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Peripheral Nerves

Intraoperative positioning nerve injuries

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Abstract Intraoperative positioning nerve injuries are regrettable complications of surgery thought to arise from stretch and/or compression of vulnerable peripheral nerves. Generally thought to be preventable, these injuries still occur in patients despite rigorous preventative measures. Sometimes injuries, initially thought to be due to malpositioning, are caused by other factors, such as retraction injury or brachial plexitis. Because of the diversity of nerves susceptible to positioning injury, the clinician must be aware of a variety of presentations and must be able to distinguish them from other postoperative complaints. Prevention remains the mainstay of the management of positioning injuries. Diagnosed and managed appropriately, these lesions typically improve completely over time.

Keywords: Anesthesia; Intraoperative complications; Peripheral nerve; Positioning; Surgery

1. Introduction

The discipline of surgery requires the choreographed manipulation of the patient by the surgical team. Unfortunately, the patient is at risk of sustaining an iatrogenic injury at any step during this process. Nerve injury may occur during placement of intravenous and intra-arterial lines, during injections, during compression of the globes of the eyes, after direct trauma in the operative field, or during the postoperative care of the patient. These forms of nerve injury are reviewed elsewhere [13,20,38,84]. Nerve injuries related to the positioning of the patient on the operating table and the positioning of the equipment vital to the operation were first described in the 1800s [15] and are the subject of this review. Such injuries are usually preventable; however, they unfortunately continue to occur.

2. Pathophysiology of nerve injury

Intraoperative positioning nerve injuries generally occur when a peripheral nerve is acutely subjected to some combination of stretch, ischemia, and/or compression at the time of surgery. Regardless of the exact mechanism of injury, there is a continuum to the severity of injuries thereby produced. Low-grade stretching of a nerve may cause physical disruption of intraneural blood vessels (vasa nervorum), causing patchy nerve ischemia [31]. More severe stretching may tear the intraneural connective tissue, resulting in intraneural hemorrhage and/or necrosis. Stretching may result in focal compression where the nerve is draped over a firm prominence. A temporary interruption of the blood supply to a nerve by mild compression results in a conduction block that may only require a few minutes of reperfusion to reverse [30,37]. More severe degrees of compression will elevate intraneural venous pressures and produce endoneurial edema [103]. The pressures thus exerted may then impair axoplasmic flow, producing a more sustained dysfunction lasting hours to weeks [27]. Continued pressure leads to Schwann cell damage and myelin displacement [30,85]. As damage to the myelin progresses, segmental or paranodal demyelination is the result. Electromyography (EMG) shows conduction slowing or even conduction block, depending on the severity of injury [103]. Remyelination recovery may require several weeks to occur [41]. Still higher pressures will result in axonal loss and wallerian degeneration [30,87]. For incomplete nerve injuries the ampli-

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tude of the compound action potential is reduced, as some axons are missing. Complete axonal loss does not conduct distal to the injury at all. After injury, persistent conduction in the distal segment of nerve occurs for several days after injury, but this ceases once the segment degenerates [67,91]. Once profound axonal loss occurs, denervational changes in the muscle appear on EMG. Recovery requires the regrowth of axons down the degenerated segment. The duration of this process is variable but requires an amount of time equal to roughly 1 mm/day of damaged nerve segment length, increasing as one progresses distally along the extremity [103]. The most severe injuries disrupt the continuity of the nerve altogether such that axonal regrowth is nearly impossible. Electromyography reveals immediate total loss of distal segment function. Such injuries have the worst prognosis but thankfully are rare complications of intraoperative positioning.

3. Predisposing factors

In an early review [44] of what was then described as "Anaesthesia-paralysis," several patients were noted to have peripheral nerve injuries after a variety of surgical procedures. Garrigues [44] noted that the selection of anesthetic was unimportant. Both long and short operations resulted in nerve injuries. The most important predisposing factor was thought to be pressure on the nerve. Thin patients were especially at risk. Later reports implicated diabetes as a predisposing factor in the development of intraoperative pressure palsies [59,77]. Sometimes these injuries occurred despite adequate padding of the affected nerve.

Pre-existing peripheral neuropathy from diabetes, peripheral vascular disease, or other causes is a predisposing factor for susceptibility to trauma [86,104]. Of a group of 42 patients undergoing open heart surgery, 11 (26%) developed a postoperative ulnar neuropathy [19]. All had evidence of conduction slowing across the elbow preoperatively. None of the patients with normal conduction preoperatively developed a postoperative ulnar neuropathy, suggesting that a subclinical neuropathy predisposed these patients to develop a symptomatic injury. Likewise, hereditary peripheral neuropathy has been reported to be a predisposing factor for positioning nerve injuries [36]. These patients developed pressure palsies after mundane activities such as crossing the legs for 30 minutes, leaning on the elbow for 10 to 15 minutes, upon awakening from sleep, or awakening from surgery. Deep hypothermia due to direct skin cooling has been proposed as a direct cause of peripheral nerve injury [102]. In addition, hypothermia has been suggested as a predisposing factor for the development of intraoperative positioning injuries [105]. Reports of open heart surgery patients have described a correlation between the use of intraoperative hypothermia and the development of peripheral nervous system injuries [69,88]. The risk of developing an intraoperative positioning nerve injury may be increased by hypotension [43], undernutrition, or malnutrition [86]. In addition to physiological derangements, anatomic variants such as cervical ribs may render the brachial plexus more vulnerable to injury [22,88].

In the normal, awake patient, extremes of body positioning or excess pressure against superficial nerves produces discomfort prompting optimization of position and relief of symptoms. The introduction of anesthetics and muscle relaxants abolishes these protective mechanisms, rendering the patient susceptible to injury [22,88,99]. Tourniquets may cause compressive nerve injuries [12,14,37,88,96]. The risk of injury seems to be greater in the lower extremity than in the upper extremity, possibly because of the increased tourniquet pressures required to achieve hemostasis [95]. Nerves may be injured by external compression from a variety of items, including table surfaces, table edges, blood pressure cuffs, tourniquets, restraining straps, retractor systems, and masks. The myriad of ways in which nerves may develop intraoperative positioning injuries will be reviewed by nerves below.

4. Injury evaluation

Crucial to the diagnosis of intraoperative positioning nerve injuries is physician awareness of these injuries and their presentation. Often patient complaints are dismissed in the early postoperative period. In the setting of potent postoperative analgesics, residual anesthesia, incisional pain, and other critical care issues, even patients are sometimes unaware of their neurologic deficits. Once a nerve injury is identified, a history with emphasis on predisposing factors should be elicited. The history must attempt as accurately as permitted to establish the timing of onset and progression of symptoms. Efforts to ascertain the mechanism of injury must be made as well. A detailed neurologic examination should then focus on motor and sensory findings. If a neurologic deficit is identified, a neurologic consultation is indicated.

Electromyography serves as a more sensitive extension of the physical examination. Its purpose is to identify and localize nerve lesions. This is accomplished by exploring sensory and motor nerve conduction velocities, the degree of muscle denervation, and the degree of muscle reinnervation. Findings may also discriminate between acute and chronic nerve injury. An EMG may be performed in the acute stages of injury to establish whether any preexisting nerve injury is present. Since denervational changes in muscle require 2 to 3 weeks to become apparent on EMG, the presence of these findings acutely suggests a preexisting nerve injury. Electromyography is also performed in a delayed fashion, commonly 3 to 4 weeks postinjury, so that denervational changes can be documented.

Once an intraoperative positioning nerve injury is suspected, it is important to distinguish it from other postoperative neurologic afflictions, including cervical spinal injuries, other musculoskeletal injuries, and autoimmune neurologic disorders [7]. An important disorder that