

WHERE CLASSICAL WISDOM MEETS INTELLIGENT LEARNING

Unit 1: Foundations of Pain and Stress

PAPER 9 — Pain Management & Stress Relief Techniques

Unit 1: Foundations of Pain and Stress

(Acute vs. chronic pain • How pain is constructed • Stress biology • Mind-body links • Key anatomy you must know)

Understanding pain: acute vs. chronic

Pain is a brain-generated experience that *usually* tracks bodily threat. **Acute pain** (minutes \rightarrow a few weeks) is protective: it accompanies fresh tissue stress (sprain, strain, bruise), warns you to unload, and fades as healing progresses. It follows fairly predictable timelines—muscle (2-4 weeks), tendon/ligament (6-12+), bone stress (6-8+).

Chronic pain (often defined as >3 months) is different: tissues may be healed or only mildly irritated, yet the nervous system stays sensitized. Inputs (touch, movement, emotion, memory) are amplified; even harmless stimuli can hurt (allodynia) and normal pain can feel excessive (hyperalgesia). Chronic pain is best viewed as a biopsychosocial condition—biology (sensitized neurons, inflammation), psychology (beliefs, fear), and social context (work, family, sleep) all weigh in. In practice: a client's pain intensity is not a direct readout of tissue damage; it's the sum of signals and meaning.

Mechanisms of pain perception and stress response

Nociception—the raw signal—unfolds in four steps:

- 1. **Transduction:** Specialized peripheral receptors (nociceptors) in skin, muscle, joint, and viscera convert mechanical/thermal/chemical stress into electrical activity (inflammatory mediators lower their thresholds).
- 2. **Transmission:** Signals travel along **A-delta** (fast, sharp) and **C** (slow, aching) fibers to the spinal cord **dorsal horn**.
- 3. **Modulation:** In the dorsal horn, interneurons and descending inputs **amplify or dampen** the message (the classic "gate control" idea: non-painful touch from **A-beta** fibers closes the gate; threat opens it).
- 4. **Perception:** Ascending tracts reach thalamus and then cortex (S1/S2, insula, anterior cingulate, prefrontal areas) where sensation, emotion, memory, and attention construct the pain experience.

Descending control from the **periaqueductal gray (PAG)** and **rostral ventromedial medulla (RVM)** can release **endogenous opioids**, serotonin, and noradrenaline to inhibit spinal transmission. This control strengthens with safety, calm breath, expectation, and soothing touch—and weakens with anxiety, sleep loss, and catastophizing.

Stress responses ride two fast lanes:

- **Sympathetic-adrenomedullary (SAM):** seconds. Adrenaline/noradrenaline increase heart rate, muscle tone, vigilance. Short bursts can produce **stress-induced analgesia** (you don't notice pain until after the fight).
- **Hypothalamic-pituitary-adrenal (HPA):** minutes to hours. **Cortisol** mobilizes fuel and modulates inflammation. Chronic dysregulation lowers pain thresholds, disturbs sleep, and stiffens muscles.

Manual therapy, slow breathing, predictable sequencing, and supportive language can shift the balance toward **parasympathetic** dominance and better descending inhibition.

Psychosomatic connections in chronic pain

"Psychosomatic" does not mean "imagined." It means the mind and body are one system:

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- Fear-avoidance loop: pain → fear of re-injury → guarding/avoidance → deconditioning → more pain with less load → more fear.
- Catastrophizing & hypervigilance: constant scanning for danger amplifies nociception; small signals loom large.
- Meaning & memory: previous injuries, clinician messages, family beliefs, and cultural frames shape expectation (placebo/nocebo effects).
- Sleep & mood: poor sleep is a powerful pain amplifier; low mood narrows movement variety and coping bandwidth.
- **Interoception:** learning to sense internal states (breath, tension) with curiosity—rather than alarm—reduces reactivity.

Kalari Uzhichil and marma-based touch help by combining **mechanoreceptor input** (A-beta "gate"), **predictable rhythm**, **breath cueing**, and **grounding rituals**, which together reduce threat and improve body maps.

Role of the nervous system and hormonal responses

- Autonomic balance: Sympathetic arousal tightens muscle tone, quickens breath, and biases attention toward threat. Parasympathetic (vagal) activity slows the heart, deepens breath, and widens perception; high vagal tone correlates with better pain modulation.
- **Neurochemistry:** Pain amplification leans on glutamate, substance P, and inflammatory cytokines; down-regulation uses **GABA/glycine**, endogenous opioids, serotonin, and noradrenaline (in specific pathways).
- **Hormones:** Short cortisol pulses help; **chronic** cortisol disturbance impairs collagen repair and sleep, raising pain persistence.
- **Touch & context:** Warm, steady touch and empathetic framing can nudge oxytocin and parasympathetic markers upward and reduce defensive muscle co-contraction—small, cumulative shifts that matter over a course of care.

Basic anatomy of pain pathways and stress centers (the "where" map)

Peripheral: free nerve endings in skin, fascia, muscle, joint capsule, periosteum.

Spinal cord: dorsal horn lamina I/II (substantia gelatinosa) and V—first major relay and "gate."

Ascending tracts: spinothalamic (sensory-discriminative) and spinoparabrachial/mesencephalic (affective-motivational).

Brain:

- Thalamus → primary/secondary somatosensory cortex (S1/S2) for location/intensity.
- Insula & anterior cingulate cortex (ACC) for feeling/affect and action readiness.
- Prefrontal cortex (PFC) for meaning, choices, and expectation.
- Amygdala & hippocampus for threat memory and context.
- Hypothalamus (paraventricular nucleus) → HPA axis; locus coeruleus for arousal; nucleus tractus solitarius integrates vagal input.

Descending modulators: PAG → **RVM** → **dorsal horn** synapses that inhibit or facilitate nociception.

Clinical translation: slow, rhythmic, **distal** → **proximal** oiling with broad contacts stimulates A-beta fibers; exhalation-timed holds at safe regulatory points (e.g., **Talahridaya** in palm/sole, **Sthāpanī** at glabella—feather contact) can assist autonomic settling; finish seated so the system integrates change safely.

Summary Tables (LMS quick-reference)

A) Acute vs. chronic pain — at a glance

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Feature Acute Chronic (persisting >3 mo typical)

Purpose Protection during tissue healing System sensitization; threat learning
Tissue status Often injured/irritated Healed or low-grade irritants may persist

Nervous system Normal gain

Best early levers Relative rest, graded reload, calm breath

Education, graded exposure, sleep, stress skills,

consistent gentle loading

Red flags Deformity, fever + hot joint, chest pain, neuro loss, DVT/PE signs → **refer** Worsening despite 2–3 weeks of graded care → **refer**

Peripheral & central sensitization

B) Nociception steps & where Kalari touch helps

Step What happens Helpful inputs

Transduction Receptors fire; threshold lowered by inflammation Cool/calm in acute heat; avoid deep/fast

Transmission A-delta & C fibers enter dorsal horn

Gentle rhythmic input nearby (not on hotspot)

A-beta stimulation (broad strokes), predictable

Modulation Gate opens/closes; descending control acts pacing, exhale timing

Perception Brain integrates sensation, emotion, meaning Safety cues, education, grounding rituals

C) Stress systems snapshot

System Speed Hormones Typical effects Chronic issue

SAM Seconds Adrenaline/NA Tense, vigilant, analgesia possible Hyperarousal, bracing

HPA Minutes-hours Cortisol Fuel mobilization, anti-inflammatory Sleep/mood disruption, low pain threshold

D) Psychosomatic loops & correctives

Loop What it looks like Corrective lever

Fear-avoidance Guarding, movement shrinkage Graded exposure, "safe but challenging" reps
Catastrophizing "This means damage" Reframe: pain = protection, not always harm
Hypervigilance Constant scanning Narrow the focus: breath, rhythm, one cue

Sleep-pain spiral Poor sleep → worse pain → poorer sleep Sleep routine; finish sessions with down-regulation

E) Pain pathway map (minimal)

Node Role One clinical cue

Dorsal horn First gate Use broad touch → avoid sharp pokes

PAG/RVM Descending brake Exhale-timed holds; calm context

ACC/Insula Feeling & action Reduce threat, increase control

PFC Meaning/choices Education; clear, kind instructions

Key take-aways

- 1. Pain is a **protect-predict** system: intensity reflects *threat*, not just tissue status.
- 2. The pain signal is **modifiable** at every step—touch, breath, and meaning all matter.
- 3. Stress biology can either numb or amplify pain; chronic arousal usually raises it.
- For chronic pain, combine graded movement, sleep hygiene, calm breath, and clear education with your manual work.
- 5. In Kalari Uzhichil, prefer **broad, rhythmic, exhale-paced** contacts; avoid deep/fast on hot tissue and **never stack heat + pressure** over high-risk marmas (heart, umbilicus, pelvis, crown).

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