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# Autonomic Hyperflexia: Current Treatment Modalities in Anesthesia

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# AUTONOMIC HYPERREFLEXIA:

# CURRENT TREATMENT MODALITIES IN ANESTHESIA

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(Chairperson)

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## ABSTRACT

Autonomic hyperreflexia is a syndrome which can cause life threatening increases in blood pressure in the spinal cord injured patient. It is estimated that of spinal cord injured patients sixty-six to eighty-five percent of them will have an occurrence of this syndrome in their lifetime. This is of great importance to anesthesia providers as the spinal cord injured patients represents only a small proportion of those patients presenting for surgery. The syndrome can present with symptoms of increased blood pressure, increased heart rate, increased respiratory rate, headache, and many others. Recognizing autonomic hyperreflexia is of particular interest because during anesthesia and surgery its symptoms can arise from several things during surgery, such as light anesthesia or not enough pain control. The problem for anesthesia providers is noticing the symptoms, identifying their root cause, and providing the proper treatment. As the condition is somewhat rare, anesthesia providers need to have a treatment plan ready for instances when this syndrome occurs. This independent study familiarized anesthesia providers from a Midwest hospital and anesthesia students from a nearby educational program on current treatment modalities for spinal cord injured patients that have an increased risk for this syndrome to occur.

In this project a review of current literature was conducted and current treatments utilized in anesthesia for autonomic hyperreflexia were identified. An informational pamphlet was developed for use by anesthesia providers to use during cases that involve spinal cord injured patients. The pamphlet can be used to properly diagnose and treat those in which autonomic hyperreflexia may occur. After researching and designing the

pamphlet an inservice on autonomic hyperreflexia was provided to anesthesia students and nurse anesthetists so as to familiarize them to the current treatment modalities for autonomic hyperreflexia.

This independent topic informed current anesthesia providers and students form a Midwestern community about a condition and its treatment that does not occur frequently during anesthesia but is very much a life threatening event if it goes unnoticed. By informing providers about the risks, signs and symptoms of autonomic hyperreflexia providers are now better prepared to properly recognize and treat patients in whom this condition may occur.

# CHAPTER I

## INTRODUCTION

Autonomic hyperreflexia has been an issue for medical providers since introduced academically and medically in 1944 by Ludwig Guttman. At this time Guttman noticed the correlation of distension of viscera in spinal cord injured patients and the response that was regulated by autonomic mechanisms which induced a profound effect on the cardiovascular system and its resultant hypotension in portions of the body that are above the level of the spinal lesion. For anesthesia providers this syndrome can be of utmost importance during surgery for the spinal cord injured patient. For as Kuczkowski (2003) stated, "for anesthesia providers knowing the difference in symptoms and the appropriate methods of treatment is imperative for optimal patient care." Jones and Jones (2002) also state, "Autonomic hyperreflexia is a common and potentially life threatening complication of chronic spinal cord injury and its control is paramount to the safe care of such patients in the surgical care environment" (p. 805).

Autonomic hyperreflexia is commonly distinguished by increases in blood pressure, reflex bradycardia, sweating, and piloerection in regions below the spinal cord injury (Hansen, 1998). These physiological symptoms may lead to further complications and possible death if not diagnosed and treated appropriately. Amzallag (1993) stated,

> "untreated symptoms such as high blood pressure lead to retinal, subarachnoid, and basilar artery insufficiency and hypertensive encephalopathy which

significantly add to rates of morbidity and mortality rates among spinal cord injured patients. The ability of the anesthesia provider to quickly recognize and treat the symptoms of this syndrome is paramount for patient safety and outcome. Also, it will empower the anesthesia provider to be able to identify the specific patient population which will undoubtedly benefit from the proper treatment and development of a preoperative plan which is specific for the spinal cord injured patient. By educating anesthesia providers with the specific signs and symptoms of the syndrome, the patient population it occurs in, and treatment for it, anesthesia care will be enhanced"(p. 87).

## Clinical Problem

Autonomic hyperreflexia is a common occurrence in patients with spinal cord injury above the thoracic sixth and seventh vertebrae. (King, Johnson, & Wood, 1999). In fact Bishop (2002) stated, "it's prevalence among the spinal cord injured population ranges from sixty six percent to eighty five percent of those individuals" (p. 485). While it may occur in the activities of daily living for spinal cord injured patients, it most often occurs either intraoperatively or postoperatively following recovery and from both spinal and general anesthesia. Surgery is a particularly potent stimulus to the development of autonomic hyperreflexia, and even patients with no history of this response may be at risk during operative procedures (Stoelting & Dierdorf, 2002). When spinal cord injured patients are anesthetized, the anesthesia provider must be knowledgeable about the pathophysiology of the autonomic hyperreflexia reflex, the signs and symptoms of the syndrome, and the proper treatment modalities for this condition. This independent study explored autonomic hyperreflexia and its treatment in addition, an informational

pamphlet and educational inservice were developed and provided for an anesthesia provider audience.

## Purpose of the Independent Study

The purpose of this independent study was to develop an educational pamphlet and inservice to be utilized by anesthesia providers for implementation during those surgeries in which spinal cord patients are given anesthesia. This study examined treatment modalities for autonomic hyperreflexia as well as familiarized anesthesia providers to the precipitating factors and signs and symptoms of autonomic hyperreflexia. Successful implementation of the pamphlet and the information therein by anesthesia providers is paramount to assist said providers to optimize anesthesia care for spinal cord injured patients.

## Conceptual Framework

A physiological framework guides this independent study. An understanding of the physiology of the spinal cord and the reflexes of it is vital to recognizing the mechanism by which autonomic hyperreflexia occurs and implementing treatment to avoid the harmful effects that autonomic hyperreflexia may occur. This said, the physiology of autonomic hyperreflexia provides the foundation of diagnosing and treatment of this condition.

## Physiology of the Spinal Cord

The spinal cord is a part of the central nervous system which also includes the brain and has as its major function the processing of information. When the body detects a sensory input the signal is received and integrated by the central nervous system and the appropriate motor responses are generated (Hansen, 1998). There is also the peripheral

nervous system which includes paired cranial and spinal nerves that function in the transmission of impulses between the central nervous system and peripheral organs. The autonomic nervous system is also a portion of the peripheral nervous system, as the peripheral system sends signals only to internal organs to maintain homeostasis of the body. Two portions of the nervous system are the peripheral and autonomic systems and are both partitions of the spinal cord.

The spinal cord extends downward from the brain through the vertebral column to the first lumbar vertebrae within the spinal column. The spinal cord consists of an inner gray matter surrounded by white matter. The inside gray matter is H shaped and the anterior portions of it contain the motor system and the posterior portions contain the sensory system. At higher levels of the intermediolateral cord, horns are present and these represent cells of the sympathetic division of the autonomic nervous system (Hansen, 1998). Within this gray matter are cell bodies of neurons that are specialized cells of the neurological system, each consisting of a cell body (soma), multiple afferent fibers (dendrites), and a single efferent fiber (axon) (Hansen, 1998). The neurons are responsible for relaying and transmitting the neural impulses that are generated by a stimulus and conducting that stimulus to the synaptic junction to further conduct the impulse to the brain. In this transmission of neural impulses neurotransmitters are released to propagate the impulse from the first order neuron to the second potentially resulting in another action potential (excitatory postsynaptic potential) or inhibiting from occurring (an inhibitory postsynaptic potential). This is the basis of an impulse generation and conduction within the spinal cord. For spinal cord injured patients, impulse conduction is severed and the reflex arc that normally exists in uninjured patients

is altered leading to increased stimulation of the sympathetic nervous system leading to the condition known as autonomic hyperreflexia (Cosman, 2005).

## Pathophysiology of Autonomic Hyperreflexia

Autonomic hyperreflexia appears following spinal shock and in association with return of spinal cord reflexes. This reflex response can be initiated by cutaneous or visceral stimulation below the level of spinal cord transaction. For as Vaidyanathan (1998) stated, "Distention of the bladder or rectum in spinal cord injured patients is a common precursor for autonomic hyperreflexia"(p. 765). Bors and French (1952), also state that, "the greatest responses of autonomic hyperreflexia were produced by stimuli with the most caudal root levels below the region of spinal cord injury. This is why pelvic visceral stimulation is most commonly implicated in autonomic hyperreflexia" (p. 807).

The incidence of occurrence depends upon the level of spinal cord transection. In spinal cord injured patients with injuries at or above the sixth thoracic vertebrae 85 percent will exhibit this reflex. Conversely, it is highly unlikely to be exhibited where transection is at or below the level of the tenth thoracic vertebrae.

Stimulation below the level of spinal cord transection initiates afferent impulses that enter the spinal cord below that level. These impulses elicit reflex sympathetic nervous system activity over the splanchnic outflow tract. In neurologically intact patients, this flow is modulated by inhibitory impulses from higher centers in the central nervous system. In the presence of spinal cord transection this outflow is segregated from the inhibitory impulses sent from the higher centers in the central nervous system thereby leading to consistent generalized vasoconstriction that persists below the level of

the spinal cord injury. Vasoconstriction results in an increase in systemic blood pressure, which is then noted by baroreceptors in the carotid sinus. As the carotid sinus senses this rise in pressure there is a decrease in efferent sympathetic nervous system activity from the central nervous system which will lead to an increase in parasympathetic nervous system activity of the heart and peripheral vasculature. This normal response to systemic increases in blood pressure will decrease blood pressure and peripheral vasoconstriction in non spinal cord injured patients. This cannot occur in spinal cord injured patients below the level of injury, as this portion of the body is neurologically isolated because of the transection of the spinal cord. Therefore, the vasoconstriction remains in areas below the spinal cord transection. Murphy (1999), supports this finding by stating,

> "Autonomic hyperreflexia occurs because the afferent impulses that produce vasodilation cannot reach the neurologically isolated portion of the spinal cord, vasoconstriction develops below the level of the spinal cord transection, resulting in systemic hypertension which left unnoticed and untreated will lead to detrimental effects to those patients that it occurs in" (p. 148)

## The Role of Noradrenaline in Autonomic Hyperreflexia

Noradrenaline plays a significant role in autonomic hyperreflexia. Levels of this catecholamine decrease greatly following a spinal cord injury and remain low. Hambly and Martin (1998) stated, "noradrenaline levels remain low in spinal cord injured patients presumably as an affect of decreased sympathetic activity of the central nervous system after the spinal cord injury" (p. 277). At times of autonomic hyperreflexia there is a noted increase of noradrenaline levels. However, during these periods of AH there is a much lower concentration of noradrenaline in spinal cord injured patients than the non

spinal cord injured counterparts. In a study by Mathias, Christensen, Corbett, Frankel and Spalding (1976) it was found that even after infusing noradrenaline in normal subjects without spinal cord injury that noradrenaline levels were many more times greater in the normal subjects than those seen during episodes of autonomic hyperreflexia. This was done in attempt to compare the ratio of noradrenaline in those with and without spinal cord injury and the resultant hypertension that occurs in the response to noradrenaline. Also, Karlsson (2006) stated, "autonomic hyperreflexia is mediated by the sympathetic nervous system as shown by a profound increase in noradrenalin spillover below the lesion level" (p. 388). This suggests that spinal cord injured patients are more sensitive to the effects of catecholamines. This statement is further confirmed by Gao, Ambring, Lambert and Karlsson (2002) in a study of spinal cord injured patients in which it was found that under resting conditions, spinal cord injured patients have lower sympathetic activity but normal cardiac control. It was also found that below the lesion level, peripheral stimulation induces vasoconstriction and profound sympathetic surge activity evidenced by an increase in total body noradrenalin spillover into the plasma.

## Significance of the Independent Study

Enhanced patient outcomes for spinal cord injured patients following surgery are dependent upon the knowledge and understanding of autonomic hyperreflexia by the anesthesia provider. To optimize patient outcome it is essential for the provider to implement the proper treatment plan for these patients. Greater yet is the estimated cost of medical care for a patient after an episode of autonomic hyperreflexia. It is estimated that the cost of medical care after spinal cord injury can be upwards of one hundred-

twenty-thousand dollars a year after the original injury and the estimated cost of caring for those affected by the complications of autonomic hyperreflexia as being upwards of twenty thousand dollars for each acute care admission related to the side effects caused by autonomic hyperreflexia (United Spinal, 2006). Most complications resulting from autonomic hyperreflexia can be avoided given the proper treatment plan and the timely recognition and treatment of autonomic hyperreflexia if it occurs unexpectedly during surgery. Through successful implementation of an evidence-based pamphlet and presentation, anesthesia providers will be more aware of autonomic hyperreflexia, its signs, symptoms and complications and will ensure that the proper care for autonomic hyperreflexia will become a reality.

## Assumptions

Core assumptions of this independent study include:

1. Anesthesia personnel would be familiar with terminology used for the pamphlets and inservice.
2. Participants partaking in the inservice would use the information to ensure the proper care of spinal cord injured patients.
3. The author assumes that the literature reviewed was an accurate portrayal of current research studies in autonomic hyperreflexia.
4. Anesthesia providers aim to provide optimal care for spinal cord injured patients when under their care.

## Limitations

The following characteristic of this independent study are limitations:

1. The final decision to utilize the pamphlet was made by the anesthesia provider or the

institution in which they are employed or attending school at.

2. Dissemination of the information was limited to the attendance at the inservice by anesthesia personnel.

## Definitions

The following definitions will assist the reader:

Spinal Shock: a state of transient physiological (rather than anatomical) reflex depression of cord function below the level of injury with associated loss of all sensorimotor functions.

Autonomic Hyperreflexia: is a syndrome of massive imbalanced reflex sympathetic discharge occurring in patients with spinal cord injury (SCI) above the splanchnic sympathetic outflow (T5-T6).

Neurogenic shock: is manifested by the triad of hypotension, bradycardia, and hypothermia. Shock tends to occur more commonly in injuries above T6, secondary to the disruption of the sympathetic outflow from T1-L2 and to unopposed vagal tone, leading to decrease in vascular resistance with associated vascular dilatation.

Catecholamines: are principally norepinephrine and epinephrine which act as neurotransmitters to produce affects in organs directly innervated by the sympathetic nervous system.

Spinal Cord Transection: damage to the spinal cord, usually due to trauma, that leads to paraplegia or quadriplegia which interrupts all nervous pathways between the brain and spinal cord.

Afferent Neurons: neurons conducting impulses inward to a central organ or section, as nerves that conduct impulses from the periphery of the body to the brain or spinal cord.

Efferent Neurons: neurons conducting impulses outwards from the brain or spinal cord to organs.

Sympathetic Nervous System: The part of the autonomic nervous system originating in the thoracic and lumbar regions of the spinal cord that in general inhibits or opposes the physiological effects of the parasympathetic nervous system, as in tending to reduce digestive secretions, speeding up the heart, and contracting blood vessels.

Parasympathetic Nervous System: The part of the autonomic nervous system originating in the brain stem and the lower part of the spinal cord that, in general, inhibits or opposes the physiological effects of the sympathetic nervous system, as in tending to stimulate digestive secretions, slow the heart, constrict the pupils, and dilate blood vessels.

Axons: The usually long process of a nerve fiber that generally conducts impulses away from the body of the nerve cell

Dendrite: A branched extension of a nerve cell that conducts impulses from adjacent cells inward toward the cell body

## Summary

The objective of the pamphlet and inservice that was developed for this independent study was to provide current research and treatment modalities regarding the use of anesthesia in spinal cord injured patients. This independent study, pamphlet, and presentation will allow anesthesia providers to be better informed about spinal cord injured patients and the possible ramifications that autonomic hyperreflexia has on this type of patient population.

# CHAPTER II

## REVIEW OF THE LITERATURE

Clinicians in anesthesia need to be aware of certain aspects of anesthesia practice that when carried out, will lead to the optimal patient outcomes. This is of utmost concern for those that have spinal cord injuries as without the proper plan of care and proper interventions detrimental side effects can and will occur. The following literature review will explain and clarify several of the key components of autonomic hyperreflexia, current methodologies in practice and the impact of the condition on healthcare and patient outcome.

### *Precursors to Condition – Spinal Cord Injury*

Most often patients that exhibit autonomic hyperreflexia have a spinal cord injury either by transection or by tumor invasion into the spinal canal. Miller (1991) stated, "At the acute time of injury following blunt trauma or compression of the spinal cord, hemorrhages are seen in the central gray matter" (p. 336). As the hemorrhages continue into the gray matter, edema and necrosis spreads from the central cord to involve, in severe injuries, the entire diameter of the cord. This occurs within six to twenty four hours from the time of injury (Miller, 1991). Damage to the gray matter typically involves two to three segments of the spinal cord at the level of injury. For as Catalana (1994) stated in a study of injuries of the spinal cord:

> As injury occurs there is an interruption of nerve conduction in the fiber tracts of the spinal cord which isolates the region of the body below the the level of injury from cerebral control. As edema and hemorrhage continue there is progressive loss of function after the initial injury for the

first twenty four hours related to the associated secondary injury, disc compression and hypoperfusion of the spinal cord. As edema subsides or circulation is reestablished, function of some areas may improve slightly (p. 22).

Injury to the patient progresses in the acute phase, as the injury causes sympathetic stimulation. Desjardins (2006) stated, "the immediate response to spinal cord compression is a massive sympathetic stimulation and reflex parasympathetic activity that usually lasts for three to four minutes and is mediated by alpha adrenergic receptors". This response causes the hemodynamic effects of severe hypertension and reflex bradycardia or tachycardia. After this primary response of the body there is a loss of neurological function below the level of injury causing spinal shock. This is the period when there is a flaccid paralysis of voluntary muscle, loss of muscle tone and sympathetic tone which creates hypotension and bradycardia in high spinal cord injured patients. These conditions then cause flaccidity of the gastrointestinal (GI) tract and bladder creating uniform intestinal ileus and urinary retention. In the days following injury sympathetic tone returns partially to control resting blood pressure. Recovery now flows to the chronic phase of the injury.

This period of chronic injury occurs after approximately four to six weeks in which time the sympathetic tone returns controlling mild hypertensive responses and various other stimuli, including pain and bladder distention below the level of injury. The hallmark of the chronic phase of injury according to Silver (2000) is when, "reflex activity returns after a period of four to six weeks" (p. 230). This phase is also characterized by spastic motor paralysis with hyperactive tendon reflexes, occasional severe autonomic hyperreflexia and some partial return of involuntary bladder function.

As the sympathetic tone recovers anesthesia providers need to be aware of the increased supersensitivity of cholinergic receptors.

When injury occurs there is also a denervation of nerve cells. The body's response to this is the proliferation of cholinergic receptors beyond the motor end plates of voluntary muscle fibers and eventually includes the entire cell membrane. The muscle then becomes very sensitive and contracts to a maximal degree in response to acetylcholine. According to Desjardins (2006), "the amount of acetylcholine required to initiate a response is only forty to fifty percent of what is needed to initiate a contraction in normal muscle." As the contraction occurs, potassium is released suddenly rather than gradually as in normal muscle. This produces a rapid increase in serum potassium levels. This sudden increase in potassium occurs also when the neuromuscular blocking agent succinylcholine is given for muscle paralysis for induction and intubation of a patient. Furthermore, succinylcholine can be used safely in the first twenty four hours but should not be used thereafter because of the risk of hyperkalemia. The latter can occur within the first week following injury and is due to excessive release of potassium secondary to the proliferation of acetylcholine receptors outside the neuromuscular junction (Morgan, Mikhail & Murray, 2002)

Succinylcholine induces an identical response of increasing serum potassium levels and may contribute to potassium increases of up to four to ten millequivalents per liter. The extent of this increase is proportional to the amount to muscle mass that is paralyzed. This is particularly important as shortly after giving succinylcholine the serum potassium levels peak and may lead to irreversible ventricular dysrhythmias and cardiac arrest. Because of the muscle sensitivity the severity of the reaction is

independent of the dose of succinylcholine given. Barash, Cullen and Stoelting (2001), state,

> "Succinylcholine produces massive and simultaneous depolarization of all of the involved muscles, within three minutes a sudden and severe increase in serum potassium occurs. Levels as high as fourteen mil-equivalents per liter may be reached, resulting in ventricular dysrhythmias and cardiac arrest. It is also best to avoid succinylcholine after the first week of injury as supersensitivity becomes evident approximately one week following injury" (p. 898)

*Causes of Autonomic Hyperreflexia*

The chronic phase of spinal cord injury in which spinal reflexes reappear is characterized by autonomic hyperreflexia. As Krassioukov, Furlan and Fehlings (2003) stated, "In a high proportion of individuals the inciting cause of autonomic hyperreflexia was due to the trigger mechanisms of somatic pain, fecal impaction, and abdominal distention" (p. 711). Colachis (2002), also stated that "urinary retention and often the urinary tract infection that accompanies it is a major contributor to autonomic hyperreflexia" (p. 233). Schonwald, Fish and Perkash (1981) stated, "Surgery is a potent stimulus for autonomic hyperreflexia and may precipitate an episode of autonomic hyperreflexia even in a patient that has never had a previous episode of it" (p. 553). There are many conditions that may lead to an incident of autonomic hyperreflexia. Pressure ulcers that often accompany spinal cord injured patients and the immobility that cause them have also been indicated as a root cause of autonomic hyperreflexia (AH) occurring (Valles, Benito, Portell & Vidal, 2005). The most often precipitant of AH is through the distention of a hollow viscus such as the rectum or urinary bladder. Although

the reflex may occur from spasm or distension of other viscera or from tactile or thermal stimulus of the skin. The magnitude of the response is generally proportional to the magnitude of the stimulus and is greater with the increasing distance between the level of the cord lesion and the level of nerve entry (Faust, 2002).

*Signs and Symptoms of Autonomic Hyperreflexia*

The presenting signs and symptoms of autonomic hyperreflexia are varied and include a throbbing headache, goose bumps, parasthesias, trembling, flushing and sweating of the head and torso, nasal obstruction, a need to void, anxiety and nausea. In addition to these symptoms hypothermia and hyperthermia may also be manifestations of the condition. There also may be a feeling of dullness in the head. Blurred vision is also a sign that is not uncommon. Kewalramani (1980) stated, "severe headache, usually of occipital, bitemporal and bifrontal location is noted in more than half of the patients" (p. 18). Most commonly the cause of headache is the increase in blood pressure created by the inability for the body to counteract the sympathetic discharge created one of the causes of autonomic hyperreflexia.

The main objective sign of autonomic hyperreflexia is a significant rise in systolic and diastolic blood pressure. Systolic blood pressure of 250-300 mg Hg and diastolic of 200-220 mm Hg has been reported (Karlsson, 1999). In those with normal physiological function the hypertension would be counteracted by the response of baroreceptors and would produce vasodilation of peripheral and arterial vessels as well as a decrease in heart rate. In those with spinal cord injuries the only way for the body to counteract the hypertension is vasodilation above the level of injury and a decrease in heart rate caused by the baroreceptors response. This vasodilation above the level of injury possibly

explains why the some exhibit the fullness of the head and nasal congestion. Karlsson, (1999) provided guidelines for clinical use:

> For clinical use the following criteria can be suggested; Increase in systolic blood pressure by at least twenty percent, combined with at least one of the following symptoms: sweating or chills or cutis anserina (goose flesh) or headache or flushing. For scientific use the increase in blood pressure of at least twenty percent ought to be combined with a visualized vasoconstriction below the lesion level, illustrated by laser doppler flowmetry of skin blood flow or saturation monitoring (p. 388).

In addition to the above described signs and symptoms, hypothermia is also a direct effect of autonomic hyperreflexia. According to Colachis (2002) patients with spinal cord injury have thermoregulatory dysfunction. The injured area creates a loss of hypothalamic control of sympathetic activity and increases the susceptibility to changes in core body temperature in response to changes in the external environment.

*Management of Autonomic Hyperreflexia*

Management of autonomic hyperreflexia is of a major concern for anesthesia providers. It is the responsibility of that provider to provide the proper care and intervene appropriately with the proper pharmaceutical agent to provide the best outcome for that patient. Management for the prevention of AH does though start at an earlier stage for the patient. In patients with spinal cord injury prevention strategies are important (a) suitable bladder and bowel routines must be maintained, (b) routine monitoring for prevention of pressure ulcer development; these are the most effective measures for prevention of autonomic hyperreflexia. However, for each individual, the identification

and elimination of specific trigger factors for AH must be employed to manage and prevent episodes of AH (Krassioukov & Claydon, 2006). Initially when an episode of AH occurs treatment should involve sitting the patient upright in order to provoke an orthostatic reduction in blood pressure. Throughout this episode blood pressure must be taken in five minute intervals. Following this, interventions must be taken to investigate the precipitating stimulus for AH and to eliminate the cause, which is most commonly bladder distention and fecal impaction in spinal cord injured patients (Mathias & Frankel, 1992). Krassioukov and Claydon (2006), also go on to state, "the use of antihypertensive drugs should be considered as a last resort, but may be necessary if the blood pressure remains elevated after initial interventions to remedy the hypertensive episode" (p. 226).

## General Anesthesia

General anesthesia is a useful anesthetic to prevent episodes of autonomic hyperreflexia from occurring. In fact, a study by Okuyama, Ueda, Morimoto, Okyyama and Kummotsu (1994) retrospectively evaluated the anesthetic management and perioperative complications of sixty nine patients with chronic spinal cord injury for genitourinary procedures. Of the sixty nine cases, thirty eight were performed under general anesthesia. Autonomic hyperreflexia was not seen in any cases with cervical cord injury in which general anesthesia was utilized. Of the occurrences of autonomic hyperreflexia in this study, two were found when spinal anesthesia was utilized and two were found when intravenous sedation and local anesthesia was used. These results note the safety of utilizing general anesthesia for prevention of autonomic hyperreflexia. In an additional study by Lambert, Deane, and Mazuzan (1982) a population of fifty spinal cord injured patients was studied in a retrospective review of seventy eight various

common surgical procedures. After review of these procedures it was found that hypertension, indicating onset of autonomic hyperreflexia, occurred more frequently and was found at a ratio of three to forty-six when compared between the general anesthesia group and the group of topical or no anesthesia respectively. Eltori, Wong, Lacerna, Comarr, and Montroy (1997) also pointed out in a retrospective review of anesthetic methods during surgery that hypertension, indicating onset of autonomic hyperreflexia, was greatest during induction, (the start of anesthesia), when no anesthesia was given as compared to when general anesthesia was given. Krassioukov, Warbuton, Teasell and Eng (2006) found that in two observational studies in spinal cord injured patients which autonomic hyperreflexia was a common complication during general surgery that ninety percent of those individuals undergoing surgery without anesthesia or with local anesthesia developed autonomic hyperreflexia. This compared to the ten percent of those patients undergoing general anesthesia that developed autonomic hyperreflexia. In further support for general anesthesia Raeder & Gisvold, (1986) stated, "A deep general anesthesia is also the recommended anesthetic plan for attenuating or preventing intraoperative autonomic hyperreflexia" (p. 672) Therefore, the aforementioned studies indicate that general anesthesia is a safer route of anesthetic management than no anesthesia or topical anesthesia alone.

## Regional Anesthesia

As mentioned earlier, autonomic hyperreflexia may occur in up to eighty-five percent of patients with spinal cord lesions above the sixth thoracic vertebrae. This said, regional anesthesia has a definite niche within anesthesia services, one of which is to optimize the outcome of those patients afflicted with spinal cord injury and the likelihood

of autonomic hyperreflexia in the perioperative and postoperative periods. Regional anesthesia is used in a variety of ways in anesthesia including spinal, epidural, regional intravenous blocks as well as interscalene blocks.

Spinal anesthesia is a method that is commonly used as a preventative method for autonomic hyperreflexia. King, Johnson, and Wood (1999) state, "the block of the afferent pathways by spinal anesthesia is considered the most effective means of preventing autonomic hyperreflexia"(p. 32). The technique that they outlined is best utilized when an isobaric solution, equal parts anesthetic and cerebral spinal fluid, is instilled into the subarachnoid space. By using an isobaric solution the migration of the anesthetic is usually insignificant and the level of anesthesia is easier to predict and control. They further maintain that of thirteen patients given this type of anesthetic management, not one has had an occurrence of autonomic hyperreflexia. The researchers further indicated that using an isobaric spinal anesthetic is a very useful method of anesthesia to control any possible occurrence of autonomic hyperreflexia.

Epidural anesthesia has been utilized in spinal cord injured patients to treat autonomic hyperreflexia in pregnancy and in the immediate postpartum period (Murphy, McGuire & Peng, 1999). This is done to allay the possibility of autonomic hyperreflexia occurring by the triggering of afferent impulses from noxious stimuli to further stimulate the spinal cord above the level of injury. Perioperatively, epidural anesthesia with the use of local anesthetics and opioids block the afferent and efferent nerves and therefore attenuates the potential for triggering the impulses for autonomic hyperreflexia (Murphy, McGuire & Peng, 1999). By blocking these nerve pathways epidural anesthesia has an important role for the anesthesia provider. This said, anesthesia personnel need to know

the justification that preempts use of epidural anesthesia. The rationale for utilizing epidural anesthesia intraoperatively and postoperatively is (a) it reduces the risk for developing autonomic hyperreflexia during the physiological stressful time during surgery, (b) to reduce the stress response induced upon the body during surgery, (c) to ensure perioperative analgesia and, (d) to enhance treatment of any hypertensive crisis brought on by an episode of autonomic hyperreflexia in the postoperative recovery period (Murphy, McGuire & Peng, 1999). Kuczkowski (2003), also lends support to epidural anesthesia in the perioperative care of obstetric spinal cord injured patients stating that "epidural management and it expeditious induction of labor analgesia may prevent or ameliorate peripartum complications such as autonomic hyperreflexia in laboring spinal cord injured patients" (p. 824). Also lending credence to the use of epidural anesthesia in the laboring patient is that control of autonomic hyperreflexia in laboring patients has been unsatisfactory and patients are placed at higher risk autonomic hyperreflexia occurring if this type of anesthesia is not utilized (Burns & Clark, 2004).

In addition to epidural anesthesia, as described earlier, there are different techniques utilized in anesthesia to minimize risk of autonomic hyperreflexia for the anesthetic provider and patient. Cosman (2005) described the use of a lidocaine anal sphincter block during anorectal surgeries that was investigated in a randomized, double-blinded study. In this study of twenty six patients, thirteen received placebo and thirteen received lidocaine. The results indicated that the mean maximal blood pressure increase was significantly lower, fourteen to twenty-two mmHg in the lidocaine group, as compared to the placebo group where the mean maximal increase in blood pressure was thirty-one to forty-seven mmHg ($p = 0.01$). Thus, the use of a lidocaine anal block

significantly limits the autonomic nervous system response in susceptible patients undergoing this type of procedure.

For patients undergoing shoulder or upper arm surgery, a regional technique can avoid the untoward side effects of autonomic hyperreflexia. An interscalene block, when local anesthetic is injected into the interscalene groove between the anterior and middle scalene muscles of the neck, can be employed to attain anesthesia for the shoulder. It is at this point that the brachial plexus, the bundle of nerves which innervate the shoulder, originate. The local anesthetic injected into this space then anesthetizes the brachial plexus. Habibi, Schmeising, and Gerancher (1999) indicated that the use of interscalene blocks is an effective way to allay the affects of autonomic hyperreflexia in spinal cord injured patients. They further stated when interscalene blocks are used in combination with general anesthesia, a decreased risk for autonomic hyperreflexia both intraoperatively and postoperatively occurs.

## Pharmacological Management

Episodes of autonomic hyperreflexia in individuals with spinal cord injury can vary in severity; from asymptomatic to very difficult to control. Also, it can be incredibly difficult to find the cause of the acute blood pressure elevation. When AH occurs immediate intervention is warranted. Blackmer (2004) recommended, "antihypertensive drugs with a rapid onset and short duration of action should be used in the management of acute episodes of autonomic hyperreflexia" (p. 1210).

One fast acting antihypertensive agent utilized in the treatment of autonomic hyperreflexia is that of Nifedipine. Blackmer (2004), recommended its use as a first line agent in treatment of AH during acute episodes. Nifedipine is a calcium channel blocker

that selectively inhibits calcium ion influx across the cell membrane of cardiac and vascular smooth muscle without changing serum calcium concentrations. This causes decreased peripheral vascular resistance and produces lowering of systolic and diastolic blood pressure. Nifedipine although, has been reported to cause adverse reactions after immediate release nifedipine is given sublingually. This has been reported in patients without autonomic hyperreflexia and has not been reported to cause adverse reactions in the hyperreflexic patient (Blackmer, 2004). In contrast, the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressures (1997) has discouraged the use of Nifedipine due to the adverse affects such as stroke, acute myocardial infarction, death, and in several instances, severe hypotension.

Captopril has also been utilized in the treatment of AH. This medication is a specific competitive inhibitor of angiotension I converting enzyme. In a study of twenty-six patients with spinal cord injury, Captopril given sublingually was effective in lowering blood pressure by a mean of forty mmHG systolically in thirty-three episodes of autonomic hyperreflexia in all twenty-six subjects. Esmail et al., (2002) point out, "Captopril appears to be safe and effective for autonomic hyperreflexia management and its use should be utilized as the primary medication in management of autonomic hyperreflexia" (p. 606). Anton and Townson (2004) also stated, "we rely on first line therapy nonpharmacological interventions but consider captopril as the first choice in those situations where drug therapy is required" (p. 1210).

In addition to the above agents utilized for treatment for autonomic hyperreflexia Nipride or Nitroprusside is also used. Nitroprusside is a potent, rapid acting intravenous antihypertensive agent. Its effect is almost immediate and usually ends when the

intravenous infusion is stopped. The brief duration of the drug's action is due to its rapid biotransformation. The hypotensive effect is augmented by ganglionic blocking agents. Nitroprusside acts independently of autonomic innervation by acting directly on peripheral blood vessels which causes them to dilate. Nitroprusside administered intravenously to hypertensive and normotensive patients produces a marked lowering of the arterial blood pressure, a slight increase in heart rate, a mild decrease in cardiac output, and a moderate diminution in calculated total peripheral vascular resistance (Krassioukov, Warburton, Teasell, & Eng, 2006). Its indication for treatment is acute hypertension that is refractory to standard therapeutic measures. This is sometimes seen in autonomic hyperreflexia when initial lines of treatment do not lower the patient's blood pressure. Intervention with the use of nitroprusside then is often warranted as it has been cited as an effective treatment of the hypertension associated with autonomic hyperreflexia.

In spinal cord injured patients that have episodes of autonomic hyperreflexia another means of controlling hypertension is the use of magnesium. Magnesium controls catecholamine induced hypertensive crisis through inhibiting the release of catecholamines from the adrenal medulla and adrenergic nerve endings. This causes a reduction in systemic vascular resistance by direct vasodilating action on vessel walls as well as direct blockade of catecholamine receptors. Jones and Jones (2002) stated, "using magnesium in the treatment of autonomic hyperreflexia lowers blood pressure on average thirty to forty mmHg and we advocate the treatment and further study of magnesium in the treatment of autonomic hyperreflexia with the use of magnesium sulphate" (p. 436).

## Summary

The review of the literature reveals the need for prompt recognition of the signs and symptoms of autonomic hyperreflexia as well as its treatment protocols. It is essential for the anesthesia provider to be aware of signs and symptoms of AH and its treatments protocols perioperatively as well as postoperatively. Providers must be prepared and act appropriately and with the proper medications so as to ensure the best outcome for the patient under their care. Integral to this care is the awareness of autonomic hyperreflexia and its possible impact on the spinal cord injured patients that anesthesia providers care for.

# CHAPTER III

## METHODOLOGY OF THE INDEPENDENT STUDY

This independent study developed a pamphlet and educational presentation for utilization in the operative setting by anesthesia providers as well as a learning tool for anesthesia students. Following this presentation and after reviewing the pamphlet anesthesia providers were asked to complete a questionnaire to ascertain its usefulness in practice and whether any information may have been omitted.

### Population

The population for which the pamphlet and educational inservice was developed was anesthesia providers and anesthesia students that worked or attended a graduate nursing anesthesia masters program in a city of approximately sixty thousand people within an upper Midwestern rural state. The anesthesia providers worked in a regional level II trauma healthcare center that is licensed for 277 beds including an operating room department that includes 11 operating rooms. The anesthesia students attended a Midwestern graduate nursing degree program with specialization in nursing anesthesia in the same Midwestern city and home base of anesthesia program. The students were in there first year of graduate education in a university setting. Education of the students is preparing them to work in the rural and urban settings of nursing anesthesia that the topic of interest may occur in.

### Methodology

For the independent study this graduate student met with the nursing anesthesia director and assistant director at a major nursing anesthesia program responsible for the education of graduate nursing anesthesia students. The topics discussed at this meeting

were the educational objectives and outcomes as well as the presentation methods that would best fit the audience that would be receiving the presentation. After this meeting and with their additional input a pamphlet and presentation was developed for use by said anesthesia students and providers to be used as a resource for those spinal cord injured patients at risk for autonomic hyperreflexia. After approval of the pamphlet and presentation by the director and assistant director of the anesthesia program the inservice was placed on the educational agenda for the didactic portion of education for 11 first year graduate nursing anesthesia students. After the inservice an evaluation questionnaire was given to those attendees to determine if the presentation was worthwhile for their educational purposes and if the teaching methods of the presenter was beneficial to their learning experience.

The powerpoint presentation outlined the root causes of autonomic hyperreflexia, the signs and symptoms of autonomic hyperreflexia, the pathophysiology of the condition as well as anesthesia management and pharmaceutical management of the condition while in the operative setting. At the end of the presentation the pamphlet developed was distributed for use by those anesthesia providers in attendance. The pamphlet was developed using multiple colors and various sized type fonts to make it easier to find specifics within the pamphlet. Within the pamphlet pathophysiology was explained as well as the precipitants, signs, symptoms and treatment of AH for the anesthesia provider to be utilized in practice.

The nursing anesthesia students were notified of the inservice by the director and assistant director of the nursing anesthesia program and that the presentation and pamphlet were partial requirements for the presenting graduate student's completion of

the nursing anesthesia masters degree program. Contact to the director and assistant director was done via email within the university system to ascertain the time and meeting place of the inservice as well as additional comments on content and delivery methods. Also, at the end of the inservice if they so desired, the anesthesia students could anonymously provide input in the students delivery, content or methods of improving the presentation so that possible successive presentations could be improved.

# CHAPTER IV

# DISCUSSION AND RECOMMENDATIONS

## INTRODUCTION

The purpose of this independent study was to explore the pathophysiology, signs and symptoms of, and treatment interventions for AH, and its resultant after affects of the reflex. In addition, it was done to prepare a presentation and pamphlet for anesthesia providers for use to better their practice in spinal cord injured patients and the possibility of AH occurrence. The ability to have this pamphlet and access to the power point presentation to aid in management of the crisis of AH is discussed with potential implications for nursing practice, education, policy and research.

## The Pamphlet and Presentation

### The pamphlet

The pamphlet begins with the discussion of what autonomic hyperreflexia is and the detrimental side affects of it occurring. The next area of discussion is the pathophysiology of AH. This was intended to facilitate the pamphlet users learning of the pathophysiology of AH and the pathways of the nervous system in which the reflex is involved. Signs and symptoms of the AH are then discussed so that the provider can realize and note changes during the preoperative, perioperative and postoperative periods during surgery. Next, the precipitating factors of the disorder are listed so that any possible event prior to surgery may be noted to provide an improved anesthetic plan by the provider. By noting prior the precipitating factors of AH and the outcomes that may occur because of it, the proper interventions to deal with it can better be plan for such an event. The pamphlet then proceeds to explain the possible interventions that can be

implemented before surgery to prevent any occurrences of AH. The pamphlet also details pharmaceutical interventions to deal with the resultant hypertension that occurs during an episode of AH. This information was included to give the provider a possible pharmacological plan to prevent the untoward effects of the hypertension from AH.

The importance of proper intervention and management of AH during anesthesia and surgery was discussed with two CRNAs, the director and assistant director of a Midwestern nursing anesthesia program, as well as an anesthesiologist. Following these discussions the pamphlet was developed and supplied to the above experts to for review. Input from the graduate students' advisor was also utilized in regards to specific font and color format as well as specific teaching points within the pamphlet. After comments were made on the information within the pamphlet the recommended changes were made and reviewed. Some of the suggestions were prioritization changes on pharmacological interventions, as well as keeping the terms the same within the text. In addition to this specific colors and font sizes were utilized to emphasize certain points within the pamphlet.

Following feedback, the information about AH was formatted into a pamphlet style using a paper size of 11 inches by 17 inches and various sized fonts and colors to emphasize important points within the pamphlet. The wording within the pamphlet was geared for anesthesia providers to inclusively include the aspects about AH that were most important on the recognition and treatment of AH. The content within the pamphlet was lengthy yet concise enough to thoroughly include the important points about AH and its management and treatment.

The presentation

The presentation begins with current case scenarios an anesthetist may encounter during the anesthetic care of a patient with spinal cord injury. After this presentation objectives were explained and reviewed. It was at this point that a general introductory overview of the condition was presented to the participants at the inservice. This was done to give a brief historical background of the discovery of AH and why it is important to anesthesia providers. Next, the problem, purpose and significance of the study were explained to give details about why the study was conducted. It was after this point that the pathophysiology on spinal cord injury and AH was reviewed. This information overviewed the inflammatory response to injury to the spinal cord and the phases of injury. Pathophysiology of AH was then explained with the use of visual aids to enhance the learning of the participants of the inservice. After this the clinical features of AH such as hypertension, bradycardia, headaches, piloerection were elaborated upon so as to better familiarize those who may encounter the condition in their practice. Following the above, management of the condition using pharmacological interventions as well as anesthesia techniques was explained. Finally, in attempt to ascertain the learning involved during the presentation the case studies viewed earlier in the presentation were covered and discussed within the group at the presentation. Also to evaluate learning a posttest was issued to participants and they were allowed 15 minutes to answer questions pertaining to the topic content. The questions were then reviewed at the end of the presentation and the participants were given time after this period to ask any questions that they may have had at this time.

## Project Outcome

The outcome of this project was the development of an educational pamphlet (see Appendix A) and PowerPoint presentation (see Appendix C) for anesthesia providers with an emphasis on current treatment modalities for AH. The pamphlet is a strategy to deliver key concepts and information in regards to cardiac rehabilitation. The initial drafts of the pamphlet were provided to experts to view and to make comments. These comments were then implemented into the final copy. This pamphlet was then provided to future and current anesthesia providers at a presentation utilizing PowerPoint (see Appendix A).

The PowerPoint presentation (see Appendix C) entailed various slides on autonomic hyperreflexia, pathophysiology of autonomic hyperreflexia, spinal cord injury, precipitating factors and pharmaceutical management of the disorder. The slides also included several case scenarios as well as a short post test after the presentation to determine the level of learning involved. To ascertain the usefulness of the presentation to the attendees an evaluation form (see Appendix D) was utilized. Factors included in the tool were, (a) the program content was current, (b) advanced my knowledge, and (c) material presented in an appropriate method. These factors then were rated by, (a) needs improvement, (b) meets expectations, and (c) exceeds expectations. The evaluation tool then allowed attendees to comment on the presentation as well as to list any possible topics that could be used as future learning opportunities. After completing the evaluation tool the forms were then reviewed after collection from the graduate student and specific content was formulated into a table (see Table 1). Comments on the

evaluation forms were: "excellent topic and delivery", "this will useful in my future practice", and "thank you".

After the presentation was given, attendees were provided a posttest of ten questions on the topic of AH. Questions were true and false and covered the topic content that was given in the presentation. After completing the post test (see Appendix B), the questions and answers were reviewed at the end of the presentation. This process enabled attendees to have the correct information regarding AH and to answer any questions that arose after the post test. Questions after the post test sparked debate on specific management styles of general and regional anesthesia. Also discussed was which pharmacological option would be best suited for the hypertension of AH. Following discussion, specific resources were then given to the participants for their further education and reference.

Table 1: Evaluation Form

| Evaluation Table | Needs Improvement | Meets Expectations | Exceeds Expectations |
|---|---|---|---|
| The Program Content Was Current | 0 | 8 | 3 |
| Advanced My Knowledge | 0 | 10 | 1 |
| Material Presented in An Appropriate Manner | 0 | 11 | 0 |

## Implications for Nursing Practice

The major implication for nursing practice is to utilize the findings within the pamphlet and PowerPoint presentation to further educate future and current anesthesia providers regarding the indications of autonomic hyperreflexia, its correct interventions during acute crisis and the implications of incorrect treatment and management of the disorder. Also, knowing the physiology of the reflex and the resultant interrupted pathway of the nervous system after spinal cord injury allows an anesthetic provider to better plan the correct anesthetic plan for the best achievable patient outcome. It is known, according to Bishop (2002), that the prevalence of AH among the spinal cord injured population ranges from sixty-six percent to eighty-five percent of individuals those individuals. This is very important in nursing anesthesia as the amount of surgeries done on spinal cord injured patients ranges around four percent. Within this four percent, eighty five percent will have an occurrence of AH (Bishop, 2002). This said, without this knowledge and education of AH among anesthesia providers AH may go unnoticed and improper care may result. Additionally, through a continued process of education on AH anesthesia providers will have the knowledge necessary to optimize the treatment of their SCI patients in the operative setting. Other areas may also benefit from this education as spinal cord injured patients are common in the emergency room, rehabilitation and long term care settings and the learning objectives and outcome goals can be manipulated to better suit there particular needs.

## Implications for Nursing Education

As anesthesia providers become more educated about AH and the implications that it has in practice education must rely solely upon the educators that provide in-

services for the providers. Ongoing education and expansion of the nurse anesthetist's knowledge base allows for a better understanding and utilization of specific treatment modalities available for SCI patients and the possible occurrence of AH. In order to identify those that can benefit from education of AH it is recommended to ask anesthesia providers what do they know about AH and how currently they would treat it. By looking at ones own practice and the possible knowledge deficits that exist, a provider can increase their knowledge through the process of continuing education. Such efforts can only positively influence their practice. It is important that anesthesia providers giving care to patients be aware of the negative affects of AH and the importance of proper treatment and management of SCI patients. With this knowledge base proper management of SCI patients can result.

This educational independent study can be a small step in informing the anesthesia providers about this sometime fatal condition that develops at times during the operative period. It is a known condition that can have fatal consequences and can occur from a variety of triggers. The knowledge base that this independent study provides can further nursing education and policy by the possible incorporation of it in future anesthesia didactics.

## Implications for Nursing Policy and Research

Follow-up studies to identify the success of education and its impact of outcomes of patients care may be a focus of future nursing research. Research studies that can identify the most beneficial treatment modality for AH may also more precisely predict future patient outcome and treatment as well as further nursing education. There is no doubt that research to identify the best modality in practice for AH would greatly enhance

nurse anesthesia and patient outcome. Future investigations could compare the usefulness of specific pharmacological agents and the affects that they have on AH. By continuing investigations on AH nursing policy and practice can be influenced by the outcomes that further investigations have. These outcomes would influence future anesthesia providers in the way that they practice and provide care for SCI patients. By doing further investigations patient care and optimal patient outcome will result.

## Summary

The purpose of this independent study was to develop an informational pamphlet and educational inservice for utilization by future and current anesthesia providers. The pamphlet and educational inservice is now a resource for those that attended the educational inservice. Its aim to provide consistent information and the possible treatment modalities for AH was explained in the pamphlet as well as in the power point presentation. With the knowledge base that this independent study provides it will further the impact that nurse anesthetists have in the operative setting. For nurse anesthetists provide the majority of anesthetic care in the operating room. By advancing their knowledge and providing the information needed about this condition patient care and outcome will be enhanced.

APPENDICES

APPENDIX A

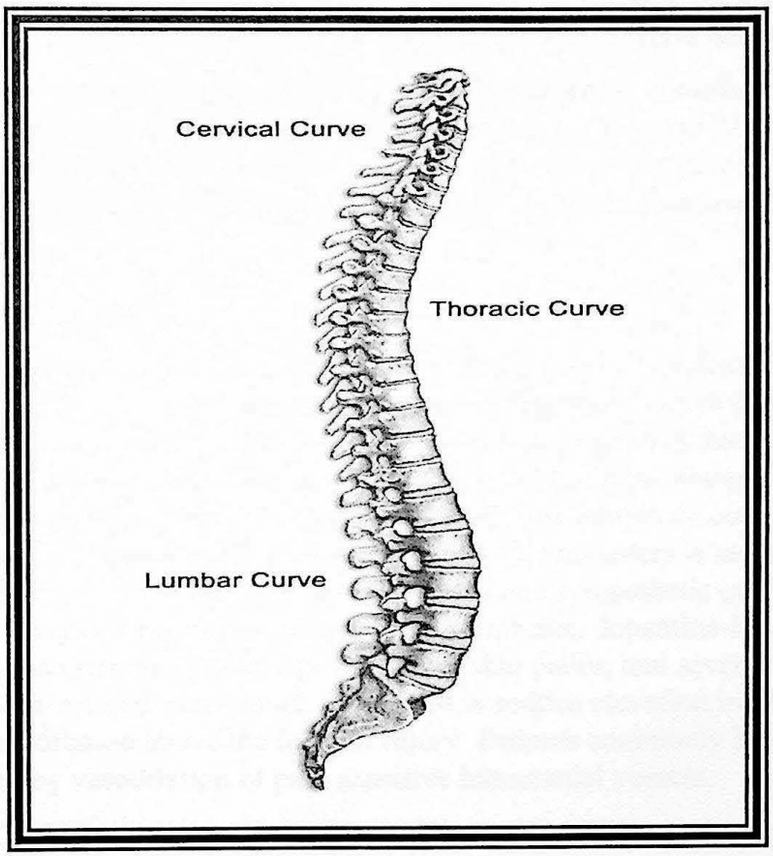
PAMPHLET

# AUTONOMIC HYPERREFLEXIA:

# QUICK REFERENCE PAMPHLET

# FOR IMPROVED

# ANESTHETIC CARE



# IMPORTANT INFORMATION REGARDING AUTONOMIC HYPERREFLEXIA

**Autonomic hyperreflexia (AH)** may occur in up to 85% of patients with spinal lesions above the level of T6. Distention of the bladder, rectum, or cervix; onset of uterine contractions; and surgical stimulation may trigger sympathetic and parasympathetic afferents below the level of the lesion that are not inhibited by supraspinal centers. This can lead to extensive sympathetic stimulation of the cardiovascular system and of the neurologically isolated adrenal medulla. Clinical manifestations of AH include marked hypertension, bradycardia, cardiac dysrhythmias, headache, piloerection, sweating, and flushing above the level of the lesion. The severe hypertension may lead to seizures or fatal cerebral hemorrhage.

**Autonomic hyperreflexia (AH)** is a potentially life-threatening complication in patients with high spinal cord injury that may present during surgery, labor and delivery or postoperatively. The use of either deep general anesthesia or neuraxial blockade to attenuate or prevent intraoperative AH has been utilized to attenuate the reflex.

### Pathophysiology

This phenomenon occurs after the phase of spinal shock in which reflexes return. Individuals with injury above the major splanchnic outflow may develop AH. Below the injury, intact peripheral sensory nerves transmit impulses that ascend in the spinothalamic and posterior columns to stimulate sympathetic neurons located in the intermediolateral gray matter of the spinal cord. The inhibitory outflow above the spinal cord injury (SCI) from cerebral vasomotor centers is increased, but it is unable to pass below the block of the SCI. This large sympathetic outflow causes release of various neurotransmitters (norepinephrine, dopamine-b-hydroxylase, and dopamine), causing piloerection, skin pallor, and severe vasoconstriction in arterial vasculature. The result is sudden elevation in blood pressure and vasodilation above the level of injury. Patients commonly have a headache caused by vasodilation of pain sensitive intracranial vessels.

### Pathophysiology

Vasomotor brainstem reflexes attempt to lower blood pressure by increasing parasympathetic stimulation to the heart through the vagus nerve to cause compensatory bradycardia. This reflex action cannot compensate for severe vasoconstriction. Parasympathetic nerves prevail above the level of injury, which may be characterized by profuse sweating and vasodilation with skin flushing

## Signs and Symptoms

- **Profuse Sweating**
- Flushing, Nasal Obstruction
- **Severe Headache**
- Difficulty Breathing
- Nausea and Vomiting
- **Shivering and Gooseflesh**
- Visual Field Defects
- Blurring of Vision

- ♥ **Severe Acute Hypertension**
- ♥ Bradycardia
- ♥ Arrhythmias
- ♥ **Vasodilation** above the level of the cord injury
- ♥ **Vasoconstriction** below the level of the cord injury
- ♥ Muscle spasm, Visceral Contraction
- ♥ Decreased LOC

**Autonomic hyperreflexia** can lead to pulmonary edema, cardiovascular collapse, retinal and/or subarachnoid cerebral hemorrhage, seizures and **death.**

**Precipitating Factors:** Episodes of AD can be triggered by many potential causes. Essentially any painful, irritating, or even strong stimulus below the level of the injury can cause an episode of AD. Although the list is not comprehensive, the following events or conditions all can cause episodes of AD:

- Bladder distension
- Urinary tract infection
- Cystoscopy
- Epididymitis or scrotal compression
- **Bowel distension**
- **Bowel impaction**
- Gallstones
- Gastric ulcers or gastritis
- **Surgical or diagnostic procedures**
- Invasive Testing
- Hemorrhoids
- Gastrocolic irritation
- **Appendicitis or other abdominal pathology trauma**
- Menstruation
- Pregnancy, especially labor and delivery
- Vaginitis
- Sexual intercourse
- Ejaculation
- Deep vein thrombosis
- Pulmonary emboli
- Pressure ulcers
- Ingrown toenail
- Burns or sunburn

- Blisters
- Insect bites
- Contact with hard or sharp objects
- Temperature fluctuations
- Constrictive clothing, shoes, or appliances
- Heterotopic bone
- Fractures or other traumas
- Pain

## Interventions

Check the patient's blood pressure. If blood pressure is elevated and the person is supine, have the person sit up immediately and loosen any clothing or constrictive devices. Sitting leads to pooling of blood in the lower extremities and may reduce blood pressure. Monitor blood pressure and pulse every 2-5 minutes until they have stabilized; blood pressures can fluctuate quickly during an AD episode from impaired autonomic regulation. Survey the person for instigating causes, beginning with the urinary system, the most common cause of AD. If the individual has an indwelling urinary catheter, check the system along its entire length for kinks, folds, constrictions, or obstructions and for correct placement of the indwelling catheter.

- If the catheter appears to be blocked, gently irrigate the bladder with a small amount of fluid, such as normal saline at body temperature. Avoid manually compressing or tapping on the bladder.
- If the catheter is draining and blood pressure remains elevated, suspect fecal impaction, the second most common cause of AH, and check the rectum for stool using lidocaine jelly as lubricant.
- Use an antihypertensive agent with rapid onset and short duration while the causes of AH are being investigated.
- Monitor the individual's symptoms and blood pressure for at least 2 hours after resolution of the AH episode to ensure that elevation of blood pressure does not recur. AH may resolve because of medication, not because of resolution of the underlying cause.

## Management & Treatment

- **STOP INITIATING STIMULUS!** (if possible)
- General Anesthetic, Spinal anesthesia
- Ganglionic blockers (trimethaphan), direct vasodilators (SNP), direct alpha-agonists (phentolamine),
- Beta-blockers for tachyarrhythmias (esmolol)
- Centrally-acting anti-hypertensives (clonidine) won't work!
- Can decrease risk (or prevent) by use of neuraxial blockade (spinal greater than epidural)

## Management & Treatment

- Dibenzyline- Long-acting, adrenergic, alpha-receptor blocking agent that can produce and maintain chemical sympathectomy by oral administration; increases blood flow to skin, mucosa, and abdominal viscera and lowers both supine and erect blood pressures. It has no effect on parasympathetic system
- Captopril – competitive inhibitor of ACE prevent conversion of Angiotension I to Angiotension II
- Nipride – causes peripheral vasodilation by direct action on venous and arteriolar smooth muscle, reducing peripheral vascular resistance

## Perioperative Implications:

- Difficult to assess height of neuraxial block due to sensory deficits - can result in inadequate coverage (to prevent AH) or too high block
- May require fiberoptic intubation if unstable Cervical spine
- May be difficult to extubate due to compromised respiratory musculature (from initial injury)
- Can occur with foley catheter placement prior to case
- Can occur postoperatively with resolution of block (secondary to pain or distended bladder/rectum)
- If difficulty awakening after severe hypertension, consider cerebral bleed
- Severe bradycardia due to post-op inability to void or defecate. Consider using arterial line, consider CVP/PA catheter if poor cardiac function/large volume changes

***The purpose of this pamphlet is to provide general information and possible treatment interventions for anesthesia providers regarding AH. The content provided is not in anyway intended to be the sole information and interventions possible for acute episodes of AH.***

APPENDIX B

Autonomic Hyperreflexia Posttest

Autonomic Hyperreflexia Posttest
May 2, 2007

- Patients with lesions below the level of T6 commonly have symptoms.
  True or False
- AH only occurs in patients with traumatic spinal cord injury.
  True or False
- Patients with incomplete spinal cord injury can be affected.
  True or False
- Quadraplegics are rarely affected.
  True or False

- Common signs and symptoms of AH include which of the following?
- Nasal Congestion
  True or False
- Hypertension
  True or False
- Tachycardia
  True or False
- Skin flushing above the level of the lesion
  True or False

- Pathophysiology of AH includes?

- AH is caused by reduced reflex activity after synaptic reorganization.
  True or False
- Autonomic imbalance leads to splanchnic vasoconstriction.
  True or False
- Parasympathetic activity increases above the lesion.
  True or False
- Goosebumps can be found below the level of the lesion.
  True or False

- Management of AH includes?

- The first stage of treatment is oral nifedipine.
  True or False
- Laying down eases the symptoms of AH.
  True or False
- 150 mm Hg SBP is normal in patients with SCI.
  True or False
- Death may occur from AH.
  True or False
- Spinal anesthesia is contraindicated in SCI patients.
  True or False

APPENDIX C

Power Point Presentation on Autonomic Hyperreflexia

Slide 1
Autonomic Hyperreflexia:
Current Treatment Modalities in Anesthesia
David Nuelle
May 2, 2007
Case scenario

Slide2
■26 year old male admitted to hospital with severe gluteal ulcers with tunneling noted. Initial blood pressure was 89/65, HR 101, RR 16 Sats 98 %. Needs I & D of ulcers and skin flaps. He sustained a spinal cord injury three years ago to the T10 level leaving him paralyzed. What is your anesthetic plan?
Case scenario

Slide 3
■44 year female in same surgery, Scheduled for cholecystectomy. Spinal cord injury at C6 with complete transection of spinal cord at that level. Has had only cervical stablization surgery and indwelling catheter surgery. During surgery you notice skin flushing, HR 38 BP 188/54. A general anesthesia was used with 2% Sevo with air/O2, you diagnose possible AH what are your interventions?

Slide 4
Autonomic Hyperreflexia
■Presentation Objectives
- Review Pathophysiology of AH.
- Review the mechanisms of spinal cord injury.
- Explain intervention taken to avoid the reflex.
- Explain pharmaceutical methods to intervene with side effects of AH.

Slide 4
Introduction
■AH was first noticed in 1944 by Ludwig Guttman
- Noted a correlation between spinal cord injured pts. & and the effect it had on the cardiovascular system. (Kuczkowski, 2003)
- Common occurrence in patients with spinal cord injury.
- Occurs most often in the perioperative or postoperative setting if SCI patient is having surgery. (Bishop, 2002)

Slide 5
Introduction
■Autonomic hyperreflexia (AH) is a condition that concerns anesthesia providers caring for spinal cord injured patients.
■It is a condition of uncontrolled sympathetic response secondary to a stimulus that usually occurs in spinal cord injured patients where injury is above or at T6.

Slide 6
Introduction
- It is estimated that of spinal cord injured patients that 50-70% with lesions at T6 or above will have at one point experience autonomic hyperreflexia.
- For SCI patients with injury at or below T10 highly unlikely to occur.
- Most prominent component of AH is a dramatic rise in blood pressure.
- Most theories of the cause of this as aberrant sympathetic nervous system over activity without normal regulation from higher brain centers after spinal cord injury.

Slide 7
Problem
- It is a condition where there is a lack of awareness of the condition among health care workers as well as anesthesia providers.
- Without knowledge of the problem, condition may go unnoticed and treated inappropriately leading to fatal complications.

Slide 8
Problem
- Autonomic hyperreflexia is a condition that occurs infrequently among surgical patients nevertheless it does occur and without knowledge of the condition and the proper treatment lethal consequences can result.

Slide 9
Purpose
- To review current literature and current treatment methods for AH.
- To review and state the pathophysiology of the AH.
- To review current treatment protocol with interventions.

Slide 10
Significance
- Is to ultimately provide better patient care to those spinal cord injured patients in which AH may occur.
- With increased knowledge of AH and its treatments practitioners are more able to diagnose and treat it further leading to improved patients outcomes.

Slide 11
Areas of Inquiry
- What is the pathophysiology of AH and its mechanisms of disease process?
- What are the common signs and symptoms of AH?
- What are the current methods of treatment?

Slide 12
Spinal Cord Injury
▪Most patients exhibiting AH have had a spinal cord injury (SCI).
•Either by transection or by tumor invasion
▪At time of injury either by blunt force or compression hemorrhages are seen in the gray matter.
▪As hemorrhages continue edema and necrosis spreads from the central cord to the entire diameter of the cord, 6 – 24 hrs.

Slide 13
Spinal Cord Injury
▪Typically damage to the gray matter occurs in two or three levels of the spinal cord. (Miller, 1991)
▪As injury occurs there is an interruption of nerve conduction fibers isolating the body below the level of injury. (Hambly, 1998)
▪Edema and hemorrhage continue → progressive loss of function r/t disc compression and hypoperfusion of the spinal cord.
▪As edema subsides circulation is reestablished.
•Can lead to reestablished function of some areas.

Slide 14
Spinal Cord Injury
Phases of SCI
•Acute phase
▪Massive sympathetic stimulation and reflex parasympathetic activity, lasting 3-4 minutes.
▪This response causes severe hypertension and reflex bradycardia or tachycardia
▪After this primary response, loss of neurological function below injury causing spinal shock.
•Flaccid paralysis of voluntary muscle and loss of sympathetic tone.

Side 15
Spinal Cord Injury
▪Acute phase cont'd
•Hypotension then occurs as well as bradycardia.
•Flaccid GI tract and bladder→ ileus, urinary retention.

Slide 16
Spinal Cord Injury
▪Chronic phase
•Occurs after 4-6 weeks after the sympathetic tone returns controlling mild hypertensive responses and other stimuli.
•Hallmark sign of chronic phase is when reflex activity returns characterized by spastic motor paralysis with hyperactive tendon reflexes, episodes of AH and some partial return of involuntary bladder function.

•After this time SCI have an increased supersensitivity of cholinergic receptors and catecholamines.

Slide 17
Spinal Cord Injury
▪After injury there is a denervation of nerve cells
•Body's response to this is a proliferation of cholinergic receptors beyond the motor end plates of voluntary muscle fibers.
•Muscle is very sensitive and contracts to maximal degree in response to Ach.
•To achieve a muscle contraction in SCI pts they need 40-50% less Ach then in the non-injured counterparts.

Slide 18
Spinal Cord Injury
▪As contraction continues $K^+$ is suddenly released in SCI pts whereas in normal muscle without injury it is gradual.
•Produces a rapid rise in serum potassium levels.
•This also occurs when Succinylcholine is given as a paralytic for induction and intubation.
•Therefore the use of Sux is only used for the first 24 hours following injury and not used thereafter for risk of hyperkalemia and the possible irreversible ventricular arrthymias and cardiac arrest.

Slide 19
Pathophysiology of AH
▪Autonomic dysreflexia is caused by spinal reflex mechanisms that remain intact despite the patient's injury.
▪A noxious stimulus (i.e., one that might be expected to cause pain or discomfort in a person without spinal cord injury) below the level of the lesion produces an afferent impulse that generates a generalized sympathetic response, which in turn results in vasoconstriction below the lesion.
▪With lesions at or above the T6 level, the splanchnic vascular bed becomes involved, which provides the critical mass of blood vessels required to cause elevation of the blood pressure.

Slide 20
Pathophysiology of AH
▪AH appears following spinal shock in association with return of spinal cord reflexes.
▪Reflex response is initiated by cutaneous or visceral stimulation below the level of spinal cord transection.

Slide 21
Pathophysiology of AH
▪Stimulation ↓ level of SCI initiates afferent impulses that enter the SC below that level→→elicits sympathetic NS activity over the splanchnic outflow tract (T4-T6).

•In noninjured patients reflex modulated by the brain and inhibitory pathways which are used to offset the impulse and maintain homeostasis.

Slide 22
Pathophysiology of AH
▪In SCI pts. the outflow is separated from the inhibitory pathways sent from the brain and CNS leading to generalized vasoconstriction that persists below the level of injury.
•Vasoconstriction and reflex bradycardia is a result of the carotid sinus baroreceptors activity and the lack of an intact neural network to offset the impulse.

Slide 23
Pathophysiology of AH
▪In a person without spinal cord injury, descending central (brain stem) inhibitory pathways would respond to the rise in blood pressure and would modulate the sympathetic response.
▪The injury to the spinal cord prevents such signals from descending to the sympathetic chain.
▪This results in peripheral and splanchnic vasoconstriction followed by development of hypertension. Excessive parasympathetic output (and lack of sympathetic tone) above the level of the lesion results in peripheral vasodilation and is thought to be responsible for the headache, flushing and sweating in the head and neck region and the nasal congestion that characterize this condition. (Dierdorf & Stoelting, 2002)

Slide 24
Pathophysiology of AH
▪In other words
•AH occurs because the impulses that produce vasodilation cannot reach the neurologically intact portions of the spinal cord → ↑BP unimpeded until death/or stroke if not treated.

Slide 25
Pathophysiology of AH

**Stimulus below level of SCI**

↓

**Activation of Preganglionic Sympathetic Nerves**

↓ ≠

**Vasoconstriction** **Vasodilation and Bradycardia**

↓ ↑

**Carotid Sinus**

↑

↓

→ → **Hypertension** → → ↑

Slide 25
Role of Noradrenaline in AH
- Plays a significant role
- Following injury there is a greatly decreased amount of this cathecholamine.
- Thought to be because of the decreased sympathetic activity of the CNS after the SCI
- Causes SCI pts. to have an increased sensitivity to catecholamines.
- At times of AH there is a noted increase of Noradrenaline and other catecholamines causing a greater response in BP as the sensitivity to catecholamines is increased following SCI.

Slide 26
Clinical Features of AH
- The presenting symptoms of AH generally include a bilateral, pounding headache along with sweating above the level of the injury, nasal congestion, malaise and nausea. Blurring of vision is also reported by some patients.
- Signs include flushed, sweaty skin above the level of the lesion and cool, pale skin below this level.
- The main physical finding is elevation of the blood pressure. It is important to remember that resting blood pressure declines after a spinal cord injury, often to the range of 90/60 mm Hg.

Slide 27
Clinical Features of AH
- Be cautious as readings of 120/80 mm Hg might be considered elevated. Systolic blood pressure as high as 250–300 mm Hg and diastolic pressure as high as 200–220 mm Hg have been reported during an episode of AH. Reflex bradycardia secondary to vagal stimulation is often seen, but tachycardia is also common.
- The differential diagnosis includes migraine and cluster headaches, essential hypertension, posterior fossa tumors, pheochromocytoma and toxemia of pregnancy.

Slide 28
Precipitants of AH
- Any noxious stimulus occurring below the level of the injury to the spinal cord can trigger AH.
- Stimulation of the lower urinary tract accounts for most episodes of AH.
- Bladder distension appears to be the precipitant in 75% to 85% of cases.
- Results from a blocked or kinked catheter or insufficient frequency of intermittent catheterization.
- Other urinary tract triggers include infection, urethral distension, instrumentation, stones and testicular torsion.

Slide 29
Precipitants
▪ 2ND most common precipitant of AH is bowel distension due to fecal impaction, which accounts for 13% to 19% of cases.
▪Other potential noxious stimuli in this area include anorectal distension triggered by manual evacuation of stool, instrumentation, hemorrhoids and anal fissures.
▪Other gastrointestinal precipitants include appendicitis, cholecystitis, esophageal reflux, and ulcer erosion or perforation.

Slide 30
Precipitants
▪Cutaneous triggers such as pressure sores.
▪Fractures - long bone of the lower extremity, and hip dislocation.
▪Menstruation, labor and delivery in women.
▪Deep vein thrombosis, pulmonary embolism, sexual activity - including ejaculation.
▪Medications may also induce AH, especially nasal decongestants and potentially misoprostol.

Slide 31
Management
General Anesthesia
•Safely used and has been found to have better efficacy and outcome than when compared to local anesthesia. (Okuyama et al., 1994)
Regional
•Spinal – utilized as it blocks the afferent pathways. Considered the most effective way of preventing AH.
▪Questions arise if you have an adequate block with a spinal cord injured patient as they are unable to ascertain level of sensory block when checking.

Slide 32
Management
▪Spinal Cont'd
•Isobaric spinal considered easier to predict and control for spinal cord injured patients and in controlled study of 13 patients none had an incidence of AH. (King, Johnson, & Wood, 1999)

Slide 33
Epidural
•Utilized to block the triggering afferent impulses from noxious stimuli to further stimulate the spinal cord above the level of injury.

Slide 34
Pharmaceutical Management
▪Nifedipine: sublingually for acute onset use with caution as AMI and CVA can result from sudden hypotension. (Note Joint Committee on HBP) Not to be used. (Anesthsiology & Critical Care Drug Handbook)

•$Ca^{+}$ Channel blocker, selectively blocks calcium ion influx across cardiac and vascular smooth muscle causing ↓ PVR and ↓ SBP and DBP. Dose 10mg SL. (Blackmer, 2003)
▪Captopril: Sublingually for acute onset as well safer to use than nifedipine as does not have the acute hypotensive results. (Anesthesiology & Critical Care Drug Handbook)
•ACE inhibitor: Competive inhibitor of angiotension converting enzyme which prevents conversion of I to II resulting in lower levels of II which causes an increase in plasma renin activity and reduction in aldosterone secretion. Dose12-25 mg SL.

Slide 35
Pharmaceutical Management
▪Nitroprusside: Vasodilator; used when previous attempts to lower BP have failed. 0.5 - 1.5 mcg/min IV initially max dose 10 mcg/kg/min do not use at max level for more than 10 minutes. Cyanide toxicity in pts with liver and kidney disease.
•Causes peripheral vasodilation by direct action on venous and arterial smooth muscle,↓ PVR, ↑CO by ↓ afterload and reduces aortal and L ventricular impedance.
Pharmaceutical Management
▪Magnesium: controls by inhibiting the release of catecholamines from adrenal medulla and adrenergic nerve endings during AH crisis.
•Direct acting vasodilator to vessel walls, also decreases Ach in motor nerve terminals and acts on myocardium by slowing rate of SA node impulse formation and prolonging of conduction time.

Slide 36
Pharmaceutical Management
▪Hydralazine: IV, direct acting vasodilator of arterioles, with little effect on veins, with decreased SVR.
•Dose: 10-20 mg IV dose every 4-6 hours, onset 5-20 minutes.

Slide 37
Methods
▪A thorough literature was done to review the pathophysiology of AH, its precipitating indicators and mechanisms of disease processes as well as the current treatment modalities in practice today.
▪Provide a seminar to current practitioners regarding the findings to provide for them a better insight of AH.

Slide 38
Case scenario
▪44 year female in same surgery, Scheduled for cholecystectomy. Spinal cord injury at C6 with complete transection of spinal cord at that level. Has had only cervical stabilization surgery and indwelling catheter surgery. During surgery you notice skin flushing, HR 38 BP 188/54. A GETA was used with 2% Sevo with air/O2, induction was non-eventful, you diagnose possible AH what are your interventions? What are your plans other than a GETA for this patient?

Slide 39
Case scenario
■26 year old male admitted to hospital with severe gluteal ulcers with tunneling. Initial blood pressure was 89/65, HR 101, RR 16 Sats 98 %. Needs I & D of ulcers and skin flaps. He sustained a spinal cord injury three years ago to the T10 level leaving him paralyzed. What is your anesthetic plan?

Slide 40
Questions?
True or False
■Patients with lesions below the level of T6 commonly have symptoms.
False
■AH only occurs in patients with traumatic spinal cord injury.
False
■Patients with incomplete spinal cord injury can be affected.
True
■Quadraplegics are rarely affected.
False

Slide 41
Questions?
True or False
■Common signs and symptoms of AH include which of the following?
▪Nasal Congestion
True
▪Hypertension
True
▪Tachycardia
False
▪Skin flushing above the level of the lesion
True

Slide 42
Questions?
True or False
■Pathophysiology of AH includes?

▪AH is caused by reduced reflex activity after synaptic reorganization.
False
▪Autonomic imbalance leads to splanchnic vasoconstriction.
True
▪Parasympathetic activity increases above the lesion.
True
▪Goosebumps can be found below the level of the lesion.
True

Slide 43
Questions?
True or False
▪Management of AH includes?

▪The first stage of treatment is oral nifedipine.
False
▪Laying down eases the symptoms of AH.
False
▪150 mm Hg SBP is normal in patients with SCI.
False
▪Death may occur from AH.
True
▪Spinal anesthesia is contraindicated in SCI patients.
False

Slide 44
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APPENDIX D

Evaluation Form

Altru Health System

## EDUCATION EVALUATION FORM

Thank you for taking time to fill out this evaluation. Your input will help us in planning future educational programs.

Program Title:_____ Date:_____

Speaker:_____ Location:_____

**Please rate the following:** (NI = Needs Improvement; M = Meets Expectations; E = Exceeds Expectations)

| | | | |
|---|---|---|---|
| The program content was current..... | ☐ NI | ☐ M | ☐ E |
| Advanced my knowledge..... | ☐ NI | ☐ M | ☐ E |
| Material presented in an appropriate method..... | ☐ NI | ☐ M | ☐ E |

Topics for future programming:_____

Comments:_____

_____

THIS FORM MUST BE COMPLETED **DURING** THE PROGRAM AND **returned to the program coordinator.**

Name_____ Ext._____

W6012-3174 NOV 05

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