MEDICAL NOTES POWERHOUSE

Medical Notes Powerhouse

The Complete High-Yield Notes for NEET SS & INI SS-ET

Cardinal Symptoms & Clinical Pharmacology

01 VOLUME
of 10 · Volume I · CH 14–72

59 CHAPTERS

10 SECTIONS

10 VOLUME SET

SECTIONS IN THIS VOLUME

- Pain & Cardinal Symptoms
- Fever & Infectious
- Neurological Symptoms
- Special Senses & ENT
- Cardiorespiratory
- Gastrointestinal
- Renal & Electrolytes
- Dermatology
- Haematology
- Clinical Pharmacology

Sources Harrison's Principles of Internal Medicine, 22nd Edition

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SECTION I

Pain & Cardinal Symptoms

Systematic approach to pain, discomfort and constitutional complaints

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CH 14 · Pain — Pathophysiology & Management

Pain mechanisms (peripheral, central, modulation), neuropathic pain, full pharmacology — NSAIDs, opioids, antidepressants, anticonvulsants. NEET SS / INI SS-ET high yield.

Pain — Definition & Duality

- Unpleasant sensation localized to body part — described as **tissue-destructive** (stabbing, burning) and/or **emotional** (terrifying, sickening).
- **Duality** = sensation + emotion. Moderate-severe pain → anxiety + escape urge.
- Acute pain → behavioral arousal + stress: ↑ **BP, HR, pupil, cortisol**; local muscle contraction.
- **Function**: protect body, maintain homeostasis. Detect, localize, identify damage.
- Quality + time + location = key diagnostic clues.

Peripheral Mechanisms

Primary afferent nociceptor

- Peripheral nerve = 3 neurons: **1° sensory afferent**, motor, sympathetic postganglionic.
- Sensory cell bodies in **dorsal root ganglia**; axons branch centrally + peripherally.
- Classified by diameter, myelination, conduction velocity.

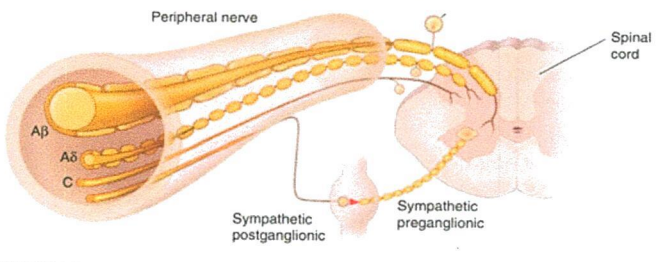


Fig 14.1 — Cutaneous nerve: Aβ, Aδ, C afferents + sympathetic postganglionic

Fiber types

Table 14.1 — Primary afferent fibers

Fiber	Myelin	Function	Pain?
Aβ	Large myelinated	Light touch / movement	No
Aδ	Small myelinated	Sharp, fast, localized	YES
C	Unmyelinated	Dull, slow, burning	YES

- **Aδ + C = nociceptors**; pain abolished if both blocked.
- **Cornea** = ONLY Aδ + C (avascular, alymphatic, all-nociceptor).

Stimuli & receptors

- Respond to: heat, cold, mechanical, ↓ pH, chemicals (**ATP, 5-HT, BK, histamine**).
- **TRPV1** = vanilloid receptor — heat, acid, capsaicin.

Sensitization

- Repeated/prolonged stimuli on damaged tissue → ↓ threshold, ↑ firing.
- Mediators: **BK, NGF, PGs, leukotrienes**.
- **Two types:**
  - **Peripheral** — at nerve terminal; ↑ ion channel insertion.
  - **Central** — at dorsal horn; ↑ spinal neuron excitability.
- **Allodynia** — innocuous stimulus → pain.
- **Hyperalgesia** — ↑ pain to noxious stimulus.
- Classic: **sunburn** — gentle slap or warm shower = severe pain.

Silent nociceptors

- Many visceral Aδ + C fibers **silent in normal tissue**.
- Become mechanically sensitive only in inflammation.
- Explains why deep structures (joints, viscera) become exquisitely sensitive in disease.

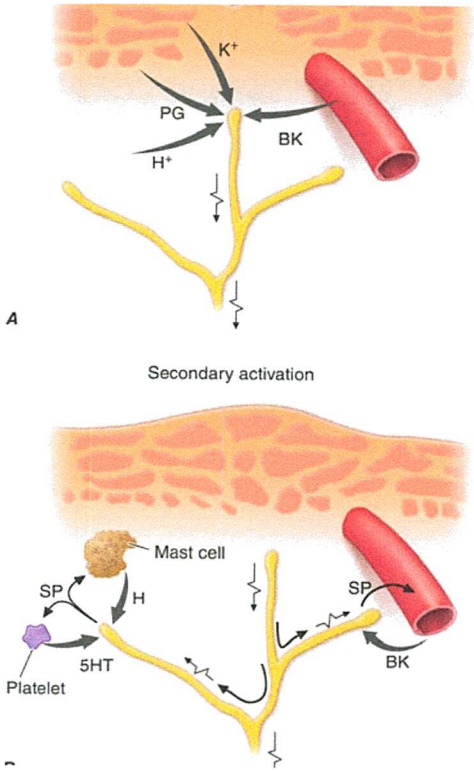


Fig 14.2 — Nociceptor activation: A) Direct (K^+, H^+, PG, BK). B) Secondary via SP → mast cells, platelets.

Nociceptor-induced inflammation

- Neuroeffector function — active tissue protection.
- **Substance P** (11 AA) — vasodilator, mast cell degranulation, leukocyte chemoattractant.
- **CGRP, cholecystokinin** also released.
- SP depletion → ↓ severity of experimental arthritis.

> ♥ **NEET SS HIGHLIGHTS**
>
> ♥ **Aβ** = touch (NO pain). **Aδ** = fast sharp. **C** = slow burning.
> ♥ **Cornea** = only Aδ + C.
> ♥ **TRPV1** = capsaicin / heat / acid.
> ♥ **SP** = vasodilator + mast cell degranulator.
> ♥ **Allodynia** = innocuous → pain; **hyperalgesia** = ↑ pain to noxious.

Central Mechanisms

Spinal cord & convergence

- Afferents enter **dorsal root** → terminate **dorsal horn**.

- Major NT = **glutamate** (rapid). SP + CGRP = slower, longer.
- Each spinal neuron = inputs from many afferents — visceral + skin same segment.
- **Convergence** determined by spinal segment of DRG.

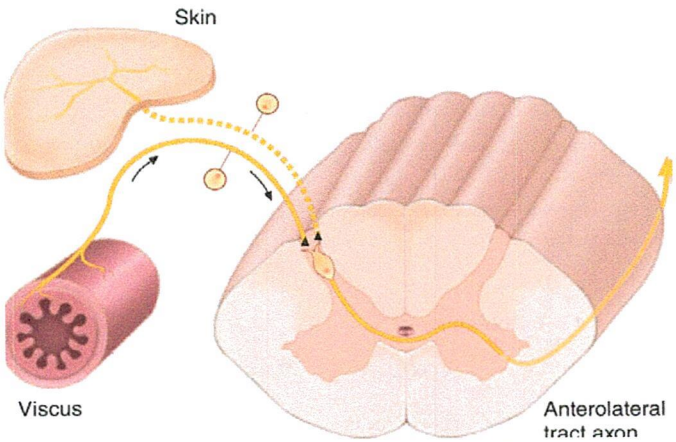


Fig 14.3 — Convergence-projection: visceral + somatic afferents share spinal neurons

Referred pain — classic

Table 14.2 — Visceral pain referral

Organ	Referred site
Central diaphragm	Shoulder (C3–C4)
Heart (MI)	Left arm, jaw, epigastrium
Gallbladder	Right shoulder/scapula
Appendix (early)	Periumbilical (T10)
Kidney/ureter	Groin, flank
Pancreas	Mid-back

Ascending — spinothalamic

- Dorsal horn neurons cross within 1–2 segments → **contralateral spinothalamic tract**.
- Anterolateral white matter → lateral medulla, pons, midbrain → thalamus.
- **Crucial for pain**; interruption = permanent pain/temp deficit.

Cortical projections

- **Somatosensory cortex** — sensory-discriminative (location, intensity, quality).
- **Anterior cingulate** — affective/emotional.
- **Insular cortex** — emotional unpleasantness.
- Frontal lesions → ↓ emotional impact, preserves localization (lobotomy effect).

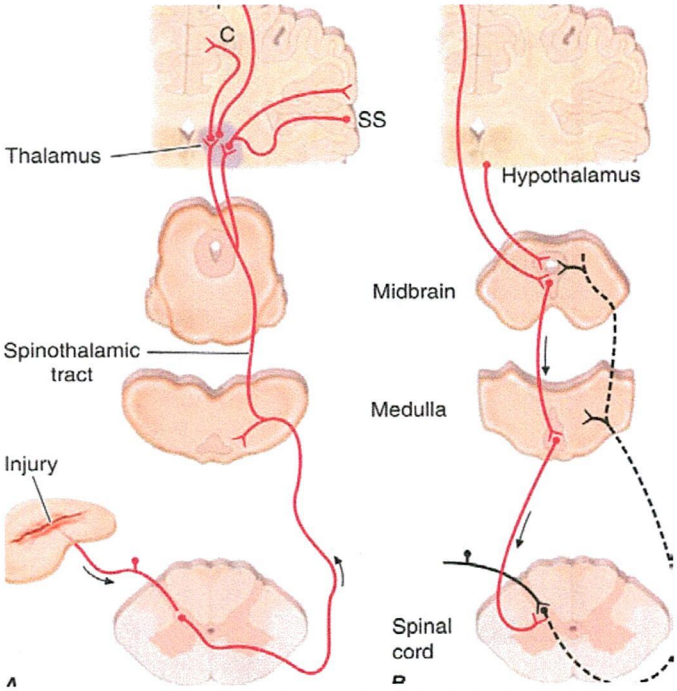


Fig 14.4 — A) Pain transmission via spinothalamic tract → thalamus → cortex. B) Descending modulation.

CLINICAL PEARLS

- Spinothalamic = **contralateral**. Cord lesion → contralateral pain/temp loss below.
- **Brown-Séquard**: ipsilateral motor + proprioception + **contralateral pain/temp loss**.
- **Syringomyelia**: cape-like dissociated loss (pain/temp lost, touch preserved).
- **Dejerine-Roussy**: severe contralateral pain after thalamic stroke.

Pain Modulation

- Pain from similar injuries = **highly variable** (Beecher's WWII soldiers).
- **Placebo** — suggestion of relief = real analgesia.
- **Nocebo** — suggestion of worsening ↑ perceived pain.
- **Descending circuit**: hypothalamus → midbrain (PAG) → medulla (RVM) → spinal dorsal horn.
- Contains opioid receptors + endogenous opioids (**enkephalins, β-endorphin**).
- **Placebo analgesia reduced by naloxone** → endogenous opioid mediated.
- Modulating circuits can **enhance OR suppress** pain.

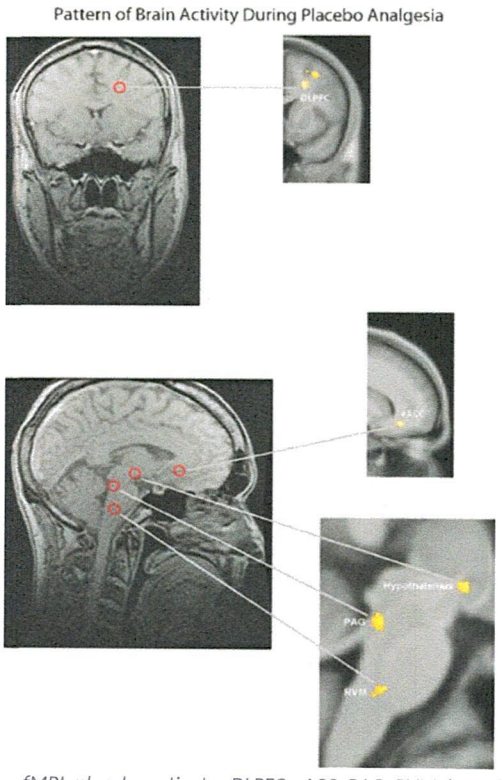


Fig 14.5 — fMRI: placebo activates DLPFC, rACC, PAG, RVM, hypothalamus. Blocked by naloxone.

Neuropathic Pain

- Pain from damage to nociceptive pathway (paradoxical).
- Causes: **diabetic neuropathy, herpes zoster, SCI, brainstem/thalamic stroke.**
- Often severe, **resistant to standard analgesics.**

Features

- Quality: **burning, tingling, electric shock-like.**
- Spontaneous OR triggered by light touch.
- Sensory deficit co-extensive with pain.
- **Hyperpathia** — exaggerated pain to mild repeated stimuli.
- **Allodynia** — light touch = exquisite pain.
- **Topical lidocaine 5% patch** — effective for PHN with allodynia.

Mechanisms

- ↑ **Na^+ channel density** in damaged fiber → spontaneous activity.
- Damaged afferents acquire **NE sensitivity.**
- Spinal neurons cut off from input → spontaneously active.
- Both peripheral + central hyperactivity.

CRPS

Table 14.3 — CRPS classification

Type	Aka	Trigger
CRPS I	Reflex sympathetic dystrophy (RSD)	No identifiable nerve injury
CRPS II	Causalgia / posttraumatic neuralgia	Identifiable nerve injury

- Burning pain + delayed onset + swelling + periarticular bone loss.
- Triggers: fractures, soft-tissue trauma, **MI, stroke.**
- **Sympathetic block** rapidly relieves acute pain + inflammation.

Acute Pain — NSAIDs / COX Inhibitors

- Ideal: **remove cause.** Begin treatment while establishing etiology.
- Rapid analgesia for: postop, burns, trauma, cancer, sickle cell crisis.
- All inhibit **COX**; all except **acetaminophen** have anti-inflammatory action.
- Best for **mild-moderate headache + MSK pain.**
- **Gastric irritation** — main dose-limiting SE; worst with aspirin.
- **Aspirin irreversibly acetylates platelet COX** → ↑ GI bleed risk.
- **Nephrotoxicity** on chronic use; avoid in volume contraction.
- Can ↑ **BP** on long-term therapy.
- **Acetaminophen** — no gastric/platelet effects, BUT **hepatotoxic at high dose.**
- **Parenteral: ketorolac, diclofenac** — can replace opioids in acute headache/MSK.

COX-1 vs COX-2

Table 14.4 — COX subtypes

Feature	COX-1	COX-2
Expression	Constitutive	Inducible
Gastric protection	YES	No
Drugs	Nonselective NSAIDs	Celecoxib (valdecoxib withdrawn)
Bleeding effect	↑	No effect
CV risk	Low (aspirin protective)	↑ MI, stroke, HF

- COX-2 = similar analgesia, less GI, NO platelet effect.
- COX-2 does **NOT** reduce nephrotoxicity vs nonselective.
- **COX-2 preferred postop**; nonselective (aspirin) **contraindicated postop.**
- **COX-2 contraindicated post-CABG** — class effect of all NSAIDs except aspirin.

Table 14.5 — Non-narcotic analgesics

Drug	Dose	Interval	Notes
Aspirin	650 mg	PO q4h	Enteric-coated avail
Acetaminophen	650 mg	PO q4h	Hepatotoxic if >4 g/d
Ibuprofen	400 mg	PO q4–6h	OTC
Naproxen	250–500 mg	PO q12h	Lowest CV risk; ↑ GI bleed
Indomethacin	25–50 mg	PO q8h	GI SE common
Ketorolac	15–60 mg	IM/IV q4–6h	Parenteral
Celecoxib	100–200 mg	PO q12–24h	COX-2 selective

Opioid Analgesics

- Most potent + broadest range. Act centrally on **μ-receptor.**
- SEs: nausea, vomiting, pruritus, sedation, **constipation.**
- **Respiratory depression** — uncommon at standard dose, life-threatening.

- Reversed by **naloxone**.
- Addiction risk small with appropriate use; ↑ with dose escalation in chronic noncancer pain.
- **Avoid co-admin with BZDs** (synergistic respiratory depression).

Meperidine — caution

- **Normeperidine** = toxic metabolite (renally cleared).
- Accumulates at **>1 g/d** or in **renal failure**.
- Causes **seizures NOT reversible by naloxone**.
- **Routine use NOT recommended.**

Table 14.6 — Opioid analgesics

Drug	Parenteral	PO	Notes
Codeine	30–60 q4h	30–60 q4h	Nausea common
Oxycodone	—	5–10 q4–6h	Often + APAP/ASA
Oxycodone ER	—	10–40 q12h	High misuse
Morphine	5 q4h	30 q4h	Reference
Hydromorphone	1–2 q4h	2–4 q4h	Shorter acting
Methadone	5–10 q6–8h	5–20 q6–8h	Long t½; start ≤40 mg/d
Meperidine	50–100 q3–4h	300 q4h	NOT recommended
Fentanyl TD	25–100 µg/h	—	72-h transdermal
Tramadol	—	50–100 q4–6h	µ + NE reuptake

✦ MNEMONIC

RESPECT *Opioid side effects*
- **R** — Respiratory depression
- **E** — Euphoria / dysphoria
- **S** — Sedation
- **P** — Pruritus
- **E** — Emesis (N/V)
- **C** — Constipation
- **T** — Tolerance / dependence

Constipation never develops tolerance — treat expectantly.

PCA + alternate routes

- **PCA:** load first, then maintain. Bolus = morphine 1 mg / hydromorphone 0.2 mg / fentanyl 10 µg.
- Lockout 10 min + hourly limit prevent overdose.
- **Intrathecal morphine 0.1–0.3 mg ≈ IV 5–10 mg.**
- Routes: intranasal (butorphanol), rectal, transdermal (fentanyl, buprenorphine), oral mucosal (fentanyl).

Peripheral opioid antagonists

- **Alvimopan** (PO, gut-restricted) — ↓ postop ileus.
- **Methylnaltrexone** (SC) — chronic opioid constipation.
- Reverse peripheral SEs WITHOUT reversing analgesia.

Chronic Pain

- Sensitization can occur without clear cause (**fibromyalgia, chronic headache**).
- Often becomes a distinct disease.
- **Multidisciplinary pain centers** + psychological/behavioral care = most effective.

Causes

1. Incurable disease — arthritis, cancer, headache, fibromyalgia, DM neuropathy.
2. Secondary perpetuating — damaged nerves, sympathetic activity, reflex spasm.
3. Psychological — exacerbate or cause pain.

Psychological red flags

- **Depression** = most common comorbidity. Use Beck Inventory.
- Pain at multiple unrelated sites; recurrent since childhood.
- Pain at time of emotional trauma.
- H/o physical/sexual abuse, substance abuse.

Chronic Pain Pharmacotherapy

TCAs

- **Nortriptyline, desipramine** preferred (better SE profile).
- Analgesic effect: **faster onset, lower dose** than antidepressant effect.
- Effective even in non-depressed patients.
- Best for **diabetic neuropathy, postherpetic neuralgia**.
- SEs: orthostatic hypotension, drowsiness, **cardiac conduction delay**, anticholinergic.

SNRIs (preferred)

- **Duloxetine, venlafaxine** — block 5-HT + NE reuptake.
- TCA-like analgesia + SSRI-like safety.
- **Largely supplanted TCAs** in chronic pain.
- SSRIs (fluoxetine) much less effective for pain.

Anticonvulsants

- Primary use: **neuropathic pain**.
- **Carbamazepine** = first-line for **trigeminal neuralgia** (lancinating).
- **Gabapentin, pregabalin** — Ca^{2+} channel $\alpha_2\delta$ ligands; broad use.
- Often **first-line** due to favorable SE profile.
- Gabapentin ≤1800 mg/d FDA-approved for PHN.

Table 14.7 — Anticonvulsants for pain

Drug	Dose	Interval	Notes
Carbamazepine	200–300 mg	q6h	Aplastic anemia, GI, hepatotoxic
Oxcarbazepine	300 mg	bid	Similar to CBZ
Gabapentin	600–1200 mg	q8h	TGN; PHN ≤1800/d
Pregabalin	150–600 mg	bid	Dry mouth, edema

Neuropathic Pain — Treatment Algorithm

1. **Topical:** Lidocaine 5% patch for PHN + cutaneous hypersensitivity.
2. **1st-line:** Gabapentin OR pregabalin OR TCA OR SNRI.
3. **Avoid** systemic antiarrhythmics (lidocaine IV transient; mexiletine poorly tolerated).
4. **2nd/3rd-line:** Opioids (sedating, escalation, may worsen pain).

5. **Tramadol** (weak opioid + NE) or **tapentadol** (stronger + NE).
6. Combine drug classes for optimal control.

Multidisciplinary

- Pharmacotherapy + counseling + PT + nerve blocks + surgery.
- **Image-guided**: epidural steroids (acute radiculopathy), RF facet joint (chronic).
- **Spinal cord stimulation** — peripheral nerve, nerve roots, dorsal columns.

✦ MNEMONIC

GPND *Neuropathic pain 1st-line drugs*
G — Gabapentin
P — Pregabalin
N — Nortriptyline / desipramine (TCAs)
D — Duloxetine / venlafaxine (SNRIs)
Opioids = 2nd/3rd line. Topical lidocaine for PHN with allodynia.

✎ MCQ CORNER

Q1. 58 y/o diabetic, burning bilateral foot pain × 6 mo, worse at night. First-line drug?
A. Oral morphine
✓ **B. Gabapentin**
C. Carbamazepine
D. Fluoxetine
► *Gabapentin/pregabalin first-line for diabetic neuropathic pain. Opioids = 2nd/3rd line. CBZ = TGN. SSRIs less effective.*

Q2. Patient on meperidine 1.2 g/d develops seizures. Cause?
A. Direct meperidine toxicity
✓ **B. Normeperidine accumulation**
C. Opioid withdrawal
D. BZD deficiency
► *Normeperidine accumulates at >1 g/d and in renal failure; seizures NOT reversible by naloxone.*

Q3. Which is TRUE about COX-2 selective inhibitors?
A. Reduce nephrotoxicity vs nonselective
B. Safe immediately post-CABG
✓ **C. Do not affect blood coagulation**
D. Lower CV risk than aspirin
► *COX-2 don't affect platelets → useful postop. Contraindicated post-CABG. Nephrotoxicity NOT reduced. ↑ CV risk.*

Q4. Referred shoulder pain from central diaphragm = convergence at:
A. T1–T2
✓ **B. C3–C4**
C. T4–T5
D. L1–L2
► *Diaphragm + shoulder skin both arise from C3–C4 DRG. Convergence-projection hypothesis.*

Q5. Postop PCA: RR 6/min, SpO_2 88%. Immediate next step?
A. Increase PCA lockout
✓ **B. Naloxone IV + ventilatory support**
C. Switch to meperidine
D. Add BZD for sedation
► *Opioid respiratory depression → immediate naloxone + ventilatory support. BZD WORSENS depression.*

◷ LAST MINUTE REVISION

► Aδ = sharp fast (myelinated); C = slow burning (unmyelinated); Aβ = touch only.
► **TRPV1** = capsaicin receptor.
► **SP** = vasodilator + mast cell degranulator.
► **Spinothalamic = contralateral**; lesion → contralateral pain/temp loss.
► **Diaphragm → C3–C4 → shoulder**.
► **Naloxone** reverses opioid AND placebo analgesia.
► **CRPS I** = no nerve injury (RSD); **CRPS II** = nerve injury (causalgia).
► Meperidine → normeperidine → seizures NOT reversed by naloxone.
► Topical lidocaine for PHN with allodynia.
► **CBZ** = TGN 1st-line.
► **Gabapentin/pregabalin** = 1st-line for most neuropathic pain.
► **SNRIs** have largely supplanted TCAs.
► COX-2: no bleeding effect BUT ↑ CV risk; **C/I post-CABG**.
► Aspirin acetylates platelet COX irreversibly → GI bleed.
► **Avoid opioid + BZD** (synergistic resp depression).
► Methylnaltrexone / alvimopan = peripheral antagonists.
► PCA bolus: morphine 1 / hydromorphone 0.2 / fentanyl 10 µg.
► CDC 2022: avoid ≥90 MME/d; consider naloxone at ≥50 MME/d.

◉ UPDATES

◉ **2022 CDC Clinical Practice Guideline** (Dowell et al) — current opioid prescribing reference.
◉ **MME**: ≥50/d → ↑ follow-up + naloxone; avoid ≥90/d.
◉ 90 MME ≈ 90 mg hydrocodone ≈ 60 mg oxycodone.
◉ **Tapentadol** — newer stronger opioid + NE reuptake.
◉ Drug-induced deaths now **2nd leading cause** of US deaths.

CH 15 · Chest Discomfort — Approach to Acute Chest Pain

Triage of acute non-traumatic chest pain — 3 buckets: ischemia, other cardiopulmonary, non-cardiopulmonary. Killers to rule out + decision-aids (HEART, EDACS). NEET SS / INI SS-ET high yield.

Epidemiology & Framing

- **One of top 3 ED visits** in US — 6–7 million/yr.
- >60% admitted; <15% diagnosed with **ACS** (range 5–20%).
- Most common dx = **GI**; only ~5% are other life-threatening cardiopulmonary causes.
- Historical **missed MI rate** after ED discharge for 'non-ischemic' pain = **2–6%**.
- Low-risk pts (no STE, no clearly non-cardiac pain): 30-day major CV event ≈ **2.5%**.
- Three triage buckets: **(1) myocardial ischemia**; **(2) other cardiopulmonary** (peri/myocarditis, aorta, PE, pneumothorax); **(3) non-cardiopulmonary** (GI, MSK, psych).

Final Discharge Dx Distribution (Fruergaard)

Cause	%
GI	42
Ischemic heart disease	31
Chest wall syndrome	28
Pericarditis	4
Pleuritis	2
Pulmonary embolism	2
Lung cancer	1.5
Aortic aneurysm	1
Aortic stenosis	1
Herpes zoster	1

Myocardial Ischemia / Injury

- **Angina pectoris** = ischemia-related chest pain (Heberden — 'strangling + anxiety').
- Mechanism = O_2 demand > supply. Demand ↑ HR, wall stress, contractility; supply = coronary flow + arterial O_2 content.
- Irreversible injury starts at **~20 min** of severe ischemia → MI.
- **Stable** = predictable, exertional, relieved by rest.
- **Unstable IHD** classified by injury + STE: Unstable angina (no injury), **NSTEMI** (injury, no STE), **STEMI** (injury + STE).
- **ACS** = umbrella for UA + NSTEMI + STEMI (acute atherothrombosis).
- **Type 1 MI** = atherothrombosis; **Type 2 MI** = supply/demand imbalance (anemia, hypotension, tachycardia).
- Other ischemic triggers: AS, HCM, dilated CMP.

Quality / location / pattern

- Quality: **pressure, tightness, squeezing, crushing, heaviness, burning**.
- Site: **retrosternal**; radiates ulnar L arm, both arms, neck, jaw, shoulder, epigastrium.
- Stable angina: gradual onset, peaks in minutes, relieved by rest/NTG.
- MI: **≥30 min**, severe, not relieved by rest.
- **Levine sign** = clenched fist over sternum.

Other Cardiopulmonary Causes

Pericarditis

- Pericardium itself **insensitive**; pain arises from **pleural inflammation**.
- Quality = **pleuritic, sharp**; worse on inspiration, cough, supine.
- **Relieved sitting up + leaning forward**.
- Refers to **shoulder + neck (trapezius ridge)** via phrenic (C3–C5) — diaphragm sensory overlap.
- **Friction rub** on auscultation.

Myocarditis / Takotsubo

- Myocarditis = variable; pain from inflammation OR ↑ wall stress.
- **Takotsubo** (stress CMP) — abrupt CP + dyspnea after emotional/physical stressor.
- ECG may show STE + ↑ biomarkers → **mimics MI**.

Acute Aortic Syndrome

- Spectrum: dissection, intramural hematoma, penetrating ulcer.
- Pain = **severe, sudden onset, 'tearing/ripping/knifelike'**; peaks immediately.
- **Ascending** → anterior midline chest; **descending** → back.
- Risks: HTN, pregnancy, **bicuspid AV, Marfan, Ehlers-Danlos**.
- Signs: **AI murmur**, asymmetric pulses, BP arm difference.

Pulmonary Embolism

- Sudden onset CP + dyspnea ± hemoptysis.
- Small PE → **pleuritic, lateral** (pleural surface near infarct).
- Massive PE → **substernal, mimics MI** (PA distension, RV strain).
- Massive: syncope, hypotension, signs of R-heart failure.
- ECG: **sinus tachy** commonest; classic **S1Q3T3**.

Pneumothorax

- Sudden pleuritic CP + dyspnea. **Unilateral ↓ breath sounds**.
- Primary spontaneous: **young, tall, thin male; smoker; Marfan**.
- Secondary: COPD, asthma, CF.
- **Tension PTX = emergency** → hemodynamic collapse.

Pulmonary HTN / pleuritis

- Chronic PHTN → substernal pressure mimicking angina (RV ischemia).
- Pleurisy / pneumonia → **knifelike, worse on inspiration**.

♥ NEET SS HIGHLIGHTS

♥ **Type 1 MI** = atherothrombosis; **Type 2 MI** = supply-demand mismatch.
♥ **Tearing pain to back** = acute aortic syndrome until proven otherwise.
♥ Pericarditis pain **relieved by sitting up + leaning forward**; refers to **trapezius ridge**.
♥ **S1Q3T3** = classic PE ECG (sinus tachy more common).
♥ **Levine sign** = clenched fist on sternum = ischemia.
♥ Chest wall tenderness on palpation does **NOT exclude** MI.

Non-Cardiopulmonary Causes

Gastrointestinal

- **Most common** overall cause of non-traumatic CP.
- **GERD**: burning, substernal/epigastric, 10–60 min, worse postprandial recumbent, relieved by antacids.
- **Esophageal spasm**: 2–30 min squeezing, **can mimic angina + relieved by NTG/DHP CCB**.
- **Mallory-Weiss / Boerhaave** (rupture) after vomiting → severe retrosternal pain.
- **PUD**: burning, epigastric, **60–90 min postprandial**, relieved by food/antacid.
- **Biliary colic / cholecystitis**: RUQ, may refer to R shoulder/scapula, post-meal.
- **Pancreatitis**: epigastric → boring to back.

Musculoskeletal & other

- **Costochondritis / Tietze**: localized tender swelling at costochondral junction.
- **Cervical radiculitis**: aching arm/chest, worse with neck movement.
- **Herpes zoster**: dermatomal burning, vesicular rash.
- **Panic disorder** = up to **10% of ED CP**.

Approach — Lee's 4 Questions

1. **Acute life-threatening?** Unstable IHD, aortic dissection, PE, PTX.
2. **Chronic with serious complications?** Stable angina, AS, PHTN.
3. **Acute, treatable?** Pericarditis, pneumonia/pleuritis, herpes zoster.
4. **Other treatable chronic?** GERD, esophageal spasm, PUD, GB disease, MSK, anxiety.

History — Key Features

Likelihood ratios for AMI (Swap & Nagurney)

Table — CP features ↑/↓ LR for AMI

Feature ↑ LR	Feature ↓ LR
Radiation R arm/shoulder	Pleuritic
Radiation to both arms	Positional
Associated with exertion	Sharp
Radiation to L arm	Reproducible with palpation
Diaphoresis	Inframammary
Nausea / vomiting	—
Worse than prior angina or like prior MI	—
Described as pressure	—

- Highly localized (one-finger) pain → **unlikely angina**.
- Pain only above mandible or below epigastrium → rarely angina.
- Fleeting (seconds) or constant for many hours w/o ECG/biomarker change → unlikely ischemia.
- **Warm-up angina** — relief continues with exertion (Ch 284).
- NTG relief in <10 min suggests ischemia OR esophageal spasm (not specific).
- **>10 min delay to NTG relief** = either not ischemic or severe (acute MI).

Associated symptoms

- Ischemia: diaphoresis, dyspnea, nausea, fatigue, faintness, eructations.
- **Anginal equivalents** (women, elderly): dyspnea / fatigue without pain.
- Sudden dyspnea → think PE / PTX.
- Syncope + CP → massive PE, dissection, ischemic arrhythmia.
- **Inferior MI** commonly has N/V (Bezold-Jarisch / vagal).

Physical Exam

- Anxious, pale, cyanotic, diaphoretic = severe cardiopulmonary disease.
- **Levine sign** = clenched fist over sternum.
- Tachycardia + hypotension → cardiogenic shock, massive PE, tamponade, tension PTX.
- Severe HTN → aortic emergency.
- Hypoxemia + tachypnea → pulmonary cause.
- S3/S4, MR murmur → ischemia / mechanical MI complication.
- AI murmur → ascending dissection.
- Friction rub → pericarditis.
- Unilateral ↓ breath sounds → PTX/pneumonia.
- Pulse deficits → dissection.
- Unilateral leg swelling → VTE.
- **Chest wall tenderness does NOT exclude MI**.

ECG

- Obtain within **10 min** of presentation.
- Primary goal = identify **STEMI** (immediate reperfusion candidates).
- **ST-depression / symmetric T-inv ≥0.2 mV** → ischemia + high risk.
- Serial ECGs **q30–60 min** in suspected ACS.
- **Right-sided leads** if suspicion + nondx 12-lead.
- Resting ECG sensitivity for ischemia **as low as 20%**.
- PE: sinus tachy commonest; **S1Q3T3**; right axis shift.
- Pericarditis vs MI: **diffuse STE + PR depression** not in coronary distribution.

Chest Radiography & CT

- Best for **pneumonia, PTX, pulmonary edema**.
- Aortic dissection: **widened mediastinum**.
- PE: **Hampton's hump** (pleural-based wedge), **Westermark's sign** (oligemia).
- Chronic pericarditis: pericardial calcification.
- **CT angiography** — preferred for low–intermediate risk; sensitive for CAD; also rules out dissection, effusion, PE.

Cardiac Biomarkers

- **Cardiac troponin (cTn)** = preferred biomarker for MI.
- Measure in **all suspected ACS**; do NOT measure without suspicion (unless for risk stratification: PE, HF).
- **High-sensitivity assays preferred** — better NPV.
- Rapid rule-out: hs-cTn at **0 + 1–3 h**; conventional at 0 + 3–6 h.
- Late presenters (>2–3 h post-symptoms): very low hs-cTn at presentation = **NPV >99%**.
- **MI definition**: acute injury (rising and/or falling pattern; at least one value >99th %ile URL) **caused by ischemia**.
- Chronic ↑ cTn = stable values; structural HD, ESRD, interfering antibodies.
- Other labs: **D-dimer** (PE r/o), **BNP** (HF).

cTn classification (Fig 15-3)

- Elevated cTn → stable = **chronic injury**; dynamic = **acute injury**.
- Acute injury + ischemia = **MI** (Type 1 atherothrombosis vs Type 2 supply/demand).
- Acute injury, no ischemia = **non-MI acute injury** (e.g., myocarditis).

Decision-Aids — HEART & EDACS

HEART score (without troponin)

Element	0	1	2
History	Slightly suspicious	Moderately	Highly
ECG	Normal	Nonspecific	Significant ST depression
Age	<45 y	45–<65 y	≥65 y
Risk factors	None	1–2	≥3

- **0–3 = low risk**; ≥4 = not low risk.
- NPV 99.55%, captures 51.8% as low risk (with hs-cTn <LoQ).

EDACS score

- Points by age (18–45 = 2; 86+ = 20).
- Known CAD or ≥3 risk factors (in ≤50 y) = +4.
- Male sex = +6.
- Symptoms: radiation arm/jaw/shoulder/neck +5; diaphoresis +3; pain on inspiration −4; reproduced by palpation −6.
- **Low risk: 0–15**; ≥16 = not low risk. NPV 99.49%, captures 60.6%.
- Integrated CDPs + hs-cTn → ACS 'miss rates' **<0.5%**.
- Differentiate dx CDPs from **TIMI/GRACE** (prognostic; for established ACS only).

Coronary / Stress Imaging

- Indication: life-threats excluded + intermediate/undetermined risk after biomarkers.
- **Coronary CTA** preferred — sensitive for obstructive CAD; **high NPV**; also r/o dissection, effusion, PE.
- Stress nuclear / stress echo — preferred if **known epicardial disease**.
- Exercise stress preferred over pharmacologic when patient can exercise.
- Stress imaging > exercise ECG alone for dx accuracy.

❦ CLINICAL PEARLS

❦ Reproducible palpation pain reduces but **does not exclude** MI.
❦ Esophageal spasm can **mimic angina and respond to NTG** — beware.
❦ Inferior MI commonly presents with N/V (Bezold-Jarisch).
❦ Marfan + sudden CP → think dissection or PTX.
❦ PE most common ECG = **sinus tachycardia** (S1Q3T3 is classic but uncommon).
❦ **Trapezius ridge radiation** → pericarditis, not angina.

✦ MNEMONIC

PE PAINS *Life-threatening causes of acute chest pain to rule out*

P — Pulmonary embolism
E — Esophageal rupture (Boerhaave)
P — Pneumothorax (tension)
A — Acute coronary syndrome
I — Infarction (MI / Type 1 vs 2)
N — Necrotizing mediastinitis
S — Stanford aortic dissection

'5 killers' classic = ACS, dissection, PE, tension PTX, esophageal rupture.

✎ MCQ CORNER

Q1. 62 y/o HTN man, sudden severe tearing CP radiating to back; BP 200/110, AI murmur, asymmetric radial pulses. Most likely?
A. STEMI
B. Massive PE
✓ **C. Acute aortic dissection**
D. Pericarditis
► *Tearing pain to back + HTN + AI + pulse deficit = ascending aortic dissection.*

Q2. 30 y/o tall thin smoker, sudden L-sided pleuritic CP + dyspnea; ↓ breath sounds L. Best next?
A. Coronary CTA
✓ **B. Upright CXR**
C. D-dimer
D. Troponin
► *Classic primary spontaneous pneumothorax — confirm with CXR.*

Q3. 45 y/o ED CP. ECG normal. hs-troponin <LoQ at 0 and 2 h. HEART score 2. Best disposition?
A. Admit for stress test
B. Coronary CTA same day
✓ **C. Discharge with outpatient follow-up**
D. Empiric DAPT
► *HEART 0–3 + negative hs-cTn → NPV >99%, ED discharge appropriate.*

Q4. Pericardial pain classically:
A. Worsens leaning forward
✓ **B. Relieved by sitting up + leaning forward**
C. Reproduced by palpation
D. Radiates to ulnar L arm
► *Pleuritic, worse supine, relieved by sitting up and leaning forward; refers to trapezius ridge.*

Q5. Most common cause of non-traumatic chest discomfort?
A. ACS
✓ **B. GI**
C. Pulmonary embolism
D. Pericarditis
► *GI causes ~42% in Fruergaard data; only ~5% are non-IHD life-threats.*

Q6. Which favors AMI on chest pain history?
A. Pleuritic quality
B. Reproducible with palpation
✓ **C. Radiation to both arms**
D. Inframammary location
► *Radiation to both arms has highest +LR for AMI (Swap-Nagurney).*

LAST MINUTE REVISION

► 3 buckets: ischemia · other cardiopulm · non-cardiopulm.
► 5 killers: ACS, dissection, PE, tension PTX, esophageal rupture.
► Type 1 MI = atherothrombosis; Type 2 = supply/demand.
► Tearing pain to back + HTN + AI = aortic dissection.
► PE small = pleuritic lateral; massive = substernal mimics MI.
► Pericarditis = pleuritic, ↑ supine, ↓ sitting forward; rub; **trapezius radiation**.
► PTX: young thin male smoker, Marfan.
► Esophageal spasm relieved by NTG — mimics angina.
► PUD pain 60–90 min postprandial; biliary colic refers R shoulder.
► Most common cause overall = GI (~42%); IHD ~31%.
► Radiation to **both arms** = highest +LR for AMI.
► Reproducible on palpation lowers but doesn't exclude AMI.
► ECG within 10 min; serial q30–60 min in suspected ACS.
► STE diffuse + PR depression = pericarditis.
► PE ECG: sinus tachy (most common); **S1Q3T3** classic.
► hs-cTn — 0/1–3 h protocol; very low at presentation >2–3 h post-Sx → NPV >99%.
► HEART 0–3 = low risk; EDACS 0–15 = low risk.
► CDP + hs-cTn miss rate <0.5%.
► Coronary CTA — high NPV; also rules out dissection, effusion, PE.
► Stress imaging preferred if known epicardial disease.

UPDATES

◉ **Universal Definition of MI** (4th UDMI) — Type 1 vs Type 2 distinction guides therapy.
◉ **hs-cTn 0/1-h algorithm** (ESC) endorsed for rapid rule-out/in.
◉ **2021 AHA/ACC Chest Pain Guideline** — risk-stratify all ED CP with validated CDP; coronary CTA Class I for low-int risk.

CH 16 · Abdominal Pain — Approach & Differential Diagnosis

Mechanisms of abdominal pain (parietal, visceral, referred, metabolic, neurogenic) + clinical approach. Pain severity ≠ disease severity. Quadrant-based differential. NEET SS / INI SS-ET high yield.

Framing — Diagnostic Principles

- Few clinical situations require **greater judgment**; catastrophe heralded by subtle signs.
- Goal = separate **urgent operative** from **non-operative manageable**.
- **'Acute / surgical abdomen' is unacceptable** as a diagnosis — misleading.
- Most common admissions: **nonspecific pain**, acute appendicitis, urologic pain, intestinal obstruction.
- **Pain severity does NOT correlate** with disease severity. Hunger reliability = poor.
- Mildest pain may need surgery; most severe may not.
- **Do NOT withhold analgesia** pending diagnosis — does not obscure findings.

Mechanism 1 — Parietal Peritoneal Inflammation

- **Steady, aching**, localized over inflamed area; **somatic nerves**.
- Intensity depends on irritant type, quantity, rate of exposure.
- Pain order: sterile acidic gastric > pancreatic enzymes > bile > bacterial > blood/urine.
- **Bacterial** contamination = low intensity until mediators build (PID, distal perforation).
- **Accentuated by pressure / cough / movement**.
- Patient **lies still** (vs colic = restless / thrashing).
- **Reflex muscle spasm** over involved segment.
- Spasm may be **minimal / absent** in: retrocecal perforation, lesser sac perforation, obtunded, debilitated, elderly, immunosuppressed, psychotic.
- Slowly developing process → attenuated spasm.

Mechanism 2 — Hollow Viscus Obstruction

- **Intermittent / colicky**, poorly localized.
- Absence of cramping can mislead — distention may give steady pain with rare paroxysms.

Bowel obstruction

- **SBO** = poorly localized, intermittent **peri-/supraumbilical**.
- Dilated atonic bowel → colicky character diminishes.
- Strangulation → spread to **lumbar region** (mesenteric root traction).
- **Colon obstruction** = lesser intensity, **infraumbilical**, may radiate lumbar.

Biliary tree

- Sudden distention = **steady pain (not colicky)** — 'biliary colic' misleading term.
- **Gallbladder**: RUQ → R posterior thorax / tip of R scapula; often near midline.
- **CBD**: epigastric → upper lumbar.
- **Gradual** biliary dilation (Ca head of pancreas) → no or mild epigastric/RUQ ache.
- **Pancreatic duct**: like CBD; **worse supine, relieved by upright**.

Urinary

- **Bladder** obstruction = dull suprapubic; restlessness only in obtunded.
- **Intravesical ureter** = severe suprapubic + flank → radiates penis/scrotum/inner thigh.
- **UPJ** = CVA pain.
- **Ureter** (rest): flank → same-side abdomen.

Mechanism 3 — Vascular Disturbances

- Misconception: vascular abdominal pain is sudden / catastrophic.
- **SMA occlusion** often = mild continuous/cramping diffuse pain for **2–3 days** before collapse.
- Early pain due to **hyperperistalsis**, not peritonitis.
- **Pain out of proportion to exam** + vascular RFs → SMA occlusion (mesenteric ischemia).
- **Ruptured AAA**: pain radiates **sacral, flank, or genitalia**; may persist days before rupture.

Mechanism 4 — Abdominal Wall

- Constant aching; worsened by movement, standing, pressure.
- **Rectus sheath hematoma** — anticoag therapy; LQ mass.
- **Carnett sign** (implied): pain persists/worsens with abdominal wall tensing → wall origin (vs visceral = relieved).
- Other muscle groups involved → myositis.

> ♥ **NEET SS HIGHLIGHTS**
>
> ♥ **Lies still** = peritonitis; **thrashing** = colic.
> ♥ **Pain out of proportion to exam** + vascular RF → **mesenteric ischemia**.
> ♥ **Cullen / Grey-Turner** = retroperitoneal bleed (pancreatitis, ruptured AAA, ectopic).
> ♥ Boas sign — R subscapular tip pain in acute cholecystitis (GB referred).
> ♥ Pancreatic / CBD pain = epigastric → back; **pancreatic relieved by sitting up**.
> ♥ Rovsing, psoas, obturator → appendicitis.

Referred Pain from Extraabdominal Source

- Always consider **intrathoracic disease** in upper abdominal pain.
- Masqueraders: MI, pneumonia, pericarditis, PE, esophageal disease.
- **Diaphragmatic pleuritis** (pneumonia/PE) → RUQ + **supraclavicular** referral.
- Subscapular pain (R) = biliary extrahepatic distention.
- Thoracic-origin referred pain: **splinting hemithorax**, ↓ respiratory excursion.
- **Abdominal wall spasm from thoracic source diminishes on inspiration**; from abdominal source persists throughout cycle.
- Palpation over referred zone does **not accentuate** or may relieve.
- Coexistence common — biliary disease + MI → epigastric pain; biliary colic referred to precordium/L shoulder in prior angina pt.
- **Spinal nerve referred**: worse with cough/sneeze/strain; **hyperesthesia** over dermatome.
- Testes/seminal vesicle referred → dull poorly localized; **↑ with slightest pressure** on organ.

Metabolic Causes

- Can simulate any intraabdominal disease.
- **Hyperlipidemia** ± concurrent pancreatitis — beware unnecessary laparotomy.
- **C1 esterase inhibitor deficiency** (hereditary angioedema) → severe episodic pain.
- **Familial Mediterranean fever** — hallmark = abdominal pain.
- **COVID-19** rare: severe abdominal pain w/o pulm symptoms.
- **Porphyria + lead colic** → mimic obstruction (severe hyperperistalsis).
- Uremia / DM → nonspecific, shifting pain.
- **DKA** may be **precipitated** by appendicitis/obstruction — if pain doesn't resolve with metabolic correction → look for surgical cause.
- **Black widow spider bite** → intense rigidity (abdomen + back).
- Other metabolic: diabetes, uremia, hyperparathyroidism, acute adrenal insufficiency.

Neurogenic Causes

- **Causalgic** — burning, peripheral nerve distribution; touch/temp triggers.
- Cutaneous 'pain spots' from old nerve injury.
- Gentle palpation triggers pain but NO rigidity, NO respiratory disturbance, NO distention.
- Pain NOT related to food.
- **Spinal nerve/root**: sudden lancinating; causes — **herpes zoster, arthritis, tumors, HNP, DM, syphilis (tabes)**.
- Worse with spine movement; hyperesthesia common.
- **IBS** (functional) — pain + altered bowel habits; brought on by stress; N/V rare; tenderness inconsistent.

Immunocompromised Patients

- Normal physiologic responses absent/masked.
- Unusual pathogens: **CMV, mycobacteria, protozoa, fungi**.
- May cause occult/overt GI perforation.
- **Splenic abscess**: Candida, Salmonella — LUQ/L flank.
- **Acalculous cholecystitis**: AIDS / IC → cryptosporidiosis, CMV.
- **Neutropenic enterocolitis (typhlitis)** — chemo BM suppression; fever + RLQ pain.
- Consider **acute GVHD**.

Differential by Location (Table 16-3)

RUQ	Epigastric	LUQ
Cholecystitis	PUD	Splenic infarct/rupture/abscess
Cholangitis	Gastritis	Gastritis
Pancreatitis	GERD	Gastric ulcer
Pneumonia / empyema	Pancreatitis	Pancreatitis
Pleurisy	MI / pericarditis	Subdiaphragmatic abscess
Subdiaphragmatic abscess	Ruptured AAA	—
Hepatitis / Budd-Chiari	Esophagitis	—

RLQ	Periumbilical	LLQ
Appendicitis	Early appendicitis	Diverticulitis
Salpingitis	Gastroenteritis	Salpingitis
Inguinal hernia	Bowel obstruction	Inguinal hernia
Ectopic pregnancy	Ruptured AAA	Ectopic pregnancy
Nephrolithiasis	—	Nephrolithiasis
IBD	—	IBS / IBD
Mesenteric lymphadenitis	—	—
Typhlitis	—	—

Diffuse non-localized

- Gastroenteritis, mesenteric ischemia, bowel obstruction, IBS, peritonitis.
- DM, malaria, FMF, metabolic, psychiatric.

Approach to the Patient

- Only **exsanguinating intraabdominal hemorrhage** (ruptured AAA) → straight to OR.
- No absolute contraindication to OR in massive bleed; patients can die awaiting imaging.
- **Chronological sequence often more important than location**.
- Don't withhold analgesia — does NOT obfuscate diagnosis.
- **Menstrual history mandatory** in females; gravid uterus alters anatomy.
- Normal pregnancy can have leukocytosis.
- Inspect: facies, position, respiration.
- Examine **gently** — once roughly examined, accurate assessment is impossible for the next examiner.
- **Rebound by sudden release = cruel + unnecessary**; use **gentle percussion** or ask patient to **cough**.
- **Pelvic + rectal exam mandatory** in every abdominal pain patient.
- **Auscultation = LEAST revealing**; normal bowel sounds with strangulation possible.
- **Silent abdomen** severe chemical peritonitis (sudden onset).

Laboratory caveats

- Labs rarely establish diagnosis.
- **Leukocytosis alone is NOT a deciding factor** for OR.
- WBC >20,000/μL: perforation, but also pancreatitis, cholecystitis, PID, intestinal infarction.
- **Normal WBC does NOT exclude** serious disease.

CLINICAL PEARLS

- Pancreatic pain — **leans forward** for relief.
- AAA pain radiating to sacrum/flank/groin → impending rupture.
- **Mesenteric ischemia** = pain out of proportion to exam in vascular pt.
- FMF / porphyria / lead → unexplained recurrent abdominal pain.
- DKA may unmask surgical abdomen — re-examine if pain persists after correction.
- Typhlitis: chemo neutropenic + fever + RLQ pain.