PAROXYSMAL SYMPATHETIC HYPERACTIVITY

Kimberly Clark, PharmD, BCCCP
Pharmacy Clinical Specialist – Critical Care, Prisma Health
Adjunct Assistant Professor, University of South Carolina College of Pharmacy
Clinical Assistant Professor, University of South Carolina Greenville School of Medicine

June 6, 2019

Disclosures

- I have no financial disclosures
- Off-label or investigational use of medications may be discussed

Objectives

- Define paroxysmal sympathetic hyperactivity (PSH)
- Explain the role of different pharmacotherapy agents in PSH
- Review the pharmacologic properties of key treatments
- Evaluate primary literature for the treatment of PSH

Traumatic Brain Injury (TBI)

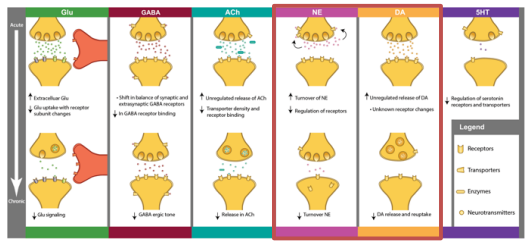
Disease Models & Mechanisms. 2013;6:1307-1315
https://www.cdc.gov/traumaticbraininjury/index.html

Injury Types

- Primary brain injury
  - Occurs at time of trauma
- Secondary brain injury
  - Injury occurs after trauma
  - Mechanisms include
    - Intracranial hypertension
    - Systemic hypotension
    - Hypoxia
    - Hypocapnia
    - Hyperpyrexia
    - Electrolyte imbalances
    - Neurotransmitter modulation

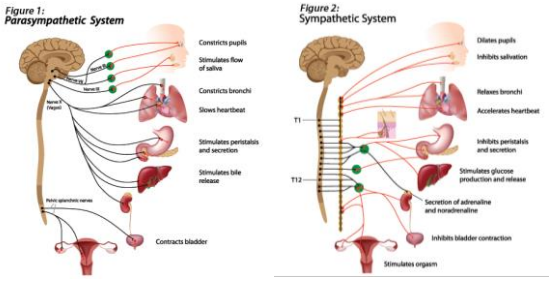
Disease Models & Mechanisms 2013;6:1307-1315.

Neurotransmitter Modulation



Mol Psychiatry 2018 Sep 13. doi: 10.1038/s41380-018-0239-6.

Autonomic Nervous System



Reyst, Heidi. *Weathering the Storm*. https://www.rainbowrehab.com/weathering-the-storm-storming/ Accessed 22 May 2019

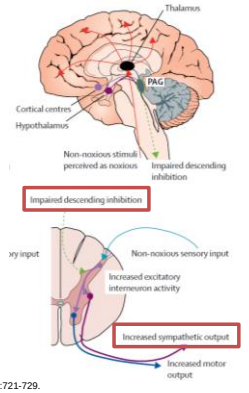
Adrenergic Receptor Review

Receptor	Location	Stimulation	Overall Effects
α 1	Peripheral vascular smooth muscle	Vasoconstriction	↑ SVR = ↑ BP
α 2	CNS	Vasodilation	↓ SVR = ↓ BP
β 1	Heart, Kidneys	Contraction	↑ HR = ↑ CO
β 2	Heart Lungs, GI	Contraction of skeletal muscle Relaxation of smooth muscle	↑ HR = ↑ CO
Dopamine	Renal, GI, Heart, Brain	Vasodilation Natriuresis Motor control	

CNS: Central Nervous System
GI: Gastrointestinal

Pathophysiology

- Imbalance of excitatory and inhibitory pathways
- Decreased parasympathetic feedback
- Increased catecholamine levels
  - Norepinephrine
  - Epinephrine



Lancet Neurol 2017;16:721-729.
Nurs Clin N Am 2018;53:459-467.

PAROXYSMAL SYMPATHETIC HYPERACTIVITY

A syndrome, recognized in a subgroup of survivors of severe acquired brain injury, of simultaneous, paroxysmal transient increases in sympathetic (elevated heart rate, blood pressure, respiratory rate, temperature, sweating) and motor (posturing) activity

Journal of Neurotrauma 2014;31:1515–1520.

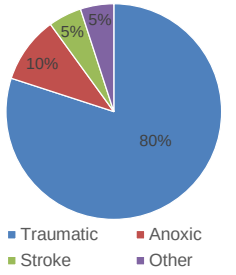
Historical Nomenclature

- Diencephalic seizure
- Diencephalic fits
- Dysautonomia
- Dysautonomic crises
- Episodic autonomic instability
- Central autonomic dysfunction
- Paroxysmal autonomic instability with dystonia
- Autonomic dysfunction syndrome
- Autonomic storming
- Sympathetic storming
- Neurostorming
- Autonomic dysreflexia
- Autonomic hyper-reflexia
- Brainstem attack
- Hyperadrenergic state
- Midbrain syndrome
- Decerebration tonic spasms
- Cerebellar tonic discharges
- Sympathetic adrenal response
- Hypothalamic-midbrain dysregulation syndrome
- Paroxysmal autonomic dysregulation
- Hyperpyrexia with prolonged muscle contraction

Med Intensiva 2019;43:35-43.

PSH

- Cause



- Incidence
  - 8 to 33% of TBI patients
- Timing
  - 5-7 days after injury
  - May persist for months to years
- Episodes
  - Last from minutes to hours (average 30 minutes)
  - Typically occur 1-5 times per day

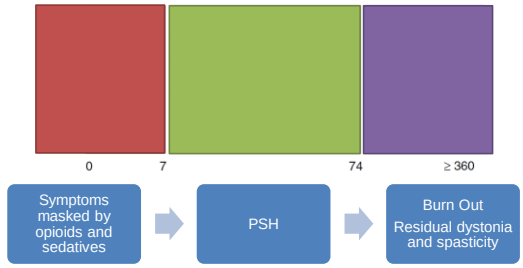
Current Treatment Options in Neurology 2008;10:151–157.
Lancet Neurol 2017;16:721-729.

Risk Factors

- Type of injury
  - Diffuse axonal injury (DAI)
- Location of injury
  - Focal parenchymal
  - Midbrain
  - Pontine
  - Periventricular white matter
  - Corpus callosum
  - Deep grey nuclei
- Higher severity of injury
- Younger age
- Male gender

Lancet Neurol 2017;16:721–729.

Phases



Med Intensiva 2019;43:35-43.

Signs and Symptoms

System	Abnormal Criteria
General	
Diaphoresis	Presence of mild to severe sweating
Hyperthermia	Body temperature ≥37.0°C
Cardiovascular	
Tachycardia	HR ≥100 beats per minute
Hypertension	Systolic blood pressure ≥140
Respiratory	
Tachypnea	Respiratory rate ≥18 breaths per minute
Musculoskeletal	
Posturing	Presence of mild to severe dystonic posturing of extremities

Nurs Clin N Am 2018;53:459–467.

Differential Diagnosis

- Infection
- Withdrawal
- Pain
- Seizures
- Neuroleptic malignant syndrome
- Malignant hyperthermia
- Lethal catatonia
- Cushing response
- Encephalitis
- Hydrocephalus
- Pulmonary thromboembolism
- Thyroid storm
- Acute myocardial infarction
- Hypoglycemia
- Serotoninergic syndrome

Journal of Neuroscience Nursing 2016;48:82-89.
Med Intensiva 2019;43:35-43.

Paroxysmal Sympathetic Hyperactivity Assessment Measure (PSH-AM)

Component	Score	0	1	2	3	Allocated score
Clinical Scale (CFS) Feature	Heart rate (per min)	<100	100-119	120-139	≥140	
	Respiratory rate (per min)	<18	18-23	24-29	≥ 30	
	Systolic blood pressure (mmHg)	<140	140-159	160-179	≥180	
	Temperature (°C)	<37.0	37.0-37.9	38.0-38.9	≥39.0	
	Sweating	Absent	Mild	Moderate	Severe	
	Posturing during episodes	Absent	Mild	Moderate	Severe	
	CFS subtotal *(Severity: 0 = Nil; 1-6 = Mild; 7-12 = Moderate; ≥ 13 = Severe)*					
Diagnosis Likelihood Tool (DLT)	Antecedent acquired brain injury					
	Clinical features occur simultaneously					
	Episodes are paroxysmal in nature					
	Sympathetic over-reactivity to normally non-painful stimuli					
	Absence of parasympathetic features during episodes					
	Features persist > 3 consecutive days					
	Features persist ≥ 2 weeks post-brain injury					
	≥ 2 episodes daily					
	Absence of other presumed causes of features					
	Features persist despite treatment of alternative differential diagnoses					
	Medication administered to decrease sympathetic features					
	DLT Subtotal *(Score 1 point for each feature present)*					
PSH-Assessment Measure (PSH-AM = CFS subtotal + DLT subtotal)						

Interpretation of PSH-AM (CFS subtotal + DLT subtotal)	Score	PSH diagnosis
	<8	Unlikely
	8-16	Possible
	≥17	Probable

Lancet Neurol 2017;16:721–729.

Component	Score	0	1	2	3	Allocated score
Clinical Scale (CFS) Feature	Heart rate (per min)	<100	100-119	120-139	≥140	
	Respiratory rate (per min)	<18	18-23	24-29	≥ 30	
	Systolic blood pressure (mmHg)	<140	140-159	160-179	≥180	
	Temperature (°C)	<37.0	37.0-37.9	38.0-38.9	≥39.0	
	Sweating	Absent	Mild	Moderate	Severe	
	Posturing during episodes	Absent	Mild	Moderate	Severe	
	CFS subtotal *(Severity: 0 = Nil; 1-6 = Mild; 7-12 = Moderate; ≥ 13 = Severe)*					
Diagnosis Likelihood Tool (DLT)	Antecedent acquired brain injury					
	Clinical features occur simultaneously					
	Episodes are paroxysmal in nature					
	Sympathetic over-reactivity to normally non-painful stimuli					
	Absence of parasympathetic features during episodes					
	Features persist > 3 consecutive days					
	Features persist ≥ 2 weeks post-brain injury					
	≥ 2 episodes daily					
	Absence of other presumed causes of features					
	Features persist despite treatment of alternative differential diagnoses					
	Medication administered to decrease sympathetic features					
	DLT Subtotal *(Score 1 point for each feature present)*					
PSH-Assessment Measure (PSH-AM = CFS subtotal + DLT subtotal)						

Interpretation of PSH-AM (CFS subtotal + DLT subtotal)	Score	PSH diagnosis
	<8	Unlikely
	8-16	Possible
	≥17	Probable

Lancet Neurol 2017;16:721–729.

Consequences

- Secondary brain injury
- Delayed neurologic recovery
- Cardiac arrhythmias
- Dehydration
- Muscle wasting
- Malnutrition
- Longer hospital and intensive care unit length of stay
- Increased cost of care

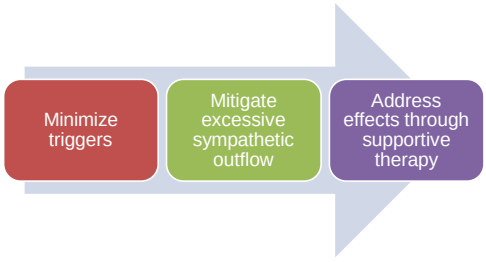
Critical Care Nurse 2007;27:30-37.

Treatment

- TBI Treatment Guidelines
  - Guidelines for the Management of Severe TBI
    - Brain Trauma Foundation
  - Evaluation and Management of Mild TBI
    - Eastern Association for the Surgery of Trauma (EAST)
- No accepted PSH treatment algorithm
- EAST Practice Management Guideline
  - Beta Blockers After Traumatic Brain Injury

Annals of Surgery 2017;266:952-961.

Treatment Goals



Lancet Neurol 2017;16:721-729.
JHN Journal 2017;12:35-37.

Triggers

- Examples
  - Endotracheal suctioning
  - Turning
  - Bathing
  - Physical exam
  - Constipation
  - Urinary retention
  - Pain
- Strategies
  - Cluster care
  - Limit visitations
  - Reduce unnecessary touch / external stimuli
- Nursing role
  - Identify
  - Mitigate
  - Caregiver education

Lancet Neurol 2017;16:721-729.

Pharmacologic Options

Opioids	• Morphine • Oxycodone	Treat / Abort
Beta-blockers	• Propranolol • Labetalol	Prevent
Alpha-agonists	• Clonidine • Dexmedetomidine	Prevent
GABA-agonists	• Benzodiazepines • Baclofen • Gabapentin	Treat / Abort Prevent
Dopaminergic	• Bromocriptine	Prevent
Calcium	• Dantrolene	Treat / Abort

Lancet Neurol 2017;16:721-729.
JHN Journal 2017;12:35-37.

Opioids

Role in PSH	Treatment and prevention; Hypertension, tachycardia, allodynia
Medications	Morphine, oxycodone
Mechanism of action	Opioid receptor agonist (Mu and Kappa)
Contraindications	None
Adverse effects	Sedation, respiratory depression, constipation, miosis, pruritus, euphoria, hypotension
Drug interactions	CNS depressants (i.e. benzodiazepines, antihistamines, antipsychotics, tricyclic antidepressants) Anticholinergics (i.e. atropine, antihistamines, tricyclic antidepressants) Monoamine oxidase inhibitors (MAOIs)
Pearls	Active metabolites Can accumulate in renal dysfunction Associated with histamine release Prolonged use will require tapering to avoid withdrawal

Current Treatment Options in Neurology 2008;10:151-157.
J Neurosci Nurs 2016;48:82-89.

Non-Selective Beta-Blockers

Role in PSH	Prevention; Hypertension, tachycardia, fever, diaphoresis, possibly dystonia
Medications	Propranolol, labetalol
Mechanism of action	Beta-blocker (peripherally diminish the effects of circulating catecholamines)
Contraindications	AV block, cardiogenic shock, acute decompensation heart failure
Adverse effects	Bradycardia, hypotension, fatigue, dizziness, shortness of breath (in patients with reactive airway disease), fluid retention, masks hypoglycemia symptoms
Drug interactions	None

AV: Atrioventricular

Current Treatment Options in Neurology 2008;10:151-157.
J Neurosci Nurs 2016;48:82-89.

Central Alpha-2 Agonists

Role in PSH	Prevention; Hypertension, tachycardia
Medications	Clonidine, dexmedetomidine
Mechanism of action	Central alpha-2 receptor agonist (inhibits sympathetic outflow and tone)
Contraindications	None
Adverse effects	Hypotension, bradycardia, dry mouth, rebound hypertension, sedation (with dexmedetomidine)
Drug interactions	Tricyclic antidepressants Cyclosporine
Pearls	Takes ~48-72h to see maximal effects on blood pressure Prolonged use requires tapering Available as oral and transdermal

Current Treatment Options in Neurology 2008;10:151-157.
J Neurosci Nurs 2016;48:82-89.

GABA AGONISTS

Benzodiazepines

Role in PSH	Treatment and prevention; Agitation, hypertension, tachycardia, and posturing
Medications	Midazolam, lorazepam, diazepam, clonazepam
Mechanism of action	Potentiate the inhibitory action of $GABA_A$ (increasing frequency of Cl- channel opening)
Contraindications	Severe glaucoma
Adverse effects	Sedation, confusion, respiratory depression
Drug interactions	CNS depressants (i.e. opiates, antihistamines, antipsychotics, tricyclic antidepressants) Diltiazem, verapamil Fluconazole
Pearls	Most useful in patients with a component of anxiety Use may slow/impair brain recovery Prolonged use will require tapering to avoid withdrawal Diazepam preferred in patients with dystonia

Current Treatment Options in Neurology 2008;10:151-157.
J Neurosci Nurs 2016;48:82-89.

Baclofen

Role in PSH	Prevention; Spasticity, dystonia
Mechanism of action	$GABA_B$ receptor agonist
Contraindications	None
Adverse effects	Sedation (less with IT), muscle weakness, elevation of liver enzymes, withdrawal with abrupt discontinuation (fever, rigidity, dystonia, or seizures) IT – cerebral spinal fluid leak, infection, pump failure
Drug interactions	Tricyclic antidepressants
Pearls	Prolonged use requires tapering to avoid withdrawal While data for IT is consistent, data for oral is limited IT only studied in later, recovery phases (>6 months after injury)

IT: Intrathecal

Gabapentin

Role in PSH	Prevention; Spasticity, allodynic responses
Mechanism of action	GABA agonist Potentiate the inhibitory action of GABA Structurally related to GABA; does not bind to GABA receptors Increasing frequency of Cl- channel opening in the dorsal horn of the spinal cord Inhibits neurotransmitter release
Contraindications	None
Adverse effects	Sedation, dizziness, peripheral edema
Drug interactions	Caution with other CNS depressants
Pearls	Alternative to oral baclofen Dose adjustment for renal impairment

Current Treatment Options in Neurology 2008;10:151-157.
J Neurosci Nurs 2016;48:82-89.

OTHER MECHANISMS

Bromocriptine

Role in PSH	Prevention; Fever, sweating, dystonia, posturing
Mechanism of action	Dopamine D2 receptor agonist Inhibits norepinephrine release Affects motor control Exact mechanism is unclear
Contraindications	Allergy to ergot alkaloids Uncontrolled hypertension
Adverse effects	Orthostasis, tachycardia, nausea/vomiting, diarrhea, discoloration of bodily fluids, hallucinations, dyskinesia, hyperprolactinemia
Drug interactions	Ergot alkaloids Cyclosporine, sirolimus, and tacrolimus
Pearls	Effect is delayed May lower seizure threshold

Dantrolene

Role in PSH	Treatment; Posturing, muscle spasms
Mechanism of action	Direct-acting skeletal muscle relaxant (no effect on cardiac or smooth muscle) that depresses the excitation-contraction coupling Inhibits the release of calcium from the SR by binding to the RyR1 receptor Decreases muscle contraction but no effect on the action potential
Contraindications	Severe liver disease (cirrhosis, hepatitis)
Adverse effects	Muscle weakness, respiratory depression, phlebitis, nausea/diarrhea, drowsiness, dizziness, confusion, hepatotoxicity
Drug interactions	Calcium channel blockers Neuromuscular blockers
Pearls	Monitor liver function tests prior to use

Medications to Avoid

- Dopamine antagonists
  - Haloperidol and atypical antipsychotics (agitation)
  - Chlorpromazine (hyperthermia)
  - Metoclopramide (gastroparesis)
- Worsen symptoms
  - Exacerbate cognitive deficits
  - Psychosis
  - Neuroleptic malignant syndrome

Medication Dosing Summary

Medication	Dose
Morphine	1-10 mg IV bolus or continuous infusion
Oxycodone	10-20 mg PO every 6-8 hrs
Propranolol	20-60 mg PO every 4-6 hr
Lorazepam	2-4 mg IV bolus
Diazepam	5-10 mg IV bolus
Midazolam	1-2 mg IV bolus
Clonazepam	0.25-2 mg PO every 8-12 hrs (Max 8 mg/day)
Baclofen	IT – test dose followed by titration per established protocol 5 mg PO every 8 hrs (Max 80 mg/day)
Gabapentin	100-300 mg PO every 8 hrs (Max 4800 mg/day)
Clonidine	0.1-0.3 mg PO every 8-12 hrs (Max 1.2 mg/day)
Dexmedetomidine	0.2-0.7 mcg/kg/hr
Bromocriptine	1.25-2.5 mg PO every 8-12 hrs (Max 40 mg/day)
Dantrolene	0.25-2 mg/kg IV every 6-12 hrs (Max 10 mg/kg/day)

LITERATURE

Case Reports

Source	No. of Subjects	Age, y	Origin	Onset, d	No. of Cycles per Day	Duration of Each Episode	Total Duration	Signs	Treatment or Drugs	Term Used to Describe
University of Virginia	7	9-29	MVC, near drowning, hanging, assault	NA	NA	NA	NA	T, 38°C-41.8°C; HR, 161-216 beats/min; BP, 123-171/78-101 mm Hg; rigid or decerebrate posturing; diaphoresis; agitation	Atenolol, lorazepam, diazepam, methadone hydrochloride, propranolol hydrochloride, clonidine hydrochloride	NA
Thorley et al,[12] 2001	1	20	MVC	1	NA	Minutes to hours	2 wk	T, 40.0°C; HR, 156 beats/min; extremity posturing; diaphoresis; agitation; CPK, 61 U/L	Midazolam, lorazepam, morphine sulfate, oxycodone, bromocriptine mesylate, dantrolene sodium	Acute hypothalamic instability
Cuny et al,[11] 2001	4	17-37	MVC	24-60	2-16	NA	1-8 wk	Hyperthermia; hypertonia; tachycardia	Intrathecal baclofen	Dysautonomia
Do et al,[9] 2000	1	21	MVC	450	3	1-2 h	NA	T, 39.1°C; HR, 140-160 beats/min; BP, 170-180/100 mm Hg; diaphoresis; flexor posturing	Labetalol hydrochloride	Paroxysmal sympathetic storm
Baguley,[1] 1999	14	NA	Hypoxia, TBI	NA	NA	NA	NA	Classic features described by Boeve et al[2] (1998)	NA	Paroxysmal sympathetic storms
Goh et al,[10] 1999	1	7	Midbrain glioma	3	NA	NA	NA	T, 38.4°C-39.5°C; HR, 150-180 beats/min; SBP, 180 mm Hg; DBP, 120 mm Hg; sweating, muscle spasms; decorticate	Phenytoin sodium, diazepam, lorazepam, clonidine	Diencephalic seizures

Arch Neurol 2004;61:321-328.

Source	No. of Subjects	Age, y	Origin	Onset, d	No. of Cycles per Day	Duration of Each Episode	Total Duration	Signs	Treatment or Drugs	Term Used to Describe
University of Virginia	7	9-29	MVC, near drowning, hanging, assault	NA	NA	NA	NA	T, 38°C-41.8°C; HR, 161-216 beats/min; BP, 123-171/78-101 mm Hg; rigid or decerebrate posturing; diaphoresis; agitation	Atenolol, lorazepam, diazepam, methadone hydrochloride, propranolol hydrochloride, clonidine hydrochloride	NA
Thorley et al,[12] 2001	1	20	MVC	1	NA	Minutes to hours	2 wk	T, 40.0°C; HR, 156 beats/min; extremity posturing; diaphoresis; agitation; CPK, 61 U/L	Midazolam, lorazepam, morphine sulfate, oxycodone, bromocriptine mesylate, dantrolene sodium	Acute hypothalamic instability
Cuny et al,[11] 2001	4	17-37	MVC	24-60	2-16	NA	1-8 wk	Hyperthermia; hypertonia; tachycardia	Intrathecal baclofen	Dysautonomia
Do et al,[9] 2000	1	21	MVC	450	3	1-2 h	NA	T, 39.1°C; HR, 140-160 beats/min; BP, 170-180/100 mm Hg; diaphoresis; flexor posturing	Labetalol hydrochloride	Paroxysmal sympathetic storm
Baguley,[1] 1999	14	NA	Hypoxia, TBI	NA	NA	NA	NA	Classic features described by Boeve et al[2] (1998)	NA	Paroxysmal sympathetic storms
Goh et al,[10] 1999	1	7	Midbrain glioma	3	NA	NA	NA	T, 38.4°C-39.5°C; HR, 150-180 beats/min; SBP, 180 mm Hg; DBP, 120 mm Hg; sweating, muscle spasms; decorticate	Phenytoin sodium, diazepam, lorazepam, clonidine	Diencephalic seizures

Arch Neurol 2004;61:321-328.

Inconsistent Results

		Ineffective	Beneficial
Opioids	• Morphine	5	11
Beta-blockers	• Propranolol	4	8
Alpha-agonists	• Clonidine	7	7
GABA-agonists	• Benzodiazepines	9	16
	• Baclofen (IT)	1	7
	• Gabapentin	0	2
Dopaminergic	• Bromocriptine	5	8
Calcium	• Dantrolene	5	2

Brain Injury 2004;18:409-417.
Ann Neurol 2010;68:126–135.

Morphine / Midazolam

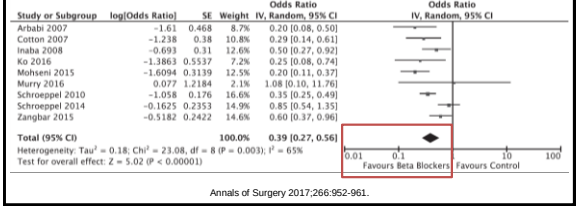
- Retrospective case control
- Severe TBI patients
- 35 cases and controls
- PSH definition
  - Simultaneous, paroxysmal increases in 5/7 reported features (HR, RR, BP, temperature, posturing, dystonia, sweating)
- PSH group
  - Majority treated with morphine (28/35) and midazolam (20/35) infusions on days 1-4

Feature	On	Off	p-Value
HR (beats/min)	108.8	120.2	<0.005
RR (breaths/min)	13.8	27.7	<0.005
Temperature (◦C)	38.5	38.3	NS

Brain Injury 2004;18:409-417.

Beta-Blockers

- Systematic review and meta-analysis
- Comparator: TBI patients who received any beta-blocker in-hospital after injury to those who did not
- Outcomes: **In-hospital mortality**, functional recovery, quality of life, cardiopulmonary adverse effects



Annals of Surgery 2017;266:952-961.

EAST Practice Management Guideline

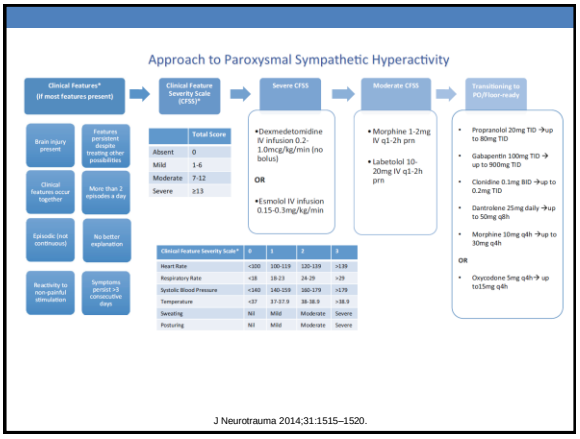
- In adults with severe TBI admitted to the ICU with no contraindications for beta-blockers, we conditionally recommend the use of in-hospital beta-blockers provided that hypotension and bradycardia are avoided
  - Hypotension: systolic blood pressure (SBP) < 90 mmHg
  - Bradycardia: heart rate (HR) < 50 with symptoms

Annals of Surgery 2017;266:952-961.

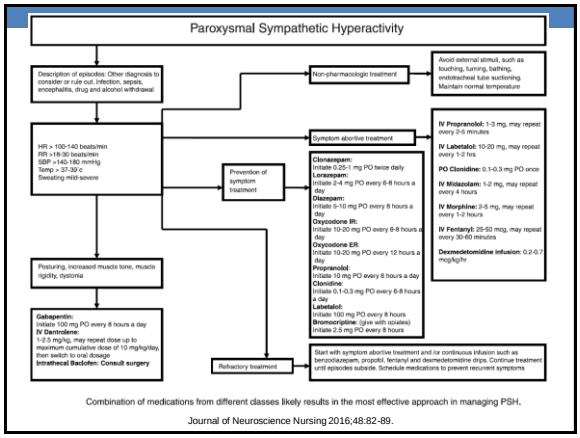
Propranolol vs. Other Beta-Blockers

Outcome	Propranolol	Other	p-Value
	(n=78)	(n=349)	
In-Hospital Mortality	3%	15%	0.002
	(n=354)	(n=766)	
30-Day Mortality	9%	16%	<0.01

J Trauma Acute Care Surg 2014;76:504-509.
J Trauma Acute Care Surg 2018;84:234-244.

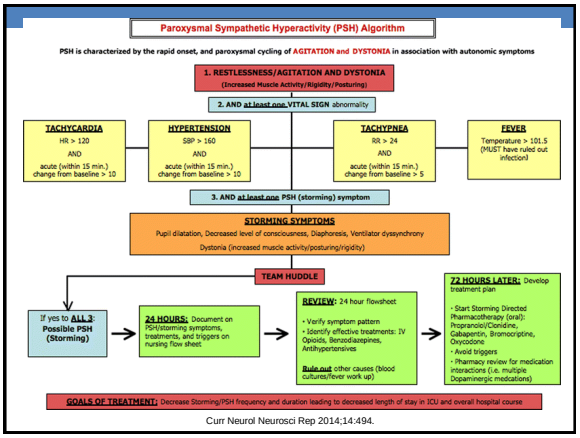


J Neurotrauma 2014;31:1515–1520.

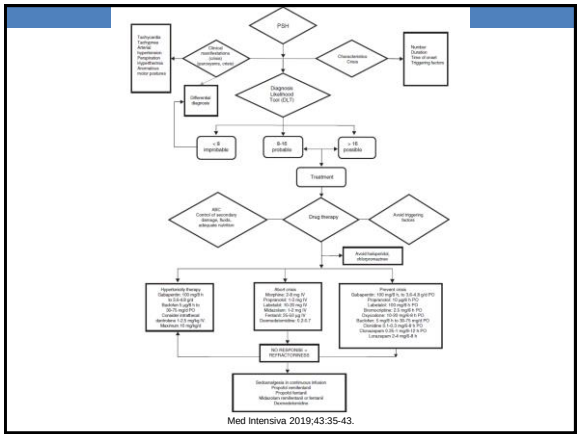


Combination of medications from different classes likely results in the most effective approach in managing PSH.

Journal of Neuroscience Nursing 2016;48:82-89.



Curr Neurol Neurosci Rep 2014;14:494.



Med Intensiva 2019;43:35-43.

Treatment Take Aways

Most patients will require a combination of drugs to target various symptoms of the syndrome

First Line	
Propranolol	Scheduled once hemodynamically stable
Morphine	As needed to abort symptoms
Lorazepam, Diazepam	As needed to abort symptoms
Second Line Add-ons	
Clonidine	Scheduled for persistent hypertension or tachycardia
Bromocriptine	Alternative in patients who cannot tolerate propranolol/clonidine
Refractory Add-ons	
Gabapentin	Scheduled for persistent spasticity, allodynia
Baclofen IT	Scheduled for persistent spasticity, dystonia
Dantrolene	Scheduled for persistent posturing

Other Considerations

- Physiotherapy
- Positioning to prevent contractures
- Temperature
- Nutrition
- Hyperbaric oxygen

Lancet Neurol 2017;16:721–729.

Take Home Points

- The accepted term for transient increases in sympathetic and motor activity is paroxysmal sympathetic hyperactivity (PSH)
- PSH is diagnosed using a two part assessment that looks at the severity and the frequency/duration of sympathetic hyperactivity symptoms
- Treatment goals include minimizing triggers, mitigating excessive sympathetic outflow, and addressing the effects of PSH through supportive therapy
- No consensus guidelines or accepted treatment algorithms for PSH exist
- A variety of pharmacologic agents can be used to achieve these goals with the most commonly used medications being opioids, benzodiazepines, and non-selective beta-blockers

PAROXYSMAL SYMPATHETIC HYPERACTIVITY

Kimberly Clark, PharmD, BCCCP
Pharmacy Clinical Specialist – Critical Care, Prisma Health
Adjunct Assistant Professor, University of South Carolina College of Pharmacy
Clinical Assistant Professor, University of South Carolina Greenville School of Medicine

kim.clark2@prismahealth.org