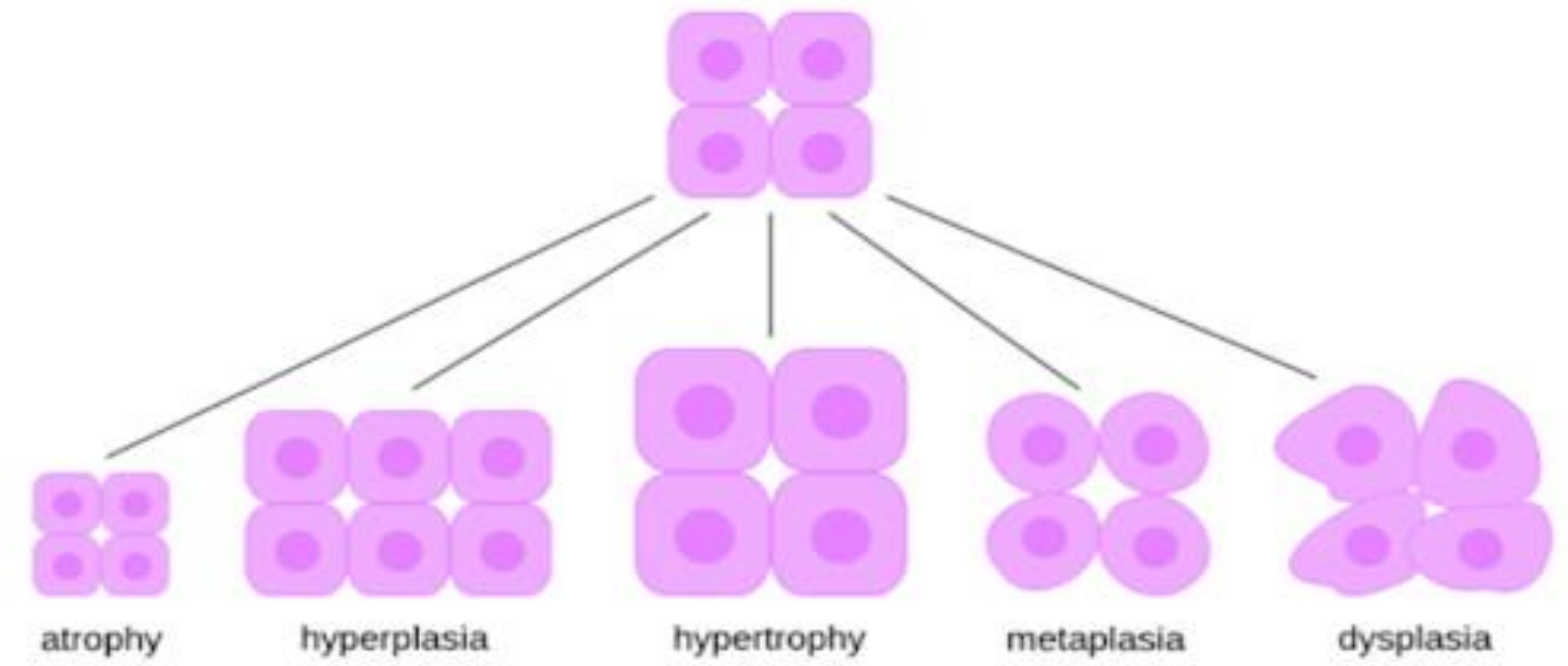
Cellular Response to Stress and Cellular Adaptations



A Quick Recap

- **What is the cell cycle?**
- Series of events that causes cell division, replication and division into two daughter cells.
- **What are the main stages of the cell cycle?**
- The main stages of the cell cycle are: **Interphase** and **M phase**
- **What happens during interphase?**
- Interphase is the longest phase of the cell cycle and includes three stages: **G1 phase**: Cell growth and preparation for DNA replication**, S phase**: DNA is replicated**, G2 phase**: The cell continues to grow and prepares for mitosis.
- **What happens during mitosis?**
- Mitosis is the process of cell division where one parent cell divides into two identical daughter cells. It consists of four main phases: **Prophase, Metaphase**, **Anaphase** and **Telophase**

A Quick Recap

- **What is cytokinesis?**
- Cytokinesis is the final step of the cell cycle, where the cytoplasm divides, and two daughter cells are formed. It usually occurs right after mitosis.
- **What are checkpoints in the cell cycle?**
- Checkpoints are control mechanisms in the cell cycle that ensure each stage is completed properly before moving on to the next stage. They help prevent errors such as DNA damage or incomplete DNA replication. **G1 Checkpoint (Restriction Point), G2 Checkpoint and M Checkpoint (Spindle Assembly Checkpoint)**

Objectives

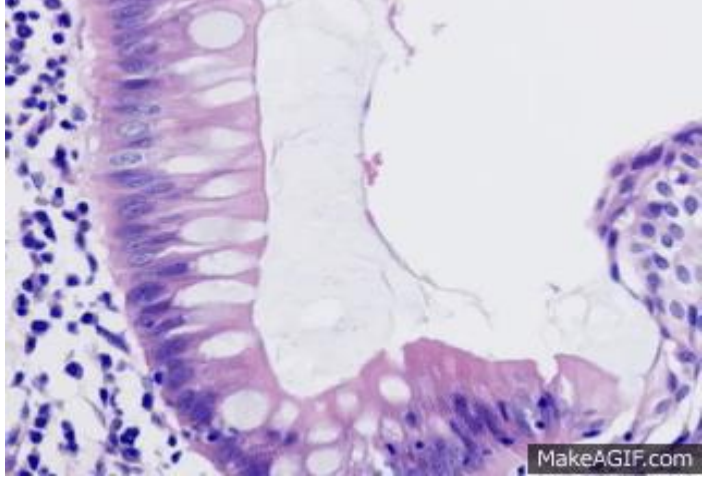
At the end of the session student's should be able to:

- Describe causes, mechanism and pathogenesis of cell injury
- Describe cellular Adaptations.
- Define with examples of:

- Hyperplasia;
- Metaplasia;
- Atrophy;
- Hypertrophy.

PATHOLOGY

Pathology is the study devoted to the study of the structural, biochemical, and functional changes in cells, tissues, and organs that underlie disease



Etiology or Cause

Genetic

Acquired

Pathogenesis

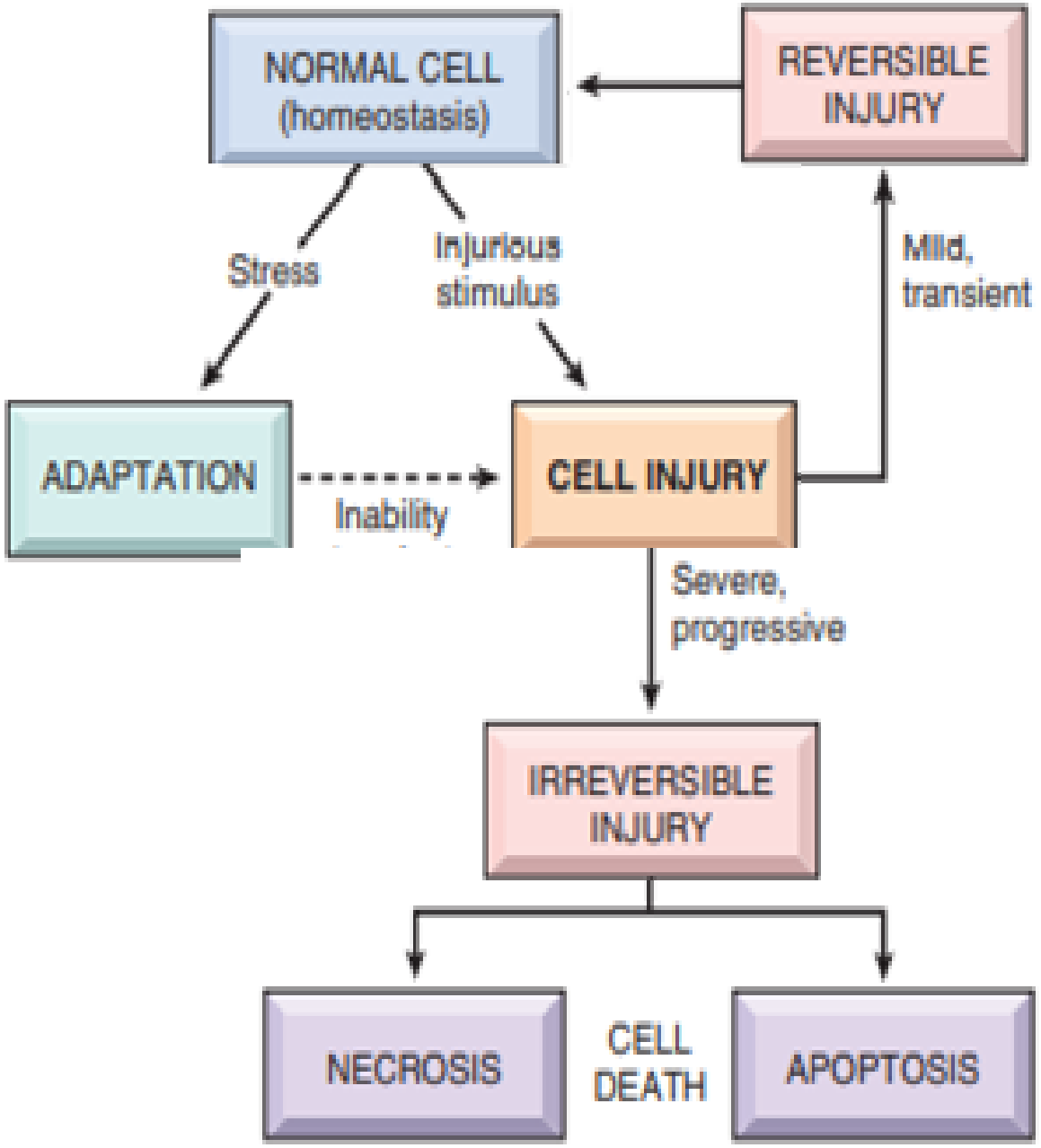
Sequence of cellular, biochemical, and molecular events that follow the exposure of cells or tissues to an injurious agent

Morphological Changes

Structural alterations in cells or tissues that are either characteristic of a disease or diagnostic of an etiologic process

Clinical Manifestations

End results of genetic, biochemical, and structural changes in cells and tissues are functional abnormalities



Homeostasis

Adaptations

Reversible functional and structural responses to changes in physiologic states (e.g., pregnancy) and some pathologic stimuli, new but altered steady states are achieved, allowing the cell to survive and continue to function

Cell injury

Reversible up to a certain point, but if the stimulus persists or is severe enough from the beginning, the cell suffers irreversible injury and ultimately undergoes cell death

Cell death

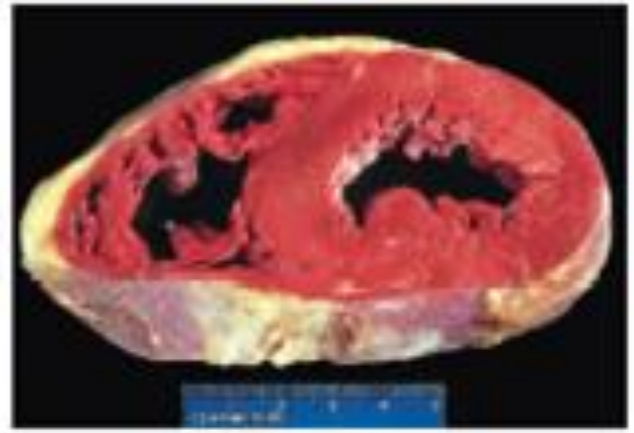
The end result of progressive cell injury, is one of the most crucial events in the evolution of disease in any tissue or organ

Table 2-1 Cellular Responses to Injury

Nature of Injurious Stimulus	Cellular Response
Altered physiologic stimuli; some nonlethal injurious stimuli	Cellular adaptations
Increased demand, increased stimulation (e.g., by growth factors, hormones)	Hyperplasia, hypertrophy
Decreased nutrients, decreased stimulation	Atrophy
Chronic irritation (physical or chemical)	Metaplasia
Reduced oxygen supply; chemical injury; microbial infection	Cell injury
Acute and transient	Acute reversible injury Cellular swelling fatty change
Progressive and severe (including DNA damage)	Irreversible injury → cell death Necrosis Apoptosis
Metabolic alterations, genetic or acquired; chronic injury	Intracellular accumulations; calcification
Cumulative sublethal injury over long life span	Cellular aging

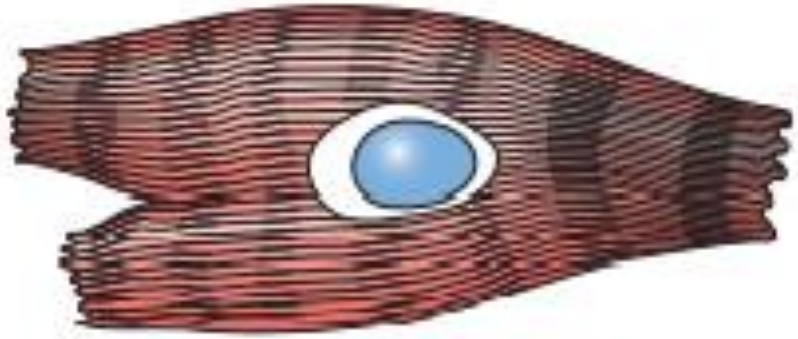
Normal myocyte

Adaptation:
response to increased load

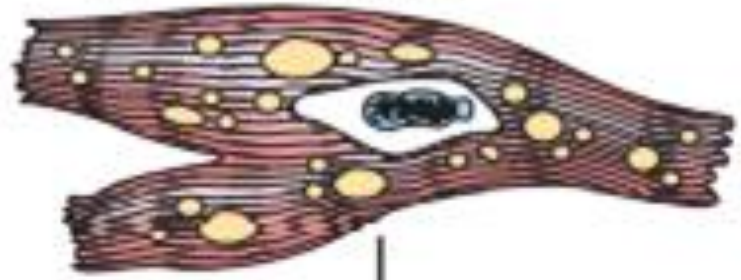


Cell injury

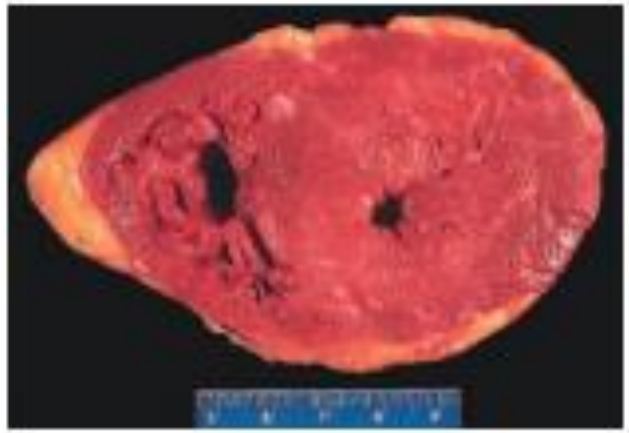
Adapted myocyte (hypertrophy)

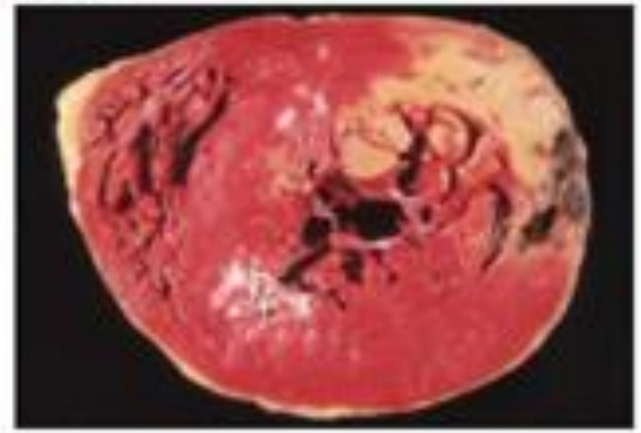


Reversibly injured myocyte



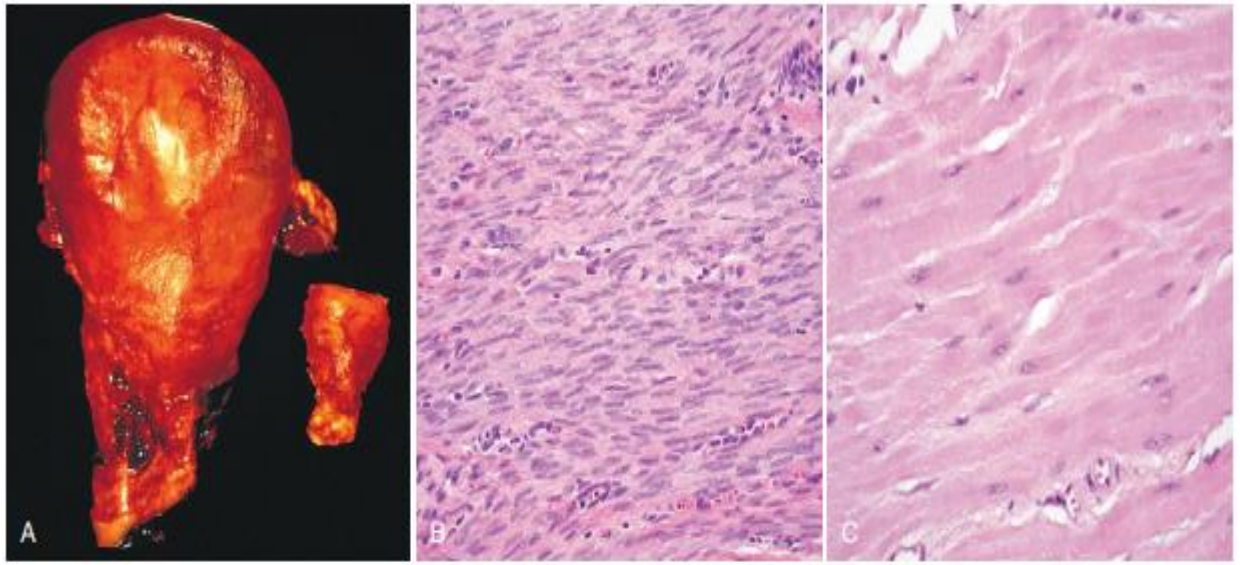
Cell death



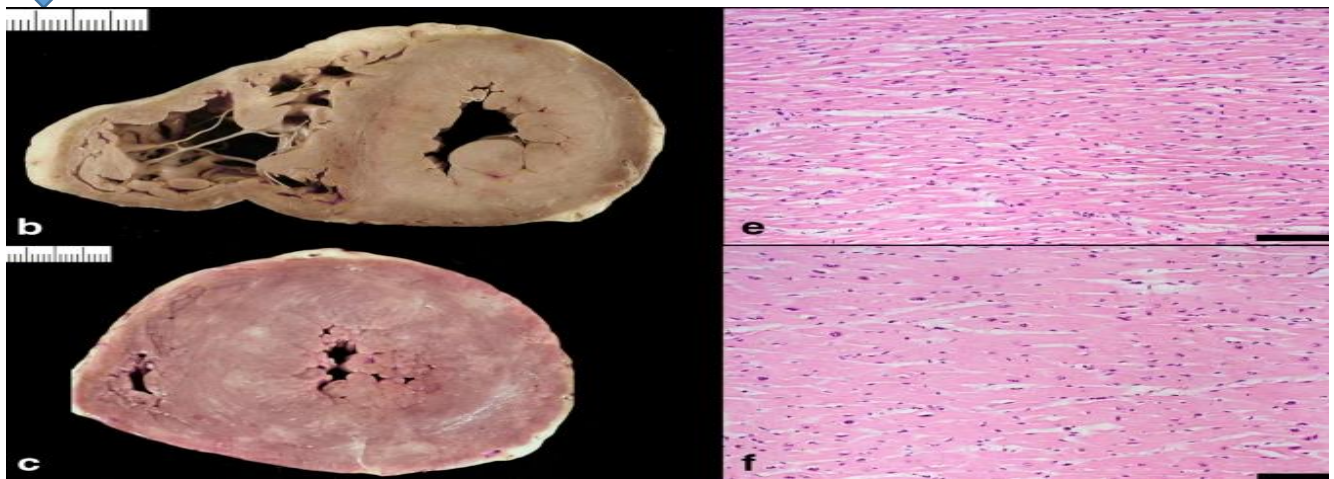


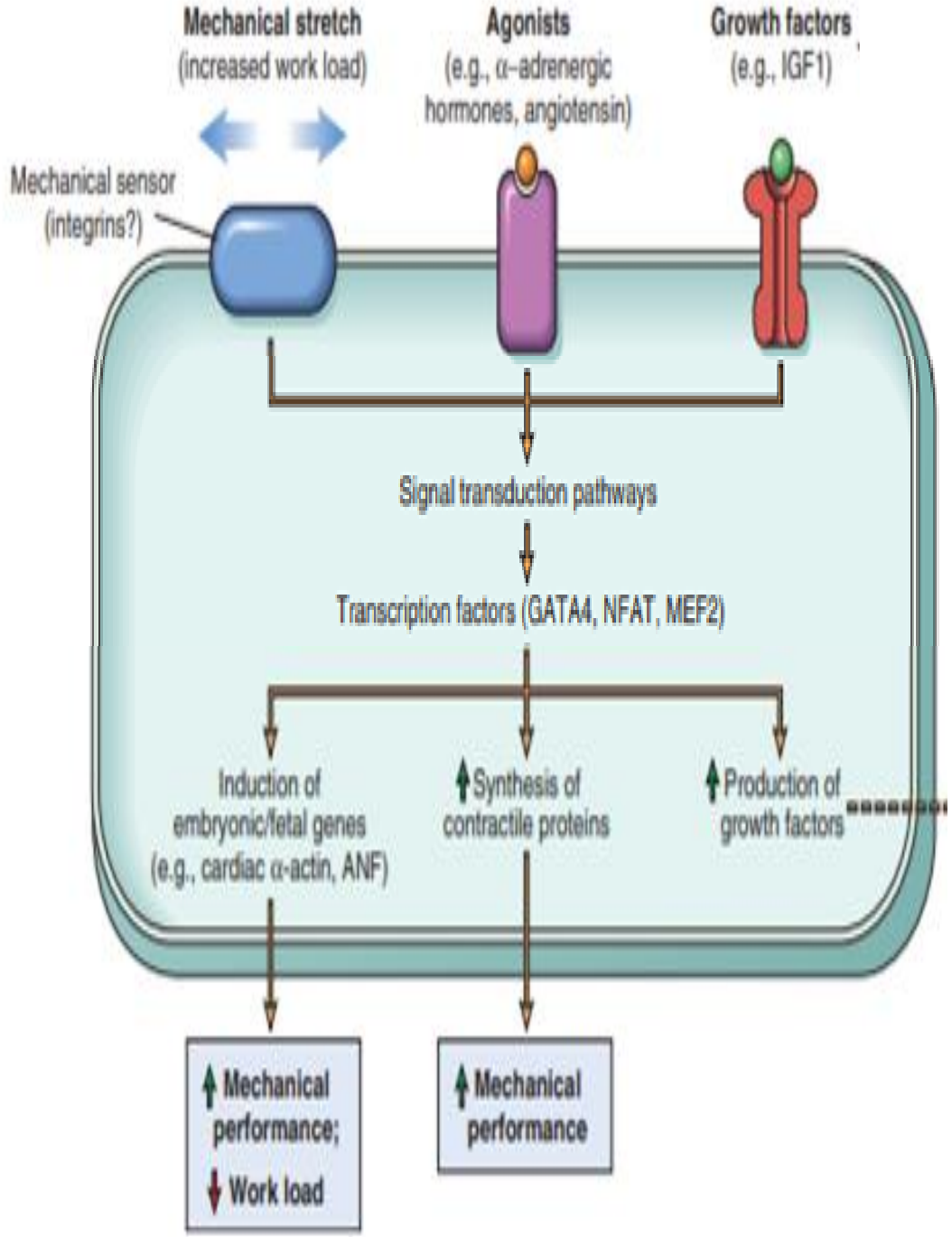
HYPERTROPHY

PHYSIOLOGICAL



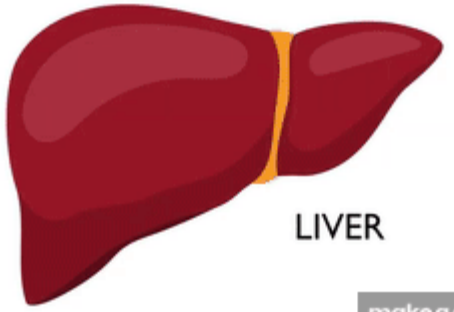
PATHOLOGICA

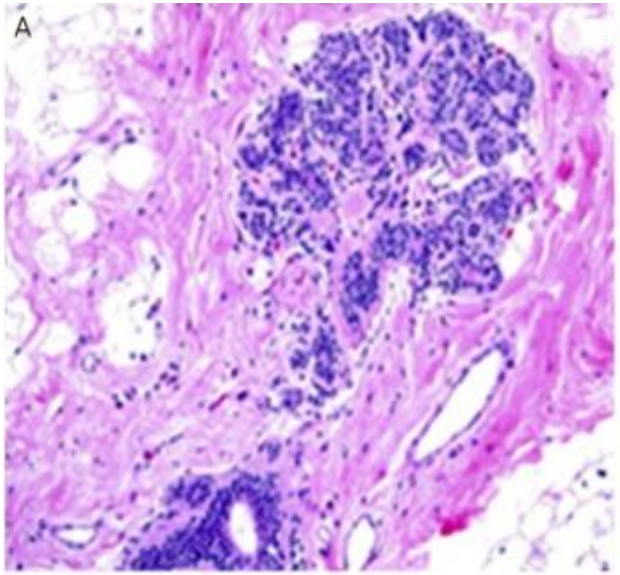


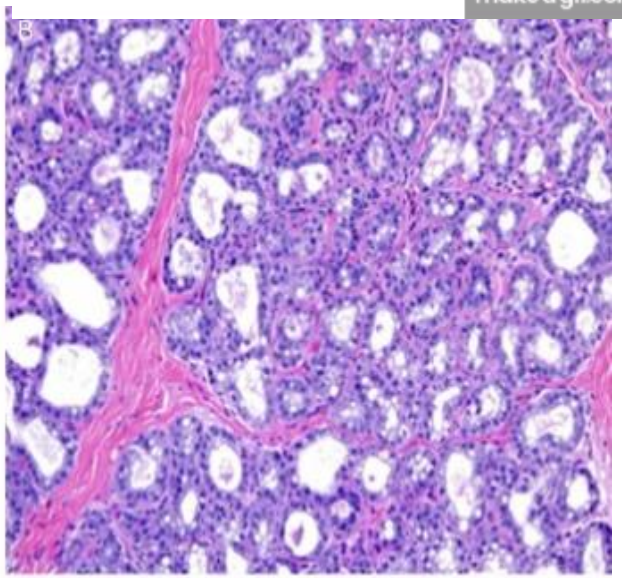


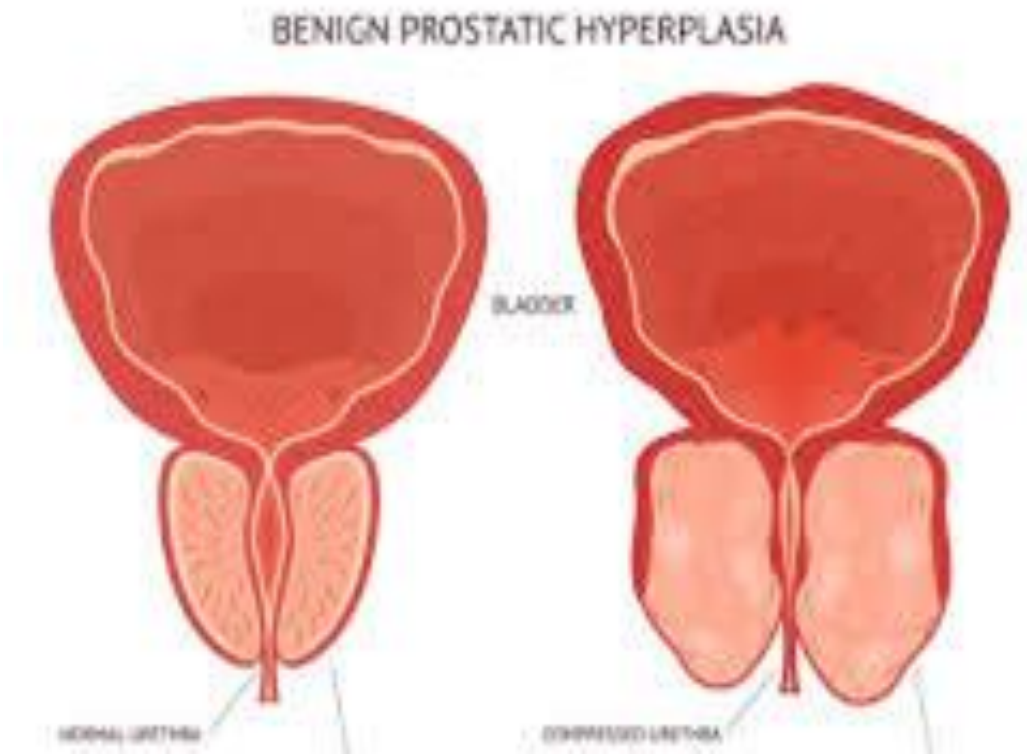
HYPERPLASIA

PHYSIOLOGICAL

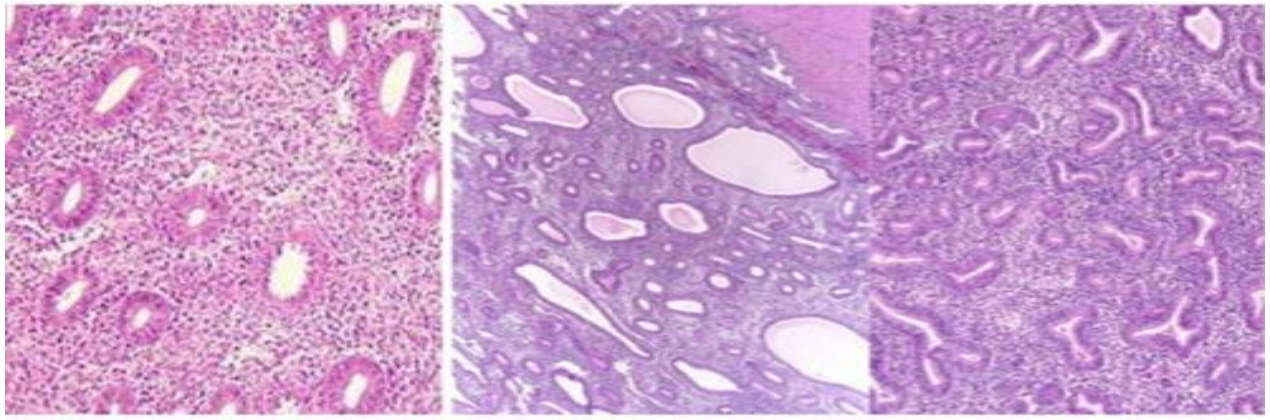








PATHOLOGICA



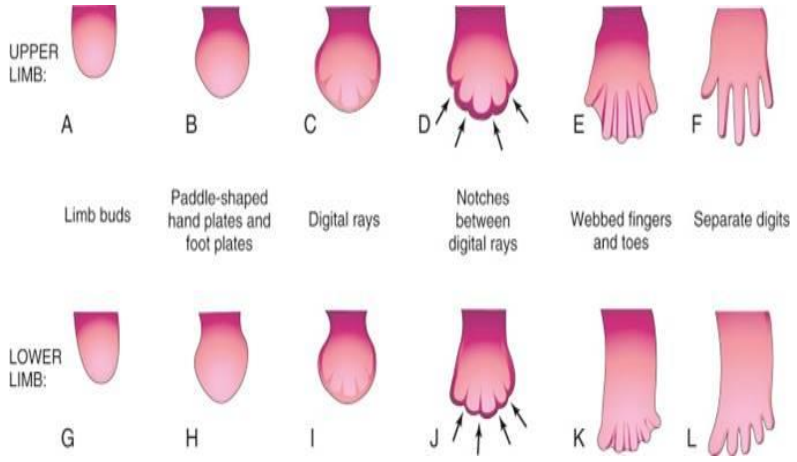
Hyperplasia if pathologic constitutes a fertile soil in which cancerous proliferations may eventually arise.

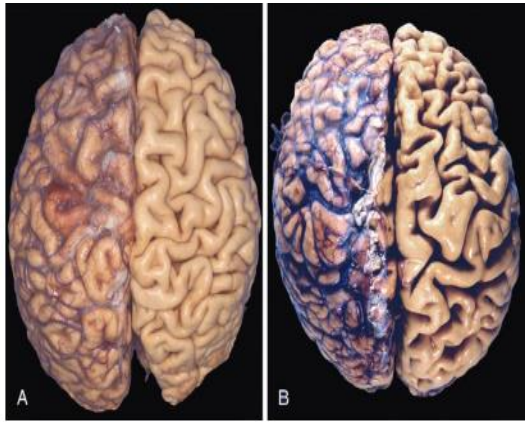
It is the result of growth factor-driven proliferation of mature cells or increase output of new cells from tissue stem cells.

ATROPHY

- Decreased workload (atrophy of disuse)
- Loss of innervation (denervation atrophy)
- Diminished blood supply
- Inadequate nutrition
- Loss of endocrine stimulation
- Pressure
- Senile

PHYSIOLOGICAL





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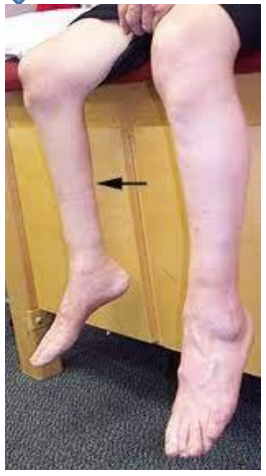
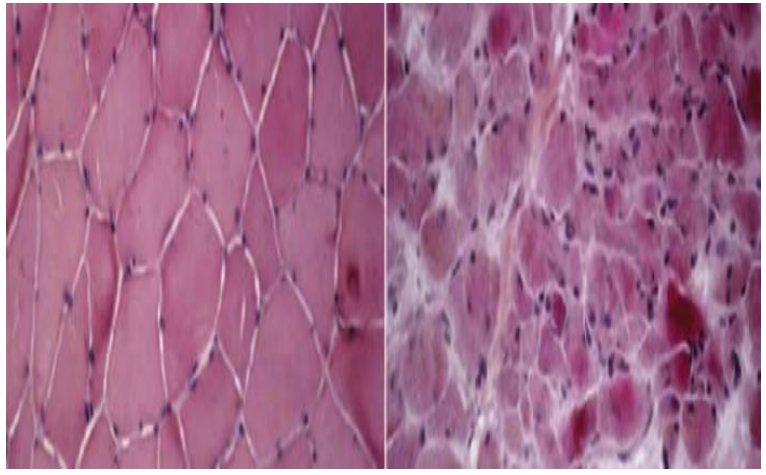
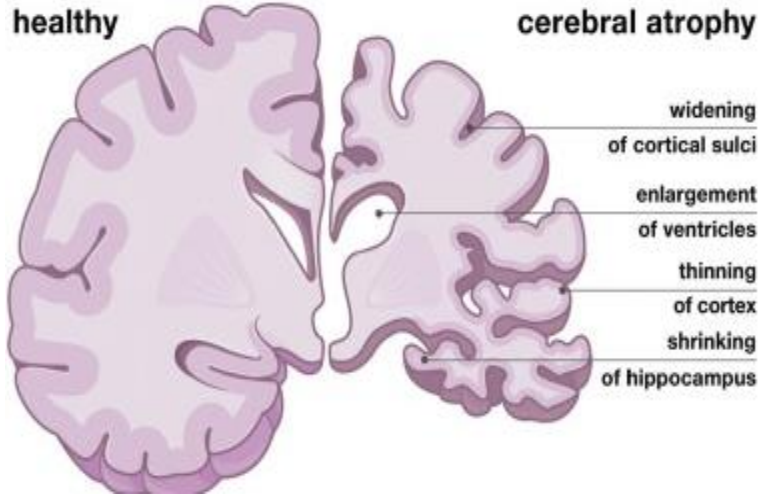


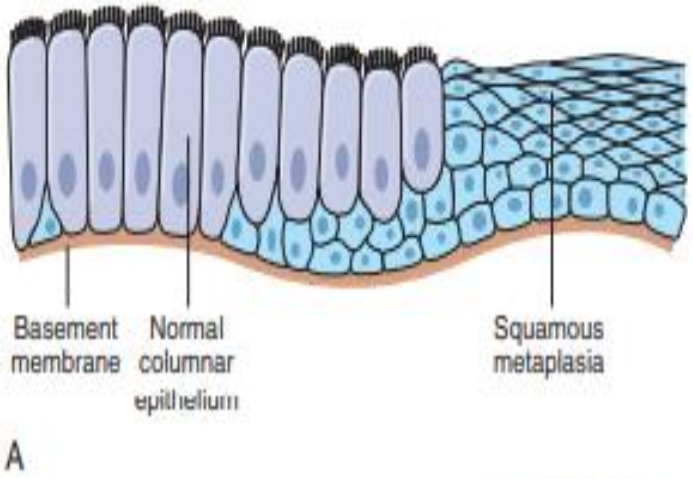




Decreased protein synthesis because of reduced metabolic activity

Degradation of cellular proteins by the ubiquitin-proteasome pathway

METAPLASIA



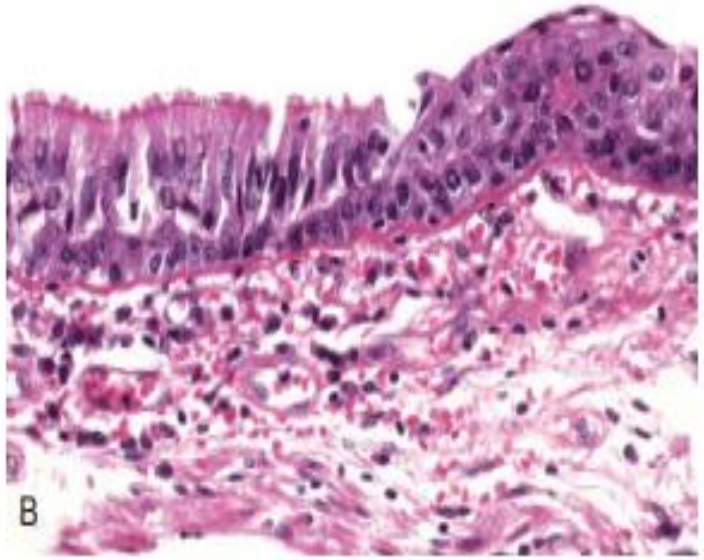
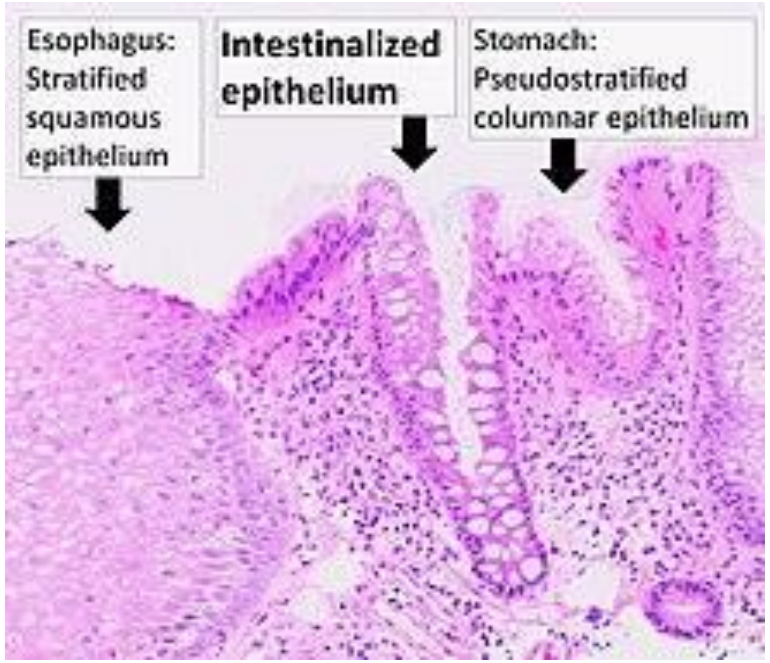
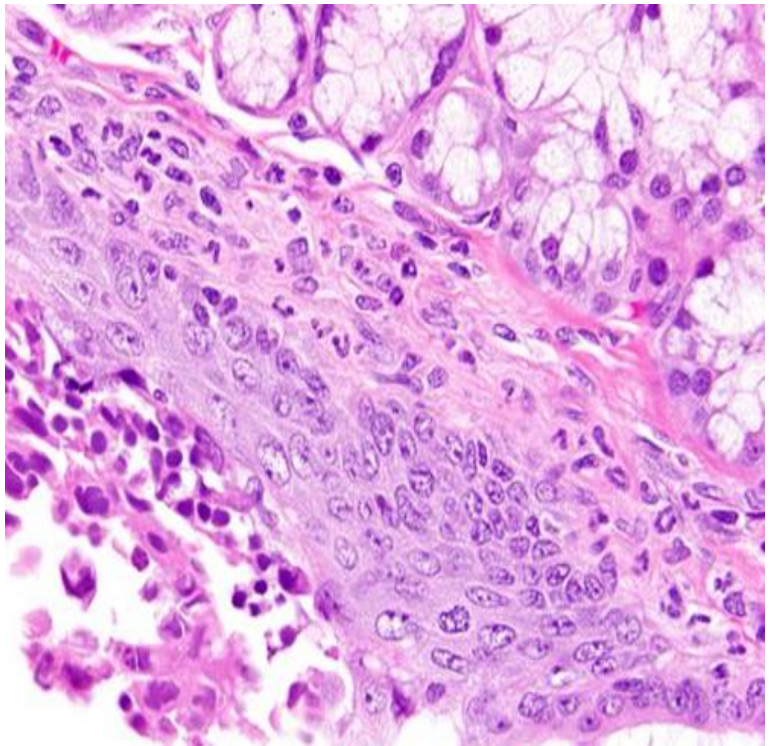


Figure 2-6 Metaplasia of columnar to squamous epithelium. **A,** Schematic diagram. **B,** Metaplasia of columnar epithelium *(left)* to squamous epithelium *(right)* in a bronchus.

Smokers/ Vit A deficiency

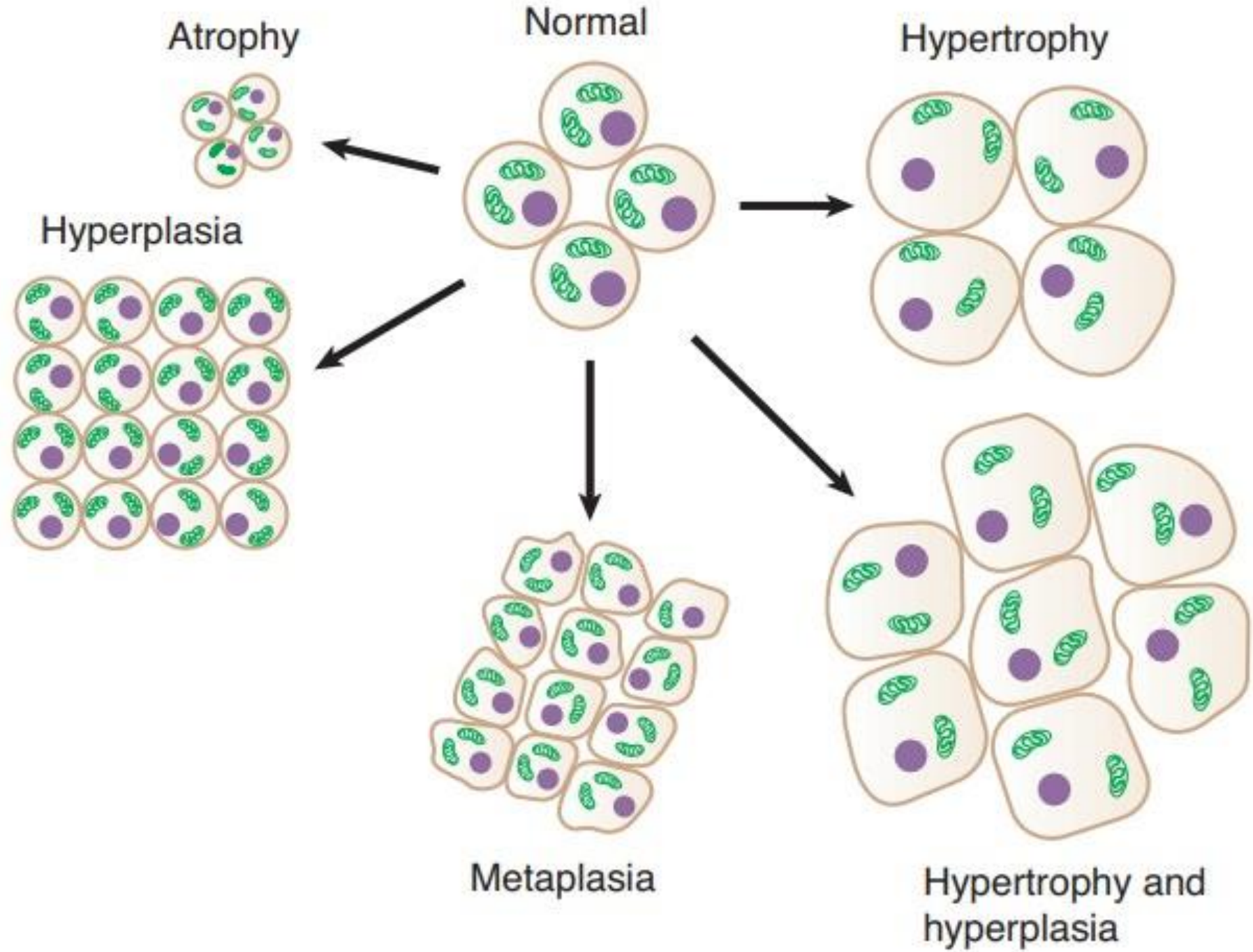




Epithelial metaplasia is a double-edged sword

With persistent stimulus, can initiate malignant transformation

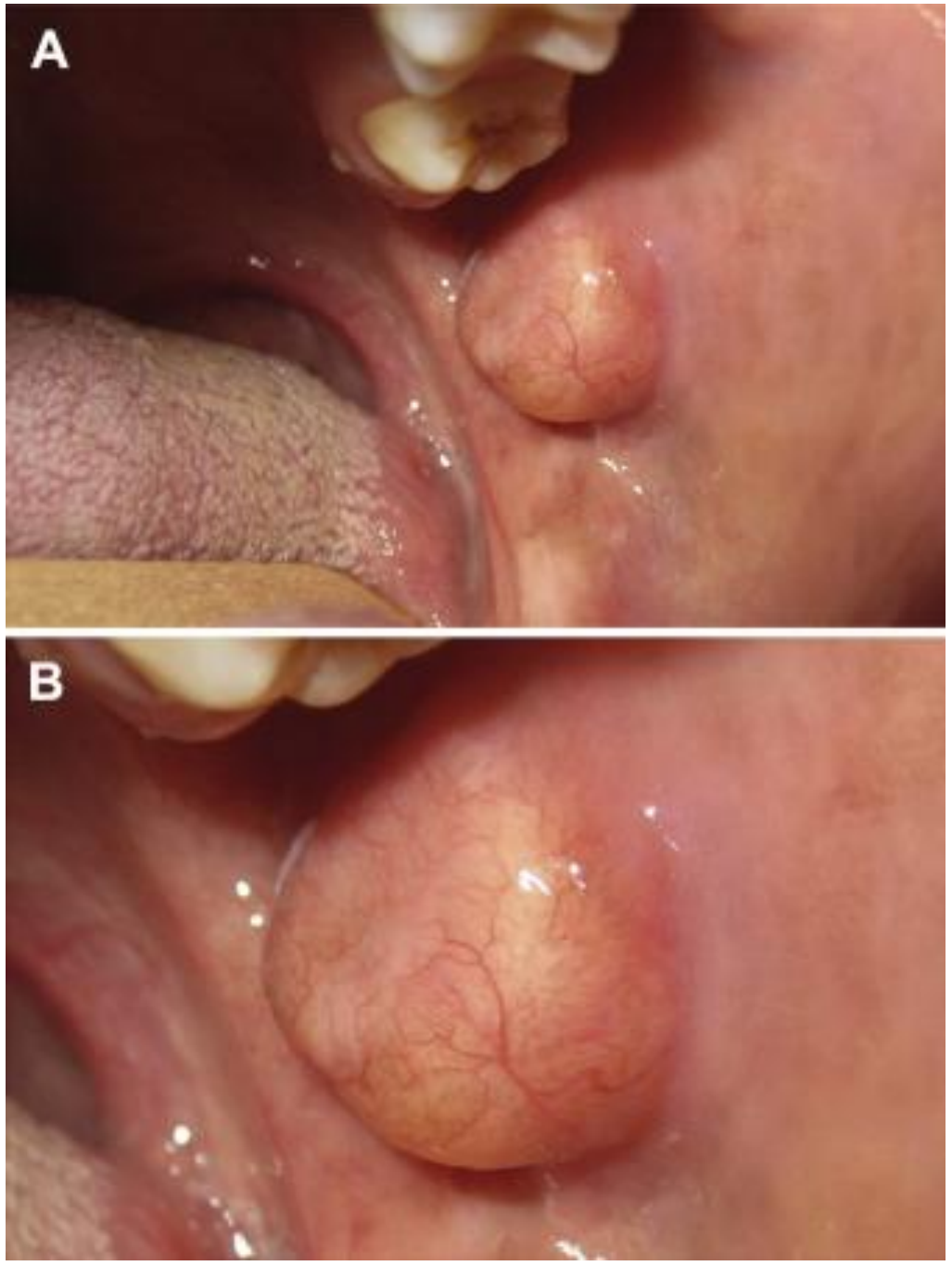
Reprogramming of adult stem cells, or undifferentiated mesenchymal cells and differentiation along a new pathway by cytokines, growth factors, and extracellular matrix components


Atrophy
Normal
Hypertrophy
Hyperplasia
Metaplasia
Hypertrophy and
hyperplasia

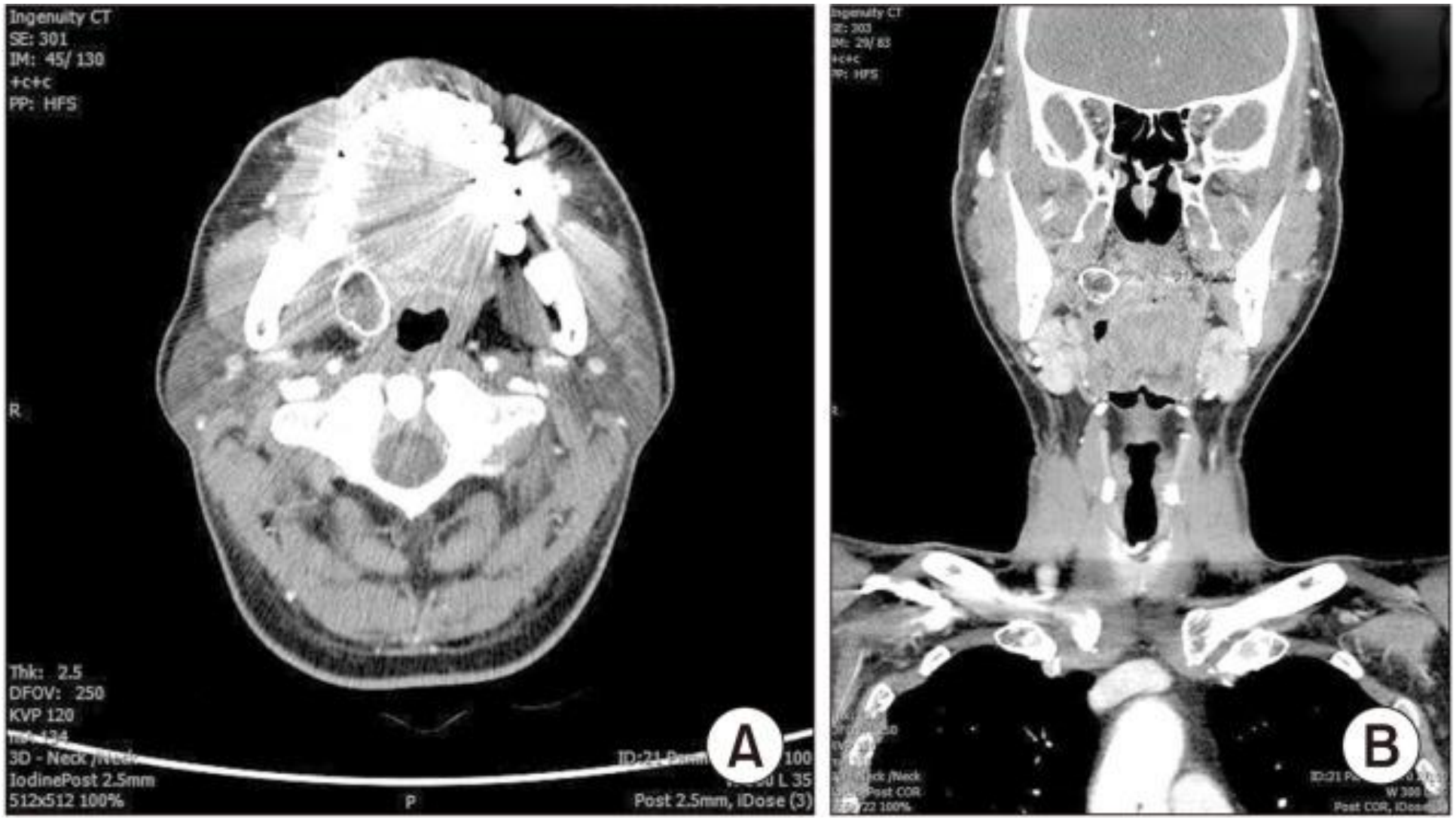
Case Study

A 60-year-old male patient visited clinic complaining of a palpable mass on the right palate area. No history of trauma and pain. The patient had no relevant diseases other than hypertension

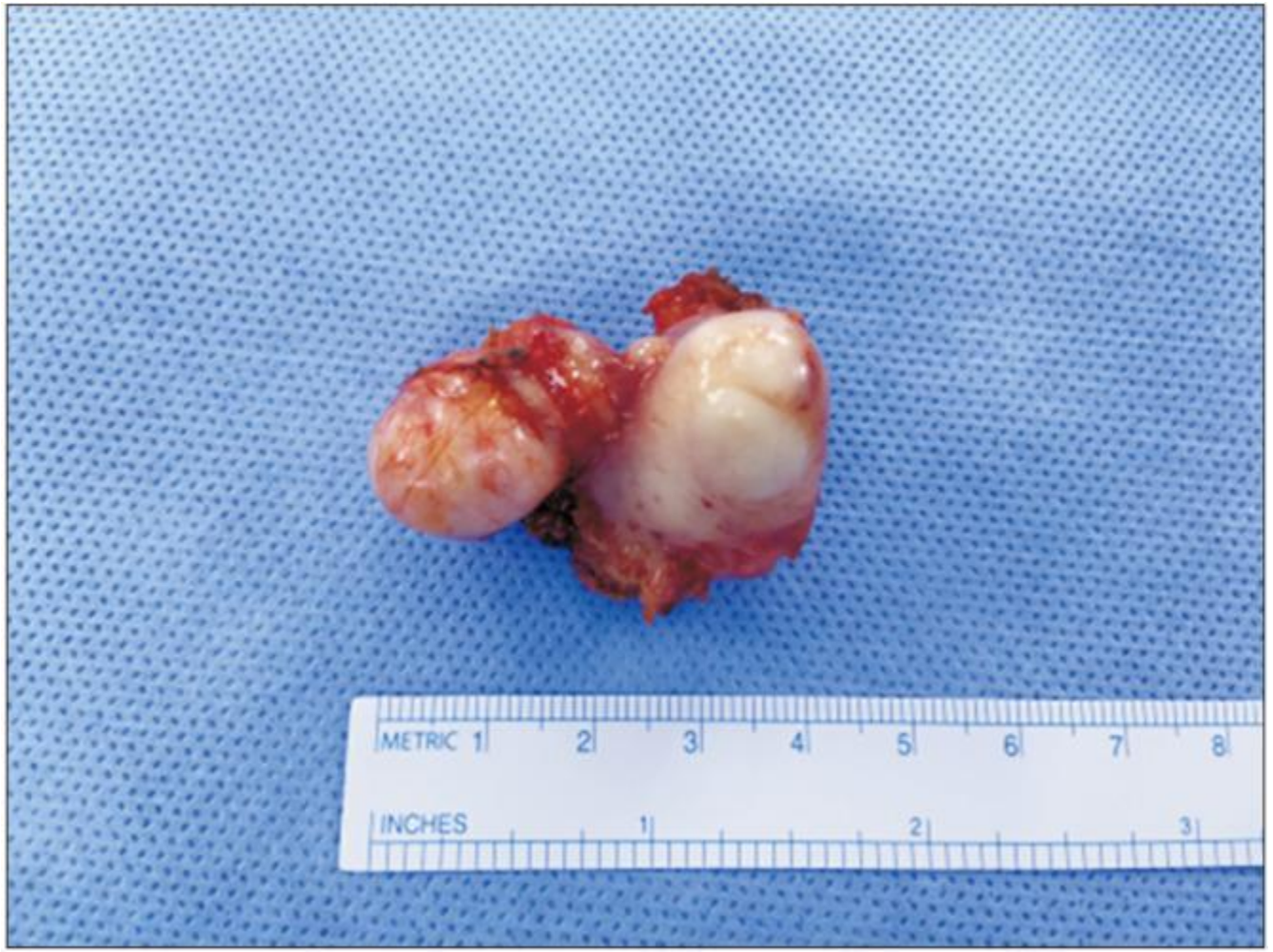
Clinical examination revealed a large, round, firm mass on the right palate area



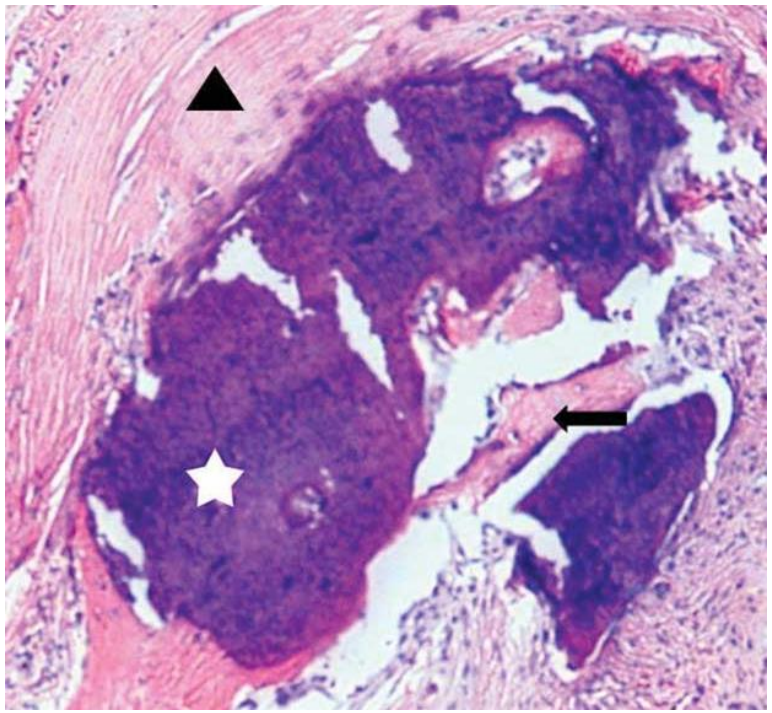
On computed tomography, there was a round hard-tissue mass approximately 2 cm in diameter on the right palate area



Excision was performed intraorally and biopsy was performed



Presence of lamellar bone inside soft tissue of palate



Osseous metaplasia :It forms as a result of the transformation of non-osseous connective tissue into mature bone

A 50-year-old woman with no previous medical or surgical history, attends a gynecological evaluation for a history of abnormal uterine bleeding

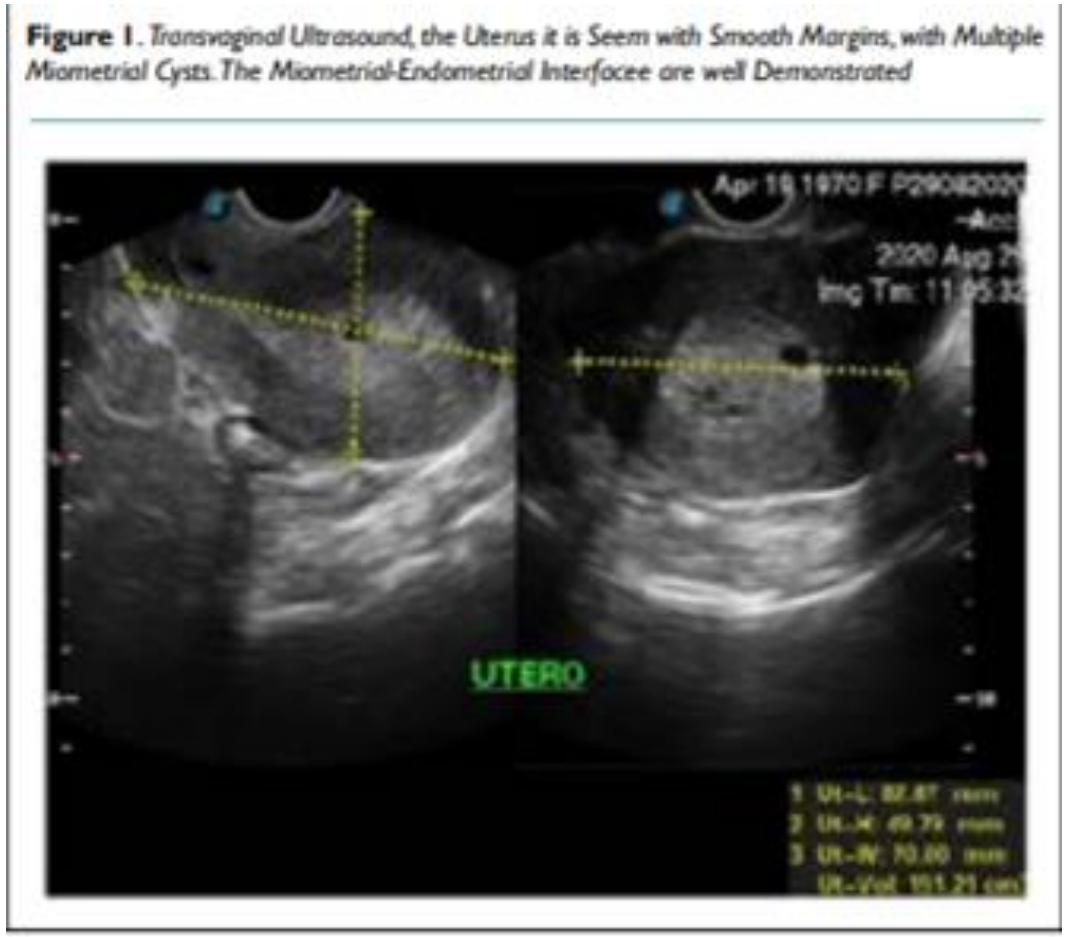
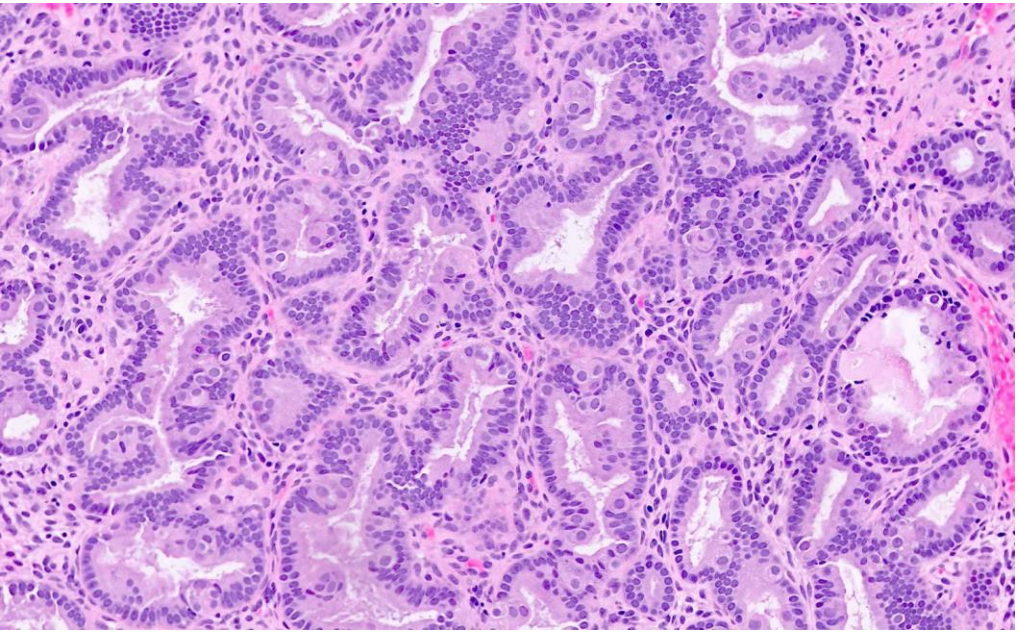
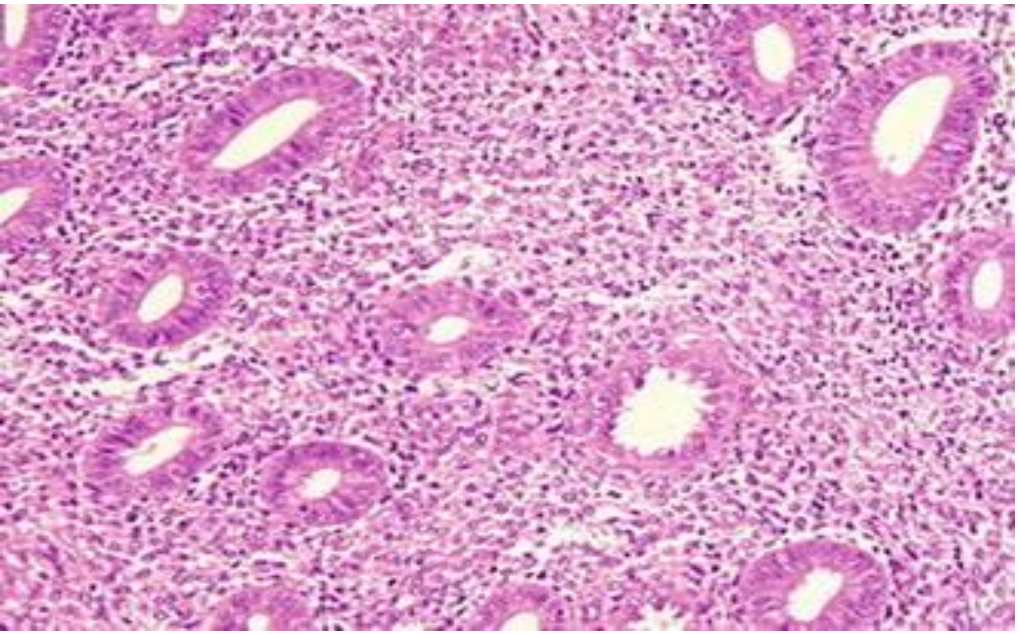


Figure 1. *Transvaginal Ultrasound, the Uterus it is Seem with Smooth Margins, with Multiple Miometrial Cysts. The Miometrial-Endometrial Interfacee are well Demonstrated*

Ultrasound showed a endometrium markedly thickened with multiple asymmetric cystic lesions



Closely packed glands with increased gland to stroma ratio
Variation in gland size with cystic dilatation



Endometrial Hyperplasia

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