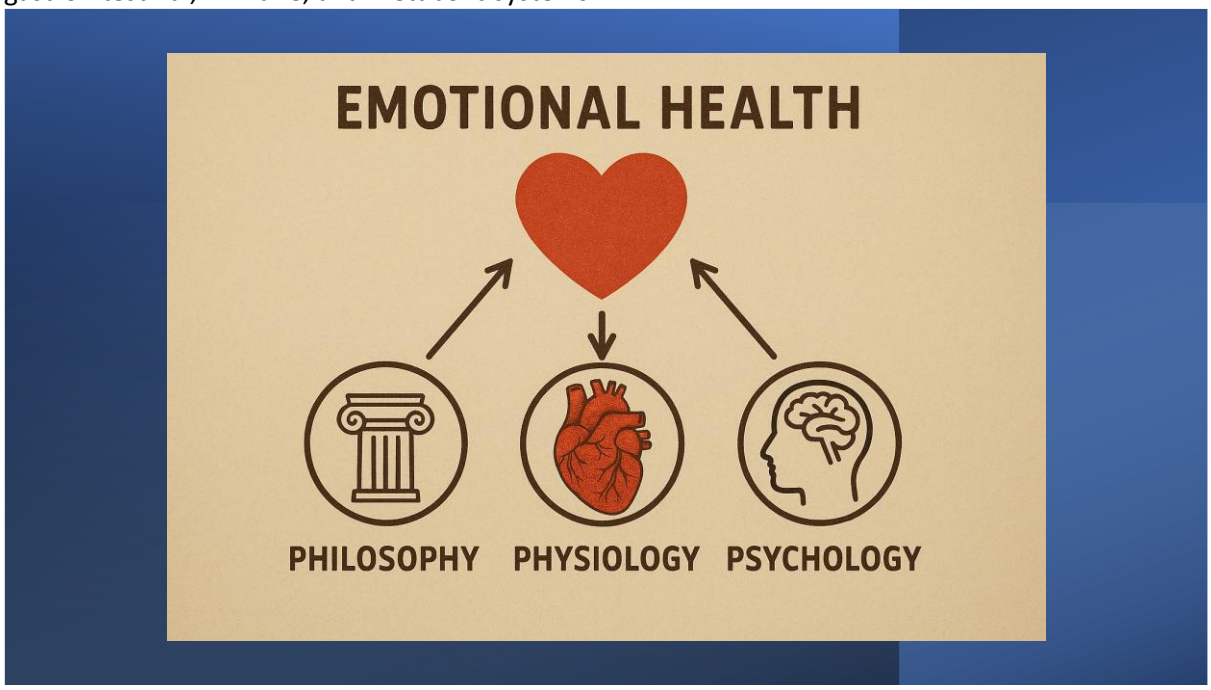
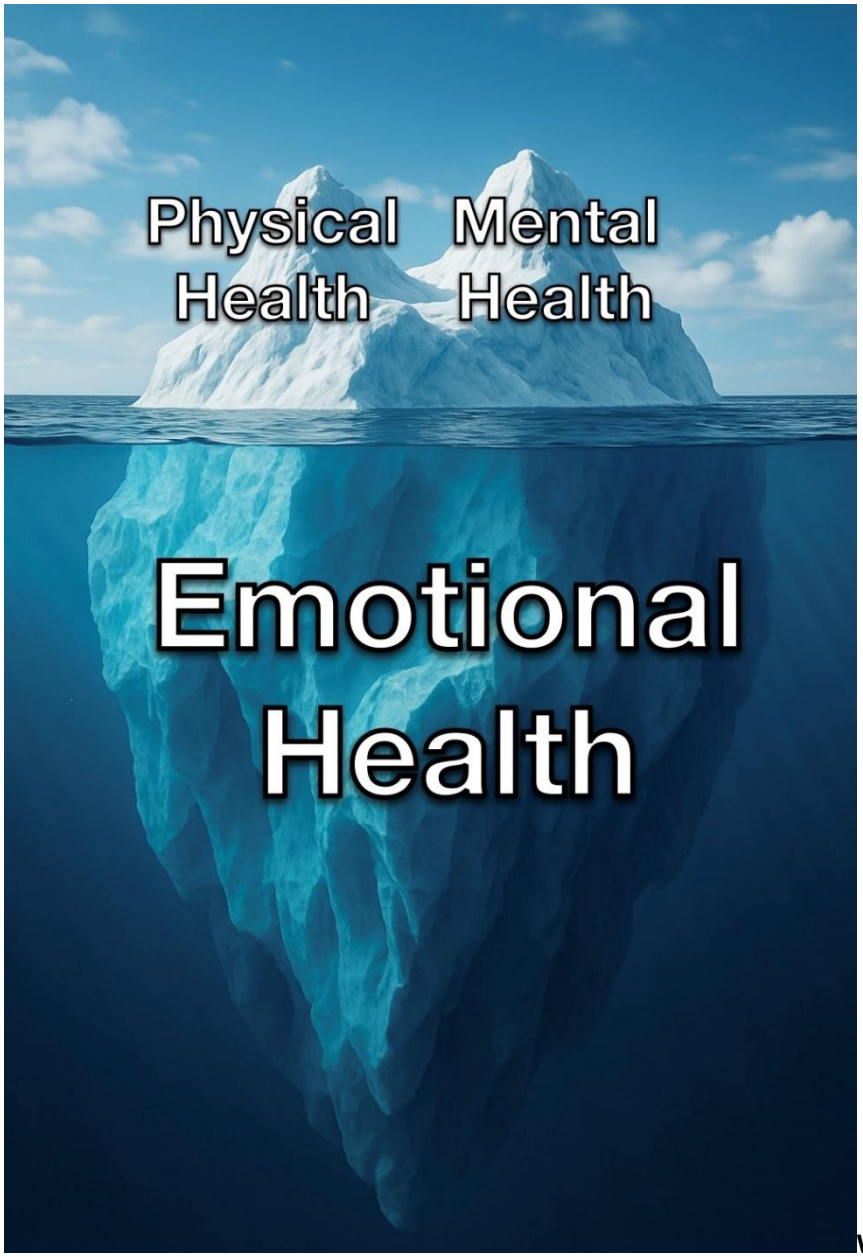
Emotional health denotes a physiological state in which distressing symptoms are **not explained by organ-specific structural pathology** but are linked to **autonomic dysregulation** driven by emotional states. The dysregulation is mediated by **neuroendocrine** and **autonomic** pathways—including altered hypothalamic–pituitary–adrenal (HPA) axis activity (e.g., cortisol) and sympatho-adrenal output (e.g., adrenaline/noradrenaline)—with downstream effects on cardiovascular, gastrointestinal, immune, and metabolic systems





Why Emotional Health Matters

Emotional Health is not "soft science"; it is grounded in robust physiology:

- **HPA Axis Activation:** Chronic stress alters cortisol rhythms, impacting cardiovascular, metabolic, and immune systems.
- **Autonomic Imbalance:** Sympathetic overdrive and vagal withdrawal manifest as palpitations, BP variability, and gut motility changes.
- **Neuroimmune Crosstalk:** Stress amplifies inflammatory signalling, influencing pain perception and fatigue.

Nosology and Relation to Existing Classifications

- **Somatic Symptom and Related Disorders (DSM-5/DSM-5-TR):** DSM-5 reframed "somatoform" conditions as *somatic symptom disorder* (SSD), emphasizing **distress and disproportionate thoughts/behaviours** rather than "medically unexplained" symptoms per

se. This reduces mind–body dualism while acknowledging that symptoms may co-occur with medical disease.

- **Functional Somatic Disorders (FSD):** Proposed umbrella term for persistent, troublesome physical symptoms across specialties, advocating a common language and neutral stance between "purely somatic" vs "purely mental."
- **Disorders of Gut–Brain Interaction (Rome IV):** Formerly "functional GI disorders," these are defined by altered **motility, visceral sensitivity, mucosal/immune function, microbiota, and CNS processing**, not structural disease.
- **Functional Neurologic Disorder (FND):** In neurology, positive *rule-in* signs permit diagnosis (i.e., not purely "exclusionary"). This coexists conceptually with the broader stress-autonomic model.

Key takeaway: Emotional-state–linked autonomic dysregulation sits compatibly within these frameworks while keeping a clinical focus on *measurable physiology* (HPA/ANS/immune) and *patient experience*.

Pathophysiology

1) Allostasis and Allostatic Load

Emotional challenges recruit *allostasis*—adaptive, brain-mediated changes in autonomic and endocrine outputs to maintain stability. When activation is chronic or poorly terminated, cumulative "**allostatic load**" leads to multisystem dysregulation (neuroendocrine, immune, metabolic, cardiovascular) and symptom expression.

2) Neuroendocrine Pathways (HPA Axis & Stress Hormones)

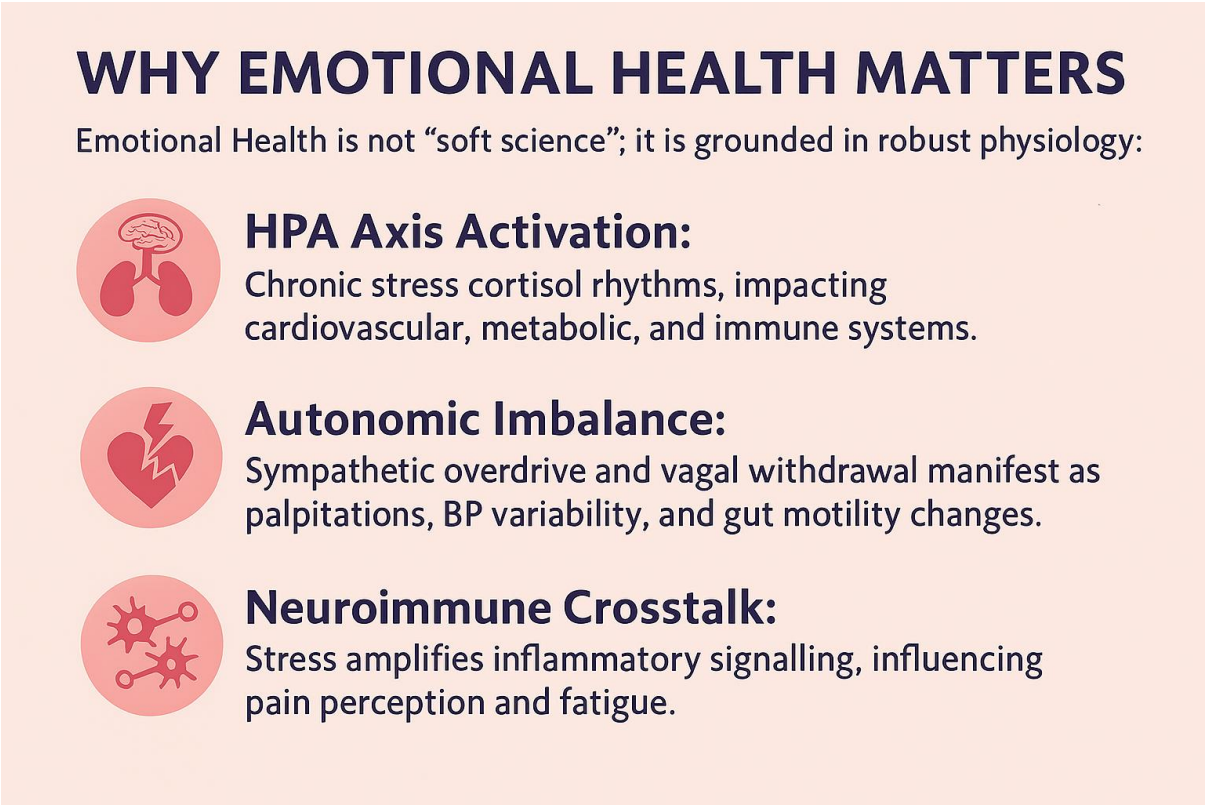
Emotional stress activates corticolimbic circuits that stimulate corticotropin-releasing hormone (CRH) → ACTH → **cortisol** secretion and co-activate the **sympatho-adrenal** system (adrenaline/noradrenaline). Persistent activation alters diurnal cortisol rhythms, glucose regulation, vascular tone, and reproductive axes, contributing to palpitations, blood pressure variability, dyspepsia/heartburn, headache, menstrual irregularity, and stress-hyperglycaemia.

3) Autonomic (ANS) Dysregulation and the Central Autonomic Network

Stress-responsive cortical and hypothalamic nodes integrate autonomic and endocrine outputs. **Reduced vagal tone and sympathetic predominance** are frequently observed, with **heart rate variability (HRV)** serving as a non-invasive proxy of autonomic balance across stress, anxiety, and depression.

4) Psychoneuroimmunology (PNI)

Chronic emotional stress can up-regulate **pro-inflammatory signalling** (e.g., IL-6, CRP), alter microglia–neuron crosstalk, and shape affective-cognitive processes (e.g., negative attentional bias), creating bidirectional loops between immune activity and mood/anxiety symptoms.



Clinical Manifestations

CopX8mmon, Organ-System–Framed Presentations (without structural disease)

- **Cardiovascular/autonomic:** Episodic **palpitations**, sinus tachycardia, labile blood pressure, presyncope; reduced HRV indicating vagal withdrawal/sympathetic dominance. Consider POTS in orthostatic symptoms.
- **Gastrointestinal:** Variable **motility** (constipation/diarrhoea), reflux/heartburn, epigastric pain or post-prandial distress consistent with Rome IV DGBI patterns.
- **Metabolic/endocrine:** Stress-related **hyperglycaemia** or metabolic derangements (via cortisol/catecholamines), **menstrual irregularity** (HPA–HPG cross-talk).
- **Neurovegetative: Sweating**, tremor, muscle tension, headache, sleep disturbance; fatigue and “brain fog” under prolonged activation/allostatic load.

Evaluation

Diagnostic Principles

1. **Safety first**—exclude structural/biochemical disease appropriate to the symptom cluster (red flags, examination, targeted labs/imaging). In the GI domain, apply Rome IV; in neurology, recognize positive FND signs where present.
2. Name and frame the problem using shared language that validates symptoms and explains brain–body mechanisms (allostasis/ANS/immune). This improves alliance and reduces over-testing.

Practical Work-Up (tailored)

- **History/psychosocial**: Triggers, temporal patterns (rest vs stress), sleep, trauma/adversity, illness beliefs and safety behaviours.
- **Basic tests** (as indicated): CBC, U&E, LFTs, TFTs, HbA1c/glucose, ferritin/B12, CRP; ECG; pregnancy where relevant.
- **Functional assessments**: Orthostatic vitals ± tilt testing when orthostatic intolerance suspected; HRV (time/frequency-domain) for research/monitoring contexts; symptom/trigger diaries.
- **Screening for SSD** (when distress/behaviours are disproportionate to symptom burden), using DSM-5 criteria pragmatically; use structured measures only if follow-up care is available.

Many presentations fall under *medically unexplained/persistent physical symptoms*. NHS guidance emphasises ruling out organic disease, validating the reality of symptoms, and offering integrated psychological support (e.g., Talking Therapies pathways for MUS/LTCs).

Management

Core Aims

1. Reduce autonomic over-arousal and restore oscillation between activation and recovery;
2. Address cognitive-behavioural amplifiers of symptom perception;
3. Minimise iatrogenesis from unnecessary tests or restrictive rest.

Interventions

- Education & reattribution: Explain brain–body integration (HPA/ANS, allostasis). Provide a simple physiological narrative for symptoms, emphasising reversibility and safety.
- Lifestyle & behavioural:
    - Sleep optimisation; graded physical activity (aerobic + strength) with pacing to remodel autonomic set-points.
    - Breathing training (slow diaphragmatic, 4–6 breaths/min), HRV biofeedback, and mindfulness to increase vagal tone and dampen sympathetic drive.
    - Nutrition: Regular meals, glycaemic stability; limit caffeine/alcohol in sensitive individuals.
- Psychological therapies: CBT (or other evidence-based approaches) targeting catastrophic interpretations, hypervigilance, and safety behaviours; increasingly offered within NHS Talking Therapies for MUS/LTCs.
- Symptom-directed pharmacology (selected cases):
    - Palpitations/anxiety-tachycardia: low-dose β-blocker if appropriate; consider SSRI/SNRI when comorbid anxiety/depression and functional impairment are prominent.

    - GI symptoms: Rome-guided use of PPIs, antispasmodics, neuromodulators; avoid long-term polypharmacy without clear benefit.
    - Orthostatic intolerance/POTS: fluids/salt, compression garments, reconditioning; pharmacotherapy (e.g., fludrocortisone, midodrine) in selected patients under specialist care.
- Care coordination: Stepped, interdisciplinary care (primary care, psychology, gastro/endo/cardio/neurology as needed) prevents "doctor shopping," reduces investigations, and improves outcomes.

Prognosis

Outcomes vary with chronicity, comorbidity, and psychosocial context. Education, early functional rehabilitation, and restoration of autonomic flexibility (e.g., improved HRV) are associated with symptom improvement and better quality of life.

Differential Diagnosis (selected "don't miss" items)

- Cardiac: Arrhythmias, myocarditis, structural heart disease.
- Endocrine/metabolic: Thyroid disorders, phaeochromocytoma, diabetes dysregulation.
- GI: Peptic ulcer disease, IBD, coeliac disease, biliary pathology.
- Neurologic: Seizure disorders, neuropathies.
- Autonomic disorders: POTS, autonomic failure, post-infectious dysautonomia (including post-COVID).

Domain	Red Flags	Initial Exclusion Tests / Actions
Cardiac	Syncope with injury; exertional chest pain; new heart failure signs; family history of sudden cardiac death	**ECG**, troponin (if chest pain), electrolytes, chest X-ray; urgent cardiology referral if abnormal
Endocrine / Metabolic	Marked weight loss; hyperthyroid signs; episodic headaches/sweats with severe hypertension	**TFTs**, glucose/HbA1c, pregnancy test; consider pheochromocytoma screen if classic paroxysms
Gastrointestinal	GI bleeding, weight loss, progressive dysphagia, nocturnal diarrhoea	FBC, ferritin, coeliac screen, **H. pylori** (if dyspepsia); endoscopy/colonoscopy if alarm features
Neurology	Focal deficits, new seizures, progressive weakness	Focused neuro exam; glucose, electrolytes; consider EEG/neuroimaging per pathway
Infection /	Fever, night sweats, focal pain	CRP, cultures, imaging as indicated

Inflammation		
Autonomic	Severe orthostatic hypotension or tachycardia	Orthostatic vitals (supine/standing), medication review; tilt testing if persistent

Targeted Work-Up (typical set; tailor to case)

- History: triggers, stressors, sleep, caffeine/alcohol, trauma/adversity; illness beliefs & safety behaviours.
- Vitals: orthostatic measurements (supine/1/3/5 min standing).
- Labs: FBC, U&E, LFTs, TFTs, HbA1c/glucose, ferritin/B12, CRP; pregnancy where relevant.
- ECG (palpitations/tachycardia), urinalysis (where indicated).
- Optional monitoring: HRV (research/monitoring contexts).
- SSD screen (DSM-5) when cognitive-behavioural amplification suspected (use only where follow-up care exists).

First-Line Management (all patients unless contraindicated)

- Explain & reassure brain–body integration (HPA/ANS; allostasis; "real symptoms, reversible mechanisms"). Your body's alarm system is stuck 'on'; we're helping it relearn calm.
- Sleep: regularity and duration; address insomnia. Sleep hygiene, graded activity
- Activity & pacing: graded aerobic + strength; avoid prolonged rest; titrate to avoid post-exertional flares.
- Breathing & vagal strategies: slow diaphragmatic breathing (4–6 breaths/min), HRV biofeedback, mindfulness.
- Nutrition: regular meals; glycaemic stability; reduce caffeine/alcohol where sensitive.
- Psychological therapies: CBT/equivalents via NHS Talking Therapies for MUS/LTCs; target catastrophising, hypervigilance, safety behaviours.
- Symptom-directed pharmacology (selected):
    - Palpitations/tachycardia: low-dose β-blocker if appropriate; treat comorbid anxiety/depression (SSRI/SNRI).
    - GI DGBI: per Rome IV (PPIs, antispasmodics, neuromodulators; avoid long-term polypharmacy).
    - POTS: fluids/salt, compression garments, reconditioning; specialist pharmacotherapy (e.g., fludrocortisone, midodrine) when needed.

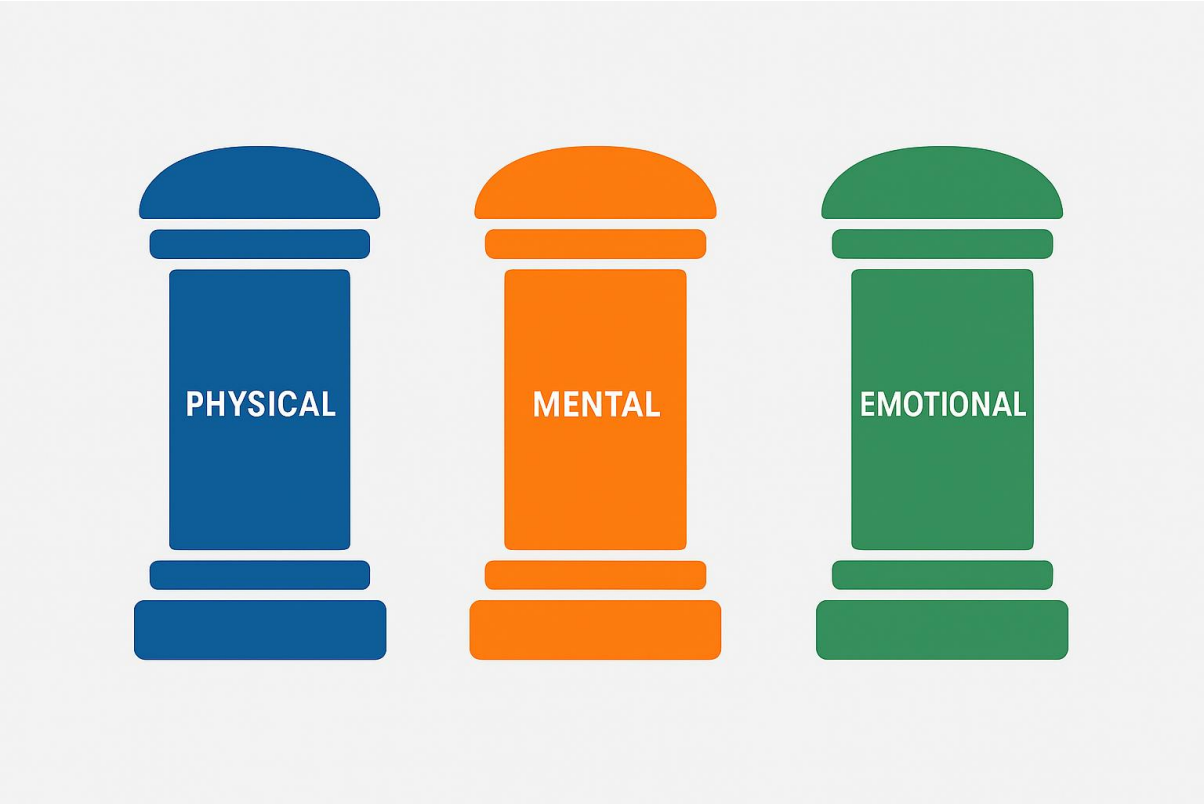
Follow-up (2–6 weeks): review symptom diary; adjust plan; escalate to interdisciplinary review if persistent impairment or diagnostic uncertainty.

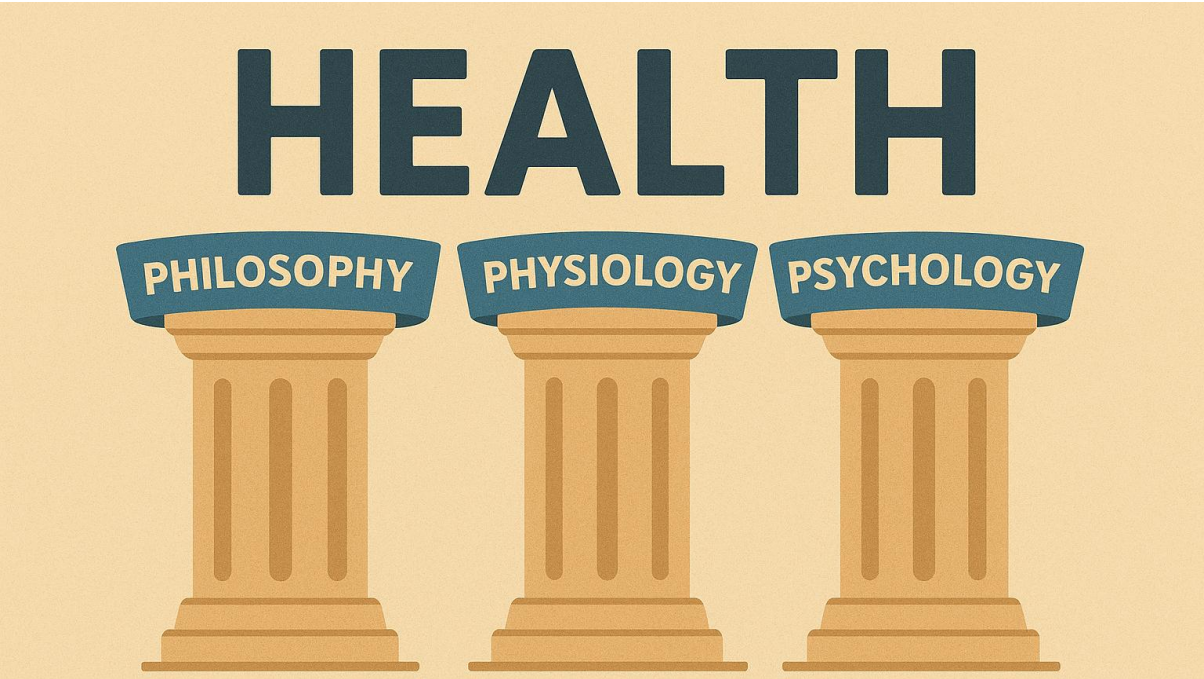
Documentation Phrase (suggested)

“Symptoms consistent with stress-linked autonomic dysregulation after appropriate exclusion of organ-specific structural/biochemical disease. Plan discussed and agreed: education on brain–body regulation, graded activity, sleep optimisation, breathing/HRV, Talking Therapies referral; symptom-directed measures as above; follow-up in 2–6 weeks.”

From Evolution to Revolution

As highlighted in *Evolution Revolution* (Dr Atul Bansal), healthcare must pivot from reactive firefighting to **anticipatory care**. EDs can lead this transformation by embracing **three pillars of health—Physical, Mental, and Emotional**—and implementing innovative models:





Emotional health is the integrity of brain–body regulation whereby emotional states modulate neuroendocrine and autonomic outputs; when dysregulated in the absence of organ pathology, this yields stress-linked symptoms across cardiovascular, gastrointestinal, metabolic, and neurovegetative domains.

Emotional Health is not an optional extra—it is the missing link in comprehensive care. Addressing it will not only ease the current burden but also transform Modern Medicine into a discipline that prevents crises before they occur by being proactive from reactive.

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