

AI-01687 DDx Stable Angina

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What is unstable angina and stable angina. Explain and deep dive. Perform a DDx of unstable angina and stable angina. What are the physiological pillars of unstable and stable angina. List 22 non medical protocols I can deploy during stable and unstable angina.

Stable vs Unstable Angina: Deep Dive with Differential Diagnosis, Physiology, and Non-Medical Protocols

Definitions

Stable Angina

- Definition: Chest pain or discomfort predictably triggered by exertion or emotional stress and relieved by rest or nitroglycerin.
- Cause: Fixed atherosclerotic plaque causing partial obstruction of coronary arteries.
- Duration: <15 minutes.
- Pattern: Consistent over weeks to months.

Unstable Angina (UA)

- Definition: New-onset, worsening, or resting chest pain not relieved predictably by rest or medications.
- Cause: Ruptured plaque with thrombus formation without complete occlusion, leading to myocardial ischemia.

- Duration: >20 minutes, increasing frequency.
- Part of Acute Coronary Syndrome (ACS): Alongside NSTEMI and STEMI.

Differential Diagnosis (DDx)

Feature	Stable Angina	Unstable Angina	Other DDx
Trigger	Exertion, stress	At rest, minimal exertion	Pericarditis, GERD, PE, Aortic dissection
Relief	Rest, nitroglycerin	Minimal or temporary relief	GERD relieved by antacids
Pattern	Predictable	Worsening/unpredictable	Costochondritis—localized pain on palpation
ECG	Normal or ST depression during stress	ST depression/T-wave inversion at rest	STEMI = ST elevation
Cardiac enzymes	Normal	Normal (unlike NSTEMI)	Elevated in NSTEMI, STEMI
Pain duration	<15 minutes	>20 minutes	>30 min in MI
Radiation	Left arm, jaw	Similar, but often more severe	PE: pleuritic pain, hemoptysis
Risk	Chronic risk of MI	Immediate risk of MI	Anxiety: vague chest tightness

Physiological Pillars

Stable Angina

1. Myocardial Oxygen Demand Exceeds Supply:
 - Triggered by exercise, stress, cold, heavy meals.
2. Fixed Coronary Artery Narrowing:
 - >70% stenosis reduces blood flow.

3. No Plaque Rupture:

- Endothelium intact; low thrombosis risk.

4. Predictable Autonomic Response:

- Increased HR and BP during activity.

5. Collateral Circulation May Develop:

- Mitigates symptoms over time.

Unstable Angina

1. Plaque Rupture with Thrombus Formation:

- But non-occlusive.

2. Increased Inflammatory Response:

- Cytokines, CRP, macrophages active.

3. Transient Ischemia at Rest:

- Mismatch between supply and demand even at low demand.

4. Impaired Endothelial Function:

- Loss of vasodilatory response.

5. Sympathetic Overactivation:

- Increased catecholamines exacerbate ischemia.

22 Non-Medical Protocols for Stable and Unstable Angina

These are supportive, not a replacement for medical care. In unstable angina, medical intervention is critical.

A.Shared Non-Medical Strategies (Helpful in Both Types)

1. Immediate rest at onset of chest discomfort.
2. Deep diaphragmatic breathing to reduce sympathetic tone.

3. Cold avoidance (vasoconstriction risk).
4. Warm compress on chest (non-medical comfort).
5. Stay in upright position (decreases preload).
6. Loosen tight clothing.
7. Mental relaxation techniques (guided imagery).
8. Mindfulness or Zen meditation daily.
9. Avoid heavy meals (especially fats).
10. Quit smoking/vaping (vasoconstriction, plaque instability).
11. Daily walking program under tolerance limit.
12. Progressive muscle relaxation routines.
13. Avoid high-stress situations (releases catecholamines).
14. High-quality sleep routine (7–8 hours).
15. Weight management via nutrition tracking.
16. Hydration (to optimize blood viscosity).
17. Limit caffeine and energy drinks.
18. Time-restricted eating (insulin sensitivity).
19. Coenzyme Q10-rich foods (spinach, broccoli).
20. Omega-3 from fish or seeds for anti-inflammation.
21. Hawthorn berry tea (traditional cardi tonic, consult doctor).
22. Develop a “chest pain action plan” with family trained to assist.

Specific Caution:

- For Unstable Angina, even non-medical measures must not delay ER visit.
- Symptoms should be documented and monitored, but not self-managed without supervision.

Summary Table

Aspect	Stable Angina	Unstable Angina
Predictable	Yes	No
Pain at rest	No	Yes
Risk of MI	Low/moderate	High
Medical emergency	No	Yes
Cause	Fixed plaque	Ruptured plaque
Relieved by rest/NTG	Yes	Not always
Cardiac enzymes	Normal	Normal
Troponin	Normal	Normal
ECG	Normal/ST changes during stress	ST depression/T-wave inversion

Would you like a one-page visual summary, or should I prepare a printable checklist for daily angina-friendly habits?

STEMI vs NSTEMI are two types of acute myocardial infarction (heart attack). They differ in severity, ECG findings, and how blood flow is blocked in the heart. Here's a clear side-by-side comparison:

Feature	STEMI (ST-Elevation Myocardial Infarction)	NSTEMI (Non-ST-Elevation Myocardial Infarction)
ECG Findings	ST segment elevation in ≥ 2 contiguous leads or new LBBB	ST depression or T-wave inversion (no ST elevation)
Cause	Complete occlusion of a coronary artery	Partial occlusion or severe narrowing of a coronary artery
Troponin (Biomarker)	Elevated	Elevated
Severity	More severe; more myocardium at risk	Less severe than STEMI
Urgency of Treatment	Immediate (within 90 mins) – PCI or fibrinolysis	Urgent but not as immediate – PCI usually within 24–72 hrs
Treatment Approach	Reperfusion therapy (PCI or thrombolytics)	Antiplatelets, anticoagulants, and possible PCI
Mortality Rate (short-term)	Generally higher due to extent of damage	Lower initially but can worsen if not managed

Key Mnemonic:

- STEMI = ST Elevation → Major blockage → Immediate intervention
- NSTEMI = Non ST elevation → Not fully blocked → Not as urgent (but still serious)

Clinical Example:

- STEMI: A 60-year-old man with crushing chest pain, ECG shows ST elevation in leads II, III, and aVF. He needs immediate PCI.
- NSTEMI: A 70-year-old woman with chest tightness, no ST elevation on ECG, but positive troponin. Treated with meds first, PCI scheduled.

Let me know if you want visuals or case studies.