Cellular Response To Injury

Lecture: 3

Dr. Payman Anwar Rashid

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Faculty of Applied Science
Physiotherapy Department

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Systemic Pathology
Second Grade

Lecture Outline:

* Overview of cellular response to stress.
* Cellular adaptation to stress.
* Hypertrophy.
* Hyperplasia.
* Atrophy.
* Metaplasia.
* Reversible & Irreversible injury.
* Causes of cell injury.

Objectives:

1. To understand concept of cellular response to stress.
2. To explain types of cellular adaption.
3. To Learn causes of cell injury

Cellular Response To Injury

- Normal cell function requires a balance between physiologic demands, the structure of cell and metabolic capacity; the result is a steady state, or homeostasis.

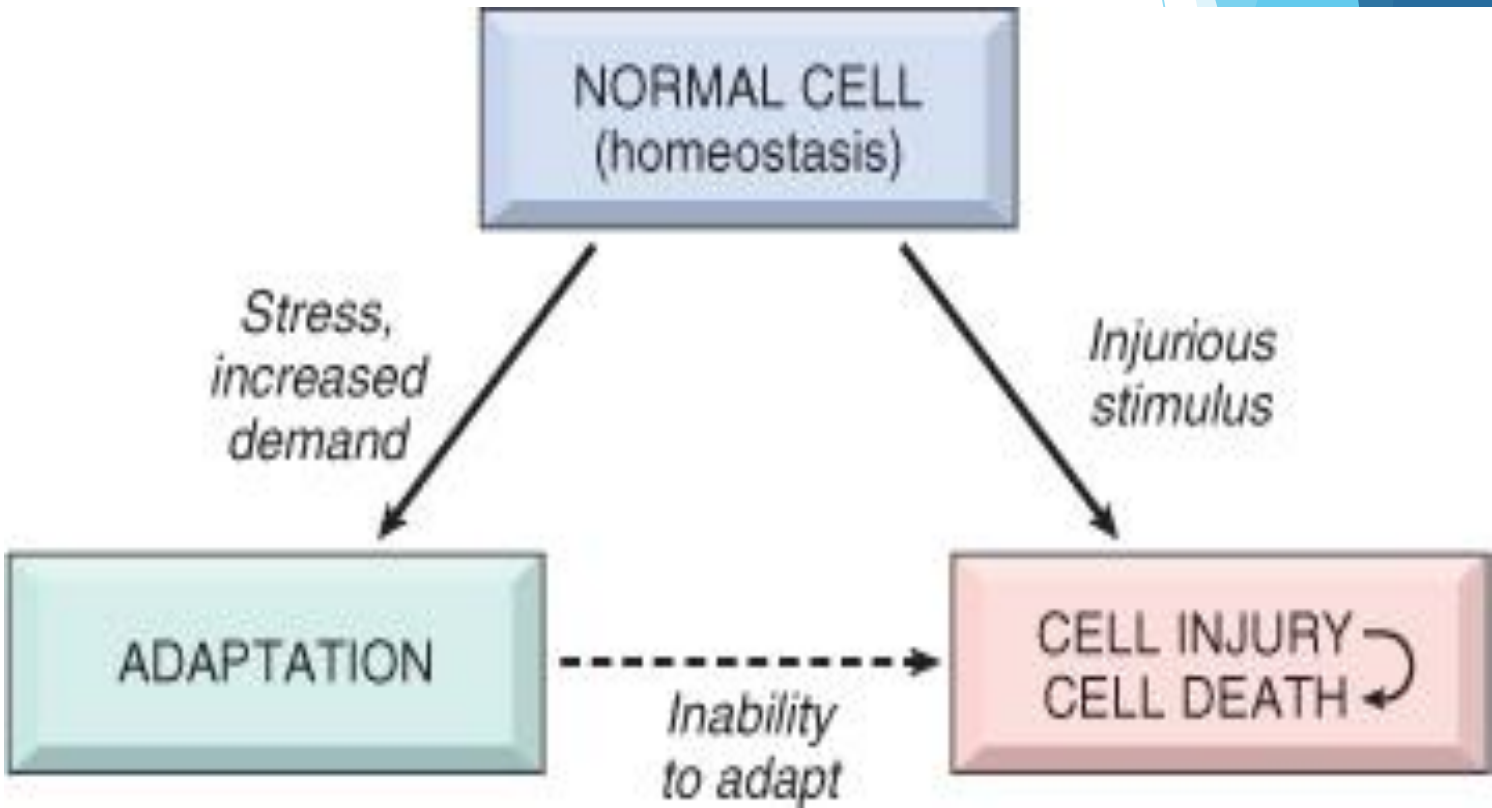
Cells can alter their functional state in response to modest stress to maintain the steady state.

More excessive physiologic stresses or adverse pathologic stimuli (injury), result in:

1. Adaptation.

2. Reversible injury.

3. Irreversible injury and cell death.



- **Adaptation:**

occurs when physiologic or pathologic stressors induce a new state that change the cell but otherwise preserves its viability in the face of the exogenous stimuli.

These changes include:

- 1. **Hyperplasia** (increased cell number).
- 2. **Hypertrophy** (increased cell mass).
- 3. **Atrophy** (decreased cell mass).
- 4. **Metaplasia**. (change from one mature cell type to another).

- **Hyperplasia:**

is an increase in the number of cells in an organ or tissue. It is usually accompanied by hypertrophy. Hyperplasia can occur only with cells capable of synthesizing DNA (such as epithelial, hematopoietic, and connective tissue cells).

Nerve, cardiac, and skeletal muscle cells have no capacity for hyperplastic growth, so muscle cells undergo pure hypertrophy when stimulated by increased functional load or hormones.

Hyperplasia can be physiologic or pathologic

- **1. Physiologic hyperplasia:**

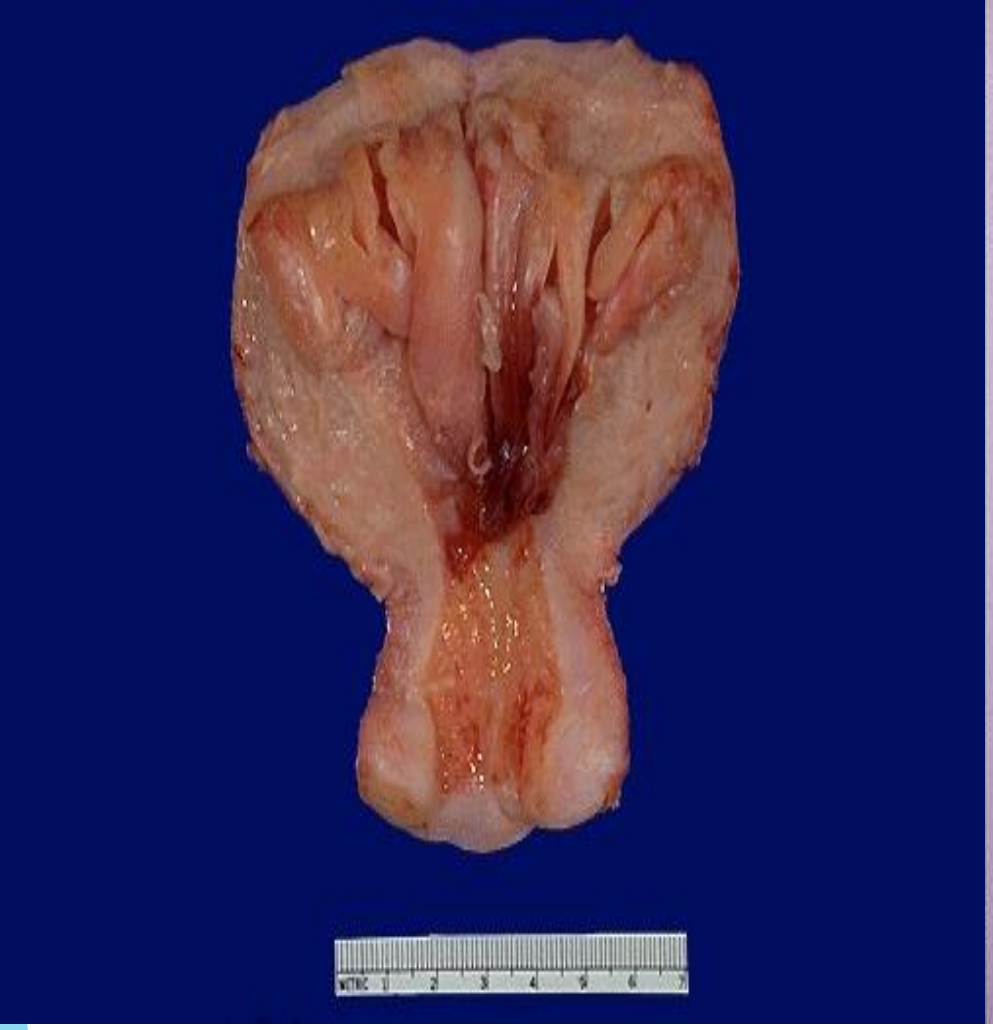
* Hormonal hyperplasia, exemplified by the proliferation of the glandular epithelium of the female breast at puberty and during pregnancy.

* Compensatory hyperplasia (e.g. hyperplasia of the liver after partial hepatectomy).

2. Pathologic hyperplasia:

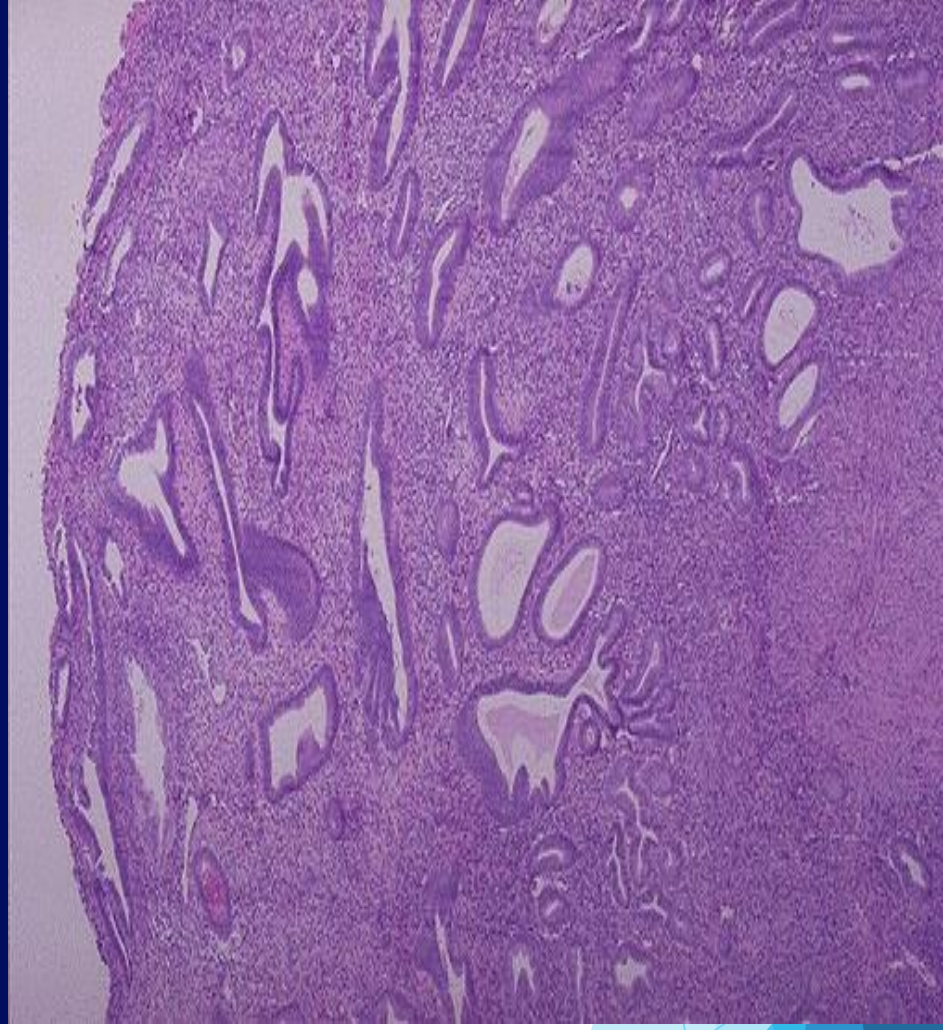
* Excessive hormonal stimulation (e.g. hyperestrinism and atypical endomaterial hyperplasia).

* Effects of locally produced growth factors on target cells (e.g. proliferation of connective tissue cells in wound healing).



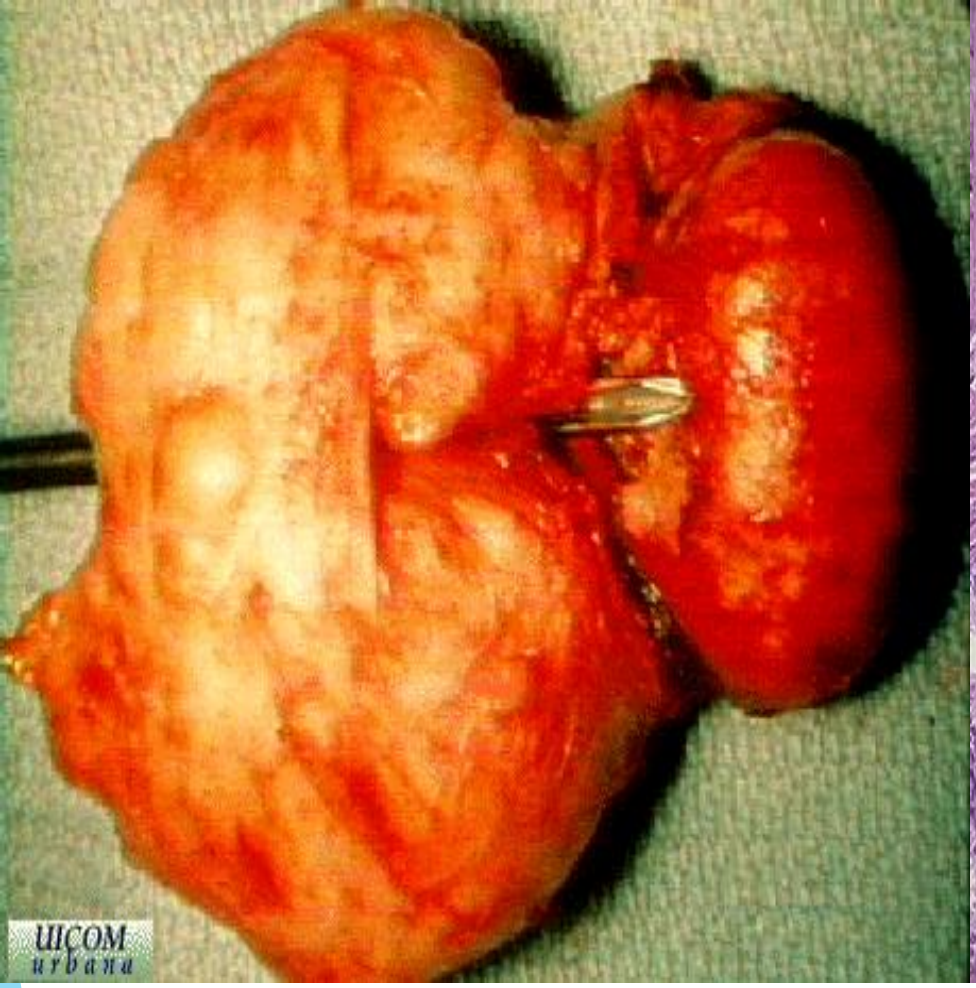
Endometrial hyperplasia

(Pathologic hormonal stimulation)

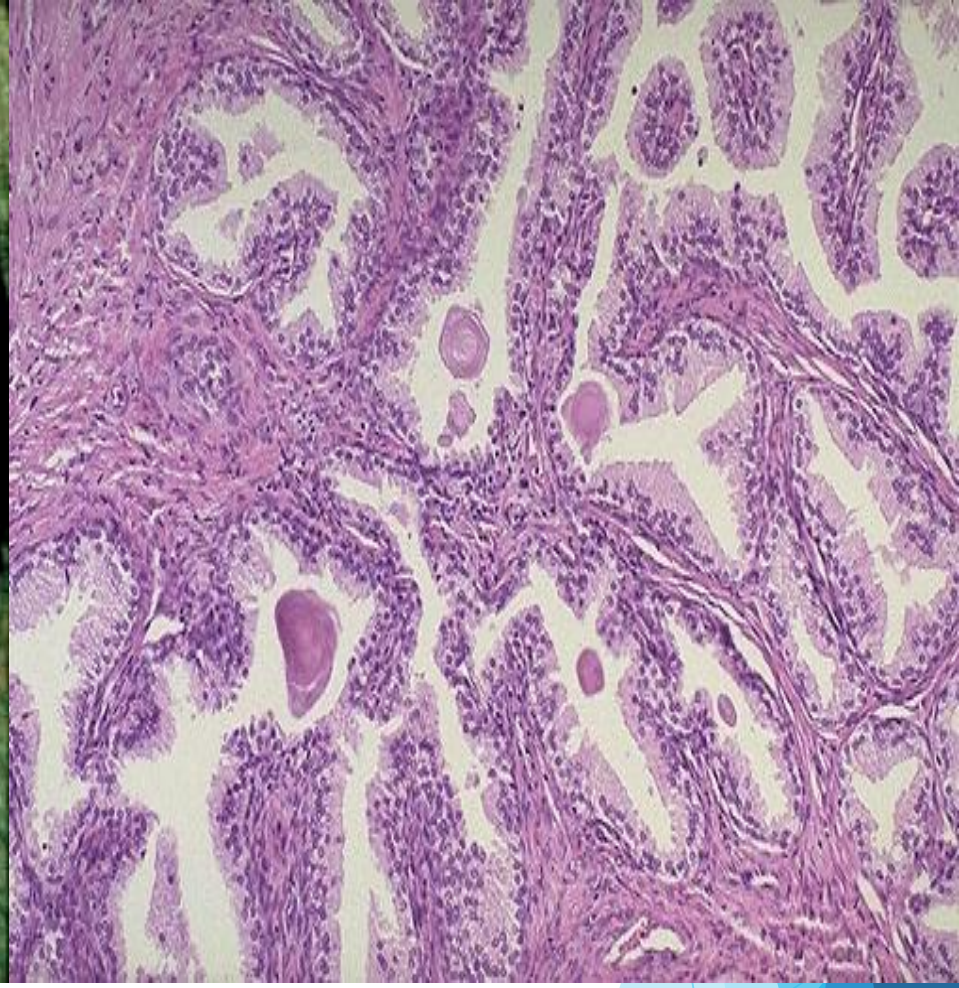


Endometrial hyperplasia

(microscopical appearance)



Benign prostatic hyperplasia
Pathologic hormonal hyperplasia



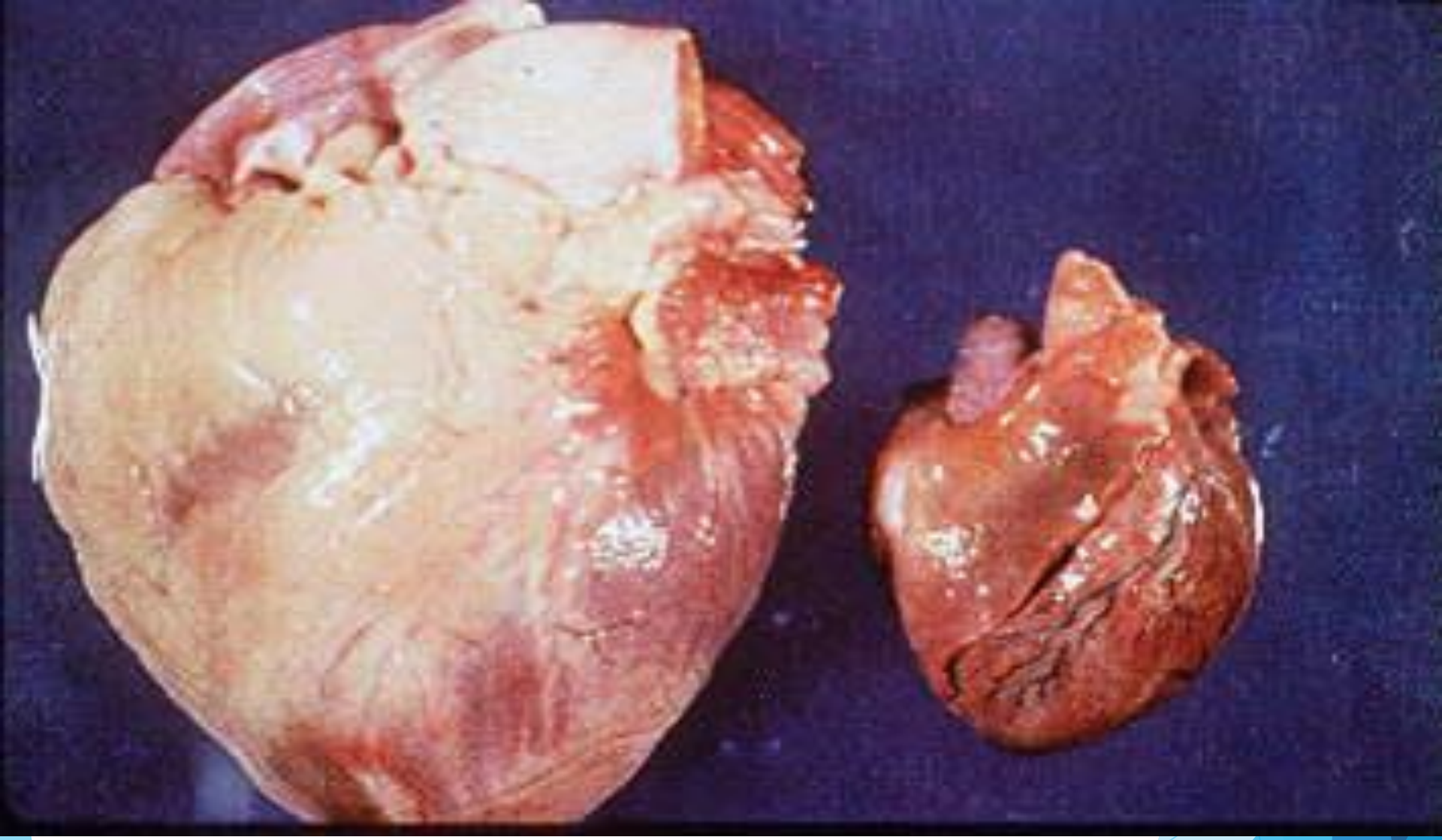
Microscopical appearance of BPH

- In pathologic hyperplasia if the stimulus removed the hyperplasia disappears.

* However pathologic hyperplasia constitute a fertile soil in which cancerous proliferation may eventually arise. E.g. atypical endometrial hyperplasia which are precursors of cancer of endometrium.

Hypertrophy:

- is an increase in the size of cells which lead to increase in size of organ.
- Hypertrophy can be physiologic or pathologic and is caused by :
- 1. Increased functional demand e.g. hypertrophy of striated muscles in muscle builders (physiologic) or of cardiac muscle in cardiac disease (pathologic).
- 2. Specific hormonal stimulation e.g. uterine hypertrophy during pregnancy.



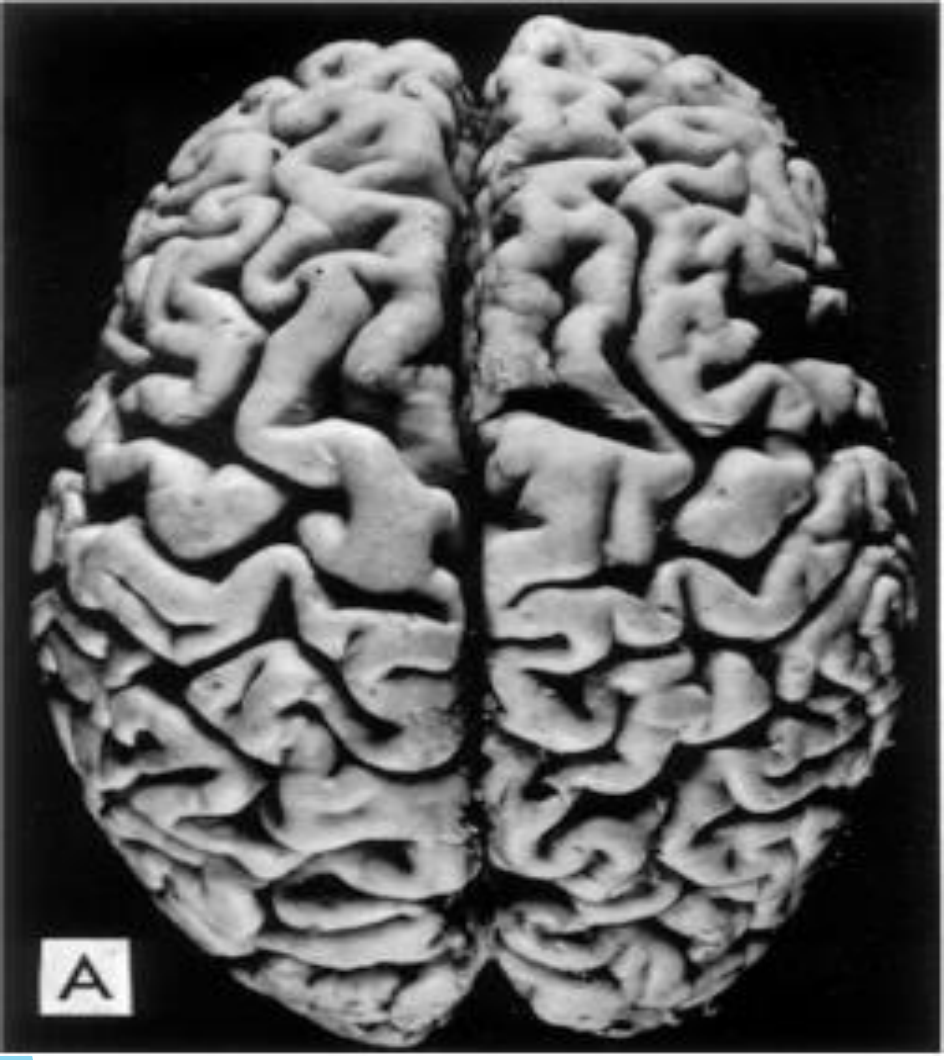
Heart hypertrophy due to work overload (as in hypertension) compared with normal heart (Rt.side) for the same age & sex.

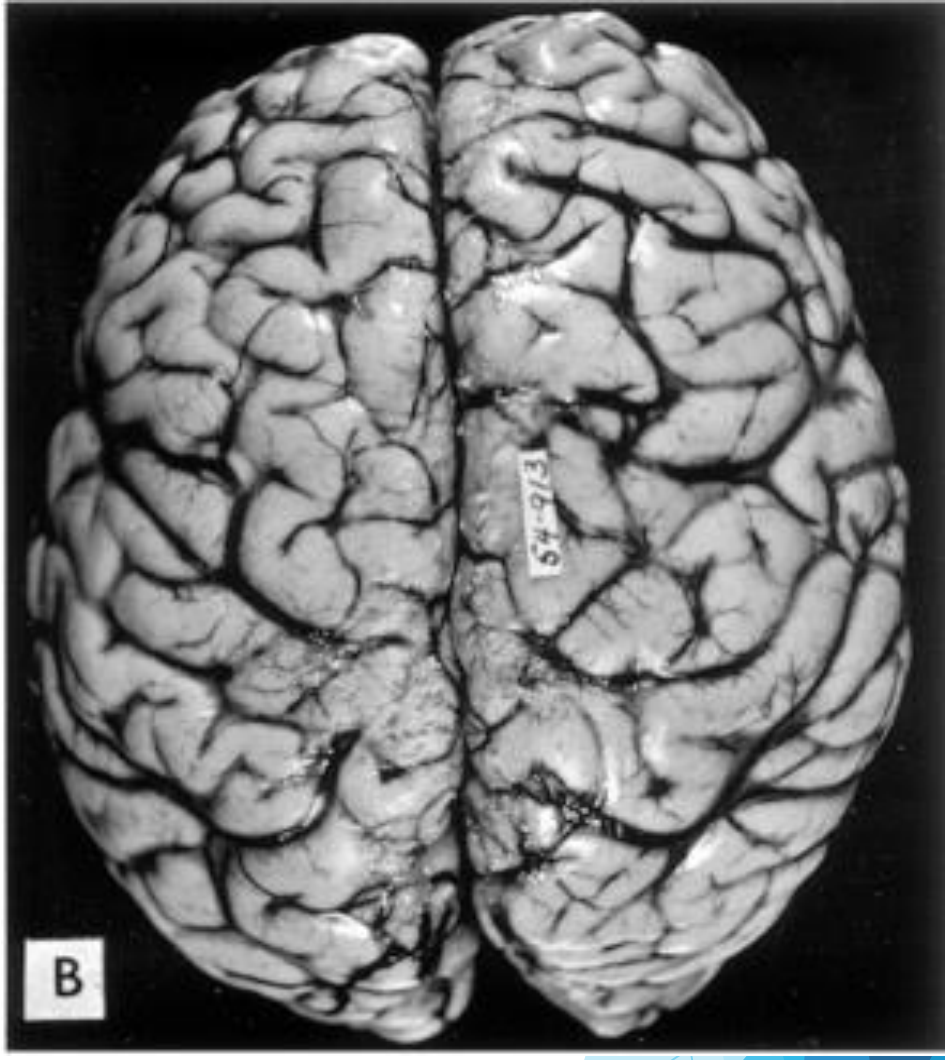
Left ventricular hypertrophy in patient with chronic hypertention

- **Atrophy:**
- is shrinkage in the size of the cell due to loss of cell substance.

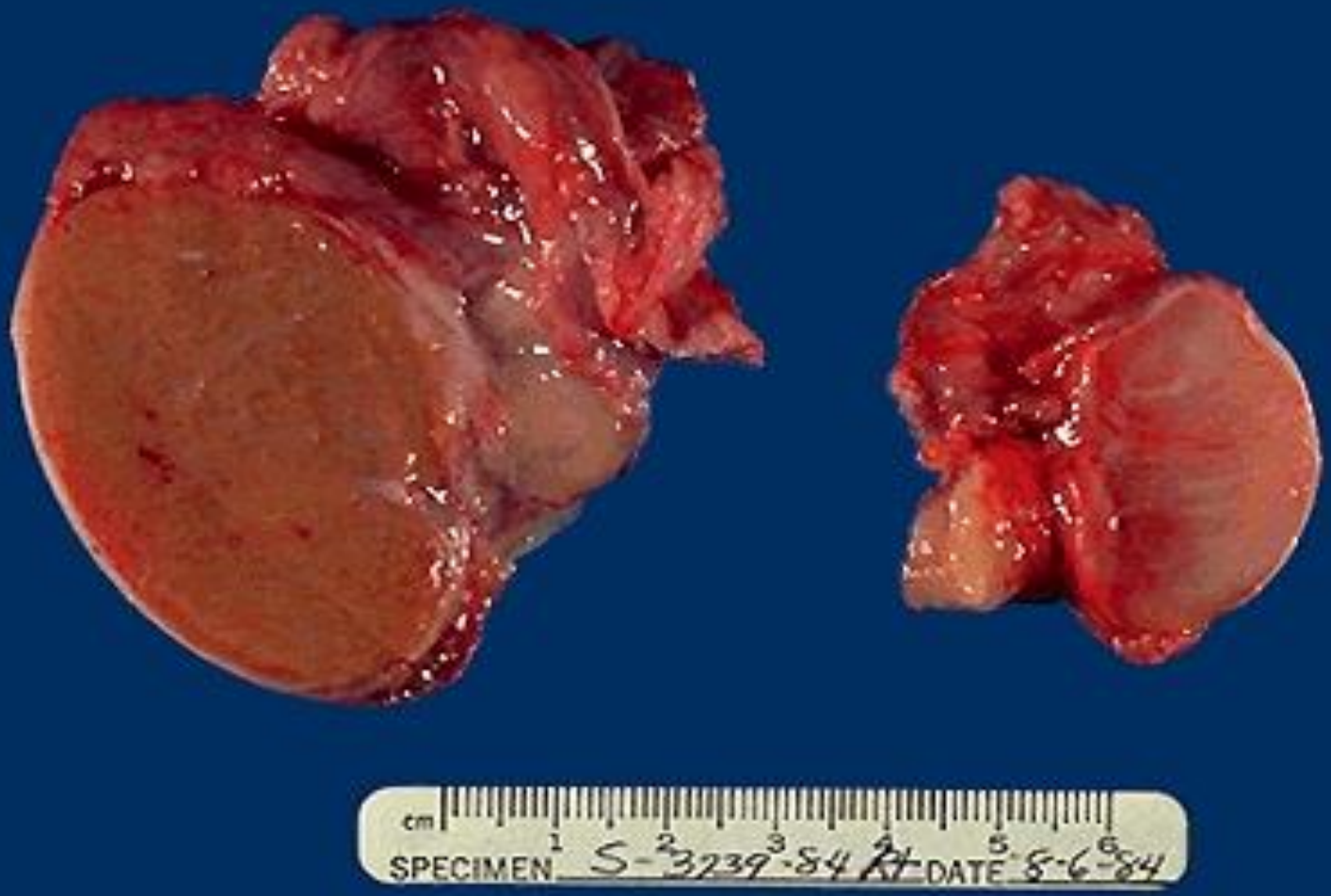
Causes of atrophy are:

- 1.decreased workload.
- 2. loss of innervation.
- 3. diminished blood supply.
- 4. inadequate nutrition.
- 5. loss of endocrine stimulation.
- 6. aging.





Brain atrophy due to aging & reduced blood supply (atherosclerosis). Note that loss of brain substance narrows the gyri and widen the sulci.



Testicular atrophy

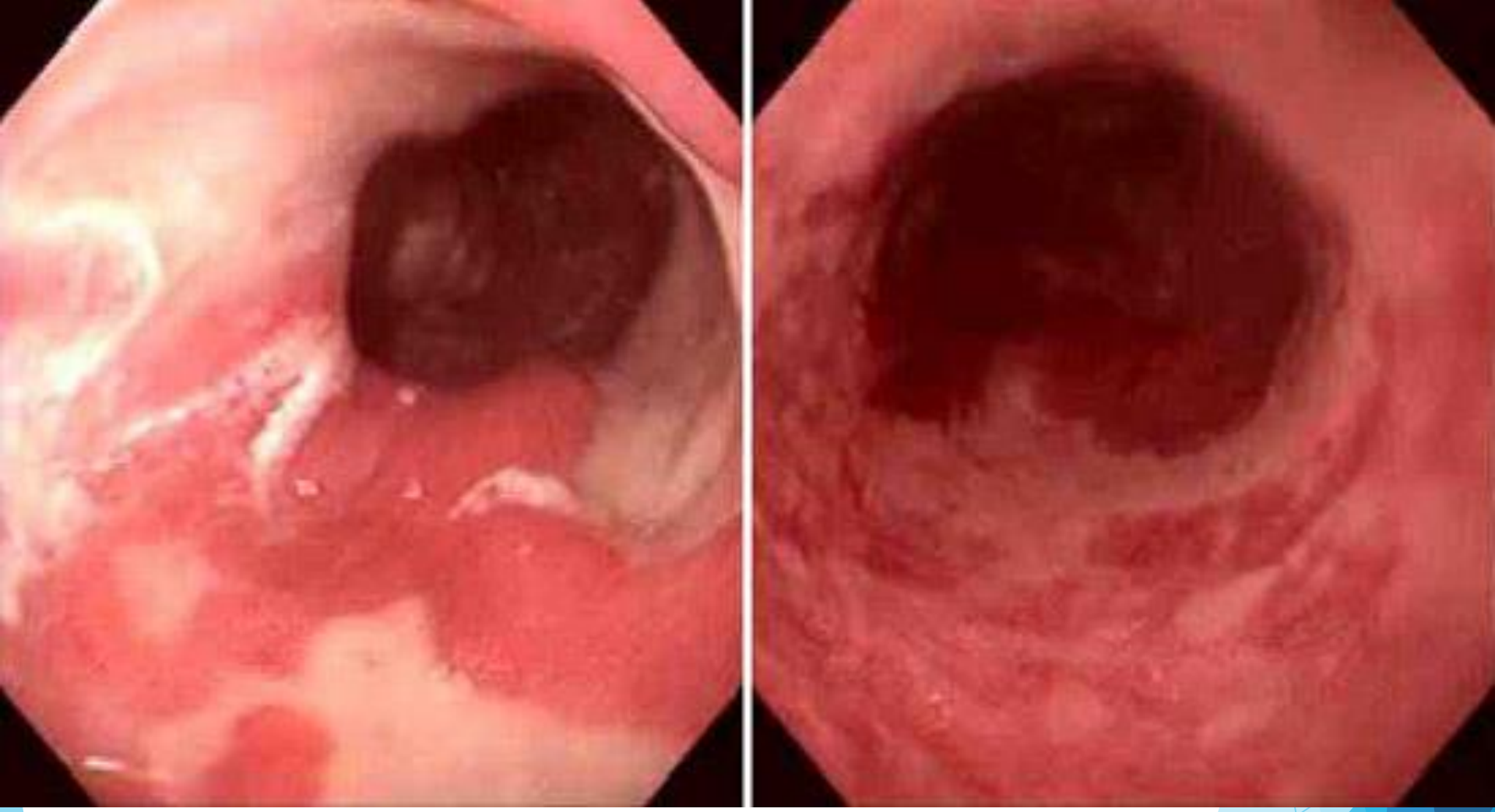
- **Metaplasia:**

is a reversible change in which one adult cell type is replaced by another (epithelial or mesenchymal).

The most common example is a change from columnar to squamous epithelium, as occurs in the squamous metaplasia of respiratory epithelium in response to chronic irritation.

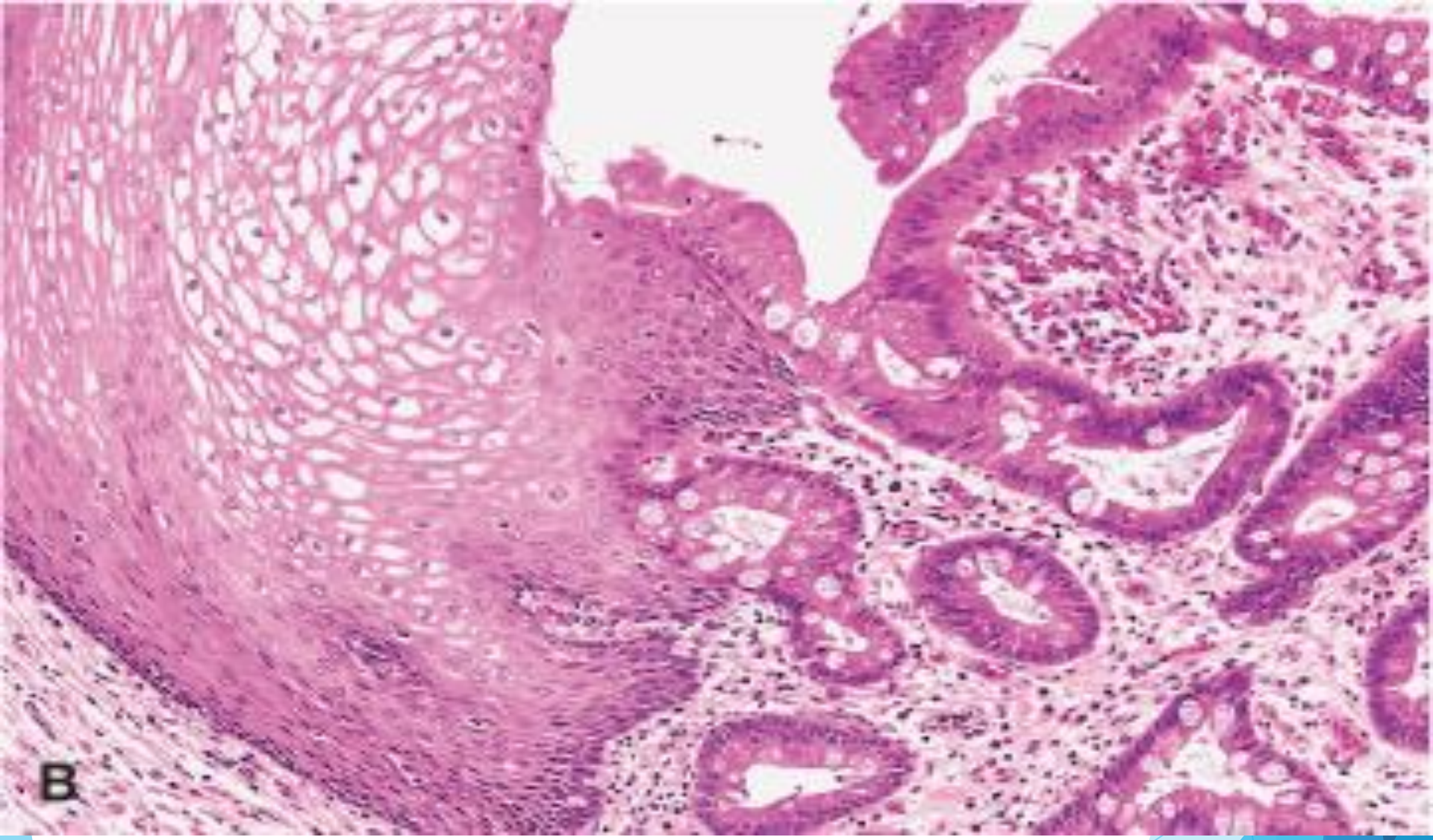
Although metaplastic epithelium is benign, the influences that predispose to such metaplasia if persist may induce atypical metaplasia which may progress to cancer transformation.

Metaplasia can occur in mesenchymal cells in which fibroblasts may become transformed to osteoblast or chondroblast to produce bone or cartilage.



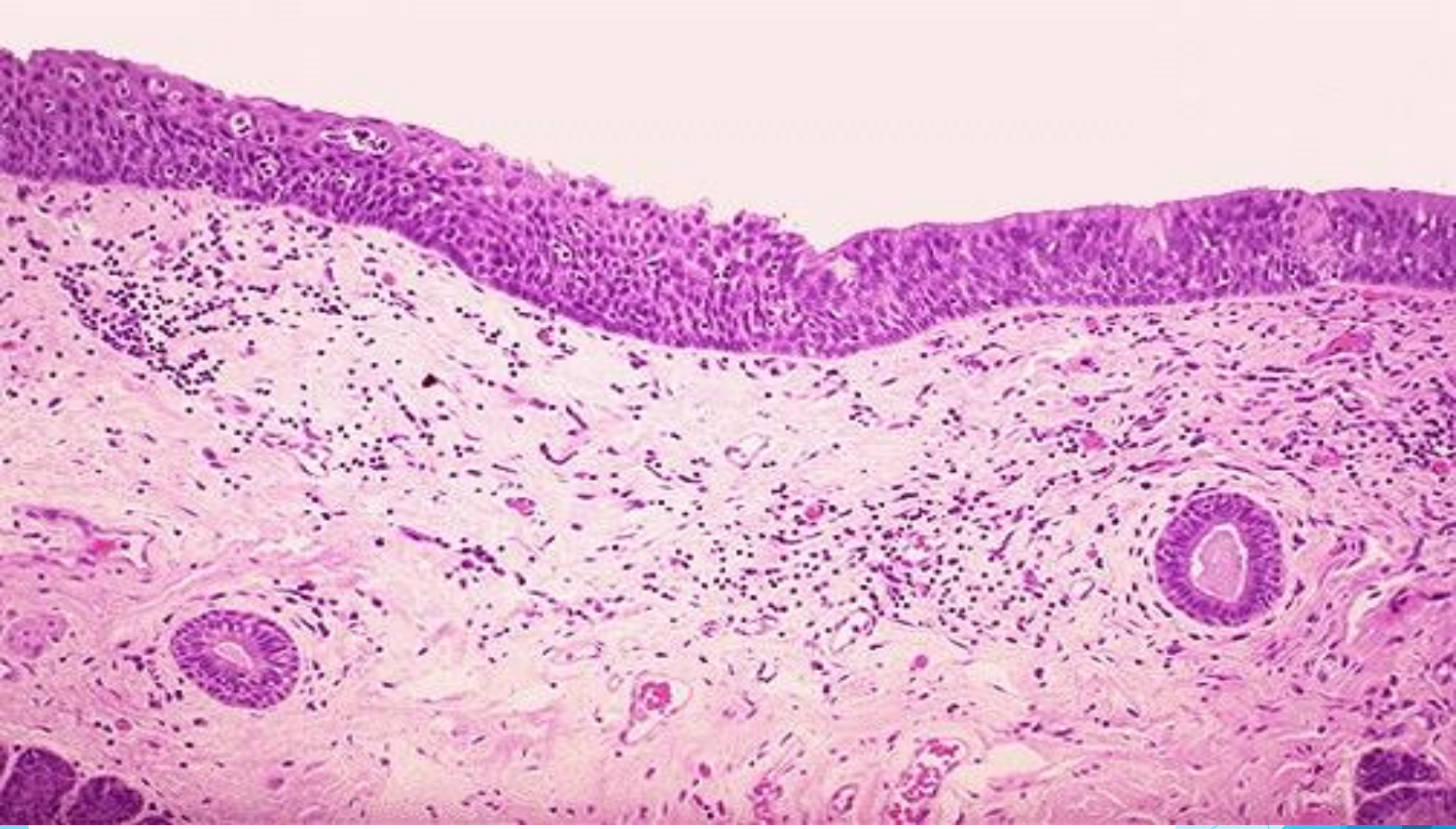
Endoscopical appearance of Barrett esophagitis

Squamous epithelium white in color while columnar epithelium red-pink in color.



Barrett's esophagitis

(squamous to columnar metaplasia)



Squamous metaplasia of respiratory epithelium. Transformation of (pseudostratified ciliated columnar epithelium (Rt.side) to stratified squamous epithelium (Lt.side)

- **Reversible injury:**

means pathologic cell changes that can be restored to normalcy if the stimulus is removed or if the cause of injury is mild.

- **Irreversible injury:**

occurs when the stressors exceed the capacity of the cell to adapt and indicate permanent pathologic changes that cause cell death.

There are two morphologic patterns of cell death:

1. **Necrosis.**
2. **Apoptosis.**

- **Necrosis:**

is the commonest type of cell death, involving severe cell swelling, denaturation and coagulation of proteins, breakdown of cellular organelles, and cell rupture.

- **Apoptosis:**

occurs when a cell dies by activation of an internal "suicide" program, involving elimination of unwanted cells with minimal disruption of the surrounding tissue.

Causes of Cell Injury:

- 1. Oxygen deprivation (hypoxia); affects aerobic respiration and therefore ability to generate adenosine triphosphate (ATP).

This important and common cause of cell injury and death occurs as a result of:

- A. Ischemia (loss of blood supply).
- B. Inadequate oxygenation (e.g. cardiorespiratory failure).
- C. Loss of oxygen-carrying capacity of the blood (e.g. anemia and CO poisoning).

- 2. Physical agents, including trauma, heat, cold, radiation, and electric shock.
- 3. Chemical agents and drugs, including therapeutic drugs, poisons, environmental pollutants, alcohol and narcotics.
- 4. Infectious agents, including viruses, bacteria, fungi, and parasites.

- 5. immunologic reactions, including autoimmune diseases.
- 6. Genetic derangements, such as chromosomal alteration and specific gene mutations.
- 7. Nutritional imbalances, including protein-calorie deficiency or lack of specific vitamins, and nutritional excesses.

References:

1. Robbins and Cotran

PATHOLOGIC BASIS OF DISEASES

By : KUMAR – ABBAS – ASTER

2. Robbins

BASIC PATHOLOGY

By: KUMAR- ABBAS – ASTER

Thank You