

SNS COLLEGE OF ALLIED HEALTH SCIENCE
Affiliated to The Tamil Nadu Dr MGR Medical University, Chennai



DEPARTMENT OF CARDIAC TECHNOLOGY

**COURSE NAME: PATHOLOGY RELATED TO CARDIAC
TECHNOLOGY**

UNIT : 1

TOPIC : CELL INJURY - IRREVERSIBLE

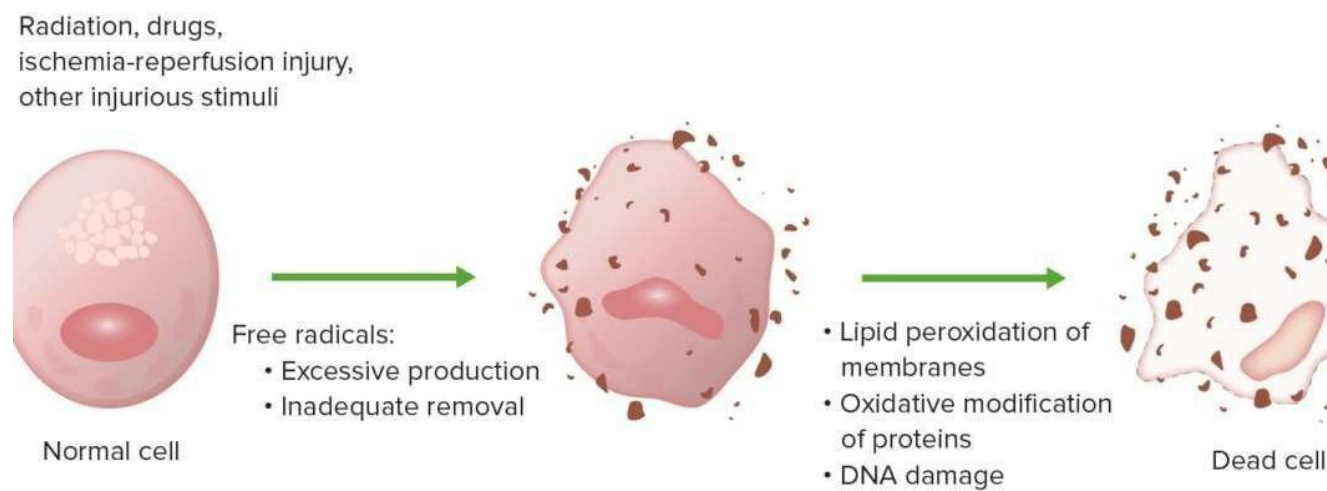
FACULTY NAME: Ms. HARSHITHA S

Definition

Cell injury occurs when cells are exposed to stress or harmful stimuli that exceed their ability to adapt. It can be:

Reversible: Cell recovers once the stimulus is removed.

Irreversible: Cell undergoes death—either by **necrosis** or **apoptosis**.

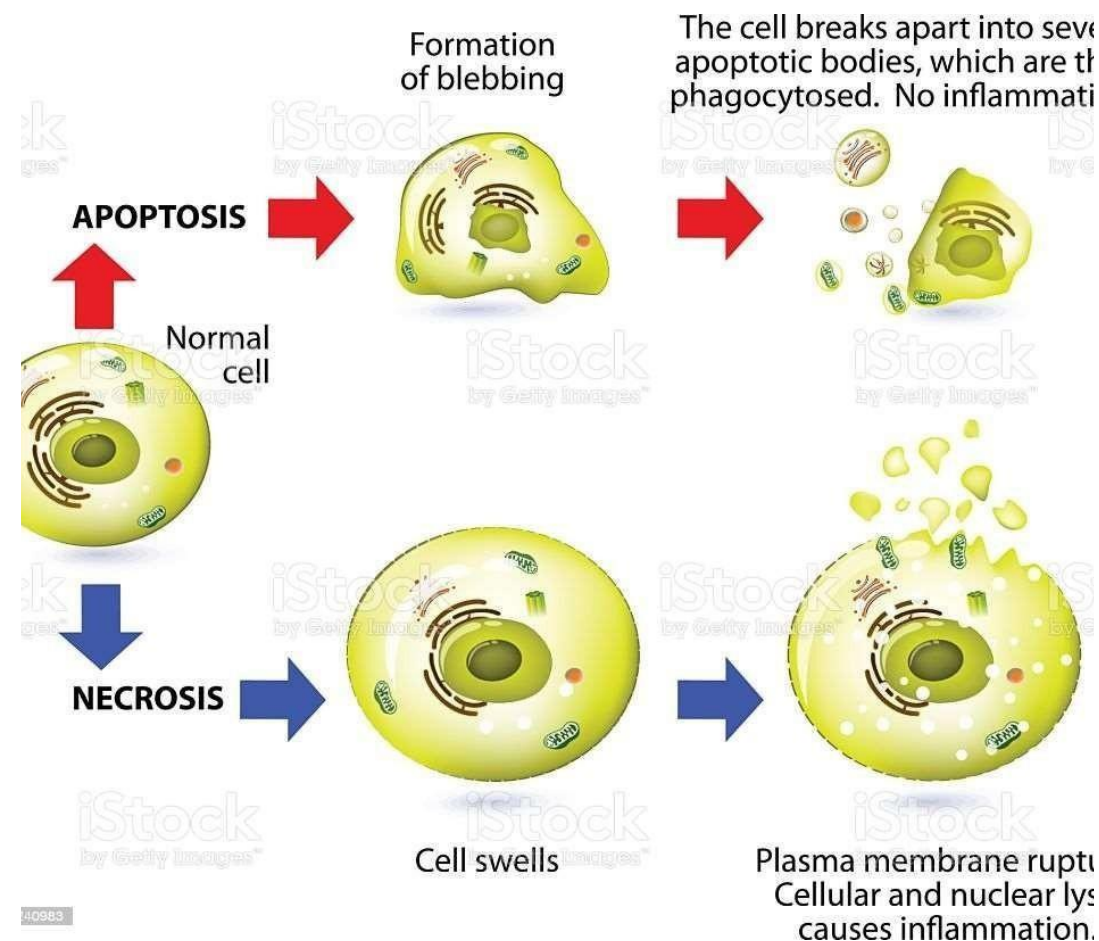


Irreversible Cell Injury

Definition:

Irreversible cell injury refers to **permanent cellular damage** where the cell **cannot recover**, even if the injurious stimulus is removed. It ultimately results in **cell death**, by either:

- **Necrosis** – uncontrolled cell death due to external injury
- **Apoptosis** – programmed, controlled cell death



Necrosis

Definition:

Necrosis is a **pathologic**, unregulated form of cell death resulting from **acute injury**, causing **cell swelling**, **membrane rupture**, and **inflammation**.

Mechanism (Pathogenesis):

Cell injury → ATP depletion → ion pump failure → **cell swelling**

Increased intracellular calcium → **activation of enzymes** (proteases, lipases, DNases)

Membrane damage → leakage of cellular contents

Inflammatory response is triggered

Morphological Changes:

Cytoplasmic:

Increased eosinophilia (pinkness) due to protein denaturation

Loss of organelles

Nuclear:

Pyknosis: Nuclear shrinkage

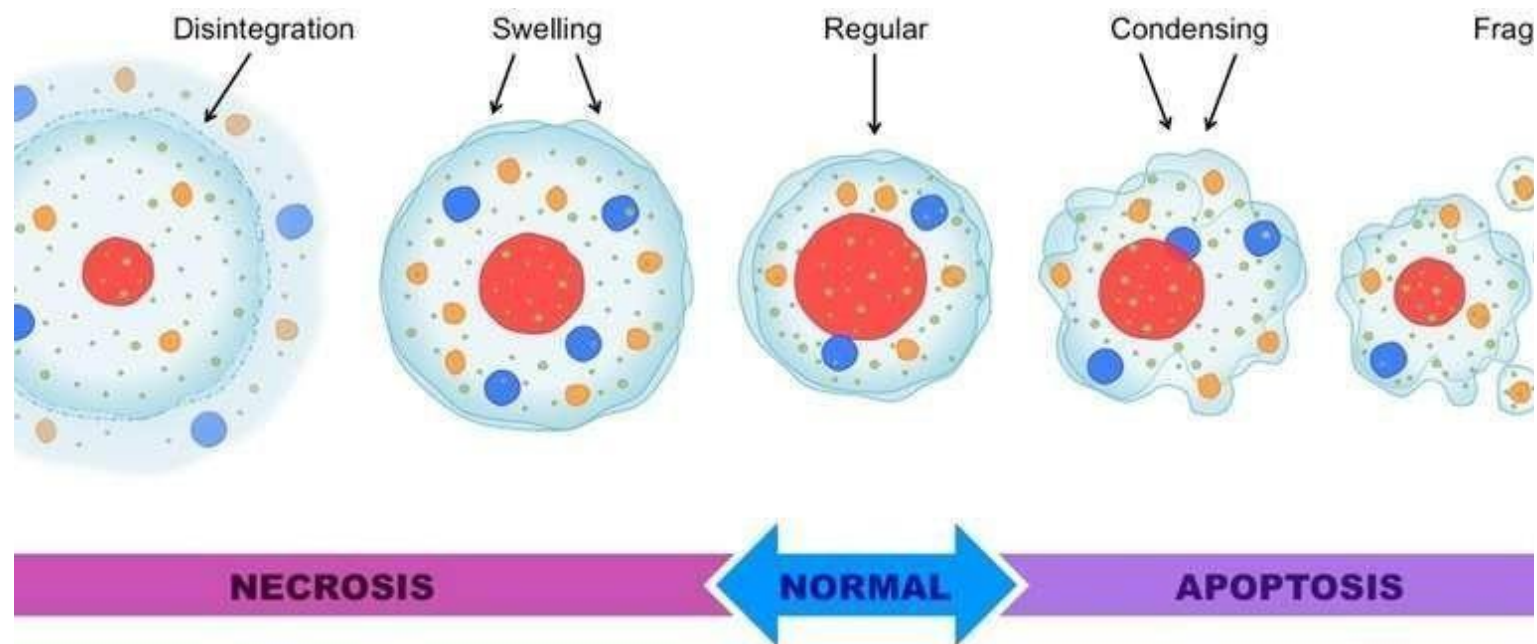
Karyorrhexis: Fragmentation of nucleus

Karyolysis: Dissolution of nucleus

Types of Necrosis

Type	Features	Example
Coagulative	Preserved tissue architecture, firm texture	Myocardial infarction
Liquefactive	Complete digestion of cells into liquid	Brain infarction, abscess
Caseous	Cheese-like, combination of coagulative and liquefactive	Tuberculosis
Fat necrosis	Chalky white fat saponification	Acute pancreatitis
Fibrinoid	Immune complex deposition in vessels	Vasculitis
Gangrenous	Death of tissue in extremities	Diabetic foot, ischemia

Apoptosis



Apoptosis

Definition:

Apoptosis is a **programmed, energy-dependent** process of **cell death** that occurs without inflammation. It plays a role in both **physiologic and pathologic conditions**.

Mechanism (Pathways):

1. Intrinsic (Mitochondrial) Pathway:

Triggered by DNA damage, oxidative stress

Regulated by Bcl-2 family proteins

Mitochondrial membrane permeabilization → Cytochrome c release → Caspase activation

2. Extrinsic (Death Receptor) Pathway:

Activation of **Fas** or **TNF receptors**

Caspase-8 activation → caspase cascade

Morphological Features:

- Cell shrinkage, intact plasma membrane
- Chromatin condensation at nuclear periphery
- Formation of apoptotic bodies
- Phagocytosis by macrophages without inflammation

Physiologic Apoptosis Examples:

- Embryogenesis
- Hormone-dependent tissue regression (e.g., endometrial shedding)
- Elimination of self-reactive lymphocytes

Pathologic Apoptosis Examples:

- DNA damage (e.g., radiation)
- Viral infections (e.g., HIV, Hepatitis)
- Atrophy after duct obstruction

Feature	Necrosis	Apoptosis
Energy-dependent?	No	Yes
Membrane integrity	Lost	Maintained
Inflammation	Present	Absent
Cell size	Swelling	Shrinkage
DNA fragmentation	Random	Ordered
Outcome	Tissue damage	Cell removal without damage

REFERENCE

Harshmohan book of pathology

<https://share.google/1czL1JEVSM9fwyEtE>