In Part III of a continuing series on neuroanesthesia, the author explores the anesthetic considerations for acute spinal cord injuries. Because of the complexity and severity of physiologic disturbances seen in patients who have such injuries, the nurse anesthetist needs an understanding of the type of injury and the surgical procedure to be performed before the proper anesthetic care can be given.

The annual incidence of spinal cord injuries involves some 11,200 people. Of that total, 4,200 die before reaching the hospital and another 1,150 die during hospitalization. About 4,000 traumatic paraplegics are added to the national number each year. However, developments in medical management have steadily decreased mortality since 1942 when the mortality rate was 60% in the first three months after injury. The leading cause of death in cases of spinal cord trauma is respiratory failure.

Spinal cord anatomy

In reviewing the anatomy of the spinal cord, one can categorize the spinal column into three broad anatomical categories.

The first is referred to as the vertebral unit. It includes the bony column as well as the spine's ligaments and muscles. This unit is responsible for the mechanical stability and mobility of the spinal column. The 26 separate vertebral bones (including 7 cervicals, 12 thoracics, 5 lumbar, the sacrum and the coccyx) provide protection for the underlying spinal cord. Strong annulus fibrous discs provide stability of the synarthroses between the vertebral bodies. The ligamentous structures including the interspinous ligaments and ligament of flavum (sometimes referred to as the yellow ligament) provide further stability.

The second unit is the cord unit which consists of the spinal cord with its associated nerve roots as well as its membranous coverings and the dura. The spinal cord is an extension of the brain stem, suspended by means of a series of nerve roots and ligaments confined by an inelastic fibrous membrane called the dura. The dura mater extends to the level of the second sacral segment where it ends in a blind sac.

The spinal cord itself ends between lumbar segments 1 and 2. The space between the vertebral column and the spinal dura is known as the epidural or extradural space. It contains a network of blood vessels, adipose, and areolar connective tissue. The subdural space is located between the inner surface of the dura and the underlying arachnoid membrane. The innermost layer of the meninges is the pia mater which contains many blood vessels and is important for nourishment of cord cells. The subarachnoid space lies between the arachnoid and pia mater and is filled with cerebrospinal fluid.

A cross section of the spinal cord shows it to
be an H-shaped mass of gray matter (unmyelinated nerve fibers) surrounded by white matter (myelinated nerve fibers). Thirty-one pairs of spinal nerves arise from the spinal cord. Each nerve rises from two roots, the dorsal or sensory root and the ventral or motor root. These nerves connect the spinal cord to the periphery.

The third anatomical category of the spine is the spinal vascular unit which includes the total blood supply of the spine. The substance of the spinal cord is supplied by branches from a number of major vessels including the vertebral and posteriocerebral arteries. Regional perfusion is provided by branches from the thoracic and abdominal aorta as well as from the deep cervical, intercostal, lumbar and lateral sacral arteries. Lateral spinal arteries originating from these vessels form the anterior and posterior radicular arteries. These arteries enter at each side of the cord by their anterior roots and join the anterior spinal artery which descends on the ventral surface of the cord. The posterior spinal arteries arise from the cerebellar arteries and run over the dorsal surface of the cord. The venous drainage to the cord is somewhat variable but its anatomical pattern is similar to that of arteries.

Pathophysiology

Injuries to the spinal cord are said to be due to flexion, extension, compression, or rotation. Rotation forces generally produce dislocation whereas compression forces produce fractures. Since it has the least support, the cervical spine is most vulnerable to injury. Compression to the spinal cord causes the greatest tissue damage. Current belief is that trauma causes a microvascular response that results in significant localized retardation of blood flow. This is a progressive reaction which leads to lethal hypoxia to the cord itself.

Two current theories have arisen concerning the precise mechanism of traumatic microcirculatory failure in the spinal cord. Some feel that injured vascular smooth muscle shortens and contracts to narrow the lumen, increase resistance and retard blood flow. This theory, however, fails to explain why direct traumatic vasospasm increases with time or why the presumably more fragile neurons are not damaged more.

The second hypothesis is that spinal cord catecholamines are responsible for vascular changes since there is a large supply of these amines at vascular receptor sites. Under this hypothesis, delayed hemorrhages are explained by the necessary lag in metabolic time before neurotransmitters can be produced in the toxic concentrations. These toxic substances may produce vasospasm, hypoxia and subsequent tissue necrosis.

Patients with a spinal cord injury present us with several potential problems. A patient with a cervical cord injury is a greater risk than one with an injury site in the lower thoracic cord because the extent of the physiologic disturbance is modified by the level of transaction.

Spinal column instability

Trauma to the spinal cord usually goes hand in hand with injury to the vertebral column. Damage could include fracture of the vertebral body or lamina or disruption of the articular facets or paraspinal ligamentous structures. Frequently, because of separation or subluxation of the spinal structures, injury of the spinal cord may be present.

Because of the need for airway management and possible early surgical intervention, anesthesia personnel may become involved in the early care of these patients. Whether the patient has suffered a complete transection or has sustained little or no injury to the spinal cord, he will usually have a stabilizing device in place to prevent any possible or further damage. Any of the common stabilizers (including the cervical collar, crutchfield tongs or vene a halo traction device) not only limit neck motion but make endotracheal intubation very difficult. Modest extension (but not flexion) of the neck is permissible to facilitate intubation in a patient whose cord is completely transected or if the fracture is present in the vertebral body or lamina.

The patient who has a fracture of the odontoid process or one who has a cervical fracture in which the cord function is intact or partially compromised presents the anesthetist with a very dangerous situation. Extension of the atlanto-occipital joint could cause the odontoid fracture to be pushed into the medulla—a situation which would be disastrous for the patient. The patient with the cervical fracture requires the most careful handling. Undue flexion or extension of the neck may cause an increase in pressure on the cervical cord, with the disastrous consequence of a minimal neurologic deficit being converted into a major one; thus, extreme care must be taken.

 Securing the airway. In order for the anesthetist to secure the airway without further risk to the spinal cord, an awake intubation by the nasal or oral tracheal route is performed. Many aspects must be considered in an awake intubation such as tube size, proper choice of sedation for the patient and a good topical anesthesia of the nose, pharynx, larynx and upper trachea.
In some instances the nasal tube may be the same size as that used through the mouth. Ordinarily, however, one size smaller is selected for the nasal tube. The nasal tube should be well lubricated with lidocaine jelly to facilitate the tube to slide easily through the patient's nostril. Selection of the nostril should be done by occluding one side at a time and asking the patient to sniff through the open one and selecting the easier of the two. The nostrils may be prepared by inserting cotton applicators which have been moistened with a 5% cocaine solution. The principal purpose of the cocaine is to achieve vasoconstriction and secondarily to provide local anesthesia. Shrinking of the mucous membranes of the nasal passage provides more room for the endotracheal tube, decreasing the likelihood of trauma and bleeding.

Local analgesia is provided below the vocal cords by transstracheal injection of 1.5-2.0 ml of 4% lidocaine. The area above the cords may be anesthetized by spraying with 4% lidocaine. Intravenous medication such as fentanyl, 1 cc at a time, may be given cautiously at this point to assist the patient's cooperation and to provide some sedation. Always remember to keep a close watch on the blood pressure.

After the endotracheal tube is in position the patient is asked to move all four extremities and then general anesthesia is induced. It should be noted that the surgeon is present in the room during intubation and induction. The surgeon should also handle the patient's head and neck if any change in position or movement of the patient is needed. Surgery should not be started until the anesthesia team is satisfied that the patient's position is correct, that the IVs and monitoring apparatus are functioning correctly and that the vital signs are stable.

Complications

The life threatening complications of the spinal cord injured patient are a considerable challenge to the anesthetist's skill and knowledge. The areas of major concern in these patients are respiratory function, cardiovascular and autonomic reflexes, temperature regulation and electrolyte disturbances.

Respiratory insufficiency. The degree of impairment of respiratory function depends on the level of spinal cord injury. Patients with an injury of the cord at the 5th or 6th vertebral segment often demonstrate severe respiratory insufficiency early after the injury. The cause is usually related to the presence of cord edema, with a transient interference of cord function. The edema usually subsides with time and frequently descends one to three levels down to the level of the injury.

With a complete lesion, there is total intercostal and abdominal muscle paralysis. With lesions at or below the 6th cervical segment level (C6), diaphragmatic innervation is intact. If the lesion is at the 5th cervical segment, there is diaphragmatic innervation. With lesions at or above C5, the diaphragm loses its major nerve supply and function is grossly impaired. In these cases ventilatory assistance is mandatory and endotracheal intubation may be lifesaving.

The respiratory impairment may be measured. Vital capacity is decreased, residual volume is increased, inspiratory force is decreased, PaO2 is decreased and PaCO2 is increased. All of these factors may lead to respiratory failure and pulmonary edema. Following guidelines of adequate ventilation will assist you in deciding whether to institute mechanical ventilation and in evaluating the need for continued support.

Alveolar ventilation is dependent on the function of both the diaphragm and intercostal muscles for its total volume. The diaphragm may be intact but the intercostal muscles are inactive in a cervical spinal cord injury. The rib cage, with descent of the diaphragm, may assume a paradoxical movement and limit alveolar exchange.

Cervical cord injury patients usually have a vital capacity of 30% of expected values while those who have a high or low thoracic injury have a 45% vital capacity of the expected value.

The surgery performed for stabilizing the neck is not extremely painful, therefore large doses of narcotics for pain relief are not usually required. If a narcotic is given postoperatively, careful evaluation of its effect on respiration is needed because atelectasis is a problem in a patient with a cervical spine injury.

Autonomic Hyperreflexia. This syndrome occurs in patients who have a transverse lesion at or above the 5th thoracic level (T5). Its symptoms include hypertension, sometimes prolonged, causing a 5-9% decrease in blood volume. Other symptoms are bradycardia, headache, and sweating, flushing and piloerection in the face and upper trunk area. The severity of the hypertension has been known to cause cerebral vascular accidents. The symptoms may be precipitated by a stimulus from below the injury site. Manipulation of the perineum, genitalia and distention of the bladder or rectum are a few of such stimuli.

It is when the autonomic afferent fibers are no longer under the control of the higher centers that
stimulation is thought to produce the autonomic hyperreflexia.

Cardiovascular alterations. A spinal cord injury from T₁ to the second lumbar space (L₂) produces an altered sympathetic outflow. Cardiac compensatory ability is lost with lesions above the level of T₃. This may be referred to as spinal shock, which is an abnormal spinal reflex mechanism. Spinal shock may vary in its duration from three days to six weeks. It is characterized by hypotension, bradycardia, hypothermia, and possible psychic disturbances. Although a transient bout of hypotension may be initially seen at the time of injury, it is followed by a prolonged period of bradycardia and hypotension. The underlying mechanism of these symptoms is that of a traumatic sympathectomy. This hypotension is a cardiovascular shock type phenomenon, not to be confused with the spinal shock.

Those patients, injured above T₁, usually have a heart rate of 50 beats per min. and a low mean arterial blood pressure of 40 mm torr. Orthostatic compensation is interfered with. Some patients can hardly tolerate even a few degrees of head up-tilt. They have only a small increase in heart rate but a greater decrease in the mean arterial pressure. Phentolamine administered intravenously in small doses can help prevent this phenomenon completely. Phentolamine is an alpha-adrenergic blocking agent. These patients seem to be able to regulate their heart rate by decreasing vagal tone and to vasoconstrict in the unsympathectomized areas.⁶

Other anesthetic considerations

Temperature. The loss of the patient’s autonomic function will cause him to be hypothermic. Monitoring devices should be employed to help in minimizing aggravation of this situation. A warming blanket, heated humidified air apparatus, warming of IV solutions and continuous monitoring of the patient’s temperature should be standard procedure.

Electrolyte imbalance. Flaccid paralysis results from the failure of nerve conduction to voluntary muscle fibers. As a result of absent muscle activity, mobilization of calcium may be increased. This produces hypercalcemia and hypercalciuria which begins about 10 days after injury.⁶ Knowing that a large increase in serum calcium may predispose the patient to ventricular arrhythmias during anesthesia, a preoperative calcium level should be measured. Antiarrhythmic agents and potassium chloride should be available during surgery.

Muscle relaxants. It has been reported that succinylcholine has induced ventricular fibrillation in spinal cord injured patients. It is recognized that peripheral nerve section or spinal cord injuries result in cholinergic receptor spread in the muscle causing a hypersensitivity to acetylcholine or drugs having a cholinergic agonist activity.⁷ The spread of the receptors begins taking place after a few days, usually two to three, after the injury and may continue for many months, up to seven or eight. The length of time is not well documented so it is advisable to avoid succinylcholine and use a safe alternative—pancuronium.

Preoperative medication. The previous reference to postoperative pain medication and its effect on respiration holds true for preoperative medication. The anesthetist also needs to evaluate if the patient’s emotional status warrants a preoperative medication. The anesthetist also needs to evaluate if the patient’s emotional status warrants a preoperative medication. The patient’s emotional status warrants a preoperative medication. The patient’s emotional status warrants a preoperative medication. The patient’s emotional status warrants a preoperative medication. The patient’s emotional status warrants a preoperative medication. The patient’s emotional status warrants a preoperative medication.

Fluid therapy. Since the heart of a quadriplegic patient cannot compensate for over-transfusion or an increase in venous return, a central venous pressure (CVP) line (or a pulmonary artery catheter) is considered a mandatory monitoring device. These patients may also have an associated pulmonary injury which could be aggravated by large volumes of solutions. Another consideration to keep in mind is the fact that the peripheral vascular bed will be dilated from the cardiac sympathectomy which occurs in a cervical cord injury. Additionally, fluid therapy may need modifications from gastric suction or intestinal dilatation.

Conclusion

From all of the knowledge pertaining to the spinal column instability, its pathophysiology, respiratory insufficiency, autonomic hyperreflexia, cardiovascular alterations and anesthetic considerations, a patient with an acute spinal cord injury must be adequately assessed preoperatively in order to provide an optimal and safe anesthetic. Communication between all members of the neurosurgical team plays a major role in reaching this goal.

REFERENCES

AUTHOR

Mary Kay Davis, CRNA, BS, received her RN from Braddock General Hospital School of Nursing in Braddock, Pennsylvania and her baccalaureate degree in Education from California State College in California, Pennsylvania. She received her anesthesia education at the University Health Center of Pittsburgh School of Anesthesia for Nurses, where she is now a didactic coordinator and clinical instructor. Ms. Davis is currently Nurse Anesthetist Coordinator of Neuroanesthesia at Presbyterian-University Hospital in Pittsburgh.

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