Central Fever & Storming (PSH)

Two things the injured brain does · that get treated as sepsis for three weeks

Temp alone = central
everything together = storming

⚠ THE THREE THAT CARRY THE PAGE

⚠ FEVER ITSELF WORSENS THE OUTCOME

After **SAH, TBI and stroke**, fever is **independently associated with worse function and higher mortality**, even after adjusting for injury severity. **The injured brain has no metabolic reserve**: raising temperature raises cerebral metabolic demand, excitatory transmitter release and ICP exactly when perfusion is most precarious. **So normothermia is a treatment goal, not a comfort measure** -treat it as actively as you would a blood pressure.

⚠ BOTH ARE DIAGNOSES OF EXCLUSION

And the expensive error runs **both ways**: calling it central on day 2 and missing **ventriculitis**, or calling it sepsis for three weeks and never treating the storm. Exclude: **ventriculitis** (any EVD, shunt or basal skull fracture -**send CSF**), VAP, UTI, line, *C. difficile*, **sinusitis from an NG tube**, **VTE** (immobile, often no prophylaxis), **drug fever (phenytoin)**, withdrawal, seizure, and **NMS / serotonin syndrome / malignant hyperthermia**.

TELLING THE TWO APART

Central fever = isolated hyperthermia. A disturbed set point, without the rest of the autonomic picture. **PSH = fever PLUS a simultaneous surge** of tachycardia, hypertension, tachypnea, drenching sweating and posturing, arriving in **discrete paroxysms** rather than as a plateau. **If the temperature rises alone, think central. If everything rises together and then subsides, think storming.**

CENTRAL FEVER *Hocker 2013 · four features together predict it with a probability of 0.90*

Item	Detail
⚠ **The four features**	**Negative cultures** · **no infiltrate on chest radiograph** · a diagnosis of **SAH, IVH or tumor** · **fever onset within 72 hours of admission**. **The practical use is permission to STOP ANTIBIOTICS in a patient who is still febrile**, which is what prevents open-ended antimicrobial courses in someone who has no infection. **A febrile patient is not automatically an infected patient**
What it looks like	Often **above 39°C**. A **plateau with loss of the normal diurnal variation** -it sits high rather than swinging, which is a useful bedside discriminator. **Poor response to antipyretics**, often the first clue. Highest risk after **SAH with intraventricular extension**, tumor, TBI, and hypothalamic or brainstem lesions
⚠ **Why antipyretics barely work** the useful physiology	Ordinary fever is **PROSTAGLANDIN-mediated**: pyrogens drive PGE2, PGE2 raises the hypothalamic set point, antipyretics inhibit COX and lower that signal. **Central fever is not generated that way** -it is **direct structural or inflammatory disruption of hypothalamic thermoregulation**, so there is little prostaglandin signal to interrupt. **A fever that shrugs off acetaminophen is a CLUE, not a dosing problem**
Treatment	**Exclude infection properly first** -a cooled patient with ventriculitis just looks better while getting worse. Trial acetaminophen anyway (cheap, many fevers are mixed), but **expect little**. **PHYSICAL COOLING is the mainstay precisely because it bypasses the set-point pathway**; feedback-controlled devices hold target far better than ice packs. If refractory: **bromocriptine, propranolol, baclofen** have the most (still weak) support, and **propranolol + baclofen** beats baclofen alone
⚠ **Shivering undoes it all**	Shivering **raises metabolic rate, oxygen consumption and ICP** -the opposite of why you cooled. Score it with the **Bedside Shivering Assessment Scale**, then step through **acetaminophen, buspirone, magnesium, SKIN COUNTER-WARMING** (warm air to face and hands while cooling the core), then opioids and sedation. **Neuromuscular blockade LAST**, since it masks seizures and the examination

PAROXYSMAL SYMPATHETIC HYPERACTIVITY (STORMING)

Item	Detail
The definition	Baguley 2014 consensus replaced **more than 31 eponyms** (sympathetic storming, dysautonomia, diencephalic seizures) with one term. **Simultaneous, paroxysmal, TRANSIENT increases in SYMPATHETIC activity (HR, BP, RR, temperature, sweating) AND MOTOR activity (posturing)** after severe acquired brain injury
PSH-AM	**Clinical Feature Scale** grades severity of the six cardinal features: **HR, RR, SBP, temperature, sweating, posturing**. **Diagnosis Likelihood Tool** grades context (paroxysmal? triggered by non-painful stimuli? simultaneous? other causes excluded?). Combined, they give the **probability** of PSH -which forces a deliberate diagnosis instead of pattern-recognition after weeks of negative sepsis workups
Who, and when	Most often **severe TBI, especially diffuse axonal injury**; also **hypoxic-ischemic injury** (often more severe), stroke, hydrocephalus. ⚠ **Emerges days to weeks later, characteristically AS SEDATION IS WEANED** -which is exactly why it is misattributed to withdrawal, pain or a new infection
⚠ **Triggered by stimulation**	**Turning, suctioning, physiotherapy, pain, a distended bladder, constipation.** This is the **strongest diagnostic clue AND a genuine treatment target** -it earns its place twice
Mimics, and the tell	**Sepsis** -storming is **paroxysmal not sustained**, provoked by handling, workup stays negative. **Seizure** -posturing looks convulsive but there is **NO EEG correlate**. **NMS** -plausible, since these patients get antipsychotics, so **review the drug chart**. **Withdrawal** -identical timing, and the two genuinely coexist. Also PE, serotonin syndrome, thyroid storm, pheochromocytoma
⚠ **Cost of missing it**	Not just unpleasant to watch: **enormous catabolism and weight loss**, dehydration from sweating, **ICP spikes**, and **contractures with heterotopic ossification** from sustained posturing. Longer stay, worse function. **Treating it is rehabilitation, not sedation for the staff's benefit**

PSH TREATMENT: THREE SEPARATE JOBS

ABORT · then PREVENT

ABORT: morphine is the cornerstone and is often dramatically effective, acting centrally and by blunting the afferent stimulus. IV **benzodiazepines** also terminate episodes.
PREVENT: propranolol -preferred because it is **lipophilic and crosses the blood-brain barrier**, unlike cardioselective agents, and it reduces the catabolism causing the weight loss. **Gabapentin** for the **background tone between paroxysms** and allodynia, where beta-blockers do little. **Clonidine / dexmedetomidine** reduce central sympathetic outflow (complementary, not duplicate). **Baclofen**, including intrathecal, for posturing and spasticity.

⚠ REMOVE THE TRIGGERS FIRST

The underrated half, and the cheapest. Since episodes are **provoked by stimulation**, **look for the provocation before adding a fifth drug**: **treat pain properly, decompress a distended bladder, treat constipation and fecal loading, CLUSTER nursing care** instead of disturbing the patient hourly, and cut unnecessary noise and handling. **This is therapeutic, not merely supportive.**
And support the consequences: feed aggressively (caloric needs are enormous), replace sweating losses, and **get physiotherapy in early** -the posturing is what produces the long-term disability.

⚠ THE EVIDENCE, STATED HONESTLY

Point	Detail
No randomized trials	**There are NO RCTs guiding drug therapy for either condition.** Everything above rests on **case series, retrospective cohorts and consensus**. That is not a reason to withhold treatment, but it is a reason to **start ONE agent at a time, state what you expect it to do, and titrate to an observed effect** -rather than layering four drugs on overnight and never learning which one worked

References: Hocker et al., indicators of central fever in the neurologic intensive care unit, JAMA Neurology 2013 · Baguley et al., paroxysmal sympathetic hyperactivity after acquired brain injury: consensus on conceptual definition, nomenclature and diagnostic criteria, J Neurotrauma 2014 · Meyfroidt, Baguley & Menon, paroxysmal sympathetic hyperactivity: the storm after acute brain injury, Lancet Neurology 2017 · published reviews of pharmacologic management of central fever.