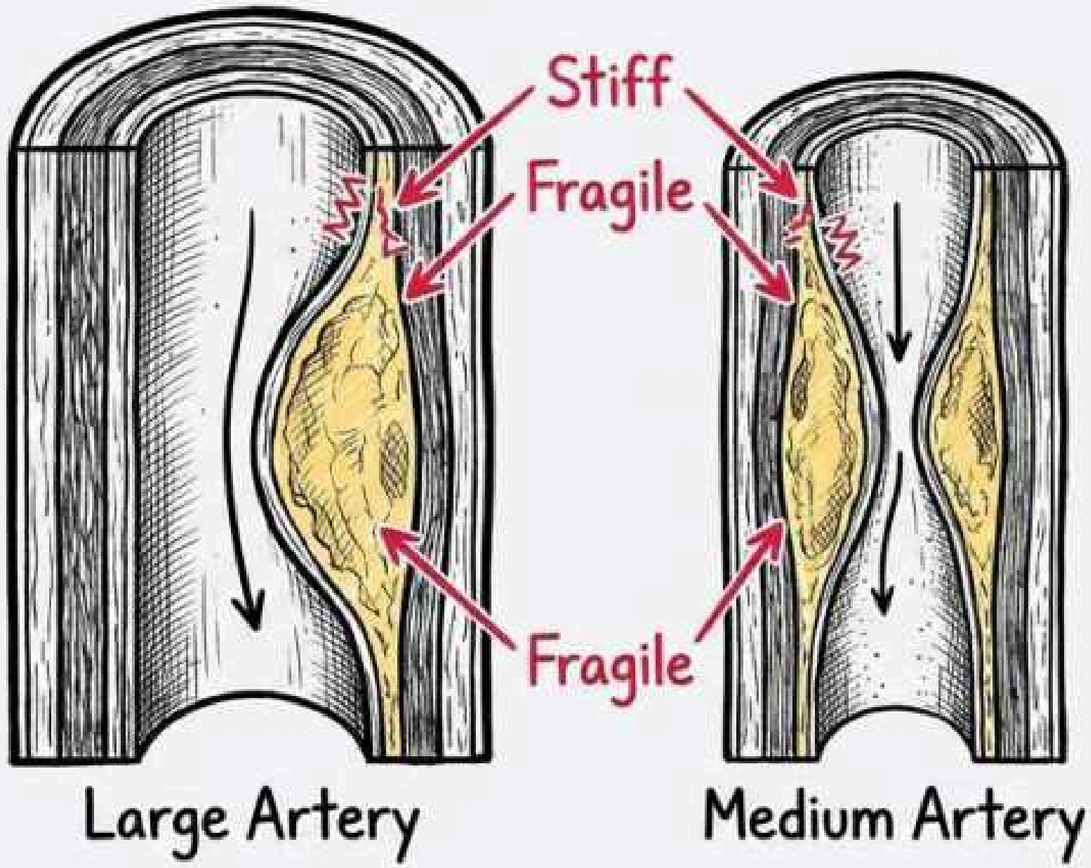
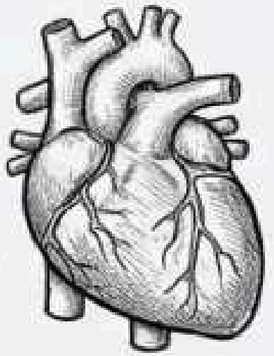
Atherosclerosis: The Foundation of Vascular Disease

¶ 3.1

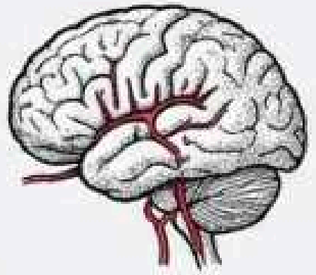
Disease of Large & Medium Arteries



Underlying Cause of:



Heart Attack



Stroke



Peripheral Narrowing

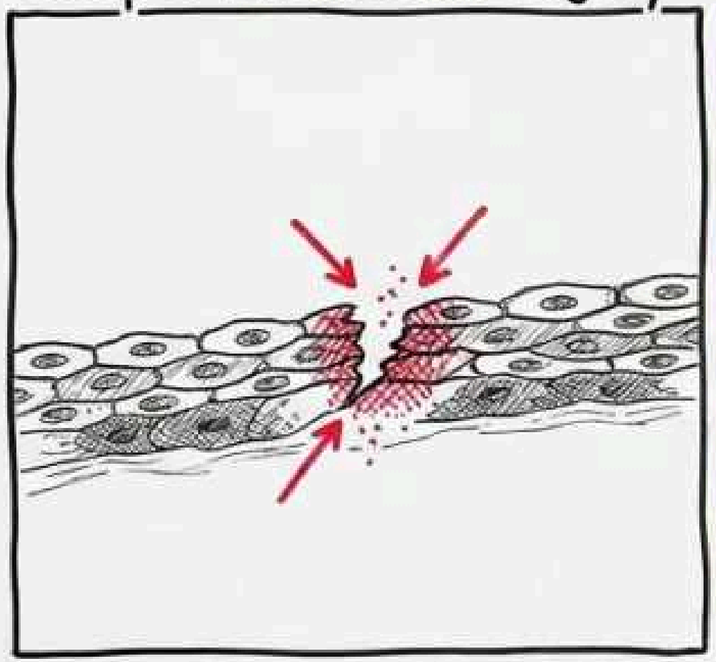
Atherosclerosis = Plaque Build-up × Decades

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Plaque Initiation: The Cellular Trigger

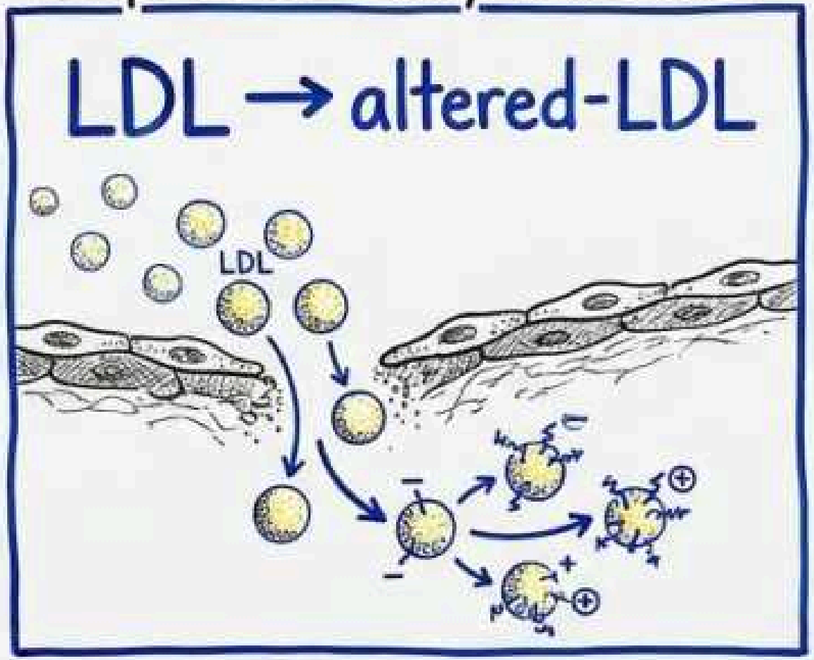
¶ 3.2

Step 1: Endothelial Injury



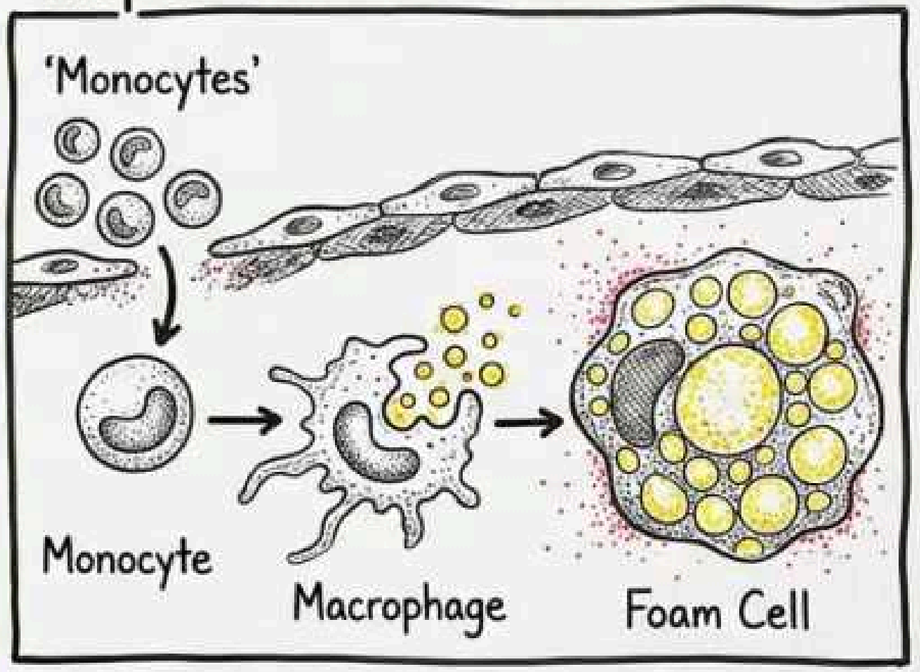
Chronic stress, hypertension, or toxins cause physical damage to the vessel lining.

Step 2: LDL Entry & Alteration



Low-density lipoprotein / "Bad Cholesterol" slips in and chemically alters

Step 3: Cellular Transformation



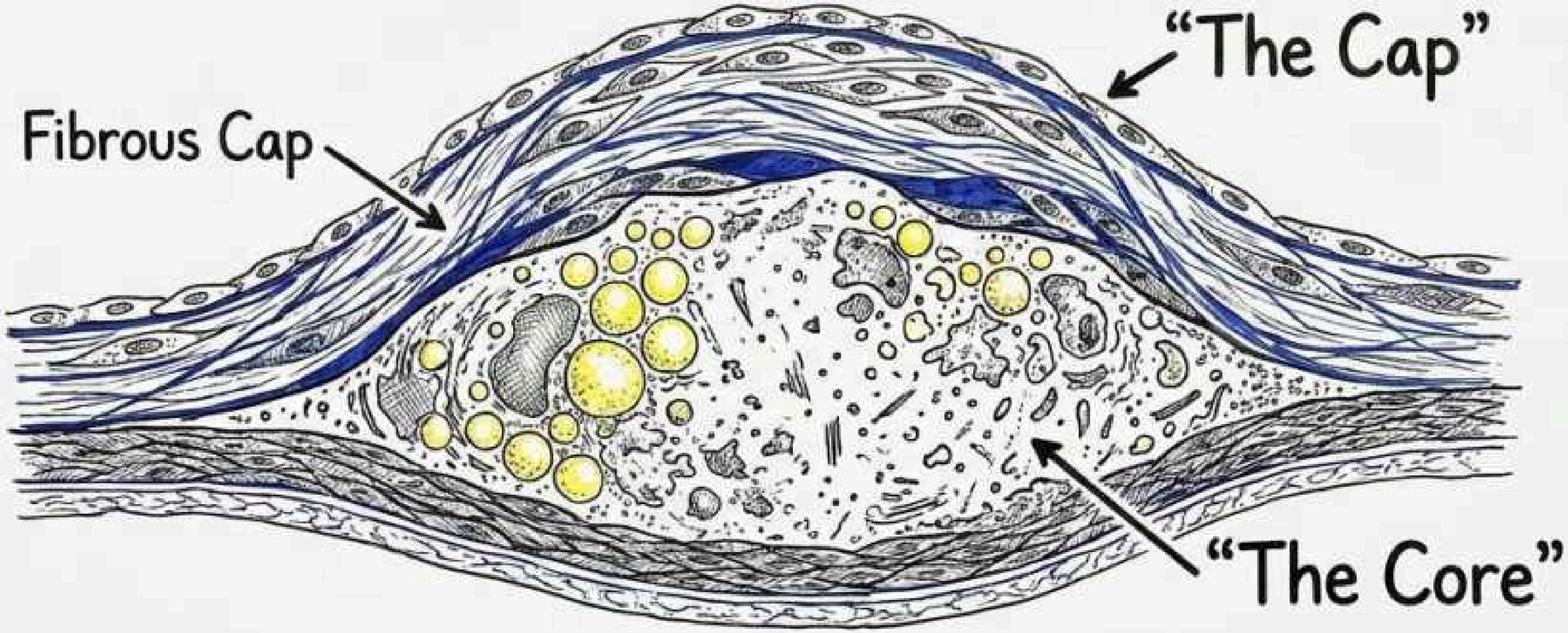
Immune cells become trapped, creating fatty streaks.

The bad cholesterol (LDL) acts as the primary trigger initiating the cellular cascade.

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Plaque Growth: The Inflammatory Paradigm

¶ 3.3



Atherosclerosis = Inflammatory Disease + Fatty Disease

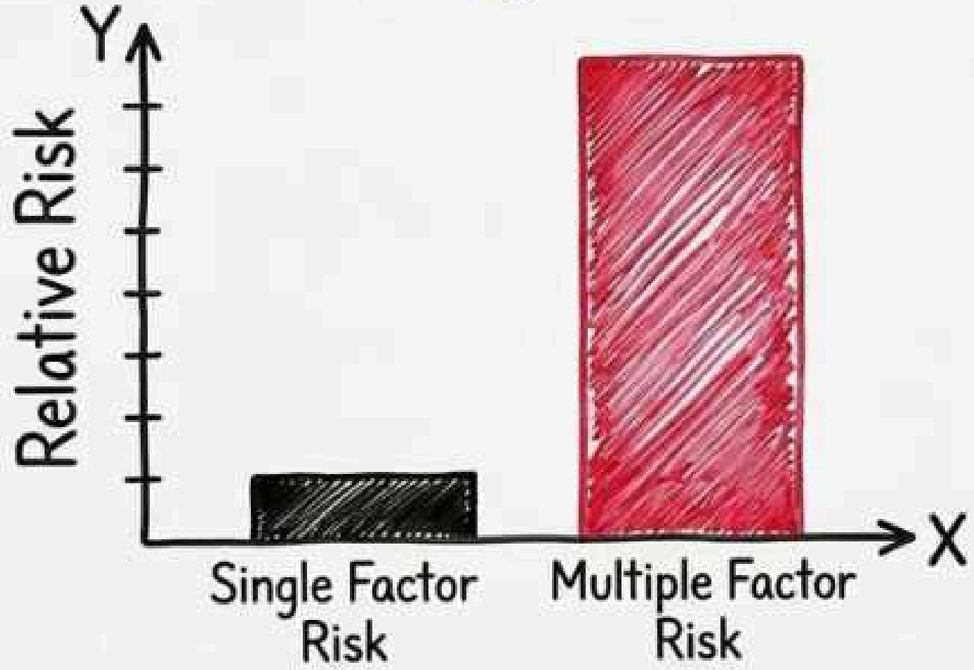
Not just a plumbing problem. The discovery of the inflammatory component fundamentally changed how modern researchers approach prevention.

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The Multiplier Effect of Risk Factors

¶ 3.4

$$Risk_Total = f(LDL) \times f(BP) \times f(Smoking) \times f(Diabetes) \times Age \times Sex_Male \times Genetics$$



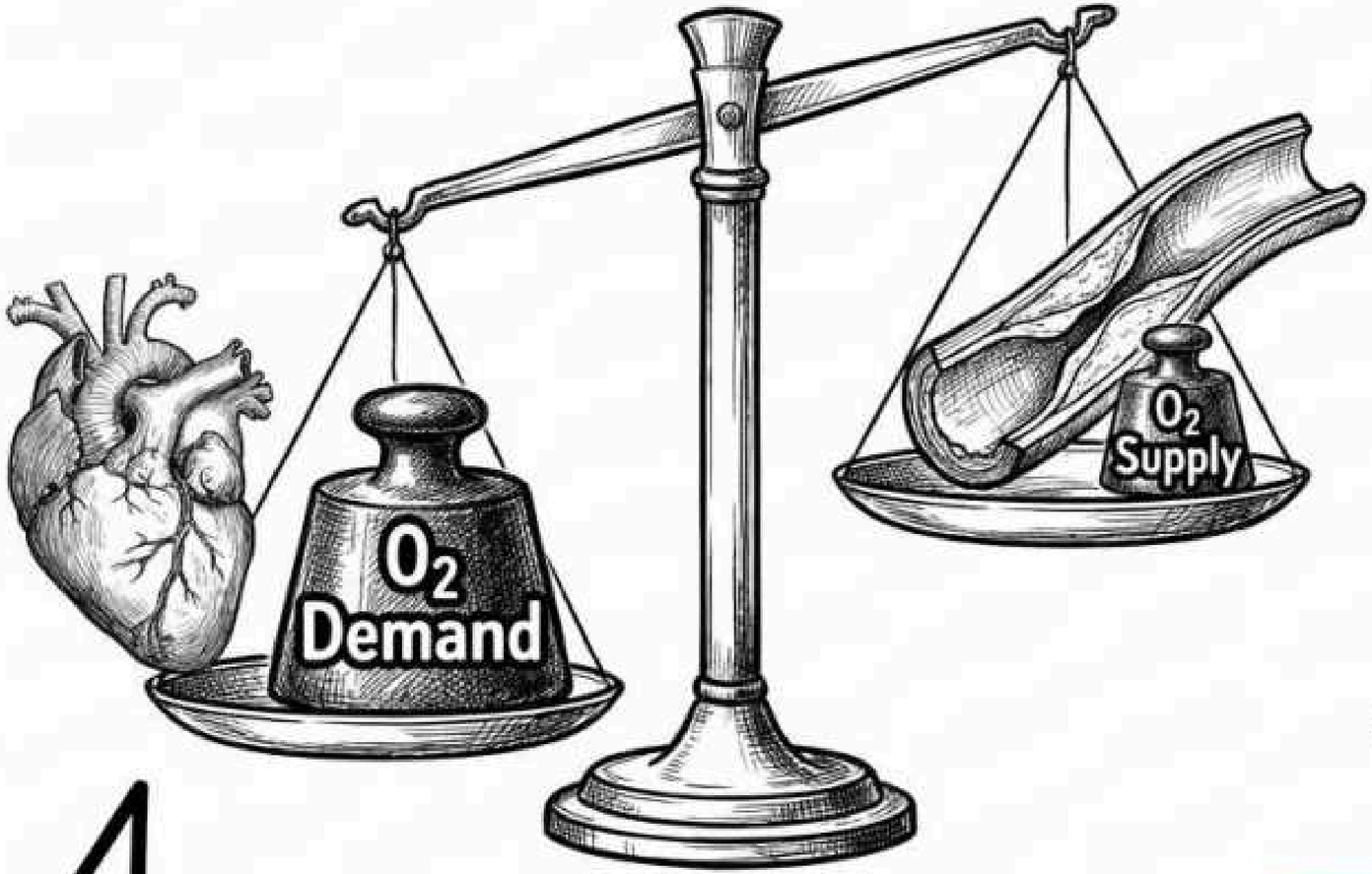
- Risk factors multiply one another's effects.
- A person with several factors faces exponentially greater risk.

As originally identified by the Framingham Heart Study (Ref: Medicine 515 B).

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Stable Angina: The Physiology of Demand Ischemia

¶ 3.5



Resting State:

Demand ≤ Supply

(Adequate blood flow through narrowed artery = No symptoms)

Exertion/Emotion State:

Demand > Supply

= Ischemia → Stable Angina

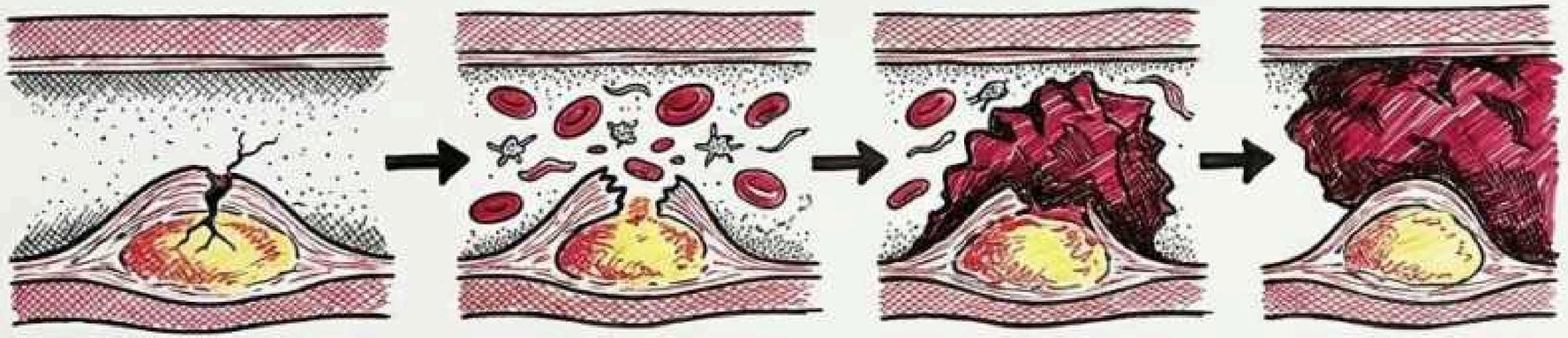
Symptom: Pressing or squeezing chest discomfort.

Rest = Relief (within minutes)

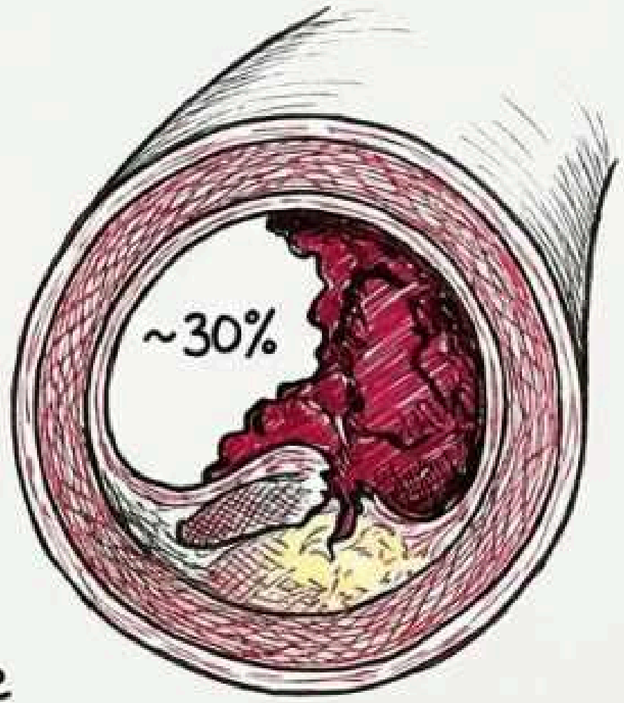
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Plaque Rupture: The Acute Crisis

¶ 3.6



Crack forms in Fibrous Cap → Soft Fatty Core exposed directly directly to blood → Clot (Thrombus) forms on top within minutes → Partial or complete arterial blockage



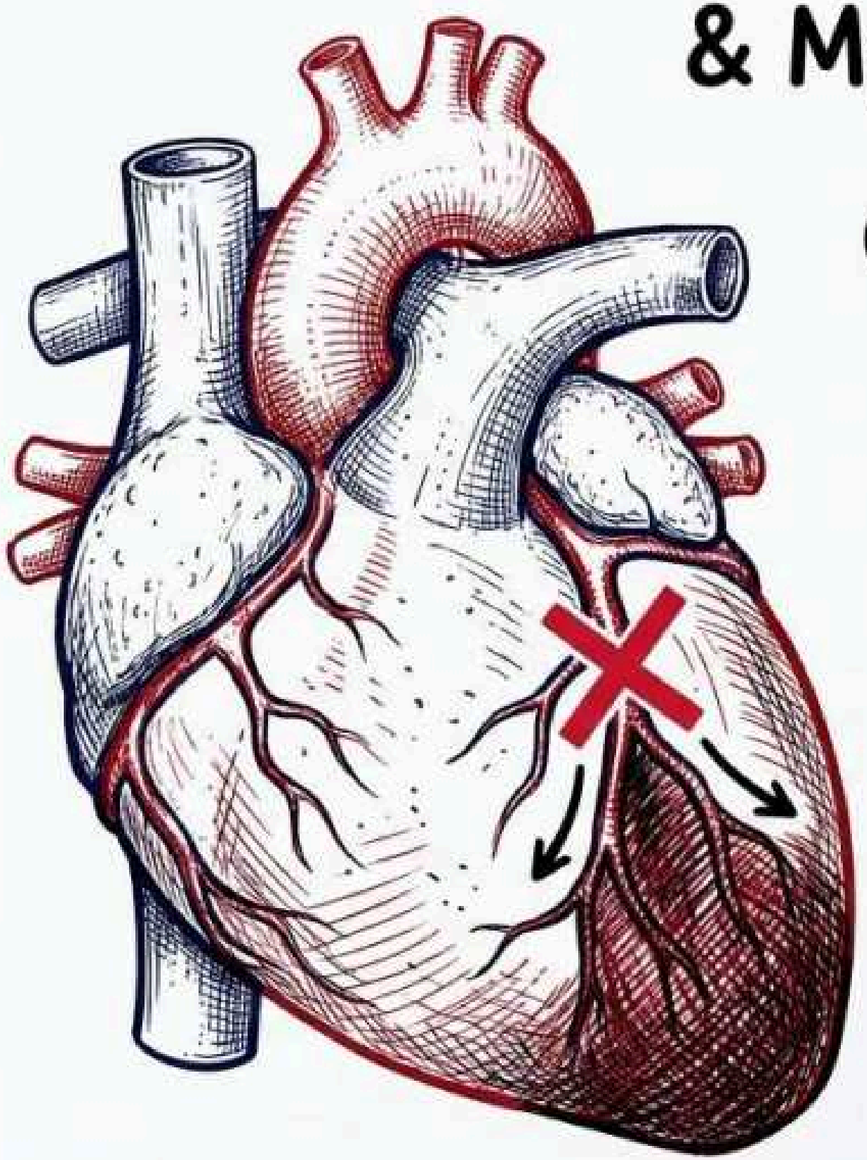
⚠ Warning: Severe narrowing is NOT a prerequisite for rupture.

This explains why many patients have zero warning symptoms before a sudden event

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Acute Coronary Syndromes (ACS) & Myocardial Infarction

¶ 3.7



Complete Blockage = Myocardial Infarction (Heart Attack)

Dying muscle is permanently replaced by rigid scar tissue → Cannot contract.

Red Flag Symptoms

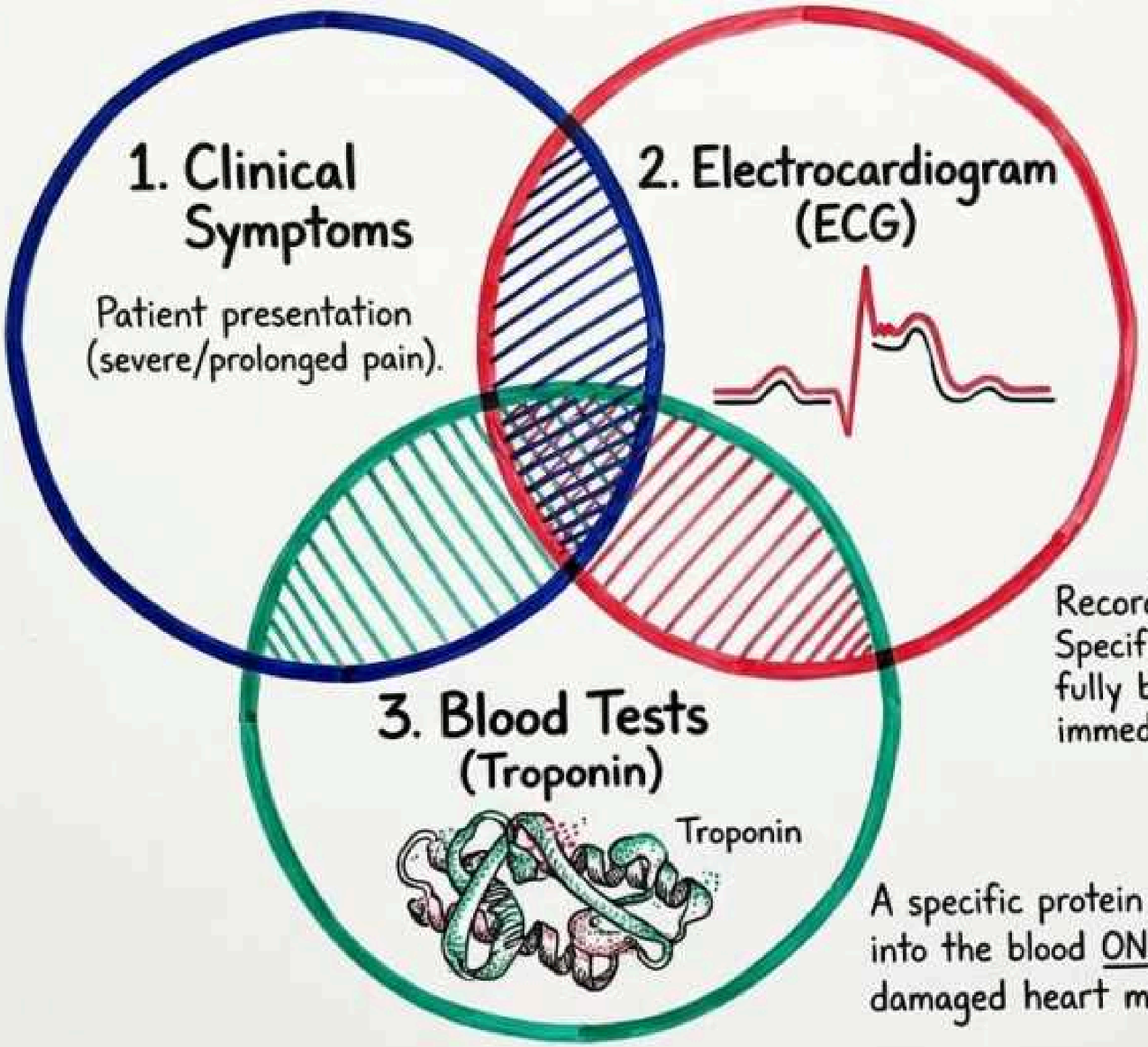
[1] Severe
[2] Prolonged
[3] Occurring at Rest

ACTION: Immediate Emergency Care Required.

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The Diagnostic Triad for Myocardial Infarction

¶ 3.8



Records electrical activity. Specific patterns identify a fully blocked artery requiring immediate reopening.

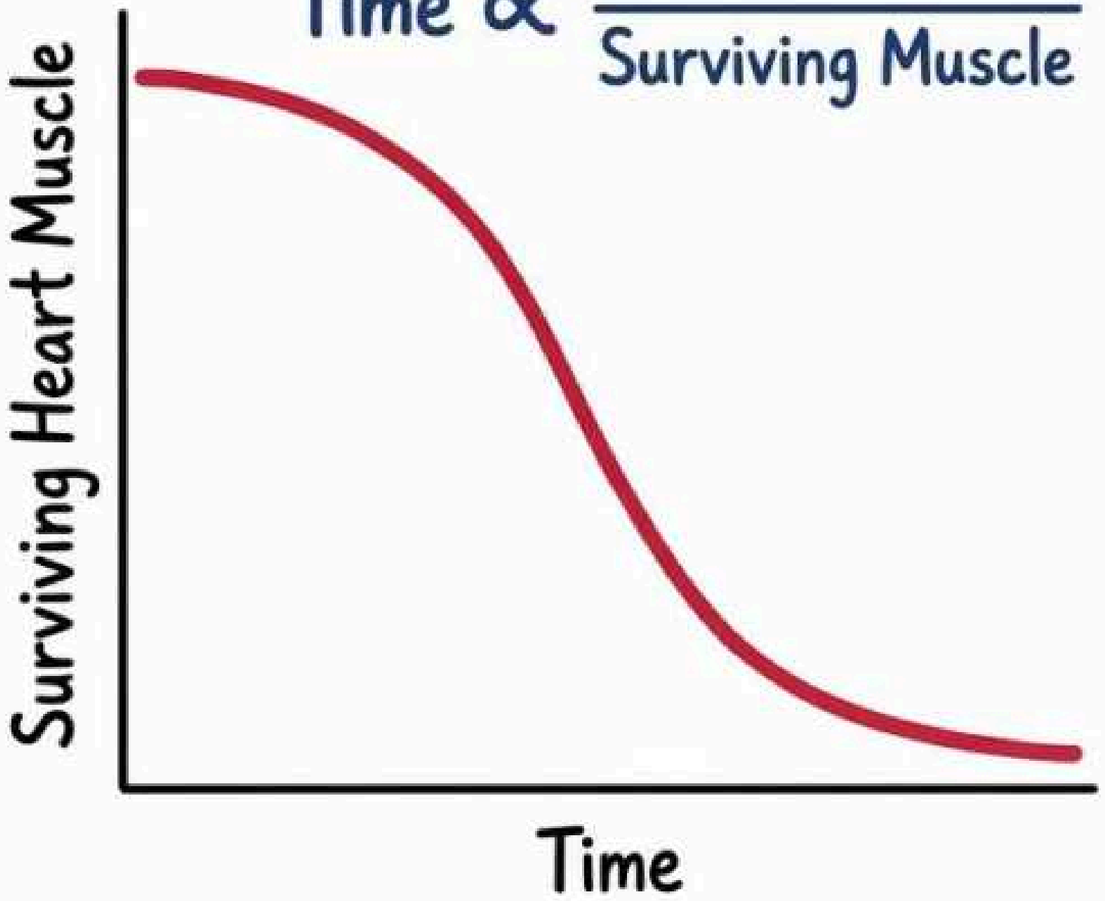
A specific protein released into the blood ONLY from damaged heart muscle.

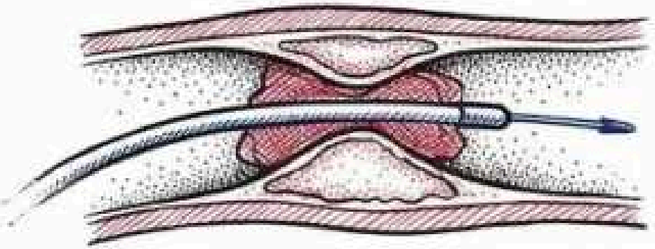
Gemini Notebook

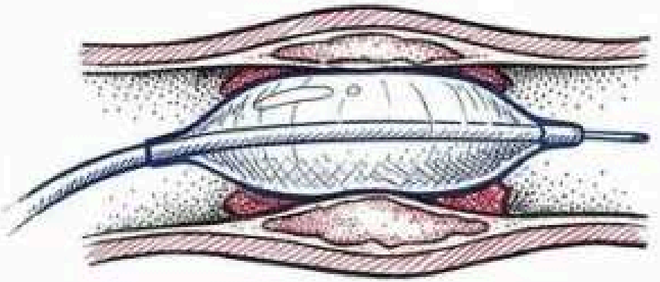
Acute Intervention: Time is Muscle

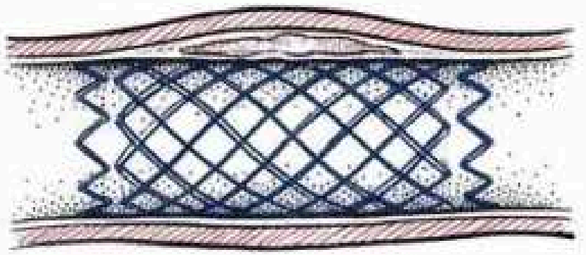
¶ 3.9

$$\text{Time} \propto \frac{1}{\text{Surviving Muscle}}$$



1. 
Pass thin tube (catheter) into artery.

2. 
Inflate balloon to crush the clot/plaque.

3. 
Leave a small mesh tube (Stent) to hold the artery open.

* Alternative Treatment **Note**: Clot-dissolving drugs are utilized if catheterization is not quickly available.

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Post-MI & Secondary Prevention

¶ 3.10

Quadrant 1 (Anti-clot): Antiplatelet drugs

Platelets - Stickiness

Quadrant 2 (Lipids & Plaque): Statins

Lower cholesterol + Stabilize existing existing plaques.

Quadrant 3 (Hemodynamics): Workload-reducing drugs

Lower the heart's baseline oxygen demand.

Quadrant 4 (Lifestyle)

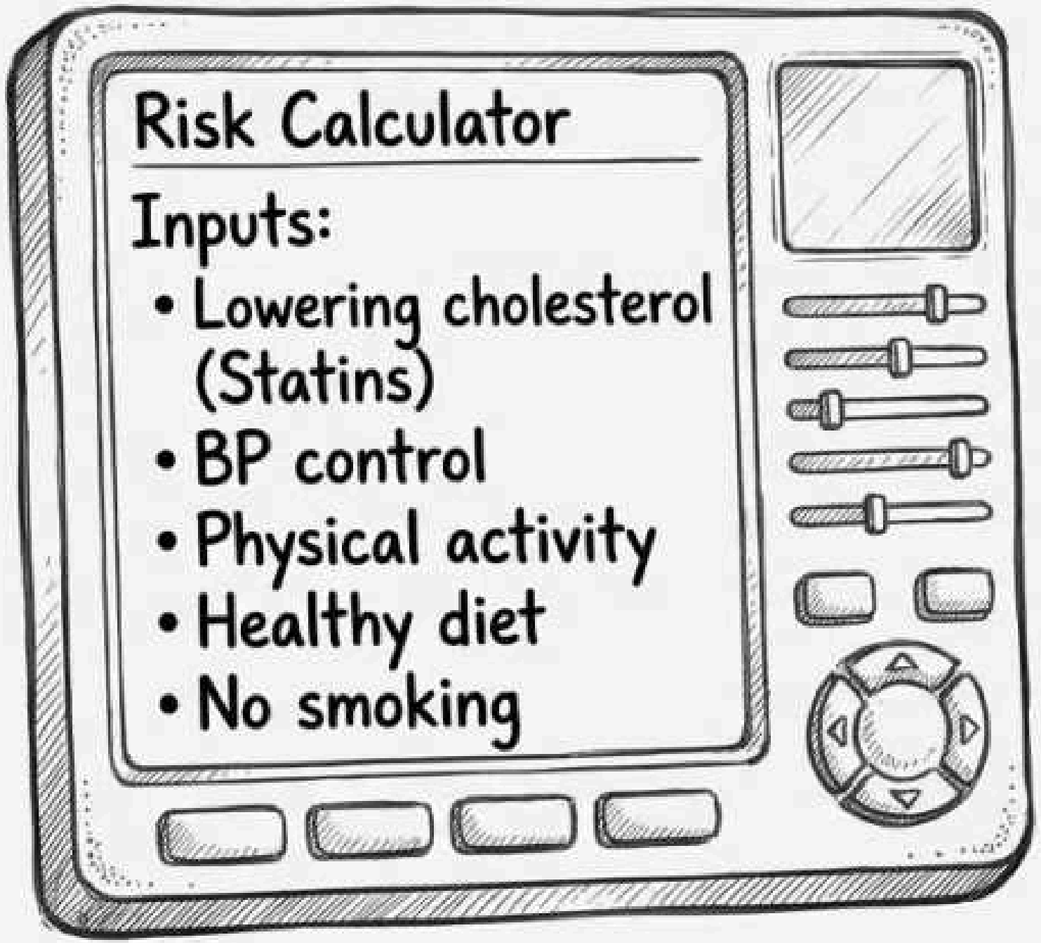
- ✓ Stop smoking
- ✓ Control Blood Pressure
- ✓ Control Diabetes

Goal: Drastically reduce the risk of further atherosclerotic events in established coronary disease.

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Primary Prevention: Intervening Before the Event

¶ 3.11



Clinical Decision Formula:

Absolute Benefit of Medicines > Risk/Cost ⟶ Intervene

- Prevention before any event is the most valuable intervention.
- Substantially reduces the risk of both heart attack and stroke.

Applying the risk calculator principles established in Medicine 514 R

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Lecture Synthesis: The Path of Atherosclerosis

¶ 3.12

Phase 1: Slow Inflammatory Build-up

↓

Phase 2: Arterial Narrowing → Angina

↓

Phase 3: Plaque Rupture → Clot → Heart Attack

→ What happens when the heart, damaged by this disease (scar tissue) or others, can no longer pump enough blood?

→ NEXT LECTURE: Heart Failure

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