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Sympathetic Storming After Severe Traumatic Brain Injury

Denise M. Lemke, MSN, APNP-BC, CNRN

Brain injury is one of the most common types of traumatic injury. In critical care units, patients with moderate to severe brain injury are often intubated and sedated in an effort to diminish the workload of the brain. Agitation or restlessness is common in these patients and can be associated with fever, posturing, tachycardia, hypertension, and diaphoresis. This exaggerated stress response, known as *sympathetic storming,* occurs in 15% to 33% of patients with severe traumatic brain injury who are comatose (score on Glasgow coma scale [GCS] ≤ 8). Sympathetic storming can occur within the first 24 hours after injury or up to weeks later.[1] The precise mechanism for the increase in activity of the sympathetic nervous system is unknown, but the increased activity is thought to be a stage of recovery from severe traumatic brain injury.[2]

Terms used to describe this phenomenon in published reports include dysautonomia,[3-5] paroxysmal autonomic instability with dystonia,[6] paroxysmal sympathetic storms,[7,8] autonomic dysfunction syndrome,[9] and diencephalic seizures.[7] Sympathetic storming has been associated not only with traumatic injuries but also with tumors,[7,9] hydrocephalus,[2,7] hypoxia,[4] and subarachnoid hemorrhage.[10-12] Multiple medications have been used to treat such episodes, although no definitive treatment protocol exists.

Signs and symptoms of sympathetic storming include posturing, dystonia, hypertension, tachycardia, pupillary dilatation, diaphoresis, hyperthermia, and tachypnea.[1-15] The episodes appear unprovoked and can last for hours or end abruptly. Sympathetic storming often occurs after discontinuation of administration of sedatives and narcotics in the intensive care unit (ICU).[3,7,13] This article reviews the pathophysiology of sympathetic storming, variations in signs and symptoms, potential treatment options, and education of patients' families and concludes with a case report.

CE Continuing Education

* This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Identify the causes of agitation in brain-injured patients
2. Describe the pathophysiological process of sympathetic storming
3. Discuss the current medical management pertaining to sympathetic storming

Author

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Pathophysiology

Sympathetic storming is theorized to be an increase in activity of the sympathetic nervous system created by a disassociation or loss of balance between the sympathetic and parasympathetic nervous systems (Table 1).[16] Theories on the specific mechanism of dysfunction include

Table 1 Effects of the parasympathetic nervous system and the sympathetic nervous system

System/function	Parasympathetic	Sympathetic
Cardiovascular	Decreased cardiac output and heart rate	Increased contraction and heart rate; increased cardiac output
Pulmonary	Bronchial constriction	Bronchial dilatation
Musculoskeletal	Muscular relaxation	Muscular contraction
Pupillary	Constriction	Dilatation
Urinary	Increased urinary output; sphincter relaxation	Decreased urinary output; sphincter contraction
Gastrointestinal	Increased motility of stomach and gastrointestinal tract; increased secretions	Decreased motility of stomach and gastrointestinal tract; decreased secretions
Glycogen to glucose conversion	No involvement	Increased
Adrenal gland	No involvement	Release epinephrine and norepinephrine

loss of cortical control,[4,6] dysregulation of autonomic balance,[1] and/or disruption of relay mechanisms.[7]

Sympathetic activity elicits an adrenergic receptor interaction that can be inhibitory or excitatory. The specific response of the target organ is determined by the category of epinephrine or norepinephrine receptor (α_1, α_2, β_1, and β_2) being stimulated (Table 2).[16] Normally the parasympathetic nervous system dampens the effects of increased activity of the sympathetic nervous system and returns the body to homeostasis. In sympathetic storming, this feedback does not occur and the individual is in an uncontrolled state of stress.

Clinical Presentation

Episodes are frequently unprovoked, catapulting a patient into a state of agitation, extreme posturing/dystonia, tachycardia, tachypnea, hypertension, diffuse diaphoresis, and hyperthermia within seconds. Signs and symptoms vary from episode to episode and from individual to individual.

Baguley et al[4] suggest that these episodes have 3 different phases. During phase 1, which lasts about a week, patients are asymptomatic while sedated or receiving paralytic agents. In phase 2, episodes of sympathetic storming occur with a mean duration of 74 days after injury. The end of this phase is defined by the cessation of diaphoresis. In phase 3, no further episodes of persistent dystonia or spasms occur. Because discontinuation of sedatives and narcotics is a common trigger, one could speculate that the episodes have only 2 phases and that the sedatives and narcotics were effectively preventing the episodes in what Baguley et al called phase 1.[3,7,13]

Triggers, events that immediately precede an episode, may include suctioning, repositioning, environmental sensory stimulation (alarms, equipment), or fever.[13] Identification of a trigger allows the patient to be pretreated in an effort to reduce the length of the episode, lessen its intensity, or even abort the episode.[13]

Differential Diagnosis

Strum[1] defined storming as a diagnosis of exclusion in patients who had recurrent spontaneous episodes of tachycardia, hypertension, and hyperthermia. Baguley et al[4] required that 5 of 7 clinical features (tachycardia, hypertension, tachypnea, hyperthermia, dystonia, posturing, and diaphoresis) be present

Table 2 Sympathetic (adrenergic) receptor interactions

	α_1 receptor	α_2 receptor	β_1 receptor	β_2 receptor
Norepinephrine	Smooth muscle, hypothalamus	Nerve endings, stomach, hypothalamus	Heart, fat cells, kidneys, brain (posterior lobe of pituitary gland)	No interaction
Epinephrine	Smooth muscle	No interaction	Heart, fat cells, kidneys	Lungs, arterioles, stomach, liver or pancreas, uterus, skeletal muscle
End effect	Vasoconstriction, elevated blood pressure, mydriasis, decreased ability to defecate and/or urinate	Vasodilatation, lowers blood pressure, constipation	Increases heart rate, increases cardiac output and force of contraction, increased conduction, lipolysis, release of renin, release of antidiuretic hormone	Relaxation of smooth muscle (vasodilatation, bronchodilatation, constipation), increased glucose production and insulin release, contraction of skeletal muscle

Table 3 Ranchos Los Amigos scale

Level	Description
I	No response to visual, verbal, tactile, auditory, noxious stimuli
II	Generalized response
III	Localized response
IV	Confused-agitated
V	Confused-inappropriate
VI	Confused-appropriate
VII	Automatic-inappropriate
VIII	Purposeful and appropriate
IX	Purposeful and appropriate (standby assistance on request)
X	Purposeful and appropriate (modified independent)

before storming could be diagnosed. For diagnosis of sympathetic storming, Blackman et al[6] required that signs and symptoms occur a minimum of 1 cycle per day for 3 consecutive days in a patient with severe brain injury (level on Ranchos Los Amigos Scale ≤ IV; Table 3). Symptoms include body temperature of 38.5°C or greater, systolic blood pressure greater than 140 mm Hg, pulse rate of at least 130 beats per minute, respiratory rate of at least 20 breaths per minute, agitation, diaphoresis, and dystonia.[6]

Documentation of elevated serum levels of epinephrine or catecholamines (sampling needed before and during episode) can confirm the suspicion of sympathetic storming, although the diagnosis is generally based on clinical examination only. No specific location of injury or pattern of neuronal injury is apparent on radiographs, although sympathetic storming is more common in patients with diffuse axonal injury.[1,3,7] Seizures were once considered a potential cause of sympathetic storming. Do et al,[8] however, reported a case study in which electroencephalography (EEG) was performed on a patient experiencing an episode. The initial EEG showed delta and theta waves without any epileptic waveform, thus confirming that the episode was unrelated to seizure activity.[8]

Even though diagnostic tests that can confirm the diagnosis of sympathetic storming are not available, further investigation into the origin of these episodes is required.[1,2,6,7] Episodes can indicate an acute change in neurological status related to an intracranial source (new or expanding lesion or edema),[1,6] seizures,[6] thyroid storming,[1] deep vein thrombosis or pulmonary emboli,[15] infectious processes,[6,11,15] neuroleptic malignant syndrome,[1,2,6] malignant hyperthermia,[2,6] central fever,[6,11,15] and drug or alcohol withdrawal.[6] Careful assessment is needed to determine the appropriate workup.

Potential Adverse Effects of Untreated Sympathetic Storms

Untreated sympathetic storming increases the risk of secondary injury to the brain.[1,4] Decreases in cerebral tissue oxygenation occur as a result of the physiological impact on the body's systems. Prolonged hypertension, arrhythmias, hyperglycemia, hyperthermia due to elevated metabolic rate, and hypernatremia from severe diaphoresis occur as a result of the sympathetic storm.

If the patient sustains uncontrolled hyperventilation, decreases in cerebral oxygenation occur because of vasoconstriction. During acute episodes, intravenous administration of sedatives or narcotics can provide immediate relief if the patient is receiving mechanical ventilation. In patients who are not receiving mechanical ventilation, additional dosing with enteric oxycodone or intravenous morphine can be used to abort the episode if care is taken to protect the airway.

Hypertension and arrhythmias are associated with storming episodes. Prolonged hypertension increases the risk of secondary injury of the brain due to increased blood flow leading to edema, risk of rebleeding, and potential cardiac dysfunction related to prolonged stress on the heart. In general, acute hypertension is not treated because it is a compensatory response. If persistent hypertension is noted, the degree of treatment or whether antihypertensive medications are instituted depends on the physician. Generally, long-term antihypertensive therapy is not needed.

Common arrhythmias include bradycardia, ectopic beats and irregular rates, atrial fibrillation, and supraventricular tachycardia.[10,12,17] Ectopic beats may be multifocal, preventricular, or nodal in origin. Ectopic beats, bradycardia, and irregular heart rates in patients with traumatic brain injury are generally not associated with clinical signs of hemodynamic instability.[17] Arrhythmias require treatment only if they are symptomatic or life threatening (eg, supraventricular tachycardia, atrial fibrillation).[12] Prolonged increases in sympathetic activity also place patients at risk for myocardial infarction.[1,9] Myocytolysis and

contraction band necrosis of the heart have been reported on autopsy in patients with severe traumatic brain injury.[10]

Neurogenic pulmonary edema may occur if circulating catecholamines cause massive fluid shifts that overload the pulmonary system.[10] Signs and symptoms of neurogenic pulmonary edema are similar to those for adult respiratory distress syndrome but can be differentiated radiographically. In general, radiographic changes in neurogenic pulmonary edema are located from mid-lung to apex rather than in the base of the lungs, as noted in adult respiratory distress syndrome.[10]

Table 4 Medications used to treat sympathetic storming

Medication	Classification	Common use	Adverse effect/ contraindications
Morphine sulfate, fentanyl, oxycodone	Opiate agonist	Analgesia	Sedation
Bromocriptine	Dopamine agonist	Lactation suppression, infertility, prolactin-secreting pituitary tumors	Seizures, use caution in patients with renal or hepatic disease
Carbidopa/levodopa		Parkinson disease	
Propranolol	Nonselective β-adrenergic antagonist	Hypertension	Hypotension, use caution in patients with asthma or bronchial disease
Clonidine	α_2-adrenergic agonist	Hypertension	
Labetalol	Nonselective β agonist Selective α_1 adrenergic antagonist	Hypertension	
Midazolam, diazepam, clonazepam	GABA-A agonist	Sedation	Sedation
Baclofen (oral/intrathecal)	GABA-B agonist	Spasms	Sedation
Chlorpromazine	Dopamine antagonist	Malignant hyperthermia	Sedation, lowers seizure threshold, extrapyramidal side effects
Phenytoin, carbamazepine	Anticonvulsant	Seizures	Sedation
Dantrolene	Other	Muscle relaxation	Hepatic disease

Abbreviation: GABA, γ-aminobutyric acid.

Increased metabolic rate elevates core body temperature, elevates blood sugar level, and increases the risk of muscle wasting and weight loss. In patients with traumatic brain injury, the most common cause of hyperthermia is infection. Fever workup and maintenance of normothermia are essential. Blood sugar levels should be tightly controlled by using a sliding scale for insulin or an insulin infusion to maintain normal levels. Increased metabolic rate and diaphoresis can lead to hypernatremia, renal insufficiency, and thickening of pulmonary secretions. A dietary consultation is important to determine the patient's requirements for energy and free water and to maintain appropriate levels. Careful monitoring of weight, input and output, serial measurements of serum levels of sodium, glucose, creatinine, and blood urea nitrogen, and findings on chest radiographs are necessary to prevent associated problems (muscle wasting, pressure sores and decubitus ulcers, renal failure, atelectasis, and pneumonia).

Treatment

Medical management of sympathetic storming focuses on treating the signs and symptoms in order to reduce the potential adverse effects of prolonged activity of the sympathetic nervous system. The choice of medications depends on the practitioner, and an effective dose is often defined through trial and error. Frequent adjustments may be required to provide adequate control of signs and symptoms. Medications that depress the central nervous system, thus suppressing the sympathetic nervous system, are most commonly used. Opiate receptor agonists, dopamine agonists, β-blockers, α-blockers, γ-aminobutyric acid (GABA) agonists, and sedatives all have been used (Table 4).

In the ICU, intravenous medications (eg, morphine, fentanyl, midazolam) are first-line drugs used to

control these episodes. Although intravenous medications offer rapid control, dosing can be extreme, thus placing the patient at greater risk for respiratory depression. Enteric medications are added to facilitate long-term management of signs and symptoms.

A frequently used medication regimen is bromocriptine and oxycodone.[13] Bromocriptine, a dopamine receptor agonist, acts at the hypothalamic level, lowering the temperature threshold, diminishing diaphoresis, and lowering the blood pressure.[1,9,11] Dosing starts at 2.5 to 5 mg every 8 hours and may be adjusted up to 30 or 40 mg daily.[1,2,11] Oxycodone, an opiate agonist, also has demonstrated effectiveness in treating episodes.[1,5,7,13,17] Scheduled dosing provides a steady serum level and can begin with 5 mg every 4 hours. Supplemental oxycodone may be required, and an order for an additional 5 mg every 4 hours as needed is recommended. If multiple additional doses are required, the dose can be increased to 10 mg every 4 hours. Medications containing acetaminophen should be avoided to diminish the risk of acetaminophen overdosing.

If the episodes are associated with severe hypertension and tachycardia, or if oxycodone and bromocriptine do not provide control, β-blockers and an α-blocker can be added.[13] Propranolol, a nonselective β-blocker, dampens sympathetic activity, thus slowing neuronal activity.[1,15] It also decreases serum levels of catecholamines,[5] reduces cardiac workload,[5,15] and inhibits central fevers by acting directly within the central nervous system.[15] Dosing starts at 10 mg twice a day and is adjusted upward with doses as high as 640 mg per day reported.[1] Bradycardia and hypotension can occur with propranolol. Caution is advised in patients with asthma or bronchial disease.

If propranolol is ineffective, clonidine or labetalol can be added.[1] Clonidine, an α_2 agonist, lowers circulating levels of norepinephrine and epinephrine, and labetalol acts as a β_1-, β_2-, and α_1-blocker.[1]

Extreme hyperthermia can be treated with chlorpromazine, a dopamine antagonist, that can be given intravenously, intramuscularly, or enterically to reduce the core temperature rapidly.[1,3,9] Chlorpromazine in low doses suppresses hypothalamic vasomotor tone, which reduces body temperature and blocks piloerection.[1,9] Given the anticholinergic activity of this medication and the risk of extrapyramidal effects, long-term use is not recommended.[1,9] Acetaminophen and hypothermia blankets are used in conjunction with chlorpromazine to control body temperature. Hyperthermia prolongs episodes of storming; thus maintaining normothermia may lessen frequency or severity of episodes. Fever workup is necessary to rule out meningitis, pneumonia, urinary tract infections, and deep vein thrombosis.

Dantrolene is added if contractures or persistent dystonia are noted.[1,2,4,6] Dantrolene suppresses the release of calcium, thus promoting relaxation of skeletal muscle, which may help to control hyperthermia.[1] Dantrolene can reduce somatosympathetic spinal reflex activity, which would in turn have an inhibitory effect on overall sympathetic activity.[6]

Other medications have been reported to treat the symptoms of sympathetic storming. Baclofen, a GABA-B agonist, has been successfully used intrathecally to control the episodes, although the precise mechanism is unknown.[5,14] The intrathecal route reduced the sedation associated with enteric baclofen but required surgical placement of the pump. Its use has been limited to Europe.[5,14] Scott et al,[9] in a single study, reported success of carbidopa/levodopa, a dopamine agonist, in treating autonomic dysfunction in a patient with "locked-in" syndrome. Cyclopropyl derivatives of oxymorphone (naltrexone) can assist in the control of sympathetic storming.[1] Medications with inconsistent or no response include antiepileptic drugs (phenytoin, phenobarbital, and carbamazepine)[4,7] and β_1-blockers (metoprolol, atenolol).[6] Theoretically, any medication that suppresses activity of the sympathetic nervous system can be used.

Education of Patients' Families

The patient's family may perceive the abrupt onset of sympathetic storming as a sign of worsening clinical status. Care must be taken to assure the family that sympathetic storming can be a normal result of brain injury. Ideally the family should be educated before they witness an episode. An educational tool (Table 5) would be helpful for preparing patients' families. Correct terminology should be used with explanations. The sheet should review pathophysiology, methods of diagnosis, treatment, and ways for family members to assist.

The informed family can alert nursing staff to the episodes, provide tips to identify triggers, and assist the heathcare team with treatment.

Table 5 Educational tool for sympathetic storming

Your brain has 2 centers called the *sympathetic (your "get up and go" system)* and the *parasympathetic (your relaxation system)* systems that keep your body at a steady level of functioning (homeostasis). When there is stress, the *sympathetic* system releases chemicals that provide the body with the needed support to respond to the stress. This is called your "fight or flight" response.

The body's response to sympathetic release of chemicals:

- Increase in heart rate (tachycardia)
- Increase in blood pressure (hypertension)
- Elevation of temperature (hyperthermia)
- Increase in breathing rate (tachypnea)
- Increase in muscle tone (dystonia)
- Pupils dilate
- Sweating (diaphoresis)
- Slowing of bowel and bladder activity

The *parasympathetic* system is responsible for "calming" this response and returning you to a normal state of homeostasis.

Occasionally in individuals who suffer traumatic injury to the brain, there may be episodes when the individual appears to be having a stress response. The heart will race, breathing becomes rapid and shallow, muscles become tight and rigid, they will sweat profusely, their temperature shoots up, and they look very uncomfortable or "stressed." There is not a clear explanation for these episodes, but it is thought that the *sympathetic* system overreacts, leading to a stress response; there is a lack of response of the *parasympathetic* system to return to a normal state of homeostasis, or a combination of the two.

These episodes can occur without warning or appear to occur spontaneously. The symptoms, as well as the duration of the episode, can be unpredictable. That is why the nurses refer to this abnormal stress response as *neuro storming* or *sympathetic storming.* It commonly appears as medications used to sedate and control pain are discontinued.

Treatment is aimed at controlling the symptoms, decreasing the frequency of the episodes, or stopping the episodes. The nurses will also try to identify "triggers" or activities that cause an episode. By identifying a trigger, the nurse can pretreat the individual before the activity or attempt to avoid the activity.

These episodes may start after your family member has been transferred to the general neurological ward. The episodes do not warrant return to the intensive care unit (ICU). The storming episodes can generally be controlled with careful adjustment of medications and care aimed at "calming" the storm. The medications used are aimed at slowing the *sympathetic* response or acting as the *parasympathetic* system.

Family can help by helping to identify triggers, alerting the nursing staff when an episode occurs, and providing calming activities (massage, relaxing music, conversation, placing cool cloths on the family member's forehead). If any of these activities cause an episode, they should be avoided. Identifying the right combination of medications and activities that help "tone" down the episodes takes time. Medications and activities need to be adjusted on the basis of your family member's response to the treatment. Generally over time the *sympathetic* and *parasympathetic* systems return to normal or a modified state and the medications can be slowly discontinued.

When the episodes occur, family members can use cool cloths, massage, quiet conversation, and soothing music. These activities provide the family with a means to help provide care for the patient and lessen the inevitable feelings of helplessness experienced by families dealing with traumatic brain injury. The following case report provides an overview of the acute management of a patient with sympathetic storming.

Case Study

Scott, a 24-year-old man, was an unrestrained driver in a motor vehicle accident that required prolonged extrication. At the scene, his pupils were fixed and dilated. His GCS score was 5 (eye opening = 1, verbal responsiveness = 1, motor responsiveness = 3), as demonstrated by no eye opening or speech and weak mixed posturing. He was intubated in the field. In the emergency department, Scott was intermittently localizing to painful stimuli, pupils were equal and reactive, there was no eye opening or speech, and his GCS score was 7 (eye opening = 1, verbal responsiveness = 1, motor responsiveness = 5). An initial computed tomography scan showed a large right-sided subdural hematoma with a significant shift from right to left. He was taken to the operating room for emergent evacuation of the subdural hematoma and placement of an intracranial pressure monitoring bolt. Intracranial pressure range was from 5 to 40 mm Hg, with a rapid increase in pressure with any activity and a fever spike to 39.4°C on the evening of admission.

An intravenous infusion of midazolam was started (2-4 mg/h) with fentanyl 25 to 50 µg administered intravenously every hour as needed

for treatment of spikes in intracranial pressure. Findings on neurological examination remained unchanged with a GCS score of 6. Within 48 hours, the intracranial pressure had stabilized. At this time, Scott's neurological status fluctuated from a GCS score of 7 (localizing) to a GCS score of 8 (following commands). The monitoring of intracranial pressure and the midazolam infusion were discontinued.

Scott was having nonstimulated episodes of tachycardia (120-150/min), hypertension (150-210/80-110 mm Hg), increased posturing, and diaphoresis consistent with sympathetic storming. Increased heart rate and blood pressure responded temporarily to high doses of intravenous fentanyl as needed (1300 µg/24 h) and midazolam (35 mg/24 h).

Storming episodes continued and administration of 12.5 mg of metoprolol twice daily was started. A magnetic resonance imaging study on day 3 showed a small amount of bleeding in the left frontal area, ischemic changes in basal ganglia on both sides, and ischemic lesions in the occipital lobe on both sides. An EEG showed diffuse intermittent slowing (greater on left side than right) and no epileptic activity. No improvement was noted with administration of metoprolol. As a result, clonidine 0.1 mg twice daily was added on hospital day 4.

A tracheostomy was performed on hospital day 6, and Scott was then weaned off of ventilatory support without difficulty. He followed commands intermittently but continued to have the storming episodes. Fentanyl as needed (625 µg/24 h) and midazolam (10 mg/24 h) lessened the response, although nurses observed that the effect was transient. Even though the EEG did not show epileptic activity, the team thought that silent seizures could not be ruled out, and phenytoin was started at 150 mg twice a day. Clonidine was increased to 0.1 mg 3 times a day and 5 to 10 mg of oxycodone was given every 4 hours as needed.

Scott's family was initially distraught over the storming episodes, which escalated when he was transferred to the general neurological ward. Frequent updates were provided to discuss the cause of the episodes, medication changes, the frequency of episodes, alternative treatments, and Scott's response to treatment. His family was encouraged to assist in the monitoring of episodes and in the use of calming techniques and cooling baths.

On day 8 after the injury, Scott was transferred to the general neurological ward after he was successfully weaned off of mechanical ventilator support. Upon transfer, the metoprolol dosage was increased to 25 mg twice daily, and the bromocriptine dosage was increased to 5 mg every 8 hours. The evening after transfer, Scott had a prolonged storming episode during which the nursing staff was able to provide only momentary relief with positioning, a cooling mattress, acetaminophen, and supplemental oxycodone. The episode was aborted after 10 mg intramuscular morphine sulfate was administered (per physician's order).

The rehabilitation service was consulted, and their recommendation was to increase the dose of bromocriptine to 10 mg every 8 hours and to discontinue the metoprolol while adding 20 mg of propranolol twice daily. At that time, Scott was having daily temperature spikes (39.7°C to 40.2°C), which appeared to aggravate the storming episodes. Cultures were negative for bacterial infection, and a chest radiograph showed no findings indicative of pneumonia or consolidation. Acetaminophen was used in conjunction with a cooling blanket to treat the temperature spikes. The storming episodes continued, although they were less frequent and shorter than before. On day 13 after the injury, the dosage of propranolol was increased to 20 mg every 8 hours.

Scott continued to follow commands intermittently (he stuck out his tongue on command and squeezed, grasped, and released his left hand). He was able to track the individuals in his environment. He exhibited increased flexor tone in his left upper extremity and increased extensor tone in his right upper extremity. His pupils were equal and reactive to light and, although he opened his eyes spontaneously, he made no attempts to speak. His GCS score was 11 (eye opening = 4, verbal responsiveness = 1, motor responsiveness = 6). The storming episodes appeared to have stabilized by this time, and Scott was transferred to a subacute rehabilitation facility on day 21 after injury.

At 7-month follow-up, Scott remained in the subacute facility. Neurological examination showed him to be alert with a flat affect, oriented times 4 with poor short-term memory and fluent speech. He followed commands and moved all 4 extremities (strength grade right 4/5; left 3/5; he remained wheelchair-dependent because of coordination deficits). Scott also exhibited a total homonymous hemianopic defect on

the left side and a partial homonymous hemianopic defect on the right side. His score on the Glasgow Outcome Scale was 3 (conscious but disabled/dependent for daily support). No further storming episodes had been noted and current medications included methylphenidate, fluoxetine, and enoxaparin.

Summary

Patients with sympathetic storming must be treated promptly. Intravenous medication can provide immediate control, although the effect is generally temporary, and dosing can be extreme, thus placing the individual at greater risk for respiratory depression. These patients already have significant cerebral compromise and must be treated promptly to ensure optimal recovery.

The onset of sympathetic storming should trigger the institution of scheduled enteric medications to provide continuous dampening of activity of the sympathetic nervous system. Multiple medications may be required, as well as a period of trial and error, before the correct medication(s) and/or dosages are determined. An effective starting point is the use of scheduled oxycodone, bromocriptine, and if hypertension is present, propranolol. If hypertension and other signs and symptoms do not improve, clonidine can be added or doses can be adjusted.

The ultimate goal is rapid control of the signs and symptoms of excess activity of the sympathetic nervous system to prevent the secondary complications of prolonged stress and to facilitate rehabilitation. Each case requires individual dosing based on signs and symptoms and response to the medication.

The nurse plays a vital role in the supportive care of patients with a severe traumatic injury and is a key player in the diagnosis and management of sympathetic storming (especially in the ICU). Initially the use of sedatives and narcotics for cerebral protection can prevent signs and symptoms of sympathetic storming, and the onset of episodes frequently coincides with weaning of patients off of these medications or with the discontinuation of these medications. The nurse can be instrumental in the coordination of intravenous and enteric medications, avoiding the adverse effects of sympathetic storming, and identifying triggers so that patients can be transferred to the general neurological ward.

Long-term use of these medications is not warranted. Generally weaning patients off the medications, one medication at a time, occurs during the rehabilitation phase. The precise timing varies, as does the decision about which medication to eliminate first.

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CE Test

Test ID C0712: Sympathetic Storming After Severe Traumatic Brain Injury

Learning objectives: 1. Identify the causes of agitation in brain-injured patients 2. Describe the pathophysiological process of sympathetic storming 3. Discuss the current medical management pertaining to sympathetic storming

1. In traumatic brain injuries, agitation and restlessness can be associated with which of the following?
a. Fever, posturing, and diaphoresis.
b. Seizures, bradycardia, and hypotension
c. Hypertension, tachypnea, and dry skin
d. Bradycardia, hypertension, and seizures

2. Which of the following have been associated with sympathetic storming?
a. Tumors and subarachnoid hemorrhage
b. Diffuse axonal injury and arteriovenous malformation
c. Subdural hemorrhages and stroke
d. Diffuse axonal injury and tumors

3. When does sympathetic storming most often occur?
a. After administering premedication for nausea and phytoin
b. After a craniotomy for evacuation of the hemorrhage
c. After discontinuing sedatives and narcotics
d. After discontinuing antiepileptic medications

4. Which of the following best describes the pathophysiology of sympathetic storming?
a. An increase in sympathetic responses in the brain creating faster synapse responses
b. A decrease in the sympathetic responses in the brain creating disassociation between synapse rates
c. Altered levels of dopamine creating excitatory responses
d. A disassociation between the sympathetic and parasympathetic nervous system

5. The end of a phase 2 episode is defined by which of the following?
a. When seizures are controlled for 6 months
b. Cessation of diaphoresis
c. When follow-up magnetic resonance imaging shows complete resolution of hemorrhage
d. When no further dystonia or spasms occur

6. Which of the following best describes the diagnosis of sympathetic storming?
a. Temperature of 37.5°C, systolic blood pressure less than 145 mm Hg, and agitation
b. Temperature of 38.5°C, diastolic blood pressure less than 80 mm Hg, and dystonia
c. Systolic blood pressure greater than 140 mm Hg, agitation, and diaphoresis
d. Systolic blood pressure less than 140 mm Hg, heart rate of at least 120 beats per minute, and dystonia

7. Prolonged hypertension should be treated because of which of the following?
a. Increased blood flow and edema, risk of rebleeding, and potential for cardiac dysfunction related to stress on heart
b. Increased metabolism, cardiac dysfunction and arrhythmias, and loss of consciousness
c. Decreased blood flow and edema, agitation, and pain control
d. Cardiac arrhythmias, increased risk of seizures, and risk of rebleeding

8. Differentiation of neurogenic versus pulmonary edema is confirmed by which of the following radiographic changes?
a. Neurogenic pulmonary edema is noted in lung bases
b. Neurogenic pulmonary edema is noted in mid-lung to apex
c. Pulmonary edema is noted in the mid-lung to apex
d. Pulmonary edema is noted throughout the entire lung fields

9. Which of the following are considered first-line intravenous medications for sympathetic storm episodes?
a. Midazolam, morphine sulfate, and phenytoin
b. Fentanyl, lorazepam, and haloperidol
c. Lorazepam, morphine sulfate, and haloperidol
d. Morphine, fentanyl, and midazolam

10. Which of the following medications can be added to the treatment regime for persistent dystonia or contractures if acetaminophen and cooling do not help for hyperthermia?
a. Dantrolene
b. Phenobarbital
c. Levodopa
d. Midazolam

11. When is the appropriate time to educate families on sympathetic storming?
a. Before a witnessed episode
b. Never because families should never witness an episode
c. After the episode has ended
d. 24 hours after an episode, and wait for them to ask the questions

12. Critical care nurse can be instrumental in the supportive care of patients with a severe traumatic injury in which of the following ways?
a. Coordinating rehabilitation facilities, educating families of rehabilitation options, and coordinating intravenous and enteric medications
b. Developing a strict care plan to follow and identifying family psychosocial issues as well as sympathetic storm triggers of the patient
c. Ensuring proper therapies are being done, identifying triggers to transfer patient to acute ward, and providing frequent medication schedules
d. Coordinating intravenous medications and identifying triggers so that patients can be transferred to the acute ward

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

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Corrections

In the December 2006 issue of *Critical Care Nurse,* the CE test following the article, "Heparin-Induced Thrombocytopenia: Advances in Diagnosis and Treatment," was incorrect. The revised CE test is available online at ccn.aacnjournals.org. If you would like to receive a faxed copy of this CE test, please contact ccn@aacn.org or call (800) 809-2273.

In the February 2007 issue of the *Journal,* Table 1 in the article "Sympathetic Storming After Severe Brain Injury (2007:30-37) contained errors in the last 4 rows. The correct Table 1 is shown.

Table 1 Effects of the parasympathetic nervous system and the sympathetic nervous system

System/function	Parasympathetic	Sympathetic
Cardiovascular	Decreased cardiac output and heart rate	Increased contraction and heart rate; increased cardiac output
Pulmonary	Bronchial constriction	Bronchial dilatation
Musculoskeletal	Muscular relaxation	Muscular contraction
Pupillary	Constriction	Dilatation
Urinary	Increased urinary output; sphincter relaxation	Decreased urinary output; sphincter contraction
Gastrointestinal	Increased motility of stomach and gastrointestinal tract; increased secretions	Decreased motility of stomach and gastrointestinal tract; decreased secretions
Glycogen to glucose conversion	No involvement	Increased
Adrenal gland	No involvement	Release epinephrine and norepinephrine