

Riding Out the Storm: Sympathetic Storming after Traumatic Brain Injury

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Abstract: Following acute multiple trauma, hypothalamic stimulation of the sympathetic nervous system and adrenal glands causes an increase in circulating corticoids and catecholamines, or a stress response. In individuals with severe traumatic brain injury or a Glasgow Coma Scale score of 3-8, this response can be exaggerated and episodic. A term commonly used by nurses caring for these individuals to describe this phenomenon is storming. Symptoms can include alterations in level of consciousness, increased posturing, dystonia, hypertension, hyperthermia, tachycardia, tachypnea, diaphoresis, and agitation. These individuals generally are at a low level of neurological activity with minimal alertness, minimal awareness, and reflexive motor response to stimulation, and the storming can take a seemingly peaceful individual into a state of chaos. Diagnosis is commonly made solely on clinical assessment, and treatment is aimed at controlling the duration and severity of the symptoms and preventing additional brain injury. Storming can pose a challenge for the nurse, from providing daily care for the individual in the height of the storming episode and treating the symptoms, to educating the family. Careful assessment of the individual leads the nurse to the diagnosis and places the nurse in the role of moderator of the storming episode, including providing treatment and evaluating outcomes.

After acute trauma, an immediate sympathetic surge provides the needed rapid response to compensate for the effects of the injury (Keller & Williams, 1993; Neil-Dwyer, Cruickshank, & Doshi, 1990; Stanford, 1994). The outward expressions of this surge are hypertension (HTN), hyperthermia, pupillary dilatation, tachycardia, cardiac arrhythmias, profuse sweating, an increased release of glucose, and an increased basal metabolic rate (Cartlidge & Shaw, 1981; Stanford). Some individuals suffering severe traumatic brain injury (TBI) have demonstrated a spontaneous episodic exaggerated stress response, or storming (Baguley, Nicholls, Felmingham, Crooks, Gurka, & Wade, 1999; Boeve, Wijdicka, Benarroch, & Schmidt, 1998; Bricolo, Turazzi, Alexandre, & Rizzuto, 1984; Bullard, 1987; Do, Sheen, & Brumfield,

2000; Hackl et al., 1991; Horntagl et al., 1980; Keller & Williams; Klug et al., 1984; Neil-Dwyer, Cruickshank, & Doshi; Pranzatelli, Palvlakis, Gould, & DeVivo, 1991; Rosner, Newsome, & Becker, 1984; Rossitich & Bullard, 1988; Russo & O'Flaherty, 2000; Strum, 2002; Thorley, Wertsch, & Klingbeil, 2001). Strum speculates that 15%-33% of individuals who suffer severe TBI will demonstrate storming, a poor prognostic indicator (Boeve et al.; Do, Sheen, & Brumfield; Pranzatelli et al.; Rossitich & Bullard; Strum).

Sympathetic storming tends to be associated with lower neurological functional level and can be caused by injury or pressure created by tumors, hydrocephalus, or subarachnoid hemorrhage, though it is most commonly seen in the TBI population (Baguley et al., 1999; Boeve et al., 1998; Do, Sheen, & Brumfield, 2000; Darnell & Arbit, 1993; Keller & Williams, 1993; Russo & O'Flaherty, 2000; Strum, 2002; Thorley et al., 2001). This article specifically addresses sympathetic storming after TBI and reviews history, proposed etiology, clinical presentation, assessment parameters, differential diagnosis, treatment, family education, and the role of the nurse.

History

Wilson (1923) introduced the term "tonic fit" when describing sudden episodes of intensification of extensor posturing. Throughout the years, terms such as diencephalic autonomic epilepsy (Penfield, 1929), central dysregulation (Bricolo, Turazzi, Alexandre, & Rizzuto, 1977), tonic decerebrate spasms (Cartlidge & Shaw, 1981), tonic cerebellar fits (Davis & Davis, 1982), sympathoadrenal response (Rosner, Newsome, & Becker, 1984), decerebrate rigidity (Klug et al., 1984), diencephalic seizures (Bullard, 1987), autonomic dysfunction syndrome (Rossitich & Bullard, 1988), traumatic apallic syndrome (Hackl et al., 1991), paroxysmal sympathetic storms (Boeve et al., 1998), dysautonomia (Baguley et al., 1999), storming (Thorley, Wertsch, & Klingbeil, 2001) and autonomic dysfunction syndrome (Strum, 2002) have been used to describe these episodes.

Theories on the cause of the episodes range from loss of cortical control over autonomic function (Horntagl et al., 1980), loss of physiological regulatory mechanisms (Klug et al., 1984), disinhibitory control of sympathetic outflow (Bullard, 1987), disruption of the autonomic relay system (Boeve et al., 1998), and dysregulation of the autonomic nervous system (Strum, 2002). Numerous sites of dysfunction have been speculated, from the

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upper brain stem and diencephalon (Bullard), brain stem (Cartlidge & Shaw, 1981), orbital frontal cortex (Strum, 2002), and, more specifically, the anterior hypothalamus or medulla (Boeve et al.; Hackl et al., 1990). Secondary to lack of diagnostic radiographic tools, early analysis of the anatomic correlate of the episodes was based on interpretation of clinical symptoms.

Recent research has looked further into identifying the possible location of the dysfunction by evaluating electroencephalogram (EEG), computed tomography (CT) and magnetic resonance imaging (MRI). In EEG testing, these individuals have not demonstrated seizure activity during storming episodes (Baguley et al., 1999; Boeve et al., 1988; Do, Sheen, & Bromfield, 2000; Pranzatelli et al., 1991; Rossitich & Bullard, 1988; Strum, 2002; Thorley, Wertsch, & Klingbeil, 2001). A consistent location of the injury has not been demonstrated on CT or MRI, though there have been several case reports of individuals with diffuse axonal injury (DAI) on MRI who exhibited storming (Boeve et al.; Klug et al., 1984; Strum). The DAI may contribute to disassociation of the sympathetic and parasympathetic systems.

Whatever the cause, there is an uncontrolled sympathetic surge that produces a stress response. In a normal state, the parasympathetic system would respond to an increased sympathetic activity by providing compensation, or an inhibitory response, and the body would return to a normal homeostatic state. In storming, there appears to be both an exaggeration of the sympathetic response with a diminished parasympathetic response or alteration in the relay system between the two networks.

Clinical Presentation

Wilson's (1923) description of "fits" elicits an image of an uncontrolled response and others have continued to use this concept in defining the phenomenon with similar terms that describe the loss of control and the unpredictability of the episodes. Clinical presentation of storming includes alterations in level of consciousness, increased posturing, dystonia, hypertension, hyperthermia, tachycardia, tachypnea, diaphoresis, arrhythmias (atrial fibrillation, supraventricular tachycardia, bradycardia, atrioventricular dissociation, nodal rhythms, and pre-ventricular/atrial beats), and agitation (Baguley et al., 1999; Boeve et al., 1998; Cartlidge & Shaw, 1981; Hackl et al., 1991; Horntagl et al., 1980; Klug et al., 1984; Neil-Dwyer, Cruickshank, & Doshi, 1990; Pranzatelli et al., 1991; Rosner et al., 1984; Rossitich & Bullard, 1988; Strum, 2002; Thorley, Wertsch, & Klingbeil, 2001). Symptoms, duration, and intensity vary from individual to individual and from episode to episode. These individuals generally are at a low level of neurological activity with minimal alertness, minimal awareness, and reflexive motor responses to stimulation and the storming can take a seemingly peaceful individual into a state of chaos.

The storming may occur as early as within the first 24 hours after injury, although frequently is delayed until the patient is transferred from the intensive care unit (ICU; Boeve et al., 1998; Cartlidge & Shaw, 1981; Hackl et al., 1991; Hortnagl et al., 1980; Klug et al., 1984; Neil-Dwyer, Cruickshank, & Doshi, 1990; Pranzatelli et al., 1991; Rosner, et al., 1984; Rossitich & Bullard, 1988; Russo & O'Flaherty, 2000; Strum, 2002; Thorley, Wertsch, & Klingbeil, 2001). The delay in clinical presentation may be related to the use of paralytic agents, sedatives, and narcotics in the treatment of increased intracranial pressure (ICP) (Russo & O'Flaherty, 2000). These treatments effectively manage storming early in the injury and it is only when status stabilizes and medications are withdrawn that the clinical symptoms may appear.

Diagnosis

The diagnosis actually becomes apparent within the clinical arena. Nursing clinical evaluation, most often over time, reveals the diagnosis. The nurse provides the most reliable data for diagnosis because the nurse monitors the individual over an extended period of time. Interpretation of this information leads to diagnosis and can provide clues into potential triggers of the episodes as well as effective treatment. Triggers are defined as anything that produces a storming episode, including simple nursing activities such as suctioning, turning, or bathing the individual. The ability to identify what triggers an episode can lead the nurse to pretreat the individual to abate or lessen the intensity of the storm.

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Strum (2002) made the diagnosis on the presence of spontaneous episodic tachycardia, hyperthermia and HTN, whereas Baguley et al. (1999) thought that an individual needed to exhibit five of seven of the clinical features (tachycardia, tachypnea, hyperthermia, hypertension, dystonia, posturing, and diaphoresis) for the diagnosis. Horntagl et al. (1980) based the diagnosis of storming solely on the ability of intravenous (IV) morphine to abate the episode because morphine is a potent opiate and suppresses sympathetic activity.

Epinephrine, catecholamine, and thyroid studies can be performed to document the sympathetic surge, although they are costly and do not change treatment (Hackl et al., 1991; Hortnagl et al., 1980; Neil-Dwyer, Cruickshank, & Doshi, 1990; Rosner, Newsome, & Becker, 1984). Radiographics, such as CT or MRI of the brain, may rule out or define changes in injury but, as previously noted, do not make the diagnosis.

Acute hyperthermia in the individual with TBI requires a fever workup. Evaluation includes blood, urine, and sputum cultures; cultures of invasive lines (arterial, ventriculostomy, and venous); chest X ray; and, as clinically indicated, lower extremity Dopplers. A positive fever workup does not negate the possibility that the individual is storming and the fever may actually be a trigger.

Additional differential diagnoses that need to be considered beyond infection are seizures, new or expanding intracranial hemorrhage or edema, pulmonary emboli, hypoxia, thyroid storm, deep vein thrombosis, myocardial infarction (MI), alcohol or drug withdrawal, pain, or anxiety. Any of these above scenarios can have a similar clinical presentation, and careful assessment of the individual can assist in differentiating the diagnosis (Table 1). Assessment of changes that need to be considered are quality and quantity of sputum, chest X ray, breath sounds, pain status, laboratory values (chemistry panel, thyroid panel, white blood cell count), 12-lead electrocardiogram (ECG), and cardiac enzymes to rule out MI, as well as history of alcohol or drug abuse to eliminate withdrawal as the etiology of the symptoms (Strum, 2002; Thorley, Wertsch, & Klingbeil, 2001). Workup should be directed at the specific suspected etiology.

Increases in ICP can be seen during the episode or an aftermath of the episode and need to be treated per ICU guidelines (Hackl et al., 1991; Hortnagl et al., 1980; Klug et al., 1984). There are differing opinions on whether elevation of ICP can be a trigger of the episodes or is just a consequence of the storm (Hackl et al.; Hortnagl et al.; Klug et al.).

Consequences of Prolonged Sympathetic Output

Prevention of secondary injury is a primary goal in the treatment of TBI. Effects of prolonged sympathetic overactivity (hyperthermia, hyperglycemia, increased basal metabolic rate, hyperhidrosis, dystonia, and increased circulating catecholamines) can increase the risk of secondary brain injury (Baguley et al., 1999; Horntagl et al., 1980; Keller & Williams, 1993; Rosner, Newsome, & Becker, 1984; Stanford, 1994; Strum, 2002). Careful monitoring of the individual is needed to prevent potential problems.

Extreme or prolonged hyperthermia and increased basal metabolic rate with hyperglycemia can result in further neuronal dysfunction, hypoxia, and possible cell death (Baguley et al., 1999; Strum, 2002). Intramuscular (IM) or intravenous (IV) chlorpromazine (Thorazine) can be helpful with severe hyperthermia. Chlorpromazine suppresses hypothalamic activity, diminishing piloerection and allowing for cooling, and can rapidly reduce the core temperature (Strum, 2002). Chlorpromazine can be used in conjunction with acetaminophen and cooling blankets (Strum). Insulin may be required to regulate serum blood glucose within a normal range, and frequent monitoring of blood glucose is required to document trends and determine insulin dosing.

Energy needs can be increased by 100%–200% in the presence of storming (Baguley et al., 1999; Horntagl et al., 1980; Rosner, Newsome, & Becker, 1984; Stanford, 1994; Strum, 2002). The drastic increase in metabolism leads to protein wasting and a catabolic state. This places the individual at risk for weight loss. Weight loss causes a decrease in muscle mass. With dystonia, there is increased risk for contractures, potential for injury, skin breakdown, and further atrophy. These can limit the potential for rehabilitation. Nursing care should be directed at measures such as providing nutritional support, use of splints, padded rails, passive range of motion, and judicious skin care to reduce the risk of problems.

Table 1. Differential Diagnosis

	Storms	Thyroid Infection	Storm	Cardio-vascular	Pulmonary	Increased Intracranial Pressure	Deep Vein Thrombosis
Hypertension	+	-	+	-	-	+/-	-
Tachycardia	+	+/-	+	+/-	+/-	+/-	+/-
Hyperthermia	+	+	+	+	+	+	-
>102°F	+	+/-	-	-	+/-	+	-
Increased posturing	+	+	+/-	-	-	+/-	-
Opisthotonus	+/-	-	-	-	-	+/-	-
Diaphoresis	+	+/-	+	+/-	+/-	-	+/-
Hyperhidrosis	+	-	-	-	-	-	-
Tachypnea	+	+/-	+	+/-	+/-	+/-	+/-
Dystonia	+	+/-	-	-	-	+/-	-
Symptoms abated with morphine	+	-	-	-	-	+/-	-
Sporadic episodes	+	-	-	-	-	-	-
Decreased level of consciousness	+	+	-	-	+/-	+	+/-
Dilated pupils	+	-	+	-	-	+/-	-
Arrhythmias	+/-	-	+	+/-	-	+/-	-
Symptomatic	-	-	-	+	-	-	-
Asymptomatic	+	-	-	-	-	-	-
Increased intracranial pressure	+/-	+/-	-	-	-	+	-
Bradycardia	+/-	-	-	+/-	-	+/-	-

Note: (+) = commonly noted; (+/-) = potential symptom; (-) = not present.

The increased metabolic rate and the presence of hyperhydrosis increase the risk of dehydration and skin breakdown. Dehydration can decrease the ability to mobilize secretions, increasing the risk for pneumonia. An electrolyte imbalance can also occur from prolonged dehydration without adequate fluid replacement. Assessment parameters include close monitoring of serum electrolytes, serum albumin, 24-hour fluid status, and weight. Ongoing dietary involvement can provide the needed adjustment of caloric, protein, and water intake to prevent problems.

Cardiovascular risks can be much higher. Prolonged sympathetic overactivity can result in ECG changes including decreased T wave amplitudes, biphasic T waves, rising ST segments, and ST depression (Horntagl et al., 1980; Keller & Williams, 1993). Sustained sympathetic stimulation can lead to heart damage, which begins as focal myocytolysis related to sustained levels of catecholamines (Horntagl et al.). This can lead to myocardial damage and, if severe, theoretically death (Horntagl et al.; Strum, 2002). Given the variety of symptoms and severity, careful assessment is needed to prevent complications, though the presence of storming does not always warrant care within the ICU setting.

Treatment

Treatment of storming is aimed at abating the symptoms and limiting the stress response. The overall goal of medication is to dampen the sympathetic outflow or act as the parasympathetic system. Thus, sedatives, opiate

receptor agonists, beta-blockers, and CNS depressants have been used. Unfortunately, these medications can dampen the level of responsiveness in an already minimally responsive individual, making assessment of neurological changes difficult.

Multiple drugs including morphine sulfate, oxycodone, midazolam (Versed), propranolol (Inderal), clonidine (Catapres), chlorpromazine, bromocriptine, dantrolene (Dantrium), atenolol, and labetalol (Normadyne) have shown effectiveness in treating storming (Baguley et al., 1999; Boeve et al., 1998; Bullard, 1987; Cartledge & Shaw, 1981; Do, Sheen, & Brumfield, 2000; Hackl et al., 1991; Horntagl et al., 1980; Klug et al., 1984; Neil-Dwyer, Cruickshank, & Doshi, 1990; Pranzatelli et al., 1991; Rosner, Newsome & Becker, 1984; Rossitich & Bullard, 1988; Russo & O'Flaherty, 2000; Strum, 2002; Thorley, Wertsch, & Klingbeil, 2001); see Table 2. Diazepam (Valium), pentobarbital, betamethasone, mannitol, lorazepam (Ativan), baclofen (Lioresal), phenytoin (Dilantin), and droperidol (Inapsine) are additional drugs identified within the literature with varied success (Baguley et al.; Boeve et al.; Horntagl et al.; Pranzatelli et al.; Rossitich & Bullard; Strum; Thorley, Wertsch, & Klingbeil).

A common starting point of treatment, advocated by Horntagl et al. (1980) is a 10-mg dose of IV morphine sulfate in ventilated individuals with an as-needed scheduling or continuous IV drip. While the patient is in the ICU, IV medication may be preferred for quick control of the storming early in the injury. Once the patient's status stabilizes, the enteric route is preferred because it decreases frequency of

Table 2. Comparison of Medications

	Baguley et al., 1999	Boeve et al., 1998	Bullard, 1987	Do, Sheen, & Brumfield, 2000	Hornagl et al., 1980	Neil-Dwyer, Cruickshank, & Doshi, 1990	Pranzatelli et al., 1991	Rossitich & Ballard, 1988	Russo & O'Flaherty, 2000	Strum, 2002	Thorley, Wertsch, & Klingbeil, 2001
Morphine sulfate	++	+	+	+	+			+	+	+	+
Propranolol	+	+		+		+	++	-		+	+
Oxycodone	+										+
Bromocriptine		+		+			-	+	+	+	+
Dantrolene		+					-	+		+	+
Chlorpromazine										+	
Haloperidol											
Midazolam	++	+/-							+		+
Droperidol					+						
Diazepam					+		+			+	
Phenobarbital		-			+			-			
Labetalol				+							
Clonidine	+						+			+	
Atenolol						+					
Betamethasone					+						
Baclofen							-				
Phenytoin		-					-	-			
Mannitol							-	-			

Note: (++) = consistent response in treating episodes; (+) = positive response in treating episodes; (+/-) = varied responses in treating episodes; (-) = no appreciative response in treating episodes.

the medication, reduces cost, and allows for eventual transfer to an acute care ward or long-term-care facility. Therefore, enteric routes should be utilized when possible.

In individuals whose symptoms are difficult to control, a starting point can be a combination of morphine or oxycodone with bromocriptine (Boeve et al., 1998). Morphine and oxycodone are both opiate receptor agonists that suppress sympathetic outflow (Boeve et al.; Strum, 2002). A continuous IV morphine drip or scheduled oxycodone may be indicated if the storming episodes are difficult to regulate. Oxycodone is used over combination medications with acetaminophen to avoid acetaminophen overdose and provide a medication that can be used for breakthrough fever.

Bromocriptine, a dopamine receptor agonist, has been effective in reducing hyperthermia and diaphoresis in individuals during storming (Bullard, 1987; Russo & O'Flaherty, 2000; Thorley, Wertsch, & Klingbeil, 2001; Strum, 2002). There may also be a mild hypotensive effect with the bromocriptine (Russo & O'Flaherty). Russo & O'Flaherty (2000) recommend starting at 0.025 mg/kg twice a day and increasing to 0.05 mg/kg three times a day as needed to control the symptoms.

As in adding medications, the withdrawal of medications can be challenging.

Propranolol, a beta-blocker, can be added if necessary and is particularly helpful if tachycardia and HTN dominate the episodes. Propranolol suppresses sympathetic outflow, slowing neuronal activity (Bullard, 1987; Strum, 2002). Bradycardia is a common side effect of propranolol, although it generally does not pose a problem with the young TBI population. In the symptomatic individual with severe hypotension or hypotension with syncope, the use of propranolol may need to be reassessed. This may be extremely difficult to assess in the minimally responsive individual, and practitioners may prefer to define a lower blood pressure limit based on their professional comfort level or switch to an alternative agent. Propranolol is also titrated up as status indicates. Clonidine and labetalol are alternatives to propranolol. Clonidine lowers circulating plasma levels of epinephrine and norepinephrine (Boeve et al., 1999; Strum), and labetalol provides alpha 1 and both beta 1 and beta 2 adrenergic receptor blockage (Do, Sheen, & Brumfield, 2000).

Again as previously noted, IM, IV, or oral chlorpromazine can be helpful with severe hyperthermia by rapidly reducing the core temperature (Strum, 2002). The ability to maintain body temperature within a normal range can lessen the severity of the storming or abate the storm. Whenever there is a temperature spike, the individual should be evaluated for potential infection and care needs to be taken that use of acetaminophen and hypothermia blankets to treat the fever do not mask an infection.

If persistent dystonia or posturing is noted, dantrolene can be added. Dantrolene decreases the release of calcium, which interferes with skeletal muscle contraction causing relaxation. There is not a clear CNS response, though drowsiness is a major side effect (Baguley et al., 1999; Strum, 2002).

The individual ends up defining the appropriate drug regime. Frequently it is trial and error before the appropriate medication or combination of medications proves effective. Inability to control the storming episodes can delay transfer to a subacute facility, because many facilities are reluctant to take an individual who requires such a high level of care.

As the storming episodes stabilize, trial withdrawal of medications is recommended, withdrawing one medication at a time by slowly decreasing the dosage of each medication (Boeve et al., 1999; Russo & O'Flaherty, 2000). The stability of the individual may signify return of regulatory control, and medications used to slow neuronal output now can dampen the functional capacity of the individual. As in adding medications, the withdrawal of medications can be challenging.

Family Education

The family is dealing with many unknowns at this time, leading to an array of emotions. The injury, hospitalization, equipment, ICU setting, and separation all play a part in their anxiety, frustration, and fears. These variables, and many more, make this time stressful. The onset of storming with its characteristic presentation of distress can signal problems to the family. Family education is an important aspect of the management of the TBI individual, especially in the individual enduring storming. This education should be geared toward reviewing the etiology of the storming, the treatment plan, and goals; clarifying that the storming may not necessarily require ICU care; clarifying potential duration of sympathetic dysfunction; and most importantly, identifying how they can help. The family can be useful in identifying triggers or treating an episode. Simple things such as applying a cool cloth to the forehead, providing a bath, assisting in monitoring response to medications, and utilizing techniques to promote relaxation can help provide a sense of security, a sense of control, and a sense that they are helping in the care of their loved one.

Summary

For any experienced nurse, the mere mention of storming provides a vivid image of the clinical scenario. The term "storming" relates the sporadic nature of the episodes as well as the variability in the severity and duration of an episode. Differential diagnosis is important in defining the appropriate treatment modality. This places the nurse in the center of the storm. Frequent assessment, monitoring of vital signs, and providing

basic care provides the nurse with the key elements needed to define the problem.

Astute nursing care can identify the triggers, reduce the occurrence, severity and duration of episodes, and alert the nurse to seek pharmacological intervention to treat the outward expression of the phenomena. The nurse not only is instrumental in the diagnosis of the storming but also is the moderator of the storm.

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