# Paroxysmal Sympathetic Hyperactivity Syndrome in the Setting of Fat Emboli Syndrome

A Case Report of Polytrauma in an Adolescent Female

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**Key Conditions**

**Paroxysmal Sympathetic Hyperactivity (PSH)**

**Complication**

**Fat Embolism Syndrome (FES)**

**Clinical Setting**

**Polytrauma**

# Introduction & Overview

## Case Background

15-year-old female with **polytrauma**

Multiple long bone fractures

Development of **fat embolism syndrome**

Complicated by **paroxysmal sympathetic hyperactivity**

## Key Topics

Background on PSH and FES

Diagnostic challenges and tools

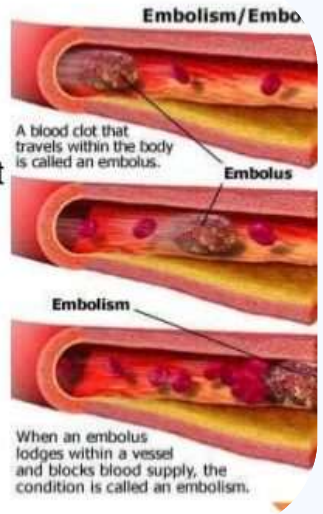
Treatment approaches

Clinical implications

**Fat Emboli:** Fat particles or droplets that travel through the circulation

**Fat Embolism**: A process by which fat emboli passes into the bloodstream and lodges within a blood vessel.

**Fat Embolism Syndrome (FES):** serious manifestation of fat embolism occasionally causes multi system dysfunction, the lungs are always involved and next is brain



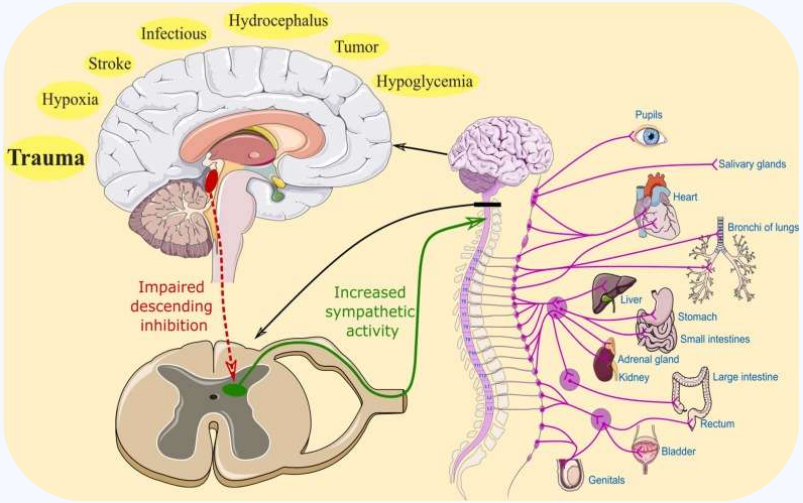
# Background on Paroxysmal Sympathetic Hyperactivity

## Definition

- Colloquially known as **"neuro-storming"**
- Potentially **life-threatening** complication after traumatic brain injuries
- Results from massive release of catecholamines in the brain

## Clinical Features

- **Tachycardia** and hypertension
- **Tachypnea** and diaphoresis
- **Posturing** and fever




**Diagnostic Challenge**

Patients may experience all or only some features, making diagnosis challenging

# Background on Fat Embolism Syndrome

## Definition

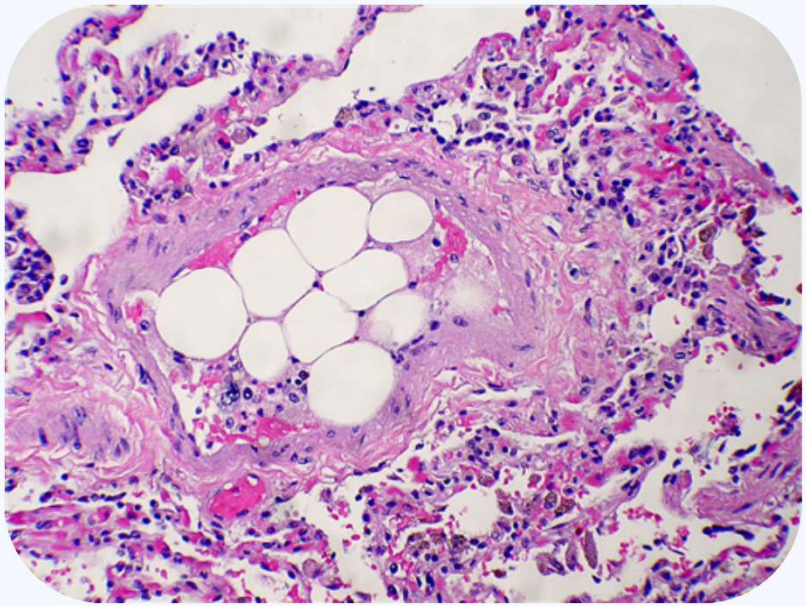
- Complication of **long bone fractures**
- Results from fat emboli entering circulation
- Can affect **pulmonary** and **cerebral** systems

## Clinical Manifestations

- **Pulmonary:** Respiratory failure, hypoxia
- **Cerebral:** Neurologic deficits, confusion
- **Cutaneous:** Petechial rash

**Risk Factors**

Multiple long bone fractures, pelvic fractures, delayed fixation, young age



# Case Presentation - Initial Assessment

**Previously Healthy Female**

15 years old

## Mechanism of Injury

- **High-speed motor vehicle collision**
- Restrained passenger
- Front-end collision with **significant vehicle damage**
- Both airbags deployed

## Initial Findings

- Glasgow Coma Scale: **15**
- Transient loss of consciousness
- Amnestic to events

**Initial Imaging**

CT head and cervical spine negative upon arrival

# Case Presentation - Injuries

## Skeletal Injuries

- **Displaced comminuted fracture** of left femur
- Multiple pelvic fractures
- Bilateral tibia/fibula fractures
- Left side: **significantly comminuted**

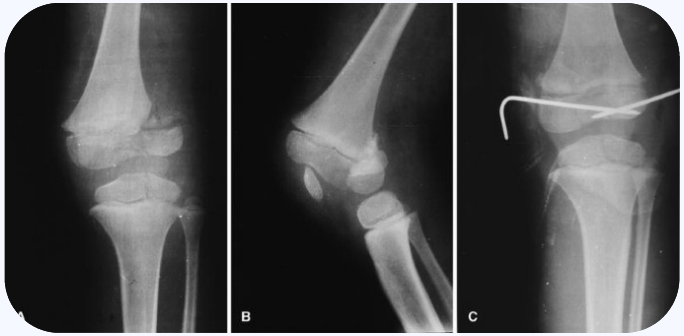
## Other Injuries

- Small left apical pneumothorax (<5%)
- Grade 2 liver laceration
- Two left rib fractures
- Multiple ecchymoses

**Initial Management**

Left lower extremity placed in traction upon admission to ICU


Displaced comminuted fracture of left femur




Tibia/fibula fractures with internal fixation

# Case Presentation - Neurological Decline

**9 hours**

### Neurological Status Change

GCS dropped from **15 to 7**

Only opening eyes and **extensor posturing** to pain

**Immediate**

### Interventions

**Intubated** for airway protection

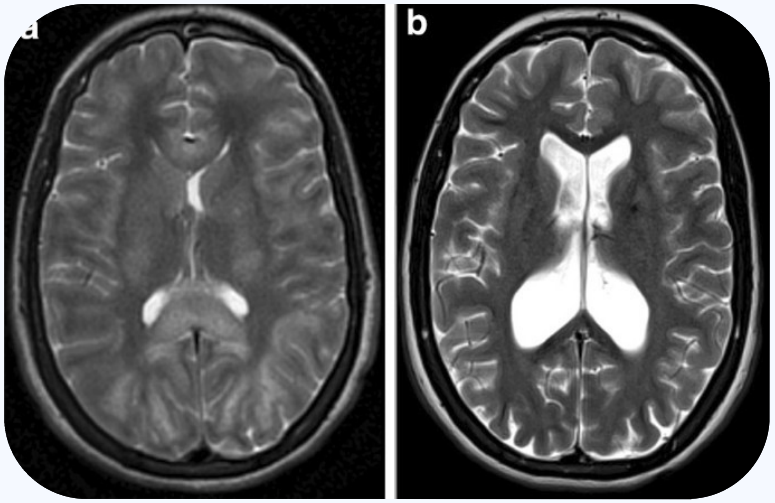
Urgent repeat CT imaging with angiography

**Results**

### Imaging Findings

No worsening bleeding or new injuries

No central pulmonary embolism




Brain imaging showing changes after neurological decline

### Laboratory Findings

| WBC | Lactic Acid | CK |
|---|---|---|
| 22.11 | 4.6 | 3,216 |

### Seizure Management

Patient loaded with Keppra and EEG ordered despite no visualized seizures

# Case Presentation - Imaging Findings

| Day 0 | Day 0 | Day 2 | Day 3 |
|---|---|---|---|
| Initial CT | Repeat CT | MRI | Echo |

## CT Scan Findings

- Initial CT: **Negative** for intracranial pathology
- Repeat CT: No new injuries or bleeding
- No central pulmonary embolism

## MRI Findings

- **Innumerable multifocal bilateral foci** of restricted diffusion
- Associated areas of cytotoxic edema
- Possibly representing **fat embolism phenomenon**

**EEG Results**

Negative for seizures or epileptiform activity

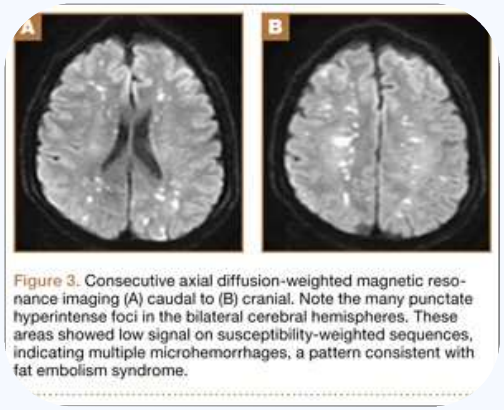
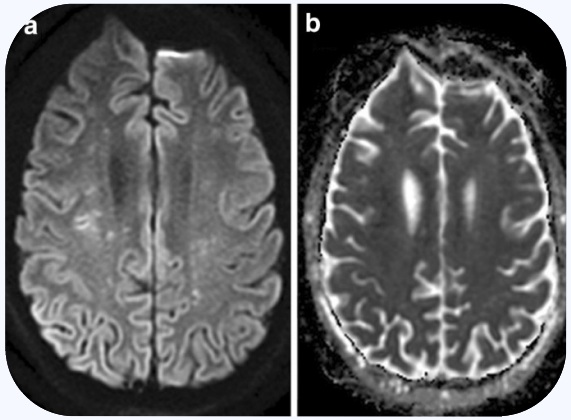



Figure 3. Consecutive axial diffusion-weighted magnetic resonance imaging (A) caudal to (B) cranial. Note the many punctate hyperintense foci in the bilateral cerebral hemispheres. These areas showed low signal on susceptibility-weighted sequences, indicating multiple microhemorrhages, a pattern consistent with fat embolism syndrome.

MRI brain showing multiple hyperintense foci representing fat emboli




MRI with DWI and ADC sequences showing fat emboli

# Case Presentation - MRI Findings

## MRI Brain Results

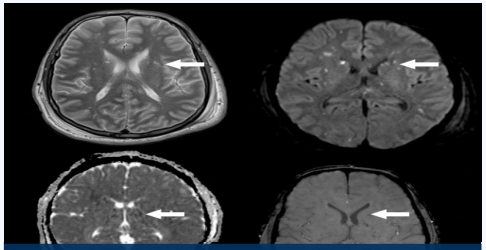
- **Innumerable multifocal bilateral foci** of restricted diffusion
- Associated areas of **cytotoxic edema**
- Findings consistent with **fat embolism phenomenon**

### Diagnostic Considerations

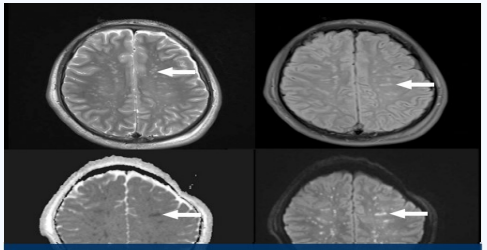
- Fat embolism syndrome - **Most likely**
- Diffuse axonal injury - Less likely
- Infection/Sepsis - No source identified

### Key Finding

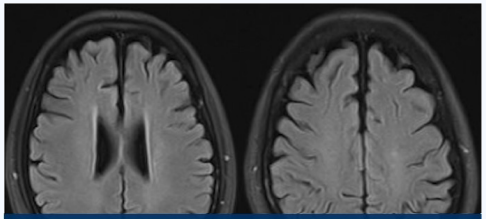
Subacute change in mental status with initial negative head CT makes FES the leading diagnosis



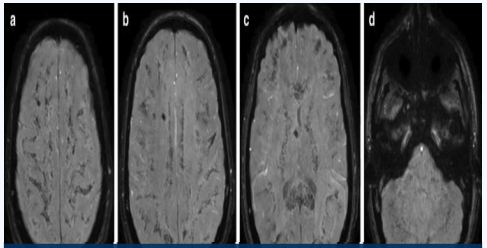
White matter hyperintense lesions



White matter lesions with arrows



FLAIR sequence: periventricular abnormalities




Multiple lesions in different sequences

### Timeline Correlation

MRI findings correlate with timeline of subacute brain injury following long bone fractures

# Case Presentation - Differential Diagnosis

### Fat Embolism Syndrome

**Most likely diagnosis** based on:

- Subacute change in mental status
- Multiple long bone fractures
- MRI findings consistent with fat emboli

### Diffuse Axonal Injury

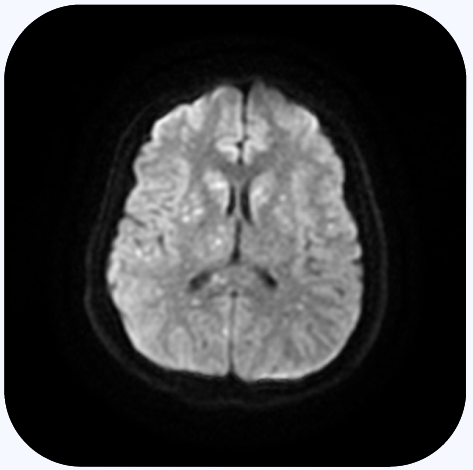
**Ruled out** because:

- Initial head CT was negative
- No intracranial hemorrhage or edema

### Infection/Sepsis

**Ruled out** because:

- No hollow viscous injury on abdominal CT
- No intra-abdominal pathology on repeat CTs
- No other infectious etiology identified



### Diagnostic Timeline

The **subacute onset** of neurological symptoms following long bone fractures with initial negative head CT strongly supports FES as the primary diagnosis

### Key Factor

The timeline of symptom onset correlated with the pathophysiology of fat emboli formation and migration to cerebral circulation

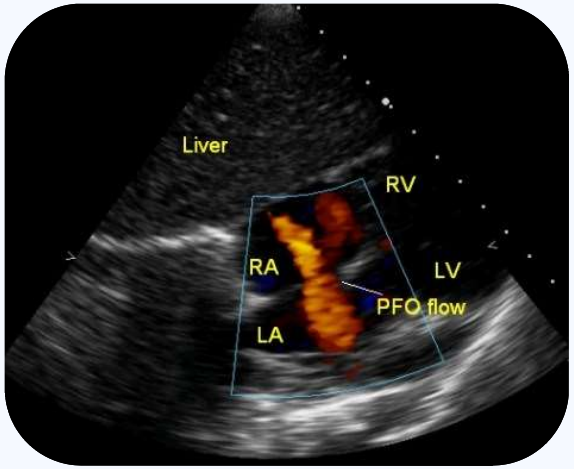
# Case Presentation - PFO Discovery

## Echocardiogram Findings

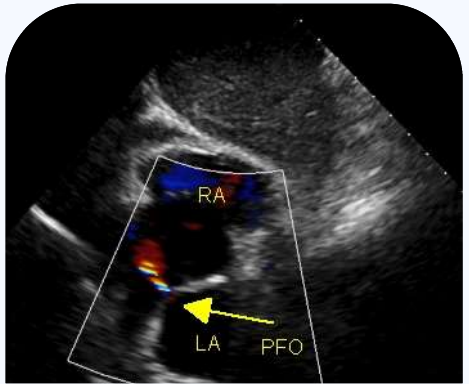
- Performed on **ICU Day 3**
- Detected **patent foramen ovale (PFO)**
- Left ventricle ejection fraction: **55-65%**
- No valvular lesions

## Clinical Significance

- PFO provides **potential pathway** for fat emboli to bypass pulmonary filtration
- May explain **cerebral fat emboli** despite absence of pulmonary embolism




Echocardiogram showing blood flow through patent foramen ovale




**12%** PFO identified in only 12% of patients with fat embolism syndrome

**Important Note**

Absence of PFO does not preclude development of FES

# Case Presentation - PSH Symptoms

**ICU Day 4** Onset of PSH symptoms

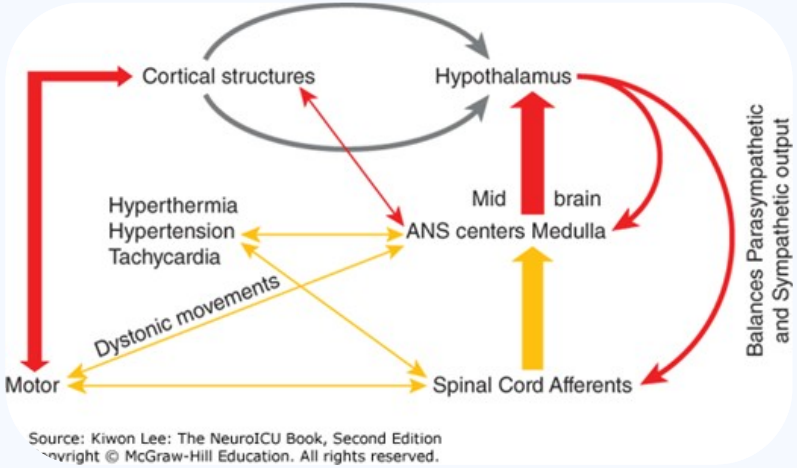
## Clinical Manifestations

**Tachycardia**
147 bpm

**Hypertension**
150/99 mmHg

**Tachypnea**
27 breaths/min

**Temperature**
37.9°C




Brain structures and autonomic nervous system interactions in PSH

## Additional Symptoms

- Mechanical asynchrony with breathing
- Muscle spasms with **extensor posturing**
- **Diaphoresis** (excessive sweating)

**PSH Diagnostic Score**

Patient scored **18** on the PSH diagnostic likelihood tool, confirming probable diagnosis

**Diagnostic Challenge**

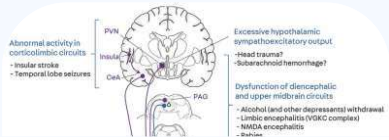
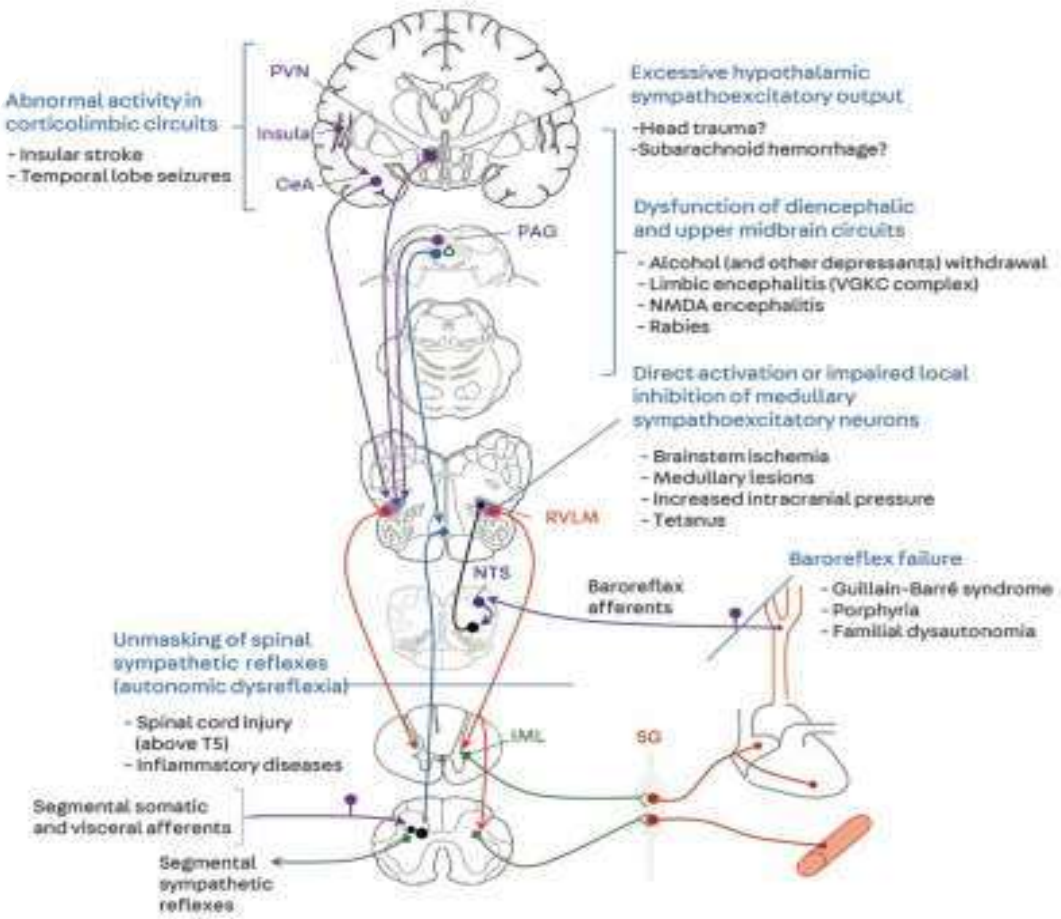


Table 1: Paroxysmal sympathetic hyperactivity (PSH) diagnostic likelihood tool [1].

| | Clinical Feature Scale (CFS) | | | | Score |
|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | |
| Heart rate | <100 | 100-119 | 120-139 | >140 | **3** |
| Respiratory rate | <18 | 18-23 | 24-29 | >30 | **2** |
| Systolic blood pressure | <140 | 140-159 | 160-179 | >180 | **2** |
| Temperature | <37 | 37-37.9 | 38-38.9 | >39 | **1** |
| Sweating | Nil | Mild | Moderate | Severe | **2** |
| Posturing during episodes | Nil | Mild | Moderate | Severe | **2** |
| | | | | CFS subtotal | **12** |
| Severity of clinical features | Nil | | 0 | | |
| | Mild | | 1-6 | | |
| | **Moderate** | | **7-12** | | |
| | Severe | | ≥13 | | |
| Diagnosis likelihood tool (DLT)—1 point for each feature present | | | | | |
| Clinical features occur simultaneously | | | | | **1** |
| Episodes are paroxysmal in nature | | | | | **1** |
| Sympathetic over-reactivity to normally nonpainful stimuli | | | | | **1** |
| Features persist ≥3 consecutive days | | | | | **1** |
| Features persist ≥2-week postbrain injury | | | | | **0** |
| Features persist despite treatment of alternative differential diagnoses | | | | | **0** |
| Medication administered to decrease sympathetic features | | | | | **1** |
| ≥2 episodes daily | | | | | **1** |
| Absence of parasympathetic features during episodes | | | | | **0** |
| Absence of other presumed cause of features | | | | | **0** |
| Antecedent of acquired brain injury | | | | | **0** |
| | | | | DLT subtotal | **6** |
| Combined total (CFS+DLT) | | | | | |
| PSH diagnostic likelihood | Unlikely | | <8 | | |
| | Possible | | 8-16 | | |
| | **Probable** | | **≥17** | | |

Based on the PSH diagnostic likelihood tool, the patient has a Clinical Feature Scale of 12 which indicates moderate severity of clinical features and a diagnosis likelihood tool of 6. The combined total scores equal 18 which indicates that PSH is the probable diagnosis. The patient's score is calculated in bold values.




**Schematic representation of the levels of neuraxial injury and proposed mechanisms for the development of autonomic hyperactivity.**

CeA = central nucleus of the amygdala; IML = intermediolateral cell column; NMDA = *N*-methyl-D-aspartate
TS = nucleus of the solitary tract; PAG = periaqueductal gray; PVN = paraventricular nucleus; RVLM =
al ventrolateral medulla; SG = sympathetic ganglion; VGKC = voltage-gated potassium channel.
d with permission from Benarroch EE.[1] © Oxford University Press.

Rabinstein AA, P

# PSH Diagnostic Tool

## Diagnostic Likelihood Tool

### Clinical Feature Scale (CFS)

### Diagnosis Likelihood Tool (DLT)

*Diagnosis Likelihood Tool (DLT)*

| | |
|---|---|
| Clinical features occur simultaneously | |
| Episodes are paroxysmal in nature | |
| Sympathetic over-reactivity to normally non-painful stimuli | |
| Features persist ≥3 consecutive days | |
| Features persist ≥2 weeks post -brain injury | |
| Features persist despite treatment of alternative differential diagnoses | |
| Medication administered to decrease sympathetic features | |
| ≥2 episodes daily | |
| Absence of parasympathetic features during episodes | |
| Absence of other presumed cause of features | |
| Antecedent acquired brain injury | |
| (Score 1 point for each feature present) | DLT subtotal |

**Diagnostic Categories**

- **Unlikely:** Score < 8
- **Possible:** Score 8-16
- **Probable:** Score ≥ 17

**Patient's Result**

With a score of **18**, the patient's PSH diagnosis is classified as **probable**

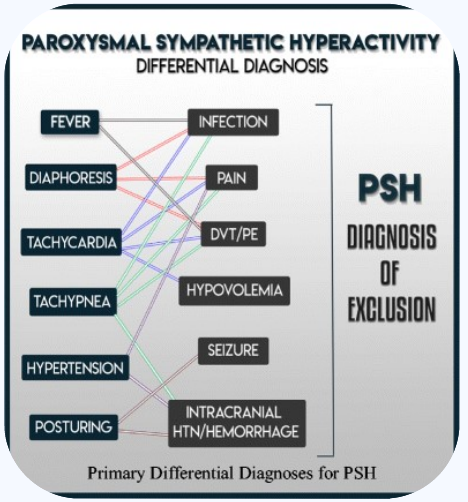
*Diagnosis Likelihood Tool (DLT)*

| | |
|---|---|
| Clinical features occur simultaneously | |
| Episodes are paroxysmal in nature | |
| Sympathetic over-reactivity to normally non-painful stimuli | |
| Features persist $\geq 3$ consecutive days | |
| Features persist $\geq 2$ weeks post -brain injury | |
| Features persist despite treatment of alternative differential diagnoses | |
| Medication administered to decrease sympathetic features | |
| $\geq 2$ episodes daily | |
| Absence of parasympathetic features during episodes | |
| Absence of other presumed cause of features | |
| Antecedent acquired brain injury | |
| (Score 1 point for each feature present) | DLT subtotal |

# PSH Diagnostic Tool (continued)

## Patient's Diagnostic Score

### Clinical Feature Scale (CFS)



Primary Differential Diagnoses for PSH

**Diagnostic Interpretation**

Score of **18** indicates **probable** PSH diagnosis (≥17 points)

### Diagnosis Likelihood Tool (DLT)

# Case Presentation - Treatment

## Pharmacological Management

### Propranolol

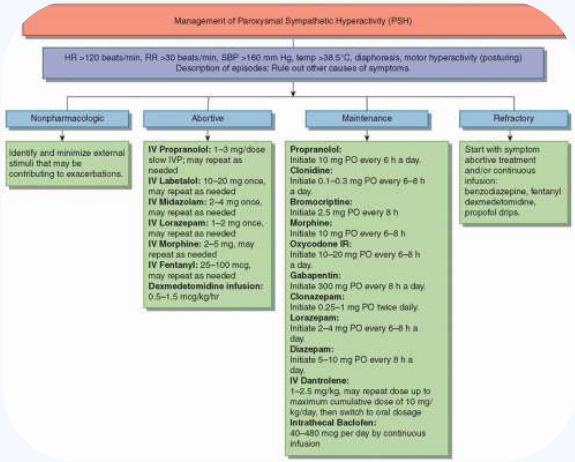
**20mg** three times daily

Beta-blocker to control sympathetic hyperactivity

### Oxycodone

Scheduled every **4 hours**

Adequate pain control to reduce sympathetic response




Medication management approach for PSH

## Additional Interventions

- **Seizure prophylaxis** with Keppra due to cerebral edema
- EEG monitoring to rule out seizure activity
- Aggressive **physical therapy** and speech therapy

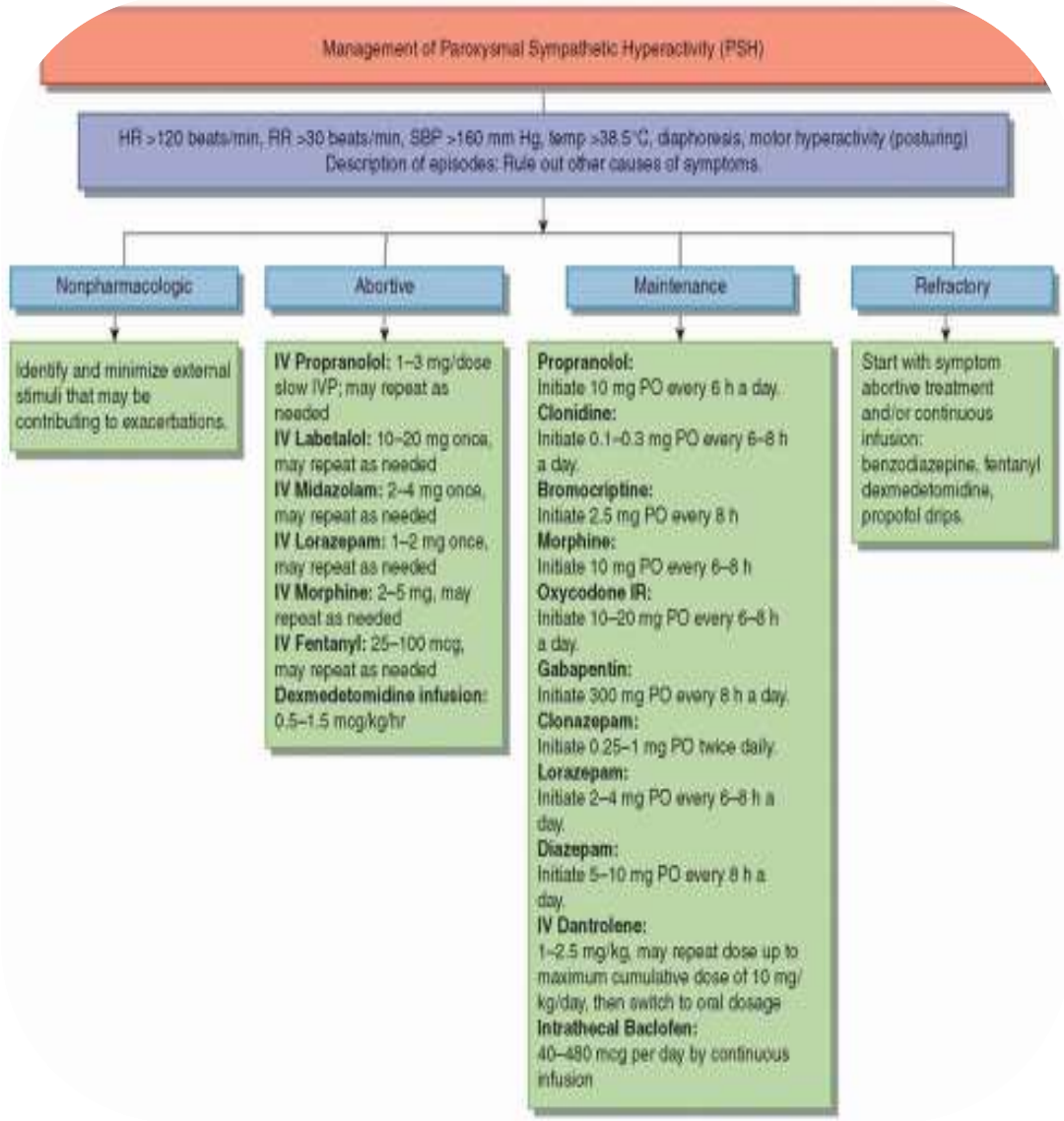
## Treatment Response

Frequency of PSH episodes **decreased** over the next few days

Patient became appropriate for definitive orthopedic fixation on ICU day 7

### Alternative Treatment Options

- **Steroids** (dexamethasone, methylprednisolone)
- **Albumin** to bind fatty acids
- **5% alcohol glucose** solution
- **Gabapentin** as adjunct therapy


Management of Paroxysmal Sympathetic Hyperactivity (PSH)
HR >120 beats/min, RR >30 beats/min, SBP >160 mm Hg, temp >38.5°C, diaphoresis, motor hyperactivity (posturing)
Description of episodes: Rule out other causes of symptoms.
Nonpharmacologic
Abortive
Maintenance
Refractory
Identify and minimize external stimuli that may be contributing to exacerbations.
IV Propranolol: 1–3 mg/dose slow IVP; may repeat as needed
IV Labetalol: 10–20 mg once, may repeat as needed
IV Midazolam: 2–4 mg once, may repeat as needed
IV Lorazepam: 1–2 mg once, may repeat as needed
IV Morphine: 2–5 mg, may repeat as needed
IV Fentanyl: 25–100 mcg, may repeat as needed
Dexmedetomidine infusion: 0.5–1.5 mcg/kg/hr
Propranolol:
Initiate 10 mg PO every 6 h a day.
Clonidine:
Initiate 0.1–0.3 mg PO every 6–8 h a day.
Bromocriptine:
Initiate 2.5 mg PO every 8 h
Morphine:
Initiate 10 mg PO every 6–8 h
Oxycodone IR:
Initiate 10–20 mg PO every 6–8 h a day.
Gabapentin:
Initiate 300 mg PO every 8 h a day.
Clonazepam:
Initiate 0.25–1 mg PO twice daily.
Lorazepam:
Initiate 2–4 mg PO every 6–8 h a day.
Diazepam:
Initiate 5–10 mg PO every 8 h a day.
IV Dantrolene:
1–2.5 mg/kg, may repeat dose up to maximum cumulative dose of 10 mg/kg/day, then switch to oral dosage
Intrathecal Baclofen:
40–480 mcg per day by continuous infusion
Start with symptom abortive treatment and/or continuous infusion: benzodiazepine, fentanyl dexmedetomidine, propofol drips.

# Case Presentation - Surgical Interventions

## Surgical Timeline

**Day 3**

### Initial Orthopedic Procedures

**Internal fixation** of right ankle

**External fixation** of left femur

**Day 7**

### Definitive Orthopedic Fixation

Performed after hemodynamic and neurologic stabilization

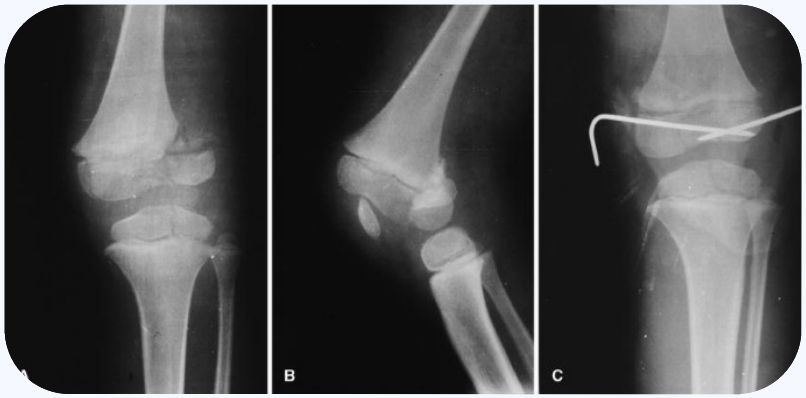
Decreased PSH episodes enabled surgery

**Later**

### Morel-Lavallée Lesion Management

**Operative evacuation** of hematoma

Placement of two JP drains




Internal fixation of orthopedic injuries

## Surgical Management Goals

- Stabilize fractures to **reduce fat emboli** risk
- Address **Morel-Lavallée lesion** from seatbelt injury
- Optimize timing based on neurological status

### Surgical Considerations

### Outcome

# Case Presentation - Recovery

## Recovery Milestones

**Day 4**

### Neurological Improvement

Spontaneous eye opening, improved movements

**Day 18**

### Successful Extubation

Airway protection maintained

**Day 23**

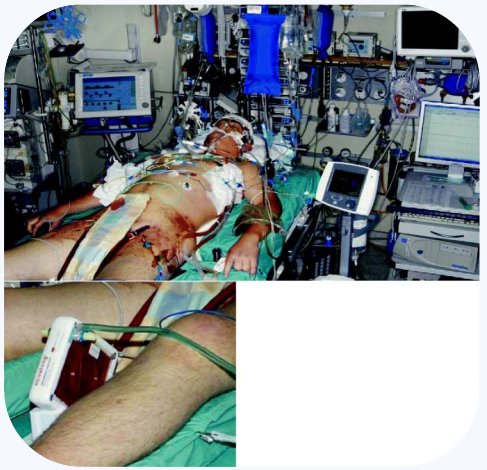
### ICU Transfer

Moved to floor for continued recovery

**Day 39**

### Discharge

Home with outpatient follow-up



## Rehabilitation Plan

**Aggressive physical therapy** for mobility
**Speech therapy** for communication
Continued **home physical therapy**

**18**
Days on ventilator

**23**
Days in ICU

**39**
Total hospital stay

### Discharge Plan

Home with outpatient orthopedic follow-up and continued home physical

# Discussion - FES Diagnosis

## Diagnostic Process

### Timeline Correlation

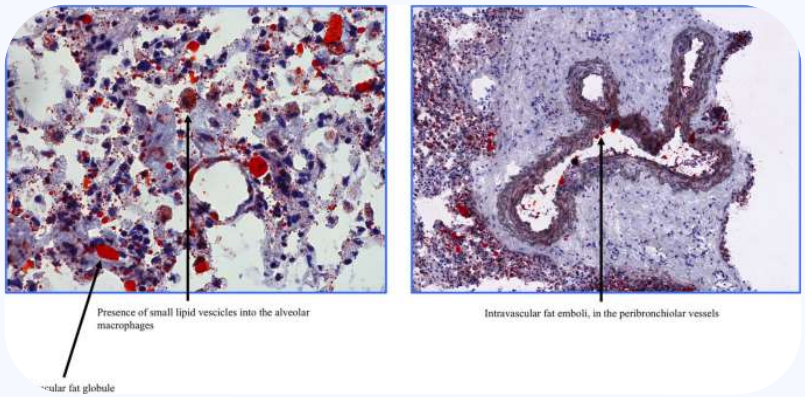
**Subacute change** in mental status following long bone fractures

### Exclusion of Alternatives

Initial negative head CT ruled out **diffuse axonal injury**

### MRI Confirmation

Multiple hyperintense foci representing **fat emboli**




Microscopic view of lung tissue showing fat emboli

## Key Diagnostic Factors

- Multiple long bone fractures
- Subacute neurological decline
- PFO facilitating cerebral emboli
- MRI with characteristic findings

### Diagnostic Challenge

FES diagnosis in polytrauma patients requires **comprehensive evaluation** of history, clinical findings, and imaging

### Role of PFO

PFO provided pathway for fat emboli to bypass pulmonary filtration, explaining **cerebral involvement** without significant pulmonary embolism

# Discussion - PSH Diagnosis

## Diagnostic Challenges

### Symptom Overlap

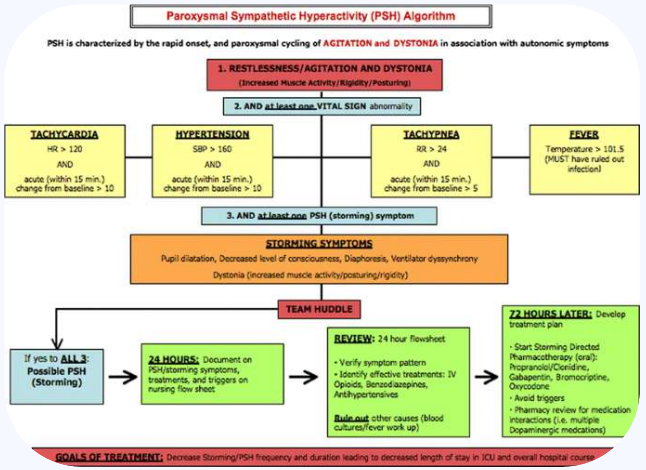
PSH symptoms similar to **pain response** in polytrauma patients

### Variable Presentation

Patients may experience **all or only some** PSH features

### Differential Diagnosis

Multiple potential causes in polytrauma: **sepsis, PE, withdrawal**




PSH diagnostic algorithm showing systematic approach to diagnosis

## Diagnostic Tool Utility

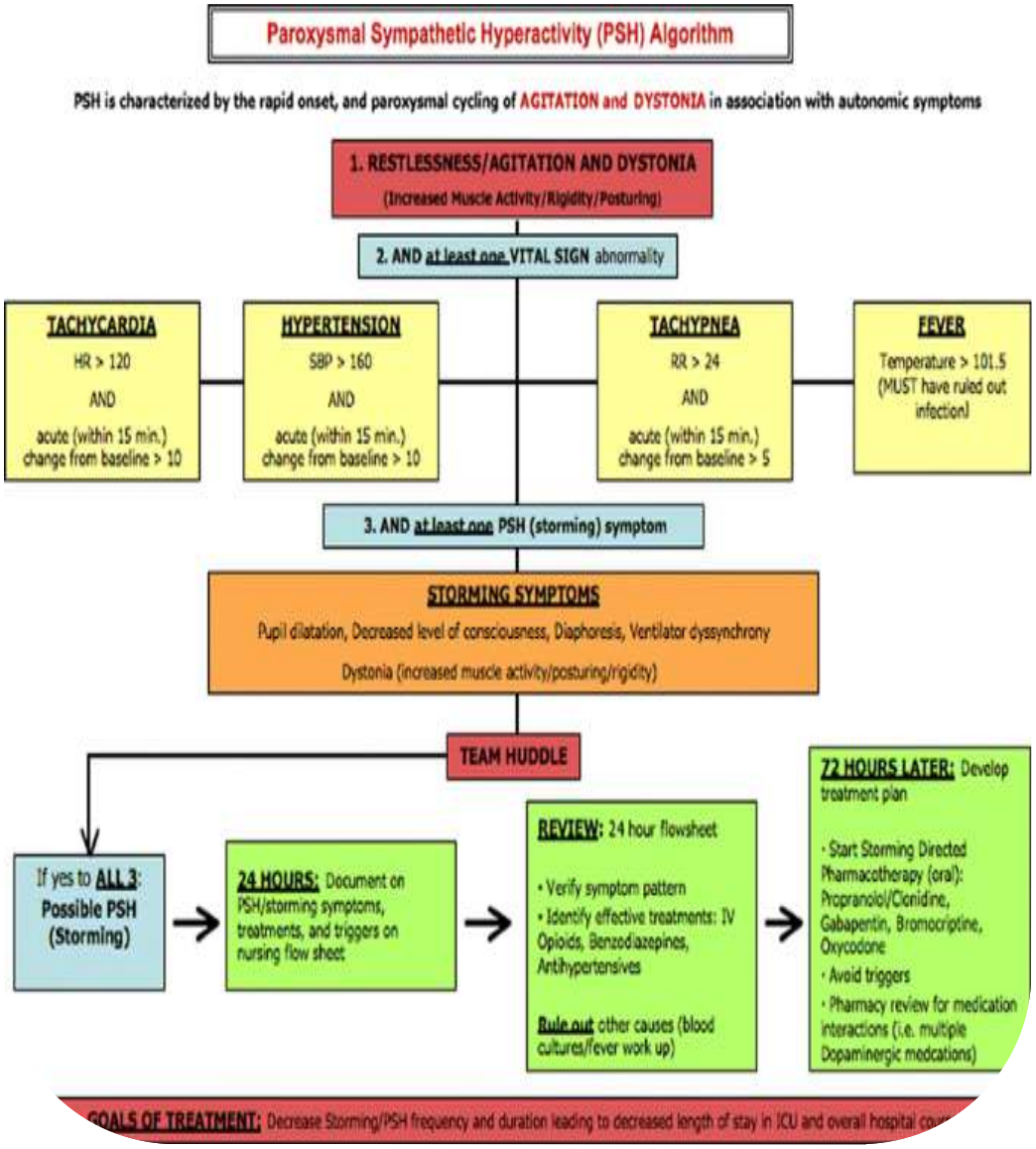
- Standardized assessment
- Quantitative scoring
- Temporal criteria
- Objective confirmation

### Case Application

Patient scored **18** on PSH diagnostic tool, confirming probable diagnosis despite initial challenges

### Clinical Impact

Early recognition enabled targeted treatment with **propranolol and pain**


Paroxysmal Sympathetic Hyperactivity (PSH) Algorithm
PSH is characterized by the rapid onset, and paroxysmal cycling of AGITATION and DYSTONIA in association with autonomic symptoms
1. RESTLESSNESS/AGITATION AND DYSTONIA
(Increased Muscle Activity/Rigidity/Posturing)
2. AND at least one VITAL SIGN abnormality
TACHYCARDIA
HR > 120
AND
acute (within 15 min.) change from baseline > 10
HYPERTENSION
SBP > 160
AND
acute (within 15 min.) change from baseline > 10
TACHYPNEA
RR > 24
AND
acute (within 15 min.) change from baseline > 5
FEVER
Temperature > 101.5
(MUST have ruled out infection)
3. AND at least one PSH (storming) symptom
STORMING SYMPTOMS
Pupil dilatation, Decreased level of consciousness, Diaphoresis, Ventilator dyssynchrony
Dystonia (increased muscle activity/posturing/rigidity)
TEAM HUDDLE
If yes to ALL 3: Possible PSH (Storming)
24 HOURS: Document on PSH/storming symptoms, treatments, and triggers on nursing flow sheet
REVIEW: 24 hour flowsheet
• Verify symptom pattern
• Identify effective treatments: IV Opioids, Benzodiazepines, Antihypertensives
Rule out other causes (blood cultures/fever work up)
72 HOURS LATER: Develop treatment plan
• Start Storming Directed Pharmacotherapy (oral): Propranolol/Clonidine, Gabapentin, Bromocriptine, Oxycodone
• Avoid triggers
• Pharmacy review for medication interactions (i.e. multiple Dopaminergic medications)
GOALS OF TREATMENT: Decrease Storming/PSH frequency and duration leading to decreased length of stay in ICU and overall hospital

# Discussion - Treatment Approaches for FES

## Pharmacological Options

### Steroids

**Dexamethasone** or methylprednisolone

Reduces cerebral edema

### Albumin

Binds with **fatty acids**

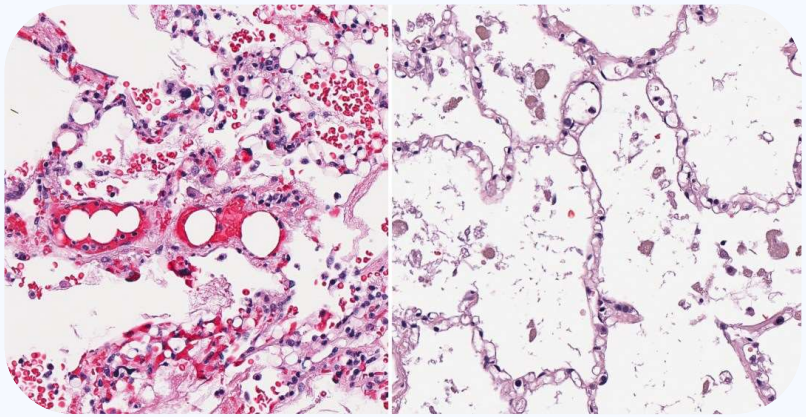
Restores blood volumes

### 5% Alcohol Glucose

Inhibits formation of **fat droplets**

### Lipid-soluble Drugs

Target fat emboli directly



Comparison of lung tissue with and without fat emboli

## Supportive Care

- **Early immobilization** of fractures with traction
- **Respiratory support** for pulmonary involvement
- **Neurological monitoring** for cerebral changes

### Treatment Goals

Reduce load of fat emboli and minimize cerebral edema to limit PSH episodes

## Mechanisms of Action

- **Anti-inflammatory** effects reduce tissue damage

### Treatment Limitations

**Methylprednisolone**

**1.5 to 2.0 mg/kg**, iv, every 8 hours for 3-6 doses


**Albumin**

**5% or 25% solution, with an initial bolus of 300-500 mL**

# Discussion - Treatment Approaches for PSH

## Pharmacological Management

### Beta-blockers

**Propranolol**

Reduces sympathetic activity

### Opioids

**Oxycodone**

Pain control to reduce sympathetic response

### Gabapentin

Adjunct therapy for **neuropathic pain**

### Benzodiazepines

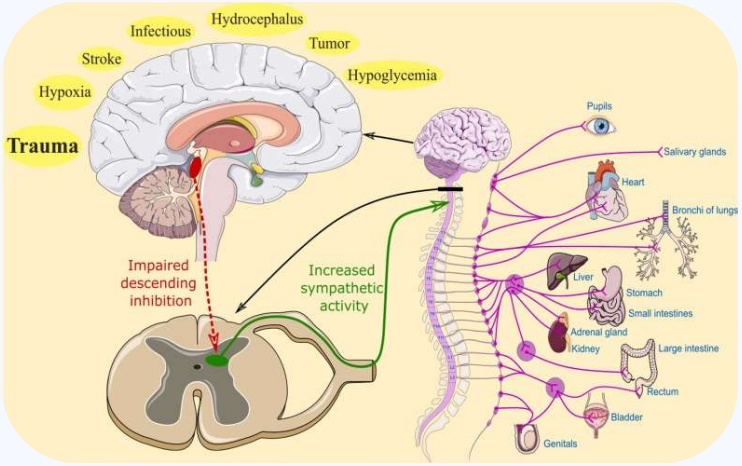
**Diazepam**

Reduces muscle spasms and anxiety

**Treatment Principles**

Control sympathetic hyperactivity to limit long-term effects on brain and body

## Mechanisms of Action




Brain injury affecting spinal cord and increasing sympathetic activity

## Patient-Specific Considerations

- **Individualized dosing** based on response
- **Combination therapy** often required
- **Regular monitoring** of vital signs

# Discussion - Patient-Specific Considerations

## Unique Patient Factors

### Adolescent Patient

**15-year-old** female with developing physiology

Different drug metabolism and dosing requirements

### Extensive Polytrauma

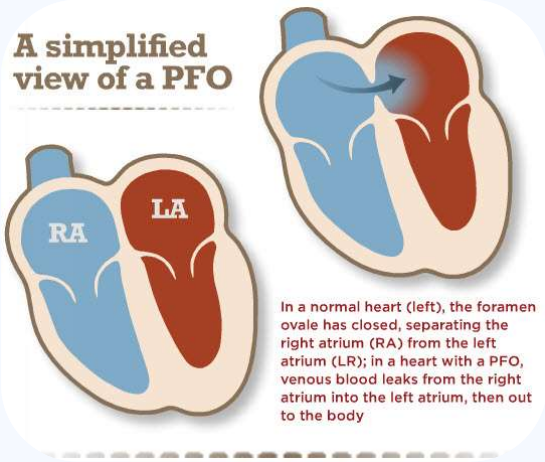
Multiple **long bone fractures** increased FES risk

Pain management complexity in PSH diagnosis

### Patent Foramen Ovale

Created **right-to-left shunt** for cerebral emboli

Explained cerebral FES without significant pulmonary involvement




Patent foramen ovale creating right-to-left shunt

## Management Implications

- **Individualized dosing** of medications based on age and response
- **Careful titration** of propranolol to avoid bradycardia
- **Balanced approach** to pain control vs. PSH symptoms
- **Multidisciplinary care** involving neurology, orthopedics, and critical care

# Discussion - Prevention Strategies

## Fat Embolism Prevention

### Early Immobilization

**Traction** of long bone fractures

Reduces fat release from marrow

### Prophylactic Corticosteroids

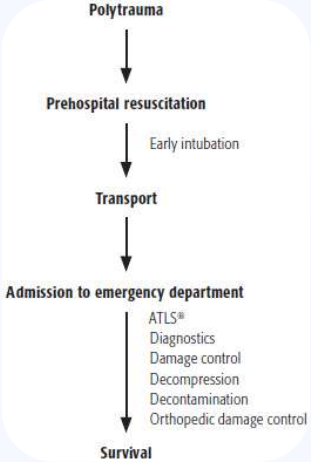
**Controversial** but may reduce incidence

Decreases inflammatory response

### Timely Surgical Fixation

**Early definitive care** when stable

Prevents continued fat release




Polytrauma management protocol emphasizing early intervention

## PSH Prevention

### Early Recognition

**High vigilance** in at-risk patients

### Adequate Pain Control

**Proactive management** reduces triggers

# Discussion - Research Gaps

## Key Knowledge Gaps

### Pathophysiology

Limited understanding of **mechanisms** linking FES to PSH

### Treatment Protocols

No **standardized guidelines** for FES or PSH management

### Population Studies

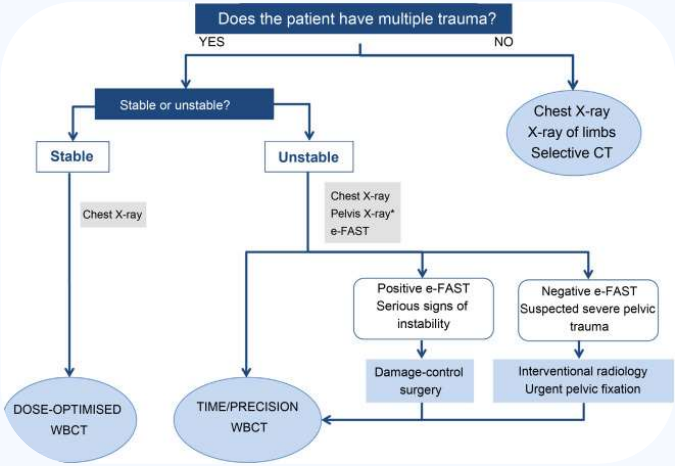
**Paucity of data** on PSH as complication of cerebral FES

**12%**

PFO in FES patients

**Limited**

Case reports available




Diagnostic flowchart for polytrauma patients

## Future Research Directions

- **Prospective studies** on FES-PSH relationship
- **Randomized trials** of treatment modalities
- **Biomarker development** for early detection

**Research Need**

# Conclusion - Key Points

## Case Highlights

### Rare Complication

**PSH in FES** is an uncommon but potentially life-threatening complication

### Diagnostic Tool Utility

**PSH diagnostic tool** enabled confirmation despite initial challenges

### PFO Significance

**Patent foramen ovale** provided pathway for cerebral fat emboli

### Critical Insight

Early recognition and targeted treatment of PSH in FES patients can significantly improve outcomes

## Clinical Takeaways

**Expedited diagnosis** leads to better outcomes

**Early immobilization** prevents FES

**Individualized treatment** for PSH episodes

**Multidisciplinary approach** essential

### Research Need

Further investigation required to establish standardized protocols for managing FES complicated by PSH

### Case Outcome

Patient achieved **positive recovery** through timely diagnosis and appropriate management

# Conclusion - Clinical Implications

## Practice Impact

### Early Recognition

**High vigilance** for PSH in FES patients improves outcomes

### Diagnostic Tools

**Standardized assessment** enables timely intervention

### Multidisciplinary Approach

**Coordinated care** essential for complex polytrauma cases

### Clinical Takeaway

PSH should be considered in all FES patients with neurological changes, even when initial imaging is negative

## Management Strategies

**Continuous monitoring** of vital signs

**Proactive treatment** with beta-blockers

**Early fixation** of long bone fractures

**Team communication** across specialties

### Research Implications

Need for standardized protocols and prospective studies on FES-PSH relationship

### Patient Outcomes

Timely diagnosis and appropriate management can lead to **positive recovery** even in severe cases

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**Note**

Additional data regarding this case report can be made upon request to the authors

# Questions & Answers

## Q&A Session

We welcome your questions and discussion

PSH Diagnosis

FES Management

Treatment Options

Patient Outcomes

# Thank You

For your attention and participation

**Early Recognition**

**Diagnostic Tools**

**Targeted Treatment**

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