POSITIONING PATIENTS FOR SPINE SURGERY: AVOIDING UNCOMMON POSITION-RELATED COMPLICATIONS

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ABSTRACT

Patient position in spine surgery is critical for optimal operating conditions and exposure to the surgical site. During spine surgery, the patient is placed in positions that are not physiologically appropriate and can lead to complications. Perioperative peripheral nerve injury (PPNI) and postoperative vision loss (POVL) are rare complications related to patient posture during spinal surgery leading to disability and loss of function. Significant for the patient. INPP is usually caused by a stretched or pinched peripheral nerve. INPP can present as brachial plexus injury or as an isolated injury to a nerve, most commonly as ulnar nerve. Understanding the etiology, mechanism, and pattern of injury with each type of nerve injury is important for the prevention of NIPP. Intraoperative neurosurgery is used to detect peripheral nerve conduction abnormalities indicating peripheral nerve stress under general anesthesia and to guide correction of upper extremity position to prevent NIPP. POVL often leads to permanent vision loss. Most cases are associated with spinal stretching procedures in the prone position under general anesthesia. The most common causes of POVL after spine surgery are ischemic optic neuropathy, and neuropathy is the most common cause of POVL after spinal surgery. It is important for spine surgeons to be ware of POVL and to participate in safe and collaborative perioperative care for spine patients. Proper training of perioperative staff, combined with clear communication and cooperation when positioning the patient in the operating room, is the best and safest approach. Prevention of rare complications of spine surgery depends primarily on identification of high-risk patients, correct placement of the, and optimal management of intraoperative physiological parameters. Modification of risk factors external to the patient may help reduce the incidence of PPNI and POVL.

Keywords: Spine surgery, Complication, Position, Nerve injury, Visual loss.

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INTRODUCTION

Progresses from the dendrite to the cell body to the axon. The axon of one neuron and the dendrite of the next The number of spine fusion surgeries performed in the United States has increased steadily over the past two decades.[14] Positioning patients for spine surgery is pivotal for optimal operating conditions and operative-site exposure. During spine surgery, patients are placed in positions that are not physiologic, would not be tolerated for prolonged periods by the patient in the awake state, and may lead to complications.

Although the incidence of complications associated with positioning patients for spine surgery is relatively low, position-related complications can be devastating and life changing to patients and their families. In this review article we discuss two of the uncommon, less recognized complications related to patient positioning during spine surgery; perioperative peripheral nerve in- jury and postoperative visual loss (POVL).

Perioperative nerve injury

Perioperative peripheral nerve injury (PPNI) is a rare but important perioperative complication resulting in significant patient disability, functional loss and the potential for litigation.[5,6] The mechanism of perioperative peripheral nerve injury is not well understood.[9] Neurosurgical and orthopedic surgical procedures have a significant association with perioperative peripheral nerve injury.[7] The normal reaction to increased loading of the peripheral nervous system (PNS) elements is progressively increasing muscle activity; this acts as a nociceptive mediated reflex to prevent further harmful elongation. But the use of muscle relaxants and inhaled anesthetics during general anesthesia may suppress this protective mechanism subjecting the PNS to greater elongation than would be tolerated in the normal awake state.[10] In an attempt to raise awareness and reduce the occurrence of PPNI, ASA formed a task force on the prevention of perioperative peripheral neuropathies. Anatomy and physiology of peripheral nerves The functional unit of the peripheral nerve system is the neuron. The neuron consists of a cell body, dendrites and a long axon.

Dendrites are attached to the cell body and carry impulses to the cell. Axons are attached to the cell body and carry impulses away from the cell. Conduction of an impulse along a neuron Portions of the cell body and the axon are covered by Schwan cells, which form myelin segments. Myelin is an insulating layer around the axons allowing quicker and more efficient impulse transmission. Once the action potential of the nerve cell reaches the threshold voltage, sodium channels in the region of the action potential open, allowing sodium to flow into the nerve cell and leading to complete depolarization of the membrane. The bundles of nerve fibers are bound together by connective tissue sheaths and form fascicles.

The endoneurium is a connective tis- sue sheath containing blood capillaries (vasa nervorum) that supply nutrients and oxygen to the nerve tissues. Epineurium is the fibrous sheath that covers the entire nerve (Figure 1). Tissue perfusion in the peripheral nerve is dependent Perfusion pressure is defined as the difference between the mean arterial blood pressure and the internal pressure within nerve. In experimental animal models, high blood flow in the sciatic nerve was observed between mean blood pressures of 80-110 mmHg.[12] Acute hypotension was associated with a de- crease in blood flow in the peripheral nerve.[13] Peripheral nerves lack vascular autoregulation.[12-14] Autoregulation is the intrinsic ability of an organ to maintain a constant blood flow despite changes in perfusion pressure. At mean blood pressures below 85 mmHg, there was marked decrease in the peripheral nerve blood flow.[12] Acute nerve ischemia leads to focal and generalized impairment of impulse conduction across the nerve that can be detected within 10 min of ischemia.[16]

Mechanism of perioperative nerve injury

Direct trauma causing disruption and destruction of nerve fibers can lead to peripheral nerve dysfunction. One of the main and crucial mechanisms of PPNI is ischemia of nerve fibers.[17,18] Slowing of nerve conduction due to ischemia of the nerve fibers is the hallmark of peripheral nerve injury. Focal demyelination may occur if local ischemia is prolonged, leading to sustained axonal damage.[19-21] Peripheral nerve studies in experimental animal model demonstrated that reperfusion injury after prolonged ischemia (3-7 h) results in endometrial edema, conduction block, bloodnerve barrier disruption, intramyelinic edema and demyelination.[22-24] Focal nerve ischemia is an important pathologic mechanism in hyperesthesia, Wallerian degeneration and axonal injury in animal models.[17] The interdependence between ischemic and mechanical factors (stretch and compression) as a cause to nerve injury is well established, although incompletely understood. Stretch of the peripheral nerve is one of the main mechanisms of peripheral nerve injury in perioperative patients.[31] During spine surgery, under general anesthesia, patients are frequently placed in positions that may stretch nerve fibers beyond their resting length. Over- stretch of the nerve can lead to direct nerve damage via disruption of axons and vasa nervosum. Peripheral

nerve injury occurs if nerves are stretched beyond 5%-15% of their resting length.[32-34] Stretch of the peripheral nerve leads to an increase in the intraneural pressure and com- pression of the intraneural capillaries and venules lead- ing to a reduction in the perfusion pressure of the nerve fibers and ischemia.[34,35] Stretch of the peripheral nerves has been shown to suppress axonal transport leading to changes in conduction characteristics.[32,33,38,39] Peripheral nerve compression is another related mechanism of PPNI.[31] Compression of peripheral nerve leads to damage of nerve fibers. in the perfusion pressure leads to ischemia and slowing of conduction in the nerve fibers. Patients with persistent postoperative neuropathy had evidence of inflammatory reaction in peripheral nerves.

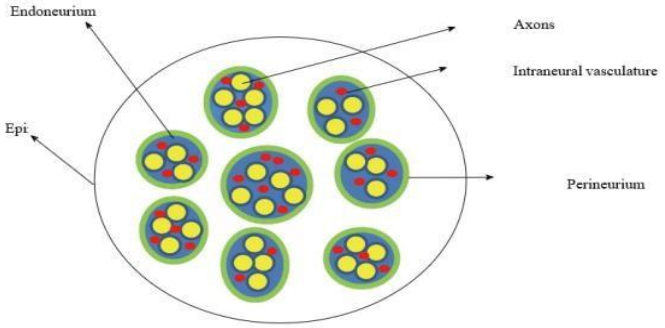

Figure 1: Schematic representation of the cross section of the peripheral nerve.

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Risk factors for nerve injury

Certain drugs and chemicals can predispose patients to peripheral neuropathy.[41] Many medical conditions and diseases can make peripheral nerves more susceptible to injury during the surgical period.[41] Diseases affecting the microvasculature and anatomical differences can contribute to nerve damage or make patients more susceptible to nerve damage. Hypertension, smoking, diabetes, general anesthesia, neurosurgical procedures, and orthopedic surgery were significantly associated with
PPNI. [7]
Advanced age is associated with peripheral neuropathy after median sternotomy.[42] Hypovolemia, dehydration, hypotension, hypoxia, electrolyte disturbances, and hypothermia are associated with neurological damage.[43] The

etiology of PPNI is multifactorial and related to the patient's predisposition to mechanical and physiological factors.

Ulnar neuropathy

Perioperative ulnar neuropathy occurred in 0. 5% of surgical patients; primarily men between 50-75 years of age.[44] Ulnar neuropathy can lead to significant morbidity and loss of function. Ulnar nerve injury results in the inability to op- pose or abduct the fifth finger and loss of sensation of the fourth and fifth fingers. who developed perioperative ulnar neuropathy had permanent neuropathy with residual symptoms beyond 2 years.[44] Perioperative ulnar nerve injury has a delayed onset, most cases manifest within 2-7 d post-operatively (median 3 d).[5,19,44-49] Perioperative ulnar neuropathy presented as sensory deficit in 47% of the cases while 53% of the deficits were mixed sensory and motor. Bilateral symptoms of ulnar neuropathy developed in 9% of the cases. Fifty-three per- cent of patients with perioperative ulnar neuropathy who survived the first postoperative year regained complete sensory and motor functions and were asymptomatic. Patient related risk factors for perioperative ulnar the ulnar nerve may be susceptible to injury due to a pre-existing subclinical neuropathy.

Pre-existing asymptomatic abnormal conduction in the contralateral ulnar nerve has been observed in patients who developed postoperative ulnar neuropathies.[46] Induced and prolonged hypotension has been associated with perioperative ulnar nerve injury.[26,51,52] Positioning during anesthesia has been related to ulnar neuropathy.[52] neatly in men[5,19,47,53,54] with 70% of perioperative ulnar nerve injury cases occurring in males.[19] The ulnar collateral artery and vein run in close proximity to the ulnar nerve and may be affected by external pressure leading to reduced perfusion, ischemia and nerve injury.[30] The forearm position is a significant factor in determining pressure over the ulnar nerve at the elbow. The study provided clear evidence that forearm supination significantly minimizes pressure over the ulnar nerve at the elbow (2 mmHg) compared with the neutral (69 mmHg) and prone (95 mmHg) forearm positions. In the supine forearm position, the pressure over the ulnar nerve was low regardless of the degree of abduction of the arm at the shoulder. In the neutral forearm position, pressure over the ulnar nerve decreased as the arm was abducted between 30° and 90°. Pronation of the forearm produced the largest pressure over the ulnar nerve regardless of the abduction of the arm between 30° and 90°.[30] Extraneural pressures recorded along the path of the ulnar nerve in fresh cadaveric arms were significantly increased with elbow flexion beyond 90°. Gelberman et al [58] investigated the relationship be- tween the ulnar nerve and the cubital tunnel during flexion of the elbow in normal human cadavers. They observed a significant decrease in the cross-sectional area of the cubital tunnel coupled with an increase in the pressure within the cubital tunnel and ulnar nerve. Intraneural pressure of the ulnar increased significantly with the elbow flexed 70° or more.

Extraneural pressure increased significantly when the elbow was flexed to 100° or more. The intraneural pressure was significantly increased at lesser degrees of flexion compared to the extraneural pressure.

The authors conclude that the increase in the intraneural pressure of the ulnar nerve is not entirely due to extraneural compression. Dynamic changes in the cubital tunnel and the cross- section of the ulnar nerve contribute to the increased intraneural pressure with flexion. Compared with full extension, the mean area of the cubital tunnel in the sub- aponeurotic region decreased by 18% and 39% and the ulnar nerve mean area decreased by 24% and 50% with elbow flexed 90 and 135 degrees respectively. Intraneural and extraneural pressures within the cubital tunnel are lowest at approximately 45° of flexion.[58] Flexion of the elbow to 135° resulted in an 18% elongation of the ulnar nerve.[59] Stretch of the ulnar nerve by elevation of the shoulder, flexion of the elbow and dorsiflexion of the wrist caused a marked increase in the intraneural pressure.[60]

Patel et al[61] assessed the morphologic changes in the ulnar nerve and cubital tunnel with elbow motion in fresh The diameter of cubital tunnel in the subaponeurotic region decreased with progressive elbow flexion.[61]

Compared to the ASA closed claims report published in 1990, the report published in 1999 showed a relative decrease in the incidence of ulnar nerve injury claims as a proportion of total nerve injury claims and a relative increase in spinal cord injury claims. In the ASA closed claims study, the mechanism of ulnar neuropathy was explicitly stated in only 9% of the claims.[6] Perioperative ulnar neuropathy is not confined to Patients commonly rest in a supine position, flexing their elbows and resting their arms on their chest and abdomen. Elbow flexion may increase pressure on the ulnar nerve in the postcondyler groove of the humerus due to stretching of the cubital tunnel retinaculum. Forearm pronation may lead to external compression of the ulnar nerve. [45]

Brachial plexus injury

Ulnar nerve injury results in the inability to oppose or abduct the fifth finger and loss of sensation of the fourth and fifth fingers. Perioperative ulnar nerve injury has a delayed onset, most cases manifest within 2-7 d postoperatively (median 3 d).[5,19,44-49] Fifty-three percent of patients with perioperative ulnar neuropathy who survived the first postoperative year regained complete sensory and motor functions and were asymptomatic. Patient related risk factors for perioperative ulnar the ulnar nerve may be susceptible to injury due to a preexisting subclinical neuropathy. Pre-existing asymptomatic abnormal conduction in the contralateral ulnar nerve has been observed in patients who developed postoperative ulnar neuropathies. [46] Positioning during anesthesia has been related to ulnar neuropathy.[52] nantly in men[5,19,47,53,54] with 70% of perioperative ulnar nerve injury cases occurring in males.[19] The forearm position is a significant factor in determining pressure over the ulnar nerve at the elbow. The study provided clear evidence that forearm supination significantly minimizes pressure over the ulnar nerve at the elbow (2 mmHg) compared with the neutral (69 mmHg) and prone (95 mmHg) forearm positions. In the supine forearm position, the pressure over the ulnar nerve was low regardless of the degree of abduction of the arm at the shoulder. In the neutral forearm position, pressure over the ulnar nerve decreased as the arm was abducted between 30° and 90°. Pronation of the forearm produced the largest pressure over the ulnar nerve regardless of the abduction of the arm between 30° and 90°.[30] Extraneural pressures recorded along the path of the ulnar nerve in fresh cadaveric arms were significantly increased with elbow flexion beyond 90°. Gelberman et al [58] investigated the relationship be- tween the ulnar nerve and the cubital tunnel during flexion of the elbow in normal human cadavers. They observed a significant decrease in the cross-sectional area of the cubital tunnel coupled with an increase in the pressure within the cubital tunnel and ulnar nerve. Intraneural pressure of the ulnar increased significantly with the elbow flexed 70° or more. Dynamic changes in the cubital tunnel and the cross- section of the ulnar nerve contribute to the increased intraneural pressure with flexion. Compared with full extension, the mean area of the cubital tunnel in the sub- aponeurotic region decreased by 18% and 39% and the ulnar nerve mean area decreased by 24% and 50% with elbow flexed 90 and 135 degrees respectively. Flexion of the elbow to 135° resulted in an 18% elongation of the ulnar nerve. [59] Stretch of the ulnar nerve by elevation of the shoulder, flexion of the elbow and dorsiflexion of the wrist caused a marked increase in the intraneural pressure.[60] Perioperative ulnar neuropathy is not confined to Patients commonly rest in a supine position, flexing their elbows and resting their arms on their chest and abdomen. Elbow flexion may increase pressure on the ulnar nerve in the post-condylar groove of the humerus due to stretching of the cubital tunnel retinaculum. Forearm pronation may lead to external compression of the ulnar nerve. [45]

Median neuropathy

Medial nerve damage is relatively rare and is responsible for only 4% of all anesthesia-related malpractice complaints. [6] The median nerve can be injured when an intravenous catheter is inserted into the anterior fossa. However, strain is the main mechanism of median nerve injury due to the surgical site.

Medial neuropathy usually presents as motor neuropathy with loss of first and fifth finger antagonism and decreased sensation on the volar surfaces of the lateral three and a half fingers. Midline neuropathy does not resolve readily, and most patients present with persistent symptoms of motor dysfunction. Elbow extension can lead to overstretching of the median nerve leading to injury. [11] Muscular patients and patients with limited elbow extensor range may be at risk for damage to the median nerve if the arm is fully extended under general anesthesia. The decreased range of extension in these patients may result in similar constriction of the median nerve, making it susceptible to overstretching.[66] Excessive extension of the elbow in the supine position should be avoided to the extent that it is uncomfortable for the awake patient.[11] Overextending the wrist to insert the arterial line can lead to temporary but significant impairment of median nerve function. Prolonged hypotension of the wrist can lead to slowed nerve conduction and median nerve damage. [67]

Radial neuropathy

The most common mechanism of radial nerve injury is direct compression at the spiral grove of the humerus. It may occur in the lateral position with abduction of the operative position, patient group and type of spine surgery.[79,83,86] Direct compression by the overhead arm board at the mid-humerus may occur in the lateral position. Injury to the radial nerve results in wrist drop, inability to extend the metacarpophalangeal joint and inability to abduct the thumb with loss of sensation from the lateral and posterior arm, posterior fore- arm and a portion of the dorsal hand.

Intraoperative neuromonitoring

Intraoperative neuromonitoring is available in most institutions in the United States and is frequently used during spine surgery.[69] Commonly used intraoperative neuro- monitoring modalities are somatosensory evoked potential (SSEP) and motor evoked potential. Neuromonitor ing is primarily used to monitor the integrity of the spinal cord during spine surgery. However, SSEP monitoring has been used to detect peripheral nerve conduction abnormalities indicating peripheral nerve stress and im- pending injury during surgery under general anesthesia in variable intraoperative positions.[70-86] Conduction changes detected by SSEP may indicate position-related impend- ing peripheral nerve injury. In a retrospective study of 1000 consecutive spine cases, position modification of the upper extremity lead to resolution of 92% of upper extremity SSEP changes.[86] Position modification strategies used in the review included correcting extreme elbow flexion and extension, decreasing shoulder abduction, re- leasing shoulder traction on tucked arms (caused by taping down the shoulder) and moving the upper extremity into the original position if the position had been modified. After position modification of the upper extremity and resolution of SSEP change, patients experienced no post-operative upper extremity peripheral nerve injury.[86] Significant SSEP change indicating impending upper extremity nerve injury is usually defined as reduction in amplitude of 50% or more and/or increase in latency of 10% or more.[73,86] Usually changes in both amplitude and latency are monitored and evaluated. Compared to laten- cy, amplitude changes may be a more sensitive and valid measure of changes in nerve conduction.[87,88] Most SSEP components are mediated by large myelinated fibers. Some secondary peaks may be transmitted by smaller fibers. Potentials recorded from Erb's point may be the most sensitive to ischemia.[28]

Intraoperative neuromonitoring

Significant SSEP changes indicate abnormal conduction and impending nerve injury. If the changes persist for a prolonged period of time, permanent nerve injury may occur.[30] The use of SSEP to monitor extremity nerve function and guide position modification of the upper extremity into a more favorable position for the peripheral nerve may protect peripheral nerves from in- jury under general anesthesia.

Postoperative visual loss

The incidence of position related significant upper extremity SSEP changes during spine surgery ranges from 1.8% to 15% depending on POVL is a rare but traumatic and devastating complication of spine surgery and general anesthesia. The re- ported prevalence rate of POVL after spine surgery is 0.0028%-0.2%.[89-92] The incidence of POVL associated with spine surgery in the prone position under general anesthesia has increased over the past several decades.[93] POVL usually results in permanent unilateral or bilateral visual loss. Most cases are associated with prolonged spine procedures in the prone position under general an- esthesia. Posterior lumbar fusion and surgery for correc tion of scoliosis were associated with the highest rate of POVL.[92] POVL has been associated with instrumented spine surgery in the prone position.[94] The most common causes of POVL after spine surgery are ischemic optic neuropathy (ION) and central retinal artery (CRA) occlusion. ION is further classified into anterior ION (AION) and posterior ION (PION). PION is the most common cause of POVL after spine surgery. In 1999, the ASA committee on professional liability established the ASA POVL registry to identify predisposing factors and intra- operative risk factors. It is important for spine surgeons to be aware of POVL and to participate in safe, collab orative perioperative care of spine patients positioned in the prone position.

Anatomy and physiology of the optic nerve

The posterior ciliary arteries are end-arteries that provide blood supply to the head of the optic nerve and the retina. The blood supply of the anterior optic nerve is derived from retinal arterioles, centripetal branches from the peri- papillary choroid and short posterior ciliary arteries (Figure 2). To summarize, the main blood supply of the anterior optic nerve is derived from the short posterior ciliary arteries and the peripapillary choroid. The main blood supply to the posterior optic nerve is derived from recurrent branches of the peripapillary choroid and pial branches of the CRA.

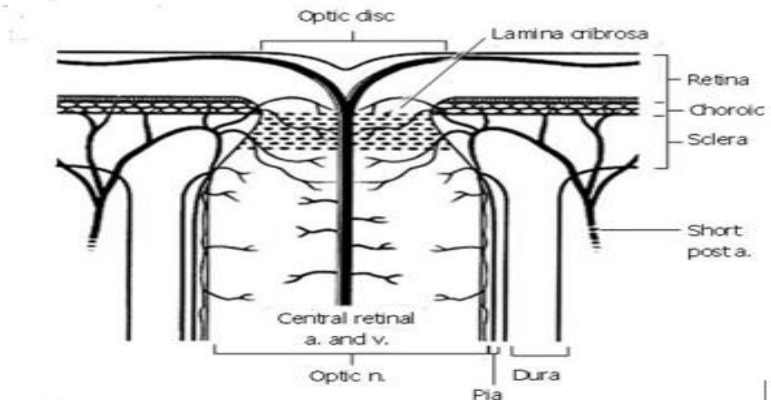

Figure 2: Diagram of the anterior optic nerve showing the Arterial and Small vessel supply to the Choroid and Optic nerve as it passes through the lamina cribrosa. Short

Blood flow to the optic nerve

The blood flow to the optic nerve head is dependent on perfusion pressure. Ocular perfusion pressure is the difference between mean arterial blood pressure and intra- ocular pressure (IOP) or venous pressure
(Whichever is higher).[101] It is important to note that the mean arterial pressure determining optic nerve blood flow refers to that of the optic nerve vasculature and not the pressure in the brachial or radial arteries. Local arteriolar vasoconstriction may reduce perfusion to the optic nerve leading to ischemia despite a normal brachial blood pressure measurement.

Thus three main factors determine optic nerve perfusion; vascular tone, arterial blood pressure and IOP.

Autoregulation

There is evidence that the optic nerve head autoregulates blood flow.[102-105] Autoregulation is achieved through alterations in resistance of the terminal arterioles. There are limitations to the degree to which arteriolar resistance can be altered to maintain perfusion. Autoregulation works within a range of mean arterial pressure, below or above which the local perfusion is dependent entirely on the difference between mean arterial blood pressure and intraocular or venous pressure. Factors leading to the breakdown of autoregulation of blood flow to the optic nerve include age, hypertension, uncontrolled blood pressure, diabetes mellitus, atherosclerosis, hypercholesterolemia, and vascular endothelial disorders.[106-109] A study of the blood flow to the optic nerve head using laser Doppler flowmetry in healthy volunteers demonstrated that blood flow was constant between ocular perfusion pres sures of 56 to 80 mmHg. Not all patients have autoregulation of the blood flow to the optic nerve.[105] A study of autoregulation of the optic nerve in.

Arterial blood pressure

Arterial blood pressure is one of the main determinants of blood flow to the anterior and posterior optic nerve. There is a progressive fall in the blood pressure from the internal carotid artery, to the ophthalmic artery, to the posterior ciliary artery and then to the small branches supplying the optic nerve. The blood pressure in optic nerve may be half or less than that measured in the brachial artery.[109] Vascular changes such as atherosclerosis, vasospasm and vasculitis may lead to further decreases in blood flow to optic nerve. Critical drops in blood pressure below the lower limit of autoregulation will lead to a reduction in optic nerve blood flow. Hypotension resulting from antihypertensive medications or shock may lead to ischemia of the optic nerve and anterior ischemic optic neuropathy.[110-113] Nocturnal hypotension has been associated with glaucomatous visual loss.[110,113,114] Nocturnal arterial hypotension may be a key factor in the development of nonarteritic AION as more than 75% of these patients discover visual loss upon awakening in the morning.[115] Arterial hypertension can decrease blood flow to the optic nerve if it is outside the upper limit of autoregulation or there is an absence of autoregulation. In this setting a decrease in the blood flow is due to
arteriolar vasoconstriction.[109]

Proper positioning

The prone surrender position: In the prone surrender (Superman) position, injury can occur along the entire length of the brachial plexus. Patients placed in the prone.

Positioning patients for spine surgery Awareness of the rare potential complications of patient

posture during spinal surgery is essential to improve care and reduce the likelihood of such complications. Complete prevention of PPNI and POVL is impractical due to the multifactorial etiology of complications and the lack of clear and definitive knowledge of the etiology. Proper education of the perioperative staff, combined with clear communication and cooperation when positioning the patient in the operating room is the best and safest approach. Prevention of rare complications of spine surgery depends primarily on identification of high-risk patients, appropriate positioning, and optimal management of intraoperative physiological parameters. Modification of risk factors extrinsic to the patient may help reduce the incidence of peripheral nerve damage and POVL during surgery.

Identifying high risk patients

High-risk PPNI patients are typically middle-aged men with extreme physical habits. Prolonged hospital stay is a risk factor for the development of intraoperative ulnar neuropathy. Certain surgical sites used in spine surgery may pose a risk of loss of nerve function in the upper extremities. The supine position has been associated with claims of nerve damage. [5] Patients placed in the supine (Superman) surrendered position and in the lateral recumbent position had a significantly higher rate of impending upper extremity nerve damage related to position than patients in the supine position. Supine position, arm supine and arm supine. Stomach. [86] Patients with a history of upper extremity peripheral nerve injury should be considered at higher risk of developing NIPP. Patients at high risk for POVL are those who are expected to undergo multiple-level vertebral lengthening procedures in the prone position with significant blood loss expected. The ASA Task Force on the Prevention of POVL considers prolonged surgery when it exceeds 6.5 hours and severe blood loss when the patient's blood loss exceeds 44.7% of the estimated blood volume.[178] POVL should be discussed with these patients when informed consent is obtained. It is important to educate patients about the multifactorial etiology of POVL, the lack of clear understanding of the etiology, the individual anatomical differences, and the very low incidence of this rare but devastating complication. This dire. Surgical staging should be considered in high-risk patients, as this may reduce the risk of POVL. [178] However, the decision to arrange for spine surgery for a high-risk patient must be individualized and weighed against other perioperative risks.

Proper positioning

The prone surrender position: In the prone surrender (Superman) position, injury can occur along the entire length of the brachial plexus. Patients placed in the prone surrender (Superman) position had a significantly higher incidence of position-related impending upper extremity nerve injury detected by SSEP compared to patients positioned in the supine arms tucked, supine arms out and prone arms tucked positions. [86] If the head is directed away from the arm this can stretch the brachial plexus, therefore lateral neck rotation should be avoided. Although patients may comfortably tolerate arm abduction greater than 90° in the prone surrender position. [11] It is advisable to limit the shoulder abducted to less than 90° to avoid overstretch of the brachial plexus. The forearm should be placed in a neutral position to minimize the direct pressure on the ulnar nerve at the elbow (Figure 3). Vigilance and frequent checking of patient positioning is important. When patients are placed in the prone position, direct pressure on the eye must be avoided as it may cause CRAO. [178] The proneview® consists of a foam cushion in a plastic frame that supports the face without applying pressure on the eyes, nose or mouth and a mirror that allows frequent examination of the eye and facial structures (Figure 5). The head should be in a neutral forward position when possible avoiding significant neck flexion, extension, lateral flexion or rotation of the brachial plexus. The longitudinal axis of the forearm should be parallel to the longitudinal axis of patient to avoid outward rotation of the arm. The longitudinal axis of the forearm should be parallel to the longitudinal axis of patient to avoid outward rotation of the arm. However, in the prone position the range of motion for the elbow extension and flexion is limited. However, in the prone position the range of motion for the elbow extension and flexion is limited. The forearm should be placed in a neutral po sition to minimize the direct pressure on the ulnar nerve at the elbow (Figure 3). The forearm should be at or be- low the table mattress surface. The elbow and the inner aspect of the upper arm should be padded with foam to avoid direct pressure on the nerves. The elbow and the inner aspect of the upper arm should be padded with foam to avoid direct pressure on the nerves. Prolonged overextension of wrist over the wrist board placed for arterial lines should be avoided as it may stretch the median nerve. Prolonged overextension of wrist over the wrist board placed for arterial lines should be avoided as it may stretch the median nerve. In a steep, prone Trendelenburg position the brachial plexus may be compressed between the clavicle and the first rib especially

with use of shoulder braces. In a steep, prone Trendelenburg position the brachial plexus may be compressed between the clavicle and the first rib especially with use of shoulder braces. Vigilance and fre- quent checking of patient positioning is important.

The use of SSEP helps to detect impending upper extremity peripheral nerve injury and guide position modification of the upper extremity. The use of SSEP helps to detect impending upper extremity peripheral nerve injury and guide position modification of the upper extremity. When patients are placed in the prone pos tion, direct pressure on the eye must be avoided as it may cause CRAO. [178]

The lateral decubitus position:

Patients placed in the lateral decubitus position had a significantly higher incidence of position- related impending upper extremity nerve injury detected by SSEP compared to patients positioned in the supine arms tucked, supine arms out and prone arms tucked positions.[86] In the lateral decubitus position compression is the main mechanism of peripheral nerve injury of the dependent brachial plexus. Placing the roll in the axilla will increase the pressure on the brachial plexus in the axilla predisposing the patient to nerve injury. In the lateral decubitus position, there is increased pressure under the dependent shoulder. The average pressure under the dependent shoulder in the lateral position is 66 mmHg (and can exceed 100 mmHg). The pressure under the dependent

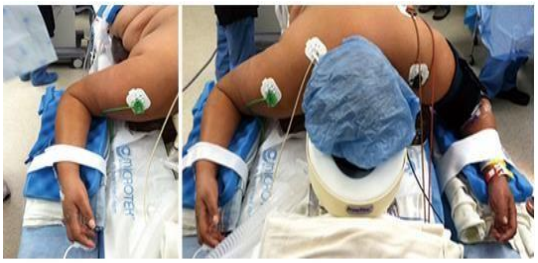

Figure 3: Positioning patient in the prone surrender (Superman) position. The head should be in neutral position on foam supporting head frame (e.g., proneview®) to avoid any direct pressure to the eye. The shoulders should be abducted less than 90°, lateral rotation of the upper arm and extreme elbow flexion should be avoided. The forearm should be positioned in the neutral position to minimize direct pressure on the ulnar nerve in the elbow. Soft foam padding should be placed under the el- bows and between the inner upper around the gel rolls (Or supporting frame) supporting the body. The level of the forearm should be at or below the

mattress surface.

The horseshoe head rest has been associated with CVAO and POVL in the prone position and therefore should be avoided if possible. The horseshoe head rest has been associated with CVAO and POVL in the prone position and therefore should be avoided if possible. Head positioning in Mayfield pins avoids direct pressure on the eye globe (Figure 4). Head positioning in Mayfield pins avoids direct pressure on the eye globe (Figure 4). The pronev iew® consists of a foam cushion in a plastic frame that supports the face without applying pressure on the eyes, nose or mouth and a mirror that allows frequent exami- nation of the eye and facial structures (Figure 5). High-risk patients should be positioned with the head above the heart when possible. High-risk patients should be positioned with the head above the heart when possible. The head should be in a neutral forward position when possible avoiding significant neck flexion, ex- tension, lateral flexion or rotation. [178]

shoulder decreased to 20 mmHg when the chest wall was elevated using an inflatable chest roll. The pressure further decreased to 12 mmHg when the head was supported by a second inflatable pillow to allow straightening of the cervical spine avoiding lateral angulation of the cervical spine. Patients placed in the lateral decubitus position had an average lateral angulation of neck of 14 degrees. After applying an inflatable chest roll, the average lateral angulation of the neck sig nificantly increased to 20°. When the neck was brought into alignment by inflating a second pillow under the head, the lateral neck angulation decreased significantly to 4°. Using inflatable pillows beneath the dependent chest was associated with significantly less pressure beneath the dependent shoulder and chest compared to a 1000 mL intravenous fluid bag or gel-pads. Prolonged lateralization of the cervical spine can stretch the brachial plexus on the nondependent side.[180] The nondependent arm rest should be positioned in a way that maintain the arm horizontal and at the same level of shoulder joint (Figure 7). Excessive

elevation of the nondependent arm at a level higher than the shoulder joint can overstretch the brachial plexus and predispose the patient to radial nerve injury in the nondependent arm. POVL has been associated with spine surgery in the lateral decubitus position.[139] Asymmetric bilateral PION with significant involvement of the dependent eye has been reported after spine surgery in the lateral decubitus position.[181] Neutral forward position of the neck should be maintained to optimize venous drainage from the eye and the orbit. The incidence of impending position-related upper extremity nerve injury detected by SSEP changes are 1. 8% and 2. 1% for the supine arms tucked and prone arms tucked positions respectively.[86] The neck should be maintained in the neutral forward position whenever possible. In the prone arms tucked position, the use of a horseshoe head rest should be avoided.

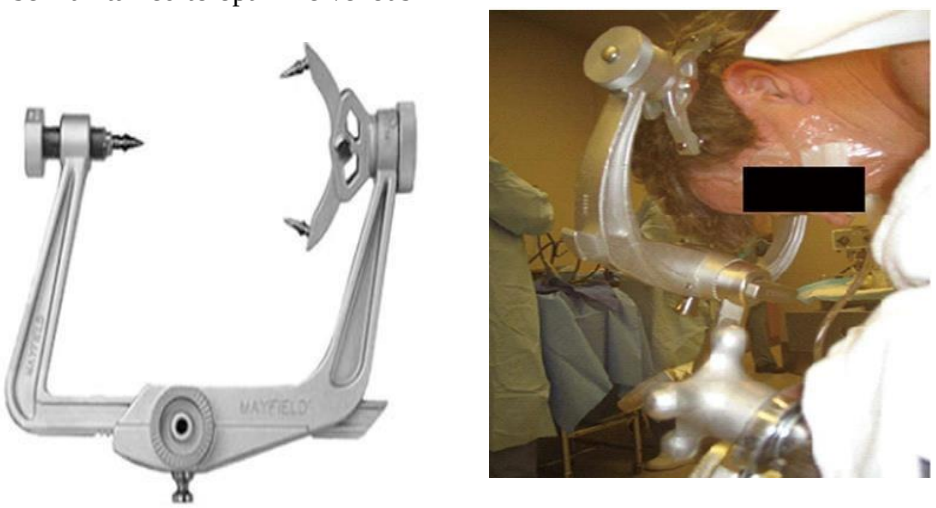

Figure 4: Mayfield (Pinned) head holder

Figure 5: The proneview® allows prone positioning without any pressure on the facial structures. The mirror provided allows frequent checking of facial structures in the prone position

Intraoperative management of physiological parameters

With the lack of this knowledge, it is advisable to optimize physiologic parameters by maintaining them close to patient's baseline values, especially in high-risk cases. Physiologic parameters determining oxygen delivery to the peripheral nerve and the optic nerve may have additive or synergistic effect in predisposed patients placed in challenging operative positions for prolonged periods. Maintaining physiologic mean arterial blood pressure parameters, avoiding severe anemia and venous congestion are important aspects of intraoperative management that may improve oxygen de livery to areas at risk. Although patient predisposition and intraoperative positioning are usually the risk factors associated with peripheral nerve injury, hypotension and anemia can affect oxygen delivery to the peripheral nerve especially in the presence of stretch or compression. The ASA task force on the prevention of POVL believes that the use of deliberate hypotension during spine

surgery has not been shown to be associated with the development of perioperative visual loss; however, it is advisable to avoid deliberate hypotension in high-risk patients (e.g., with preoperative chronic hypertension). If deliberate hypotension will be used in patients without preoperative hypertension, the blood pressure should be maintained on average within 24% of baseline MAP or with a minimum systolic BP of 84 mmHg. Central venous pressure monitoring should be considered in high-risk cases. Hemoglobin should be monitored periodically in high-risk cases with significant blood loss. Until we have a better understanding of the effects of hypotension and anemia on PPNI and POVL it is advisable to maintain intraoperative mean arterial blood pressure and hemoglobin levels close to preoperative levels in patients at high-risk for PPNI and POVL.

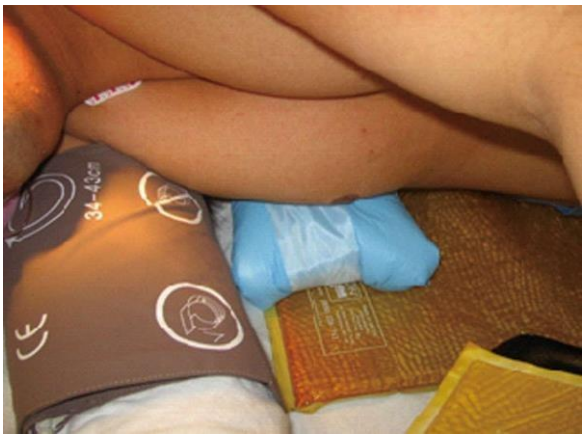

Figure 6: Proper placement of chest roll under the dependent chest in the lateral decubitus position. The chest roll should not be placed under the dependent axilla.

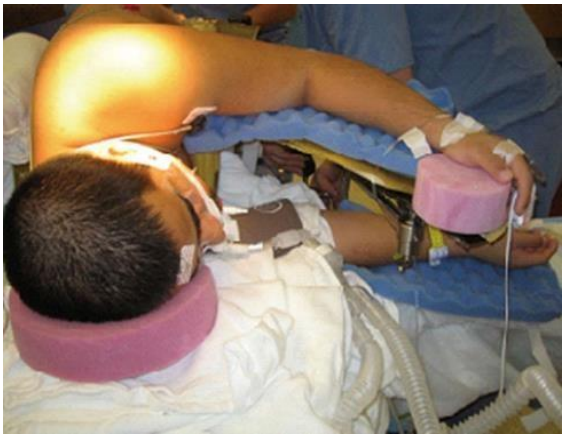

Figure 7: Positioning the upper extremity in the lateral decubitus position. The shoulder abduction more than 90°, extreme elbow flexion and forearm pronation should be avoided in the nondependent arm. The nondependent and dependent elbows should be padded with foam. Placing foam or blankets under the dependent hand and foreram to avoid full extension may reduce the likelihood of median nerve injury. Head and neck should be in neutral forward position avoiding neck flexion extension, lateral rotation and lateral flexion.

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