Anesthetic Considerations in Rehabilitative Surgery

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Introduction

The field of rehabilitative surgery has grown considerably over the past decade. Care of the neurologically injured patient previously entailed providing support and comfort care. We now aim to restore functionality for these patients and repair their injuries. Patients with brain and spinal cord injury (SCI) have unique challenges because of their injuries. This chapter will discuss the anesthetic management of these patients, including preoperative evaluation and preparation, intraoperative management, and postoperative care, including acute pain management.

SCI is associated with reduced life expectancy among survivors. Mortality is highest in the first year. After the first year, life expectancy is approximately 90% of normal [1–3]. Higher level of injury, i.e., cervical or high thoracic, and advanced age are negative risk factors. The most common causes of death are pulmonary complications followed by cardiovascular events [1, 2]. The rate of suicide is also higher than in the general population [3].

Autonomic Dysreflexia

Autonomic dysreflexia (AD), seen in SCI above T6, is a manifestation of the loss of coordinated autonomic response to stimuli on cardiac function and vascular tone [4, 5]. Exaggerated sympathetic response to stimulation below the level of the injury leads to diffuse vasoconstriction and hypertension. A compensatory parasympathetic response above the level of the injury leads to vasodilation and bradycardia. This does not allow for enough "runoff" to reduce elevated blood pressure. Lesions lower than T6 do not have this problem as the neurologically intact splanchnic vascular bed provides compensatory dilatation. Although any stimulation below this level can cause this syndrome, typical sources include bladder distention, stool impaction, pressure ulcers, bone injury, or even positioning on the operating room (OR) table [6]. It can also complicate the peripartum period.

Symptomatic manifestations include headache, diaphoresis, elevated blood pressure, flushing, nausea, and blurred vision. Severity ranges from asymptomatic to severe cardiac and/or neurological events. The higher the level of SCI is, the greater the frequency and severity of the attacks. Acute management of AD attacks involves:

- 1. Monitoring blood pressure (BP) and heart rate (HR).
 - Prompt reduction in BP and correction of HR abnormalities with short-acting agents

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A.I. Elkwood et al. (eds.), Rehabilitative Surgery, DOI 10.1007/978-3-319-41406-5_3

- Common agents include:
 - Nitrates (1" Nitropaste[®])
 - Enalapril (1.25 mg IV)
 - Sublingual nifedipine (10 mg)
 - IV hydralazine (10 mg)
- 2. Reverse Trendelenburg positioning (placing the head of the bed below the foot) to induce orthostatic hypotension.
- 3. Look for and correct noxious stimuli.

Preoperative Evaluation

As with all patients, preoperative evaluation begins with a systemic analysis, with special emphasis on the areas of increased risk in SCI (Table 3.1). These patients may have been disabled for many years, requiring an anesthesiologist to have a higher level of vigilance for earlier onset of disease states. SCI patients often have undergone multiple surgical procedures to address urological issues. Integument and gastrointestinal pathology may occur at a higher rate than in the general population.

Cardiovascular System

Patients with high spinal lesions lack innervation to the sympathetic splanchnic outflow. Lack of basal sympathetic tone to peripheral blood vessels results in vasodilation and postural hypotension. This is modulated over time by an increase in the renin–angiotensin system compensation resulting in a higher capacitance vessel tone. Intravascular volume is often decreased in these patients, and it is critical to ensure adequate volume resuscitation prior to induction of anesthesia. In addition, they have inadequate norepinephrine release that often magnifies the hypotension on induction.

Table 3.1 Expected functional recovery following complete spinal cord injury by spinal level

| Spinal level | Activities of daily living | Mobility/locomotion |
|--------------|---|--|
| C1C4 | Feeding possible with balanced forearm orthosesComputer access by tongue, breath, voice controlsWeight shifts with power tilt and recline chair Mouth stick use | Operate power chair with tongue, chin, or breath controller |
| C5 | Drink from cup, feed with static splints and setup Oral/facial hygiene, writing, typing with equipment Dressing upper body possible Side-to-side weight shifts | Propel chair with hand rim projections short distances on smooth surfaces Power chair with hand controller |
| C6 | Feed, dress upper body with setup Dressing lower body possible Forward weight shifts | Bed mobility with equipment Level surface transfers with assistance Propel indoors with coated hand rims |
| C7 | Independent feeding, dressing, bathing with adaptive equipment, built-up utensils | Independent bed mobility, level surface transfers Wheelchair use outdoors (power chair for school or work) |
| C8 | Independent in feeding, dressing, bathing Bowel and bladder care with setup | Propel chair, including curbs and wheelies Wheelchair-to-car transfers |
| T1 | Independent in all self-care | Transfer from floor to wheelchair |
| T2-L1 | | Stand with braces for exercise |
| L2 | | Potential for swing to gait with long leg braces indoors Use of forearm crutches |
| L3 | | Potential for community ambulation Potential for ambulation with short leg braces |
| L4-S1 | | Potential for ambulation without assistive devices |

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As mentioned above, lesions above T6 interrupt the cardiac accelerator fibers resulting in bradycardia and a diminution in inotropy. As a result, patients present with high vagal tone with conduction defects, heart block, and arrhythmias. A preoperative electrocardiogram (ECG) should be routine regardless of the patient's age.

Respiratory System

Respiratory compromise is common not only in patients with lesions above C6, the level of innervation of the diaphragm, but in those with thoracic lesions as well. In thoracic lesions, abdominal muscle activity may be absent and intercostal activity, minimal. This impairs coughing, deep breathing, and clearing secretions [7]. Upper accessory muscles, e.g., the sternocleidomastoid and trapezius, may play a larger role in breathing. Gastric and bowel distention from autonomic dysfunction may further impair diaphragmatic excursion and increase atelectasis, as well as increased risk of regurgitation and aspiration. Kyphoscoliosis, an abnormal curvature of the spine in both a coronal and sagittal plane, is common in these patients and may aggravate these issues. Pulmonary function tests (PFTs) often show a decrease in vital capacity, functional residual capacity, and expiratory flows as well as a decrease in PaO₂ and increase in dead space and $PaCO_2$ secondary to the increase in atelectasis. Management may include placement of a nasogastric tube to decompress bowel, chest physiotherapy, and tracheal suctioning.

Genitourinary System

Chronic infection and colonization of the urinary tract develops early in the spinal cord-injured patient. Chronic infection leads to proteinuria, hypocalcemia, and renal insufficiency. A main cause of death in chronic spinal cord lesion patients is renal failure [8]. Patients also develop amyloidosis and hypoalbuminemia secondary to albuminuria. This can lead to significant peripheral edema and skin breakdown. These patients often need prophylactic corticosteroid administration secondary to adrenal cortical dysfunction. Adrenocorticotropic hormone (ACTH) levels can be measured to assist in monitoring adrenal function [11].

Chronic renal insufficiency also leads to a decrease in hemopoietin hormone production causing chronic anemia. This may require blood transfusions prior to surgery. The resultant renal insufficiency can also result in electrolyte and acid–base imbalances often exacerbated by treatment for constipation with enemas, diuretics for peripheral edema, and low-salt diets.

Calcium and potassium imbalances can put the patient at risk for cardiac arrhythmias if not corrected preoperatively [9].

Musculoskeletal System

SCI patients develop osteoporosis and muscle very quickly after injury [10]. wasting Hypocalcemia from renal insufficiency accelerates mobilization of calcium from bones resulting in a high propensity for pathologic fractures from simple movement or positioning on an operating room table. Pressure injury to the skin below the level of the SCI occurs commonly. Decubitus ulcers can develop after 2 h of continuous pressure on a skin area. The chronic anemia and hypoalbuminemia of renal insufficiency make para- and quadriplegic patients even more susceptible to this kind of injury. Secondary osteomyelitis develops at these areas, particularly the ischium, sacrum, and heels. Ulceration at the site of possible injection, fever, elevated white count, or untreated infection are contraindications to regional anesthetic block [11]. Skeletal muscle spasm can also occur after a stimulus below the level of the spinal cord lesion. This involves a spinal reflex arc. This can occur intraoperatively and interfere with the surgical procedure.

Thermal Regulation

The SCI patient is poikilothermic, having a body temperature that varies with the temperature of the surroundings below the level of the spinal cord injury with loss of autonomic control cutaneous vasoactivity, sweating, and shivering. These patients are particularly susceptible to the changes in ambient temperature in an OR. They are not able to efficiently get rid of heat in a warm OR or develop hypothermia quickly in a cold OR [12].

This requires close monitoring of temperature with either an esophageal or bladder temp probe. Forced air warmers and warmed intravenous (IV) fluids should be immediately available.

Psychological Issues

Depression is common in these patients, particularly at the time of the acute lesion. Emotional strains are compounded by the work of rehabilitation economic pressures and recognition of their permanent handicap. Frequently, these problems are compounded by alcohol and drug dependence. It is important to elicit any drug or alcohol use in the preoperative interview as well as any herbal or homeopathic remedies [13].

Intraoperative Considerations

Intraoperative Monitoring

Routine monitoring, including ECG, BP, pulse oximetry, and $EtCO_2$, as well as temperature measurement, should be used on all cases. A urinary catheter is necessary as these patients have no control of bladder function. More invasive monitors, including intra-arterial monitors, should be used if there are expected fluid shifts, a prolonged case, or a patient with a history of severe autonomic hyperreflexia because blood pressure changes may be unpredictable. This also allows for regular measurement of hematocrit, electrolytes, and arterial blood gases.

Central venous pressure monitoring also is recommended when significant fluid shifts are expected because urine output in these patients is not always a good marker of fluid status. It also allows for vasoactive drugs to be given directly into the central circulation to treat episodes of hyper- and hypotension.

Anesthetic Considerations

Patients with SCI require anesthesia for procedures because of the occurrence of autonomic hyperreflexia in otherwise insensitive areas (Box 3.1). It is not recommended to perform surgery on these patients, including common urinary tract work (calculi, fistulas, or bladder work), debridement of decubitus ulcers, or even minor procedures, without an anesthetic that blocks reflexes at the spinal cord level. An episode of hyperreflexia can provoke life-threatening hypertension and bradycardia. Furthermore, topical anesthesia does not block afferent transmission to the spinal cord. Attempting to perform a procedure under inadequate, local anesthesia in a patient with SCI may be extremely dangerous and should not be attempted [14].

General Anesthesia

Induction

Induction of anesthesia in these patients is full of challenges. Not enough anesthesia can induce a hyperreflexic crisis, but too much and profound hypotension and tachycardia ensues. A slow and gentle induction in these patients is required along with the availability of agents to treat wide swings in blood pressure. It is often valuable to monitor BP with an arterial line in these patients so beat-to-beat changes can be treated quickly. Direct-acting agents, like epinephrine, norepinephrine, and phenylephrine, and adequate fluid

Box 3.1: Common Symptoms and Signs of AD

- Headache
- Hypertension (note: comparatively lowresting blood pressure in SCI)
- Flushing/blotching of skin above level of injury
- Sweating above level of injury Reproduced from *Autonomic dysreflexia: a medical emergency*, J Bycroft, I S Shergill, E A L Choong, N Arya, P J R Shah, 81, p. 232–235, 2004

resuscitation should be used. Indirect-acting agents such as ephedrine should be avoided [15]. Depolarizing paralytics, e.g., succinylcholine, should not be used in the first 18 months after injury, as it can cause release of large amounts of potassium into the bloodstream causing ventricular fibrillation. A defasciculating dose does not mitigate this risk. Non-depolarizing agents should be used exclusively in these patients [16].

These patients remain at high risk of aspiration during anesthesia and should always be considered "a full stomach." As discussed earlier, they often do not have adequate respiratory function and reserve and, under anesthesia, are unable to develop adequate inspiratory pressures. For this reason, laryngeal mask airways should be avoided. Endotracheal intubation is the preferred method of protecting the airway. A rapid sequence induction with a non-depolarizing agent, Sellick maneuver (cricoid pressure), and preoxygenation is standard. Awake intubation is an option in patients with an unstable neck or a fixed and fused spine. Newer intubation tools such as video laryngoscopes also have a role. Asleep intubation with a fiberoptic scope is another safe and useful way to secure the airway. The key is a thorough and careful airway examination.

Maintenance

Inhalational anesthetics, particularly agents such as desflurane and sevoflurane, allow for careful titration to stimulus. Maintaining adequate levels of anesthesia allows for attenuation of hypertensive response to stimulus. This often means keeping the patient "deep" until the end of the operation. The choice of inhalational anesthetic is dependent on the underlying hemodynamics of the patient and comorbidities. Renal disease is common in patients with SCI; avoiding sevoflurane in these patients would be appropriate. On the other hand, desflurane can cause tachycardia in therapeutic doses, making hemodynamic control more difficult. Narcotics should be used judiciously in these patients both intra- and postoperatively as they contribute to ileus, which is already common in these patients. One approach to pain management is the use of multimodal therapy, including intravenous (IV) nonsteroidal antiinflammatory drugs, IV acetaminophen, steroids, gabapentins, and judicious use of narcotics.

This helps speed return of bowel function and prevention of ileus.

Use of non-depolarizing muscle relaxants is important to prevent mass muscle reflex occurring during abdominal surgery and facilitates mechanical ventilation in a patient with limited functional reserve capacity. This helps with pulmonary toilet and helps to prevent hypercarbia, atelectasis, and respiratory fatigue in patients with limited reserve [17]. However, mechanical ventilation causes diminished preload by impeding venous return to the heart contributing to the hypotension from the inhalation anesthetics. This is best treated with increased fluids and repairing preload [18].

Increased bleeding is noted in these patients, even in the face of normal clotting parameters. This may be related to the loss of sympathetic arterioles and smaller venules. in tone Hypertensive responses will increase this bleeding, and early transfusion is recommended [13]. Integument protection and temperature control are also critical in these patients. They are far more prone to decubitus ulcers and skin degradation or abrasion. In addition, temperature regulation is significantly more difficult to control in these patients without any compensatory vasoconstriction or dilatation, even if depressed by anesthesia. Forced air warming of these patients as well as using warmed IV fluids can help. The key is temperature monitoring as overenthusiastic warming can lead to hyperthermia [15].

Emergence

Because these patients are being kept "deep" to avoid blood pressure swings during the procedure, it is important to have a controlled emergence and loading of adequate amounts of narcotic to prevent a hypertensive crisis on emergence. The challenge remains to weigh the return of adequate respiratory function [15]. The ability to clear secretions, adequate tidal volume, and minute ventilation all contribute to the decision of when to extubate. The right balance between residual narcotic, inhalation anesthesia, and muscle relaxant all make the decision to extubate often a difficult one.

Spinal and Epidural Versus Regional Anesthesia

Spinal and epidural anesthesia have been used successfully in patients with SCI without any adverse effects on their injury [14]. There is no evidence that it worsens the neurological state of a chronic and stable spinal cord injury. Peripheral nerve blocks can be used as well in patients with nerve injuries, both of the lower and upper extremities. As we have already discussed, the greatest challenge with these patients is their autonomic hyperreflexia. One of the best ways to prevent this is to block the afferent pathways at the level of the spinal cord, so the reflex loop is cut. Both epidural and subarachnoid blocks can achieve this reliably, as opposed to topical or local anesthesia that does not impact the afferent loop at all. Spinal anesthesia is particularly useful in blocking stimulus at the sacral roots, commonly stimulated in urological procedures. Lumbar epidural anesthesia does not provide consistently reliable relief at the sacral levels but can be more effective at higher levels and is slowly titratable to the required level [19].

The respiratory and cardiac difficulties that are the hallmark of general anesthesia are often easier to manage under a regional block. These blocks also provide good muscle relaxation with the use of paralytics. For abdominal operations, an epidural needs to be used in conjunction with a general anesthetic for patient's comfort and safety. The use of epidural or spinal narcotics also helps to block opiate receptors that inhibit autonomic reflexes.

Regional anesthesia is not without its challenges. It's often difficult to determine the level of the block because of the sensory deficit so it is difficult to ascertain if block is at an appropriate level to sufficiently counteract autonomic reflexes until the procedure starts. A test dose through the epidural is difficult to evaluate for subarachnoid injection and intravascular injection putting the patient at risk for total spinal and hemodynamic instability. Neuraxial anesthetics result in often complete sympathetic blockade, making them difficult to manage in patients with SCI who are often hypovolemic, anemic, brady- or tachycardic, and hemodynamically unstable. Patients will typically need fluid resuscitation and vasoactive agents immediately available. It is also often technically difficult to do neuraxial anesthesia in these patients. They often have integumentary issues like decubitus ulcers and excoriations on the lumbar spine area, muscle spasm, and distortion of the vertebral column. Osteoporosis and previous spinal fusion can also be a problem along with difficulty in positioning.

Spinal and epidural anesthesia can be very effective in preventing autonomic hyperreflexia but can be problematic. Assessment of anesthetic level can be difficult; in addition, a test dose during epidural anesthesia is not useful. Technical difficulties are common. Vertebral column deformities, surgery, and infection may make access to CSF difficult [14].

Regional anesthesia has little or no role in the care of spinal cord-injured patients. It neither blunts an autonomic hyperreflexive response nor provides analgesia to an already denervated area.

Treatment of Autonomic Hyperreflexia

Autonomic hyperreflexia needs to be treated immediately and aggressively as soon as it occurs. It can cause increased morbidity and mortality both intraoperatively and postoperatively (Box 3.2). Stroke remains the second most common cause of death in these patients [20]. Treatment starts with the removal of the precipitating stimulus. This may require deepening the level of general anesthesia, raising the level of an epidural, or placing a catheter to empty the bladder. The underlying cause must be identified and treated. Pharmacologic agents should be used simultaneously to lower the blood pressure until the inciting cause is

Box 3.2: The Main Precipitants of AD

- Urological
 - Bladder distension, urinary tract infection, urological procedures (e.g., cystoscopy, urodynamics), and genital stimulation (including assisted ejaculation)
- Gastrointestinal
 - Rectal distension, anorectal conditions, anorectal procedures, and acute abdomen
- Musculoskeletal
 - Fractures, dislocation, and heterotopic ossification
- Others
 - Skin problems (e.g., ulceration, infection), pregnancy, and labor Reproduced from *Autonomic dysre-flexia: a medical emergency*, J Bycroft, I S Shergill, E A L Choong, N Arya, P J R Shah, 81, p. 232–235, 2004

identified. The agents should be easily administered, rapid in onset, and short in duration of action as well as easily titratable.

- Hydralazine [21]
 - Very useful agent in this scenario
 - Pure arteriolar vasodilator, with quick onset and easily titratable
 - Often associated with reflex tachycardia that helps alleviate the bradycardia
 - Dosed in 5–20 mg IV
- Enalapril
 - ACE inhibitor that dilates blood vessels by inhibiting the conversion of angiotensin I to angiotensin II (vasoconstrictor)
 - Causes a gentle vasodilation generally without a reflex tachycardia
 - Dosed in increments of 1.25 mg, up to 5 mg IV
- Nitroprusside
 - Direct arteriolar and venous vasodilator, easily titratable and always effective

- Requires an arterial line for titration
- Prolonged administration can result in cyanide toxicity
- Clevidipine
 - Intravenous calcium channel blocking infusion
 - Arteriolar vasodilator
 - Start with doses of 1–2 mg/h and titrate up to 1 mg/h every 90 s until desired BP
- Nifedipine
 - Also calcium channel blocker that can be used sublingually as well as orally
 - Rapid onset and short duration of action
 - Can also be used prophylactically when given prior to a procedure
- Labetalol
 - Commonly used antihypertensive, but its role in this situation is limited because bradycardia from beta-blocker effect can make the reflex bradycardia from autonomic hyperreflexia worse

Special Cases: Peripheral Nerve Reconstruction

When performing surgery for peripheral nerve reconstruction, there are a few considerations from an anesthetic perspective. Frequently, the surgeons will use intraoperative nerve monitoring, and care must be taken not to interfere with the signal. This means limited use of neuromuscular blocking agents for intubation. Due to muscle spasm, rigidity, previous surgery, or injury, these patients often have a difficult airway, and succinvlcholine is the muscle relaxant of choice. A limited dose of a nondepolarizing muscle relaxant can be used as long as it has worn off before nerve monitoring commences. Inhalation anesthetics provide amnesia, analgesia, and hypnosis but need to be kept to less than half-MAC, standard dosing measure of inhalation anesthetics, to avoid interference with neuromuscular monitoring. Total intravenous anesthesia (TIVA) can either be used alone or in conjunction with small doses of inhalation agents. Often the surgeon requests limited movement on emergence to prevent injuring a fresh nerve repair.

Suctioning the airway while the patient is still deep and ensuring adequate analgesia when awakening help reduce "bucking" and straining on the endotracheal tube.

Postoperative Considerations

Postanesthesia Care

In the postanesthesia care unit, the patient still remains at risk for autonomic hyperreflexia. Care must be taken to prevent bladder distention and distended rectum.

The biggest concern remains in the respiratory system. Inadequate respiration can cause atelectasis and hypoxia. Inability to handle secretions is common in this patient population who often require suctioning [14]. It is important to insure that all muscle relaxants are reversed and patients meet all criteria prior to extubation. Frequently these patients need prolonged ventilation after major surgery. Do not hesitate to leave the patient intubated if it is not clear that he or she can protect his or her airway, clear secretions, and adequately ventilate.

Long-Term Care

Frequent episodes of autonomic hyperreflexia can be managed with nifedipine as well as removing the causative stimulus [22]. Occasionally, cordectomy or neurectomy of pudendal or pelvic nerves is necessary. Selective dorsal rhizotomy can relieve spasticity in intractable cases [23]. Bilateral paravertebral blocks can also be used to block the response. Since bladder distention is the most common cause of autonomic hyperreflexia, efforts should be directed to relieving and preventing this.

Acute Pain Management

The postoperative pain management of these patients starts with a thorough history of pain management medications and muscle relaxants as

well as all medications of which the patient does not tolerate the side effects. Some narcotics are better tolerated than others and this varies from patient to patient. This is particularly important in these patients who often have problems with ileus secondary to denervation or are on narcotics and other medicines preoperatively that slow bowel motility. A thorough history of the pain medicines, both narcotic medications and those used to treat neuropathic pain, that the patient is already on preoperatively is critical to knowing the patient's baseline need for pain medication and muscle relaxants. For example, it would be an error to put a patient on what appears to be an appropriate amount of pain medicine for the procedure and then realize the patient takes more than that dose at home prior to the procedure. It is important to understand the patient's chronic pain needs before treating acute pain.

When thinking about acute pain management in patients with SCI or nerve damage, it is important to think first about multimodal therapy [23]. This is the approach of using nonnarcotic analgesics, muscle relaxants, and anti-inflammatory medications to reduce the amount of narcotic that is required to keep a patient comfortable. Narcotics can commonly cause nausea, ileus, and dysphoria and often require larger doses when chronically used to release pain. The short-term addiction potential is small when used to relieve pain but can create dependency over time.

When thinking about multimodal therapy, we use different medications that work on different areas of the nociceptive pathways. Below is a list of some of the drugs that can be used and suggested doses. These drugs are meant to be used in combination, reflecting a larger goal in pain management.

- Acetaminophen
 - Mechanism thought to be inhibition of COX2 enzyme centrally
 - No local anti-inflammatory effects peripherally
 - Administered IV or orally
 - IV dose is more efficacious immediately post-op in the face of poor GI motility and absorption.

- IV can reduce narcotic requirements of 40–60% in most patients when used on a regular basis as opposed to as needed.
- After 48–72 h, transition to oral doses.
- Dosage and safety concerns
 - Up to 4 g/day in patients with normal liver function.
 - Dosage reduced in patients with impaired liver function demonstrated by elevated bilirubin or prolonged PT in the face of no anticoagulation.
 - Care needs to be taken to consider other combination agents (Percocet, Lortab, etc.) that contain acetaminophen when calculating the total dose.
 - We recommend not using combination agents and giving acetaminophen around the clock to a total of 4 g/day and supplement with narcotics as needed.
- Nonsteroidal anti-inflammatory drugs (NSAIDs)
 - Peripheral anti-inflammatory effect.
 - Analgesic effects both centrally and peripherally.
 - They work mainly by inhibiting cyclooxygenases (COX1 and COX2).
 - Side effects
 - Increase bleeding by inhibiting platelet aggregation
 - One of the leading causes of GI bleeding from ulcers and erosive gastritis
 - Cause acute renal failure if used in the face of impaired renal function or dehydration, particularly in the elderly
 - Important to follow the BUN and creatinine tests and keep the patients well hydrated
 - Despite these drawbacks, extremely efficacious in the treatment of acute pain in the perioperative setting
 - Multiple oral versions of these drugs with different strengths and duration of actions as well as different preferences for COX1 or COX2 receptors
 - IV formulations used in pain management
 - Ketorolac (Toradol).
 - Inexpensive agent
 - Duration of action of 4–6 h and dosed q6 h

- Not recommended to be used for more than 5 days.
- Other IV NSAIDs are diclofenac and naproxen.
 - Similar concerns and varying degrees of COX1 vs. COX2 effects
- NSAIDs, in combination with acetaminophen, provide most of the narcotic dose reduction in multimodal therapy.
- Corticosteroids
 - Reduce inflammation at the site of injury
 - Reduce concentration of cytokines and other mediators of pain in the inflammatory reaction
 - Single dose at the time of the procedure or a short course after a procedure
 - GABA analogs: gabapentin and pregabalin
 - Used to treat neuropathic or "burning" pain
 - All pain from injury, particularly nerve injury, has a component arising from the nerve itself.
 - Possibly works through an inhibitory effect on nerve transmission.
 - Evidence that using these agents in multimodal therapy reduces the incidence of chronic pain syndromes after acute pain episodes.
- Ketamine
 - Works through the NMDA receptor
 - Both dissociative and analgesic
 - Occasionally used in low doses intraoperatively to reduce narcotic requirements
 - Usually 0.5 mg/kg/h loading and 0.1 mg/ kg/h infusion

Narcotic use in these patients is frequently required. A background level is obtained by using a long-acting agent, whether by the oral route or as a patch supplemented by as needed doses of IV or oral agents. A patient-controlled analgesia pump can be used with morphine or dilaudid to allow for pain control and determination of the patient's narcotic requirements. The dose of the long-acting agents can be adjusted to reflect additional requirements and converted to oral form or weaned as the patient improves. A background of multimodal therapy reduces the narcotic requirement significantly, making fewer complications and higher patient satisfaction.

Summary

Anesthesia for rehabilitative surgery requires taking into account the unique challenges of SCI. The loss of internal autonomic function can cause wide blood pressure swings as well as exaggerated response to a severed spinal cord reflex loop. These patients also present positioning problems secondary to contractures, previous surgeries, and muscle spasm. They are often dehydrated with both chronic and acute renal problems, including infection and calculi.

Chronic pain syndromes are common in these patients, who are frequently on narcotics preoperatively that need to be considered in determining their intra- and postoperative pain regimen. Temperature measurement and regulation while these patients are under anesthesia can be a challenge as they are poikilothermic.

References

- 1. National Spinal Cord Injury Statistical Center, Birmingham, AL. Annual report for the model spinal cord injury care system. Birm; 2005.
- Frankel HL, Coll JR, Charlifue SW, et al. Long term survival in spinal cord injury: a fifty year investigation. Spinal Cord. 1998;36:266.
- Hagen EM, Lie SA, Rekand T, et al. Mortality after traumatic spinal cord injury: 50 years of follow-up. J Neurol Neurosurg Psych. 2010;81:368–73.
- Bycroft J, Shergill IS, Chung EA, et al. Autonomic dysreflexia: a medical emergency. Postgrad Med J. 2005;81:232–5.
- Karlsson AK. Autonomic dysreflexia. Spinal Cord. 1999;37:383–91.
- McKinley WO, Jackson AB, Cardenas DD, De Vivo MJ. Long term medical complications after traumatic spinal cord injury: a regional model system analysis. Arch Phys Med Rehab. 1999;80:1402–10.

- Bellucci CH, Wollner J, Gregorini F, et al. Acute spinal cord injury- do ambulatory patients need urodynamic investigations? J Urol. 2013;189:1369–73.
- Chiodo AE, Scelza WM, Kirshblum SC, et al. Spinal cord injury medicine. 5. Long-term Medical Issues and Health Maintenance. Arch Phys Med Rehabil. 2007;88:S76–83.
- Lazo MG, Shirazi P, Sam M, et al. Osteoporosis and risk of fracture in men with spinal cord injury. Spinal Cord. 2001;39:208.
- Desmond J. Paraplegia: problems confronting the anaesthesiologist. Can Anaesth Soc J. 1970;17:435–51.
- Vandam LD, Rossier AB. Circulatory, respiratory and ancillary problems in acute and chronic spinal cord injury. In: Hershey SG, editor. Refresher courses in anesthesiology. Philadelphia: JB Lipincott; 1975. p. 171–82.
- Rocco AG, Vandam LD. Problems in anesthesia for paraplegics. Anesthesiology. 1959;20:348–54.
- Marz DG, Schreibman DL, Matjasko MJ. Neurologic diseases. In: Katz J, Benumof JL, Kadis BL, editors. Anesthesia and uncommon diseases. 3rd ed. Philadelphia: Saunders; 1990. p. 560–89.
- Giffin JP, Grush K, Karlin AD, et al. Spinal cord injury. In: Newfield P, Cottrell JE, editors. Handbook of neuroanesthesia: clinical and physiologic essentials. Boston: Little Brown; 1991. p. 338.
- Gronert GA, Theye RA. Pathophysiology of hyperkalemia induced by succinylcholine. Anesthesiology. 1975;43:89–99.
- Welply NC, Mathias CJ, Frankel HL. Circulatory reflexes in tetraplegics during artificial ventilation and general anaesthesia. Paraplegia. 1975;13:172–82.
- Schonwald G, Fish KJ, Perkash I. Cardiovascular complications during anesthesia in chronic spinal cord injured patients. Anesthesiology. 1981;55:550–8.
- Fraser A, Edmonds-Seal J. Spinal cord injuries. Anesthesia. 1982;37:1084–98.
- Raeder JC, Grisvold SE. Perioperative autonomic hyperreflexia in high spinal cord lesion: a case report. Acta Anesthesiol Scand. 1986;30:672–3.
- Lindan R, Joiner E, Freehafer AA, Hazel C. Incidence and clinical features of autonomic dysreflexia in patients with spinal cord injuries. Paraplegia. 1980;18:285–92.
- Steinberger RE, Ohl DA, Bennett CJ, et al. Nifedipine pretreatment for autonomic dysreflexia during electroejaculation. Urology. 1990;36:228–31.
- Mulcahy JJ, Young AB. Long-term follow-up of percutaneous radiofrequency sacral rhizotomy. Urology. 1990;35:76–7.
- Gordon DB, Dahl JL, et al. American pain society recommendations for improving the quality of acute and cancer pain management. Arch Intern Med. 2005;165:1574–80.
- Randall B, editor. Physical medicine and rehabilitation. 2nd ed. Philadelphia: WB Saunders Company; 2000.