medical history of morbid obesity, is scheduled for a C3–C5 ACDF (right-sided approach) for intermittent and nonreproducible radiculopathic symptoms in her left upper arm. Her clinical examination is not consistent with any myelopathy, and a cervical MRI seems to confirm this (no spinal cord impingement). Of note, on her physical examination, the patient has a Mallampati Class IV airway with a thyromental distance of 4 cm. Previous anesthetic records indicate that she was easy to ventilate by bag/mask but difficult to intubate, requiring fiberoptic intubation.

How should the airway be secured in this patient? Should an awake or asleep technique be used? Would neuromonitoring, after induction but prior to intubation, be of any value in this case? What neuromonitoring modalities should be used for this case?

Based on the patient's previous airway history, an awake or asleep fiberoptic technique would seem prudent. The advantage of an awake fiberoptic intubation, besides maintaining spontaneous ventilation, would be to retain the ability to examine the patient for evidence of new radiculopathic/myelopathic symptoms during and after intubation. An asleep fiberoptic intubation could also be performed, with SSEPs and EMG acquired pre- and postintubation (under "steady state" anesthesia), to confirm that neurologic injury from intubation had not occurred. Whether intubation is performed awake or asleep, the use of flexible fiberoptic bronchoscopy should limit the amount of neck movement and cervical subluxation compared to a direct laryngoscopy.

For this surgical procedure, any combination of the neuromonitoring modalities mentioned above could be used depending on the level of concern for spinal cord, nerve root, or peripheral nerve injury.

Because no myelopathy is suspected in this patient, and because of a known ability to mask ventilate her in the past, an oral asleep fiberoptic intubation is chosen to secure the airway. SSEPs of the median and posterior tibial nerves are obtained as well as EMG of the deltoid, biceps, and triceps muscles. All of these neuromonitoring modalities remained unchanged before and after intubation under a "steady state" of anesthesia, being careful to record signals after recovery from the succinylcholine (i.e., no residual effect on EMG) used to facilitate intubation. A small amount of rocuronium (20 mg) was then given to assist during positioning and exposure. Anesthesia was maintained with propofol, 100–150 μg/kg/min, and fentanyl, 1–5 μg/kg/h (TIVA), which were infused through a dedicated intravenous (IV) catheter placed in the left arm. Fluids and bolus medications were injected into the IV catheter placed in the right arm. SSEP baselines were obtained from all four extremities and were found to be robust and reproducible. Before surgical incision, the neuromonitoring technologist reports a greater than 50% decrease in the amplitude and an increase in the latency of the SSEP signals recorded from the right arm (Fig. 31.3).

What could be the cause of these right arm SSEP changes? What should be done to correct these changes and avoid injury?

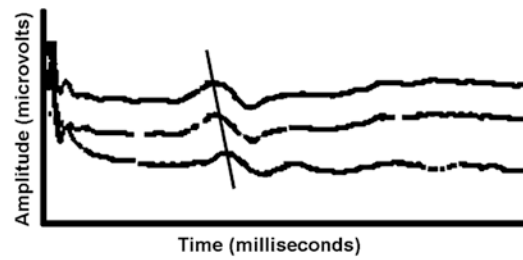

Fig. 31.3 SSEP changes at the cervical level due to excessive traction on the shoulder caused by taping too tightly

Since surgery has not yet begun, surgical causes for the observed changes are eliminated, and because this is a unilateral change, anesthetic and physiologic causes are unlikely. The possibility of a technical cause exists (e.g., due to a decrease in stimulation intensity caused by partially dislodged stimulating pads). All stimulating and recording pad placements were checked, and all other technical parameters were within normal limits. On further evaluation of the right shoulder position, the shoulder was found to be taped down to the table rather tightly, placing it in undue traction. The tape was somewhat released, which resulted in the return of the SSEP signals to baseline. Presumably, the observed change was related to stretching of the brachial plexus, and if left uncorrected might have led to a longer-lasting neurapraxia. A similar effect could result from straps attached to the wrists to allow traction, which would improve visualization of the spine under fluoroscopy. Other causes of a change in the SSEP responses from the upper arm were also excluded (such as a tourniquet effect of the noninvasive blood pressure cuff or drapes used to hold the arm or a cold arm from infusing cold intravenous fluids).

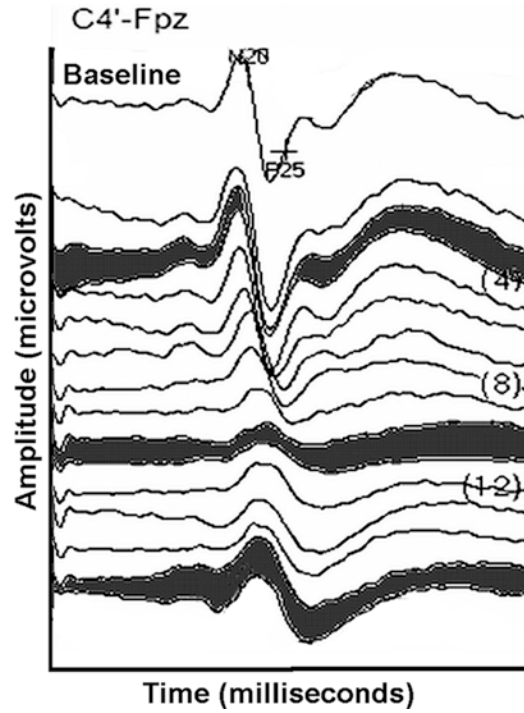

Fig. 31.4 SSEP changes during intravenous infiltration of the extremity

As surgery proceeded, the anesthesiologist noticed a few episodes in which the blood pressure and heart rate suddenly and inexplicably rose in association with an elevated Bispectral Index (BIS) value, seeming to indicate periods of "light" anesthesia. These were treated with IV boluses of medication. However, after three such episodes, the anesthesiologist added desflurane, 6.6 % end-tidal concentration (1 MAC), to control these episodes. The patient's vital signs and BIS value promptly returned to those of a "normally" anesthetized state, and the case continued. The surgeon continued to work near the spinal cord and a few minutes later the neuromonitoring technologist noticed a decrease in the amplitude of all the cortical SSEPs without any changes to the cervical or Erb's point SSEP waveforms and without any EMG discharges.

What could be the cause of this global change in cortical SSEPs alone?

This is a global change that is isolated to the cortical leads with normal signals from both Erb's point and the cervical spinal cord. Technical, positional, and surgical causes for such a change are unlikely because of the global nature of the signal aberrations. Physiologic factors are a possibility, but none can be identified, as blood pressure and temperature were found to be within normal limits. Hence, an anesthetic cause, namely the addition of a volatile agent at 1 MAC, seems to be the most likely candidate for the observed changes. Inhalation agents administered at less than 0.5 MAC can be used in most patients with good SSEP signal acquisition. Levels higher than this may be problematic in some patients, especially in those patients who have pre-existing diminished baseline SSEPs. In this case, the inhalation agent was decreased to 0.5 MAC and the SSEP signals recovered to baseline.

Near the completion of surgery, the cortical, cervical, and Erb's point SSEP responses from the left arm began to deteriorate, while the right arm signals remained stable (Fig. 31.4).

What might be the cause of this type of change in SSEPs?

Since this was a unilateral change, it is unlikely to be caused by anesthetic agents or physiologic alterations (with the possible exception of regional hypothermia causing a cold arm). A positional cause might be possible, but the timing of the changes does not support this as there was no recent change in the patient's position. Technical causes for the change were checked by the neuromonitoring technologist and were ruled out. Furthermore, a surgical cause for the changes was deemed unlikely as the surgery was at the stage of closure with no direct manipulation of the spinal column, and no bleeding or hematoma on the spine was visualized.

In this case, the disappearance of the left upper extremity SSEP waveforms seems to coincide with "light" anesthesia, as evidenced by the previously mentioned changes in hemodynamic vital signs, necessitating the addition of a volatile agent. These changes, when taken together, should prompt the anesthesiologist to examine intravenous lines, drug infusion pumps, and so on for appropriate drug delivery. If drugs or fluids have extravasated into an extremity, a decrease in SSEP signals from that extremity might be expected. This would be due to expanding tissue planes and, subsequently, to a greater distance between the stimulating electrode and the peripheral nerve being stimulated.

To counteract this problem, stimulation intensity can be increased at the peripheral nerve site that is stimulated. Another option to improve signal strength is to exchange the surface transcutaneous stimulating electrodes for needle stimulating electrodes, which can be quite helpful in cases in which there is a significant amount of adipose tissue or edema (or extravasated fluid), causing the nerve to be more distant from the skin surface.

The left upper extremity was examined and appeared to be in a good position without any excessive extension, abduction, or external pressure. The extremity, however, was noted to be somewhat tense in the forearm, and drug extravasation was suspected. The pulse oximeter was placed on the left hand and obtained a strong signal. Radial and ulnar pulses were confirmed as being present on the affected side. The intravenous catheter on the left arm was removed, and the intravenous medications were subsequently connected and delivered to the IV catheter in the right hand. The transcutaneous stimulating electrodes were exchanged for needle stimulating electrodes in the affected extremity, and the SSEP signals gradually but dramatically improved. The remainder of the case was uneventful.

Conclusion

An ACDF is a commonly performed surgical procedure with a generally low, but finite, incidence of nervous system injury. Neurophysiologic monitoring is often employed for these cases, especially in complex operations so as to avert neurologic injury. Because of the potential for central as well as peripheral nervous system injury during these cases, it is important to apply a systematic approach when troubleshooting changes in neuromonitoring signals, paying close attention to how the different modalities of the neuromonitoring interact to paint a picture of the status of the nervous system at any given point in time.

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Questions

1. What is the value of recording the Erb's point waveform on SSEPs when performing an ACDF?
2. Why might MEPs be an especially useful monitoring modality, in addition to SSEPs and EMG, in ACDF?
3. What differences exist in the anesthetic regimen that can be used when SSEPs are used alone versus when SSEPs are used in conjunction with MEPs?

Answers

1. Changes in the Erb's point waveform (increased latency or decreased amplitude) without changes in cortical or subcortical signals may

signal a problem with conduction through the upper extremity due to localized effects.
2. MEPs will monitor the anterolateral spinal cord (corticospinal tracts) for ischemia, which may be more at risk than the posterior spinal cord (dorsal columns) during anterior cervical surgery.
3. The use of MEPs prohibits or severely limits the amount of muscle relaxant that can be used, whereas with SSEPs alone, muscle relaxant may actually help to improve the signal obtained. In both cases, 0.5 MAC or less of volatile anesthetic should be used to allow signal acquisition.