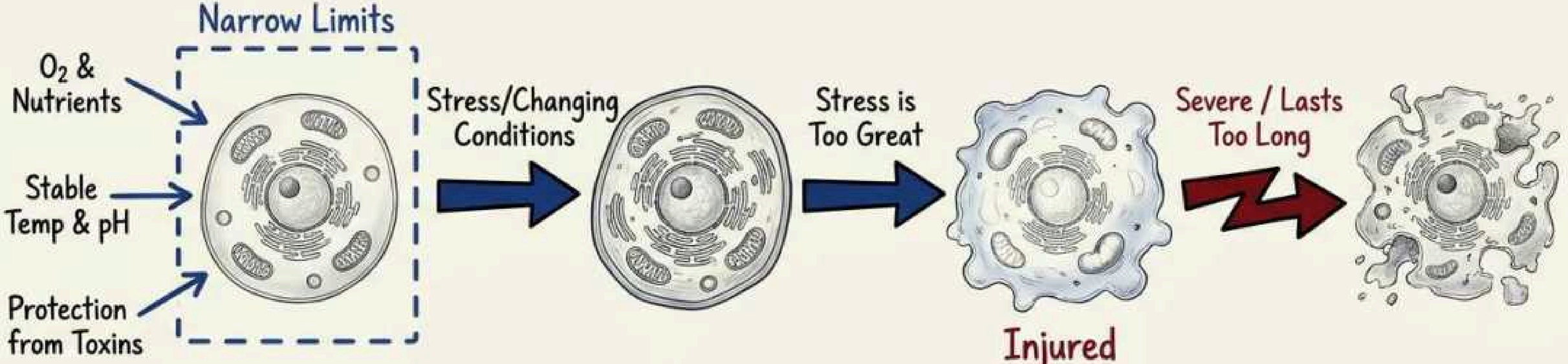
MED 505 B Lecture 1:
Cell Adaptation, Injury and Death

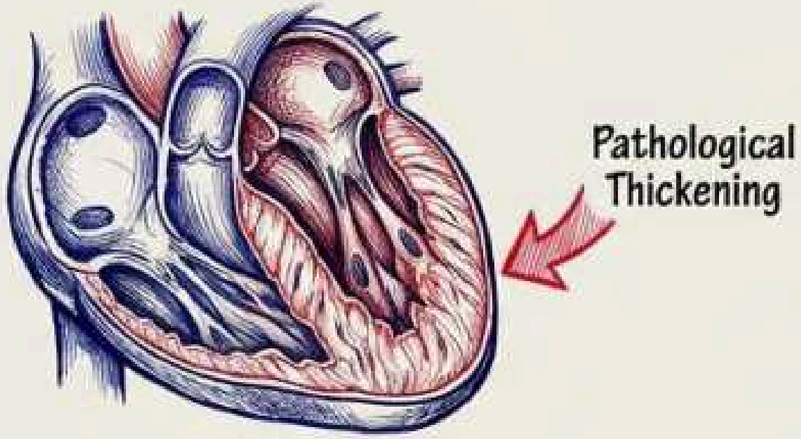
¶ 1.1



The starting point of all pathology.

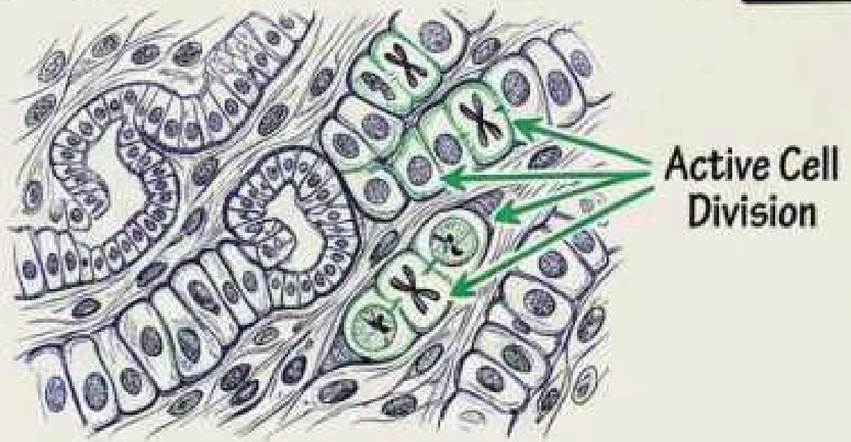
¶ 1.2

Hypertrophy (Increased Size)



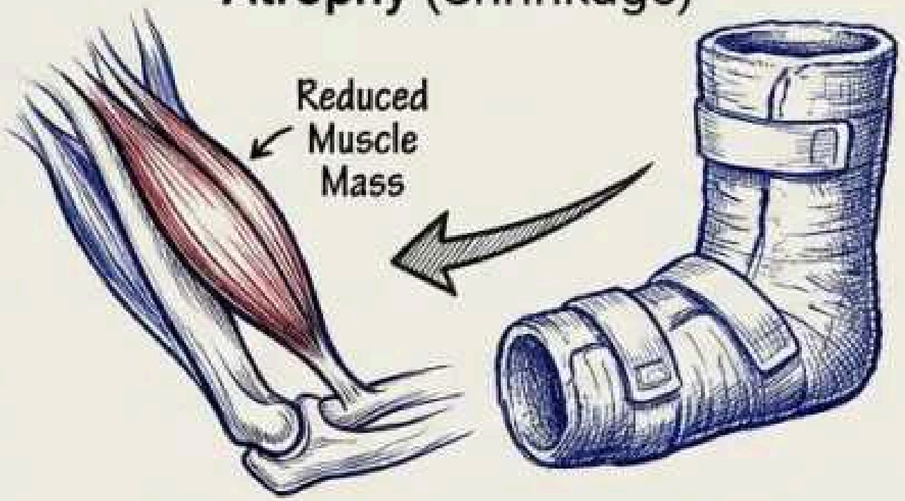
Cells grow larger (e.g., heart muscle responding to high blood pressure [in red]; skeletal muscle with exercise [eed]).

Hyperplasia (Increased Number)



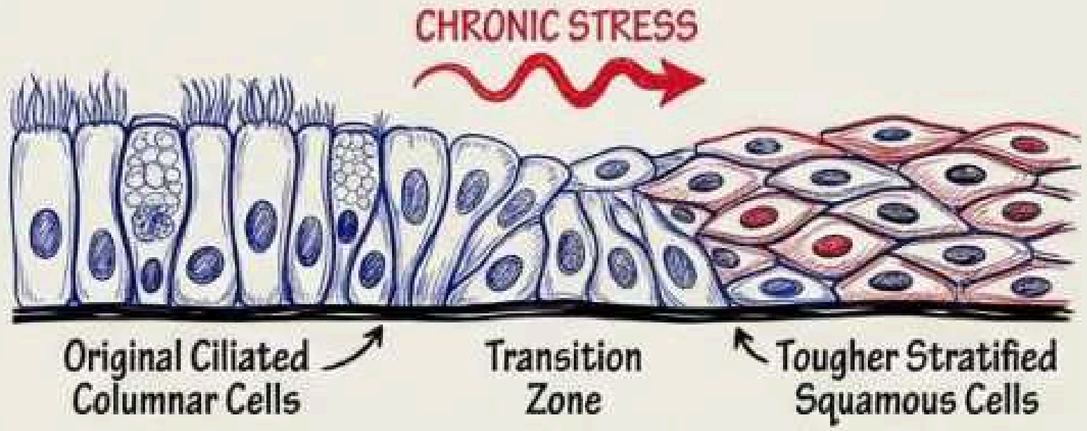
Cells increase in number (e.g., womb lining thickening during menstrual cycle]).

Atrophy (Shrinkage)



Cells shrink (e.g., limb muscle loss in a plaster cast).

Metaplasia (Replacement)

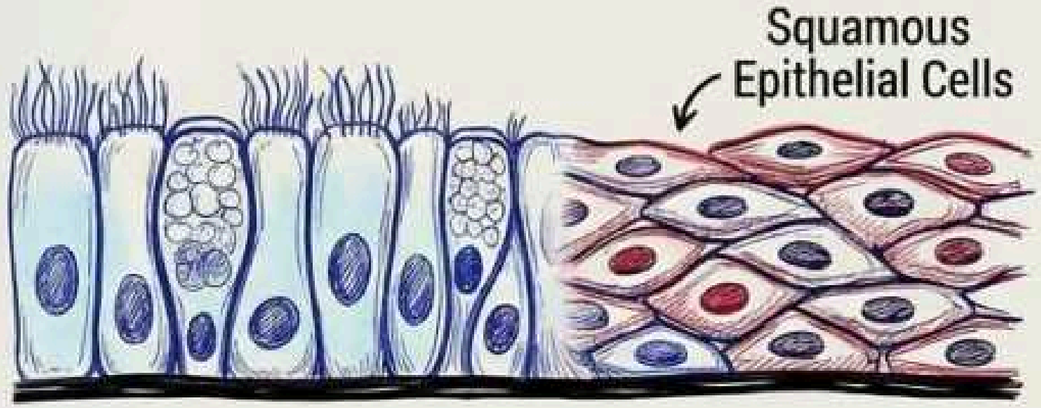


One mature cell type is replaced by another better suited to withstand stress.

The Hidden Cost of Metaplasia

¶ 1.3

Smoker's Airway

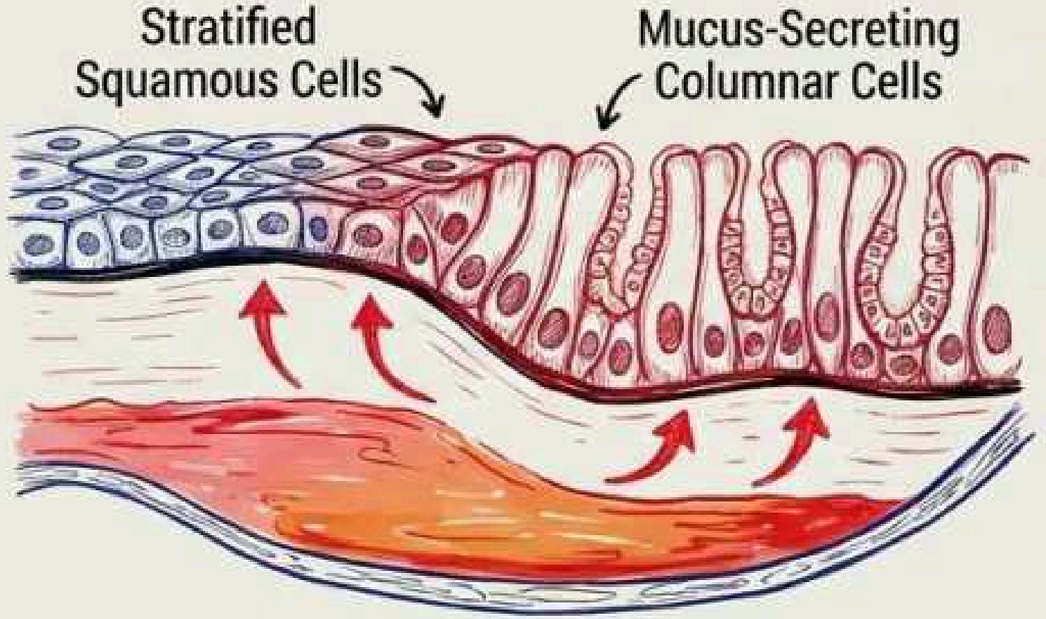


(+) Resists smoke better.

(–) Cannot sweep away mucus.

(–) Step on the road to cancer.

Lower Oesophagus

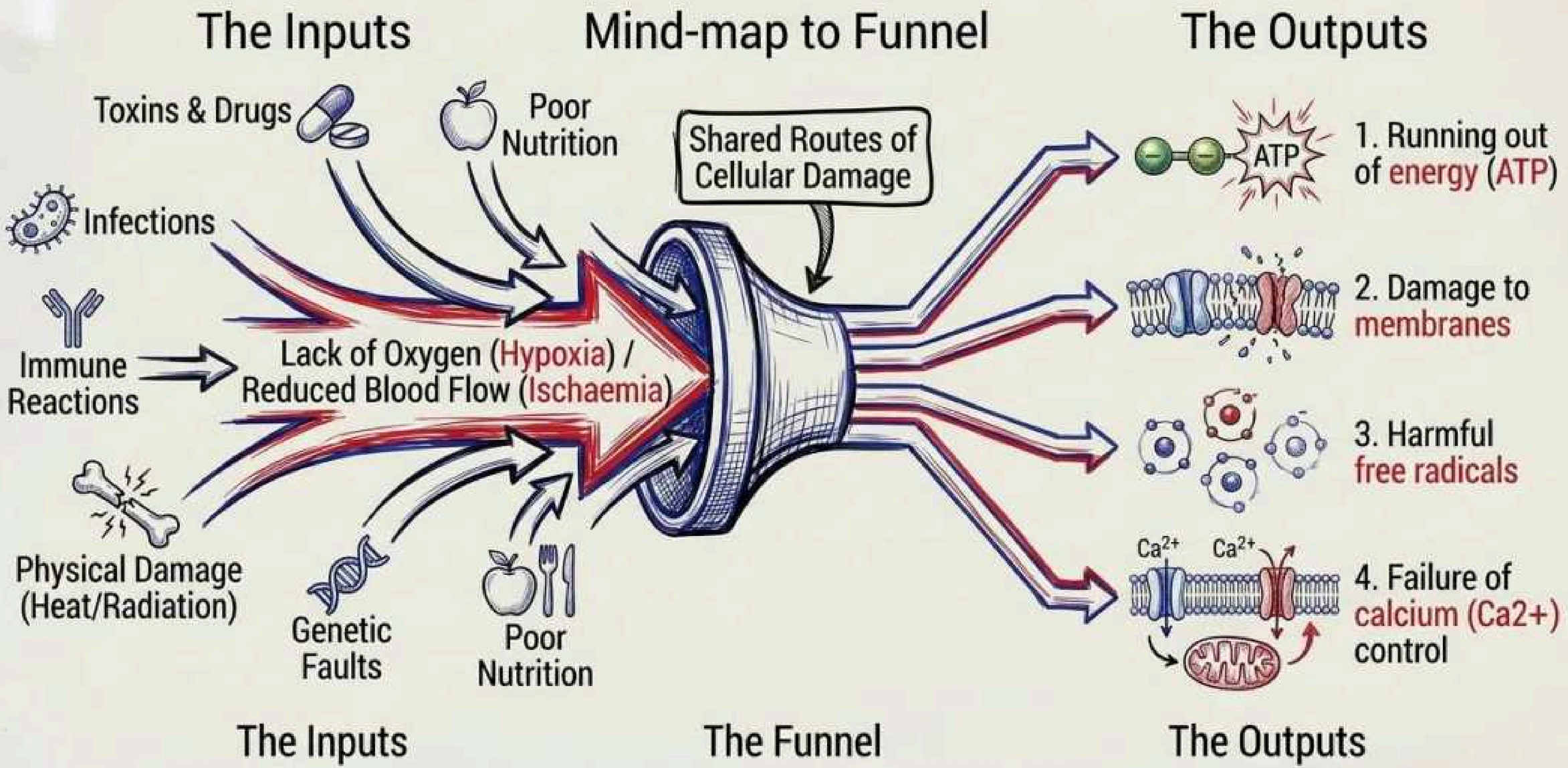


Long-term acid reflux causes similar protective cell replacement, raising cancer risk.

Adaptation provides *immediate* survival but carries long-term hidden costs.

When Adaptation Fails: Causes and Routes of Injury

¶ 1.4



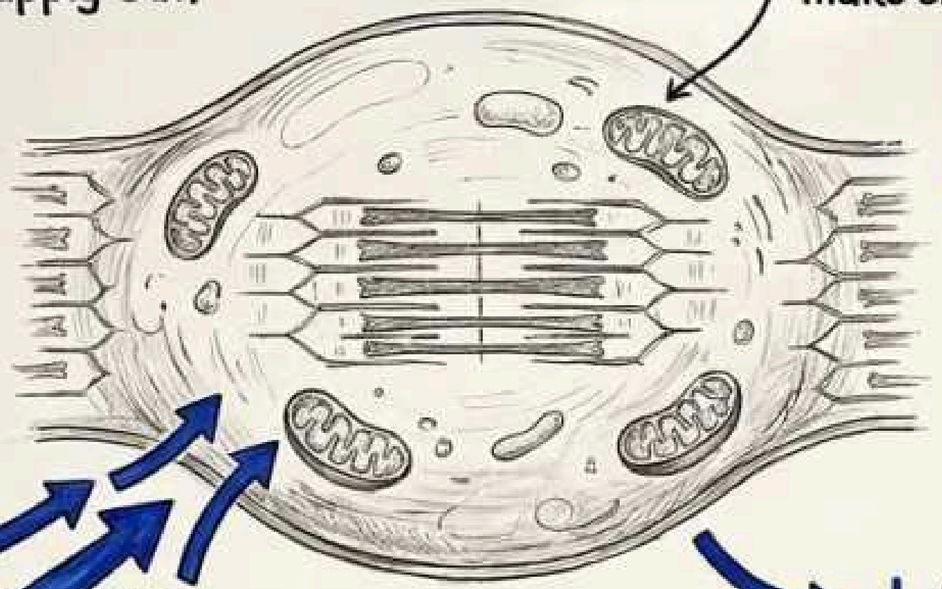
Reversible Injury: The Energy Crisis

¶ 1.5

Ischaemia
(Blood Supply Cut)

→ ↓ O_2

Recall MED 502 B:
Mitochondria cannot make energy.



Within Seconds → Cell stops contracting

H_2O floods into the cell → Cellular Swelling

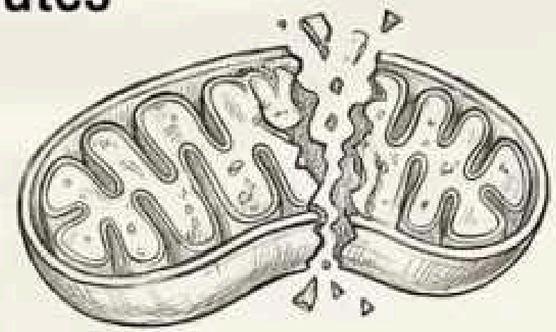
↓ ATP Production → Ion pumps fail

Damage is **REVERSIBLE**. Restoring blood flow fast allows recovery and saves heart muscle.

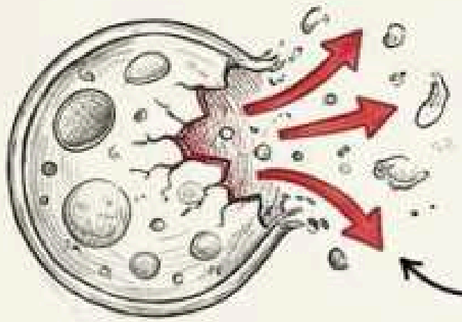
Irreversible Injury: The Point of No Return

¶ 1.6

20 to 40 minutes



Severe damage to mitochondria.



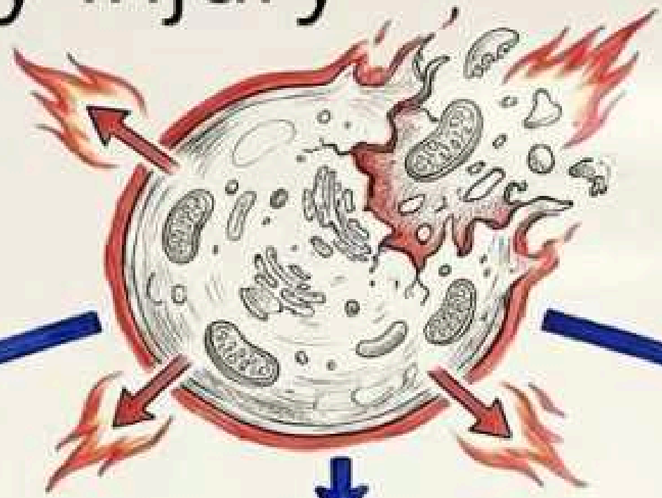
Membrane breakdown.

Digestive enzymes start to digest the cell from within.

After this point, the cell will die even if blood flow returns.

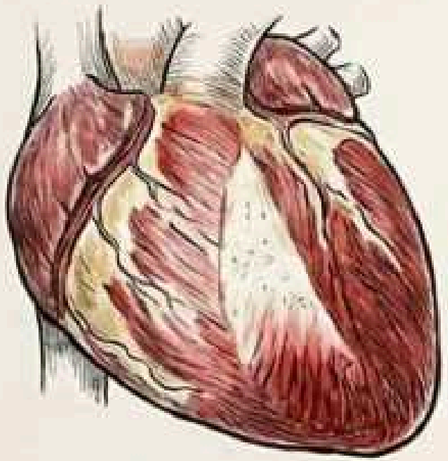
Necrosis: Death by Injury

¶ 1.7



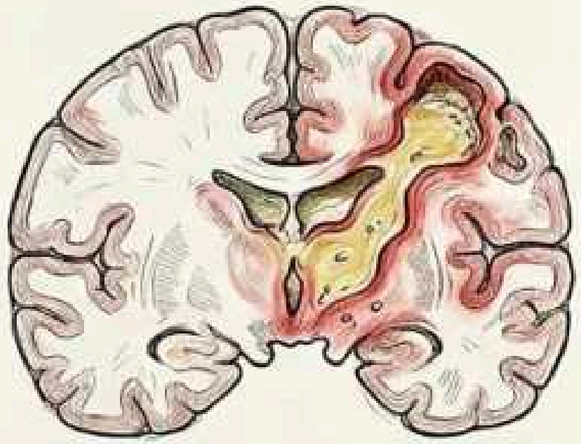
Cell swells and bursts
↳ Spills contents
↳ Provokes INFLAMMATION.
Always a sign something has gone wrong.

Coagulative Necrosis



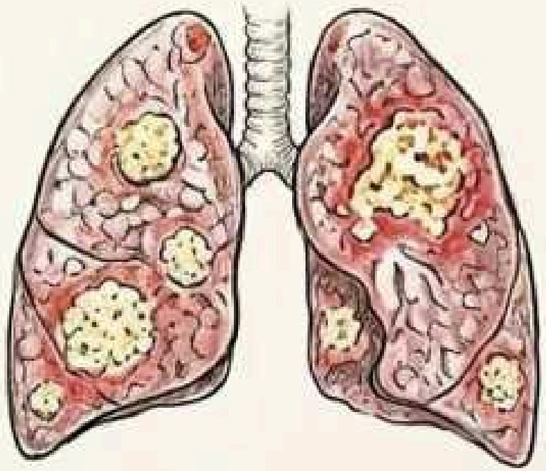
Dead tissue keeps its outline for a while.

Liquefactive Necrosis



Tissue turns to liquid.

Caseous Necrosis



Looks crumbly, like soft cheese.

Apoptosis: Programmed Cell Death

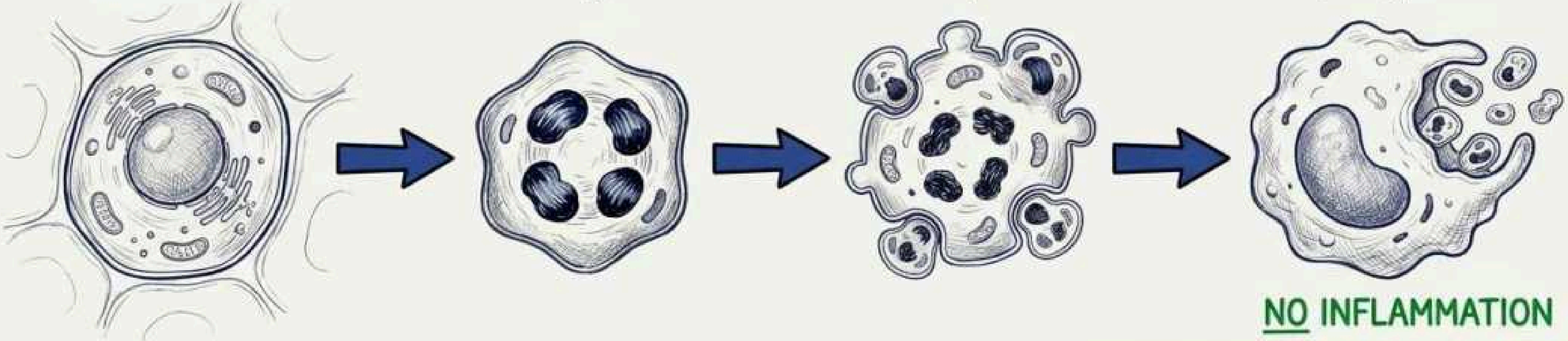
¶ 1.8

Enzymes switched on to dismantle from within.

Nucleus breaks into fragments.

Packaged into small pieces.

Swallowed quietly by neighbors.



NO INFLAMMATION

Normal Life Roles

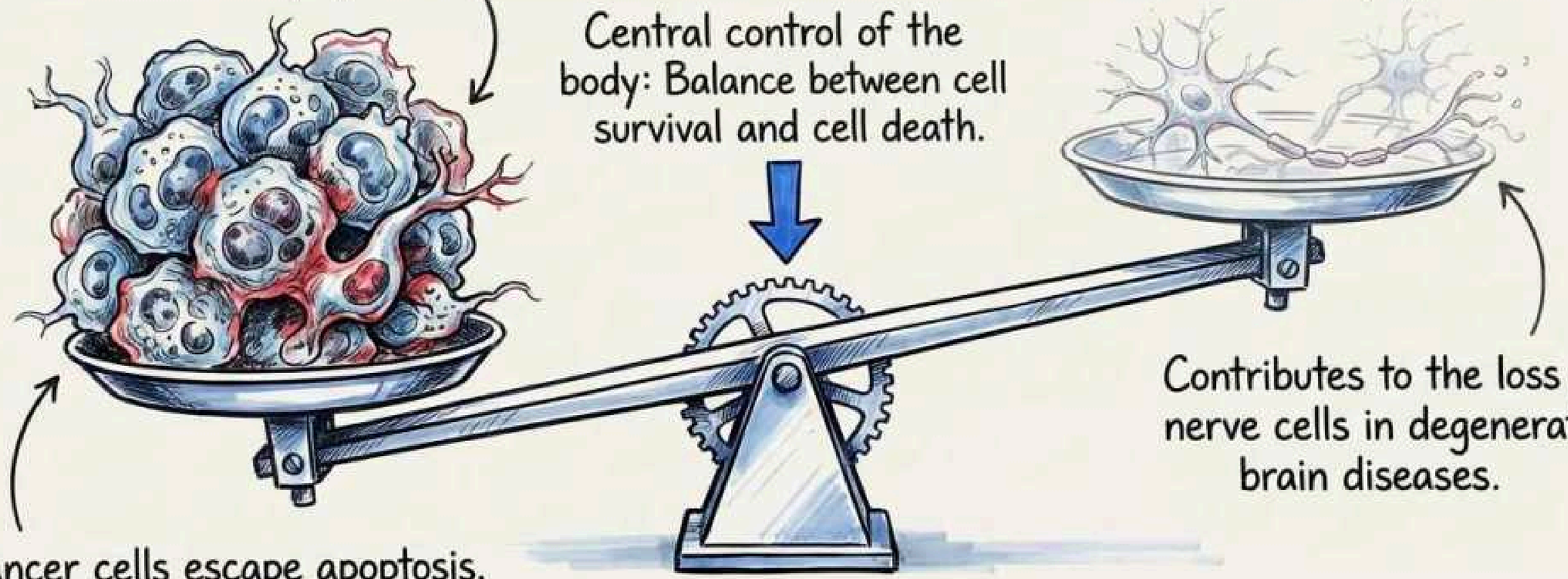
1. Shapes embryo fingers (removing tissue).
2. Removes worn-out cells.
3. Eliminates cells with damaged DNA.
4. Triggered by Killer T cells to destroy infected cells (Recall MED 504 B).

The Apoptosis Balance

Too Little Apoptosis

Too Much Apoptosis

Central control of the body: Balance between cell survival and cell death.



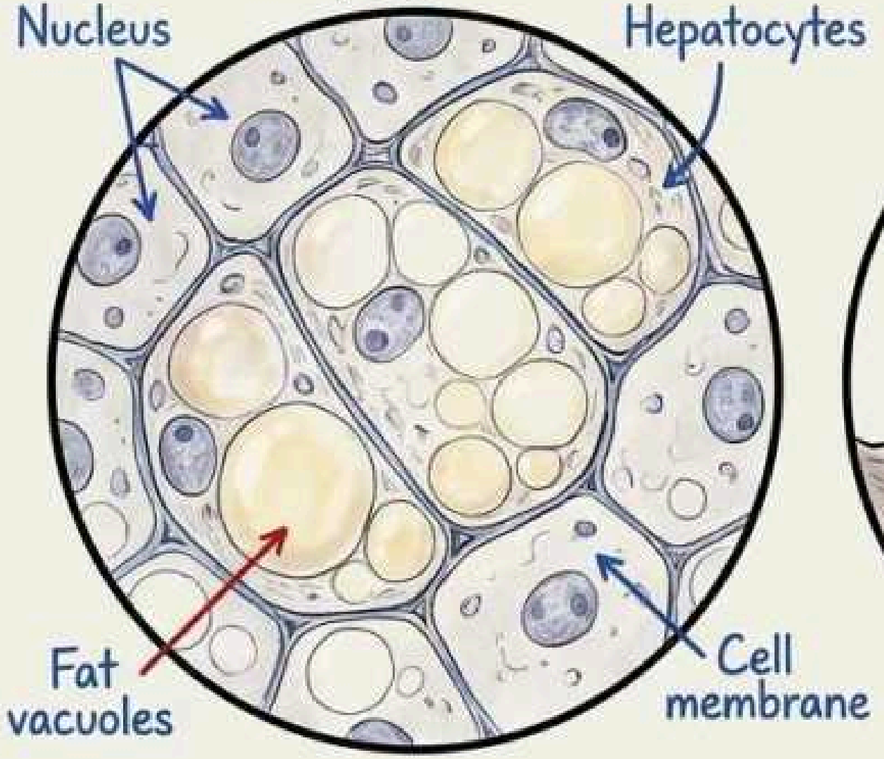
Cancer cells escape apoptosis, surviving when they should die (Theme of Lecture 4).

Contributes to the loss of nerve cells in degenerative brain diseases.

Pathological Accumulations: Clues to Tissue History

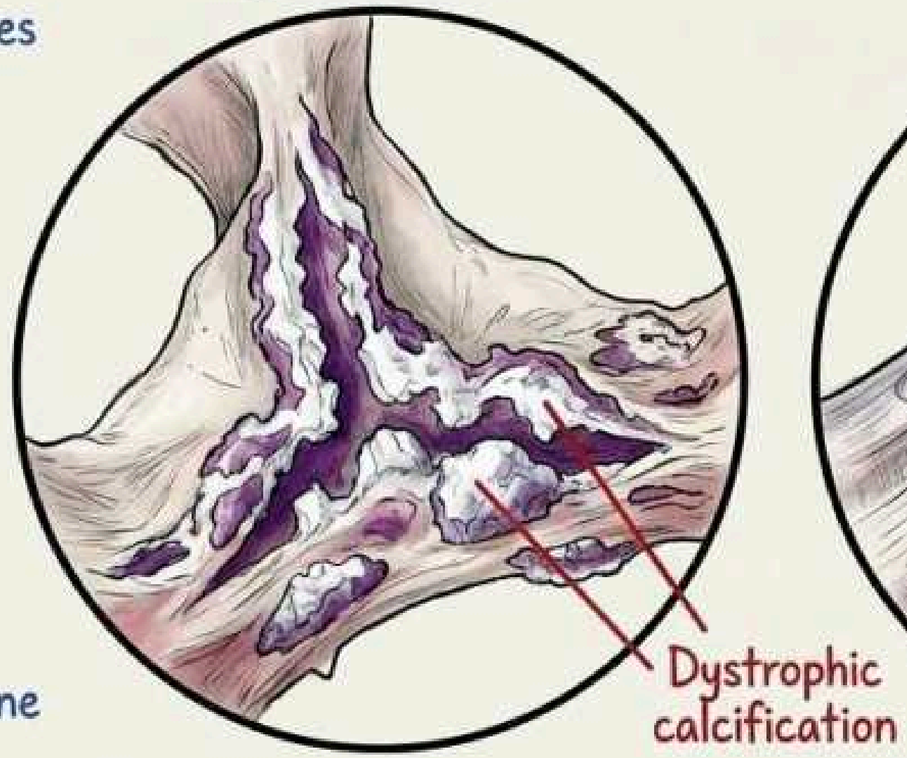
¶ 1.10

Fat



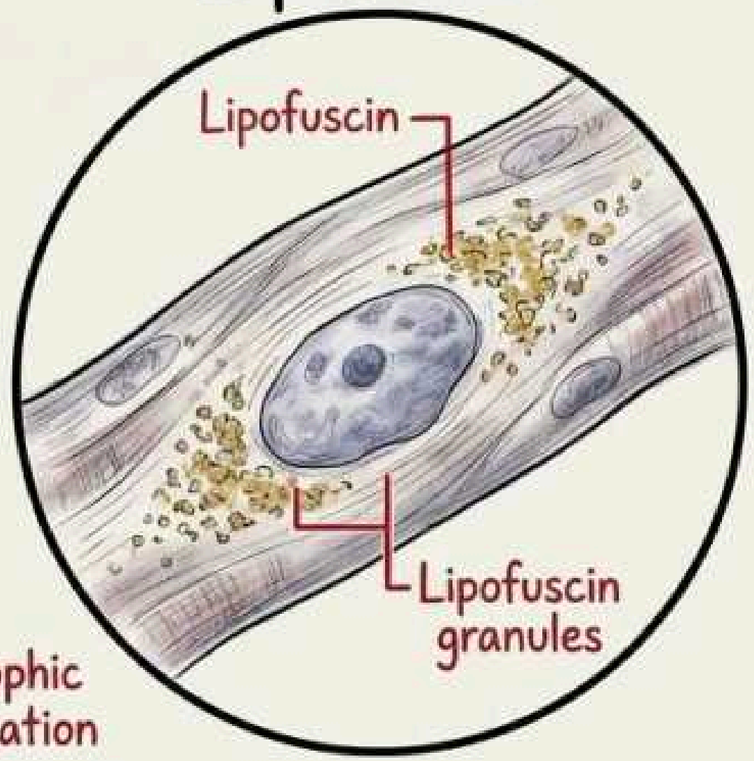
Fat builds up in liver cells (Fatty liver disease).
Linked to alcohol, obesity, or diabetes.

Calcium



Dystrophic Calcification.
Calcium deposited in dead tissue (e.g., damaged heart valves, old tuberculosis areas).

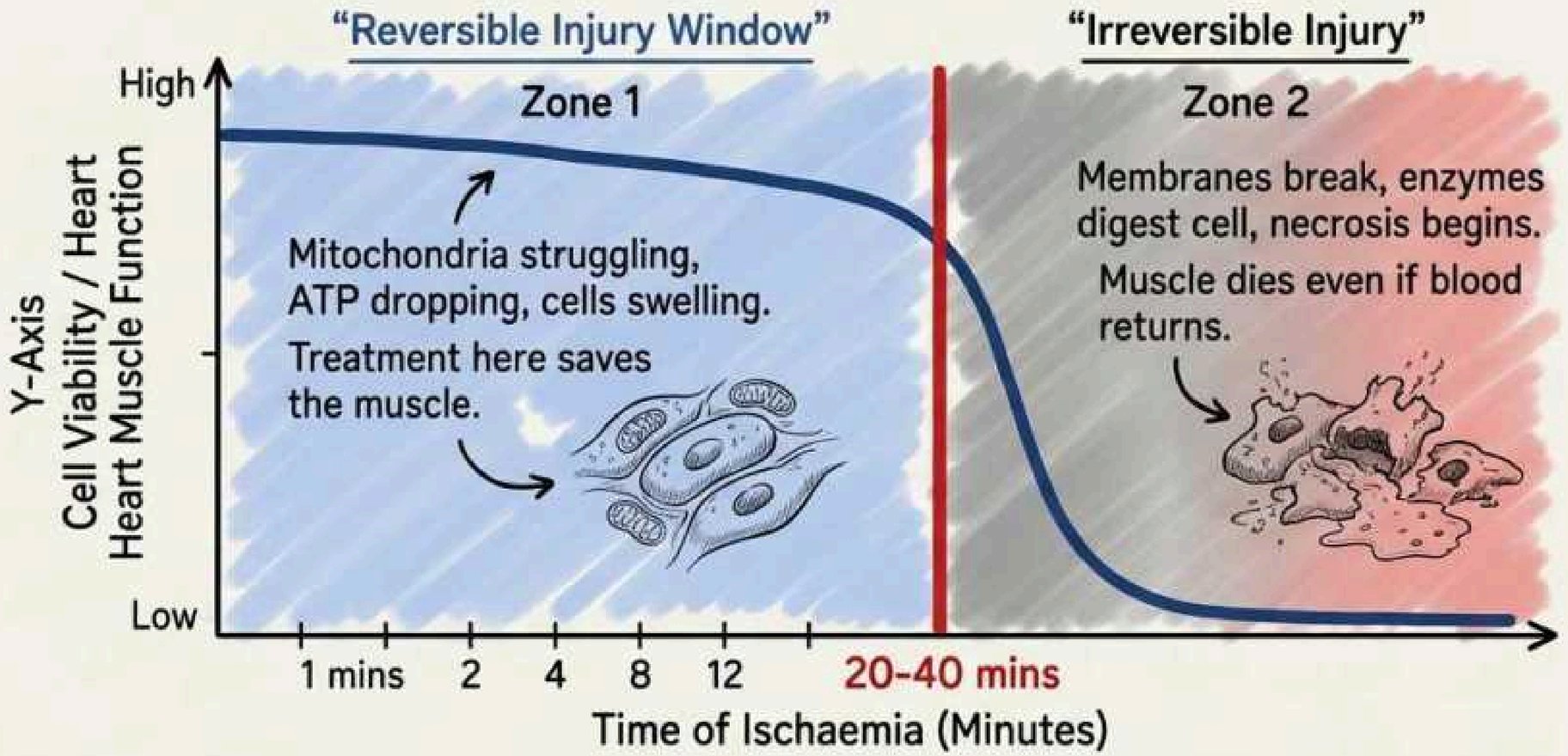
Lipofuscin



Brown pigment in long-lived cells.
A sign of aging and cellular wear and tear.

Clinical Synthesis: The Heart Attack Timeline

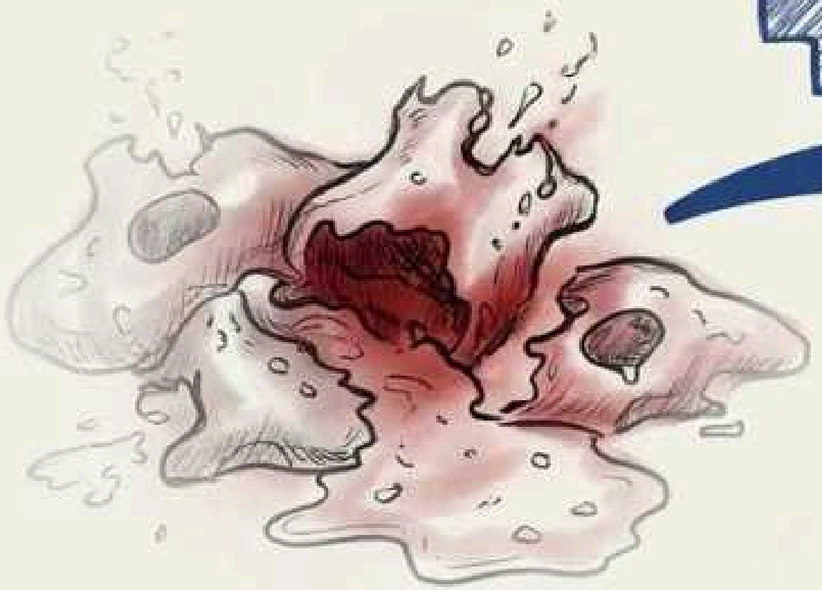
Why Speed of Treatment Matters



The difference between life and death of the tissue is defined by beating the 20-40 minute window.

Next Steps: The Pathologist's Point of View

¶ 1.12



Triggers...

Inflammation

Tissue Repair

Coming up in Lecture Two.