Pathophysiology lab 2

Cellular injury and adaptation

Adaptation

Cellular changes that aim to preserve cell viability and prevent cell injury. The adaptive responses include:

1. ***Atrophy***
2. ***Hypertrophy***
3. ***Hyperplasia***
4. ***Metaplasia***
5. ***Hypoplasia***
6. ***Dysplasia***

***Hypertrophy*: increase in cell size only ie cells will be bigger.**

Hypertrophy could be:

A. *Physiological* as in uterine hypertrophy during pregnancy and skeletal muscles in athletes.

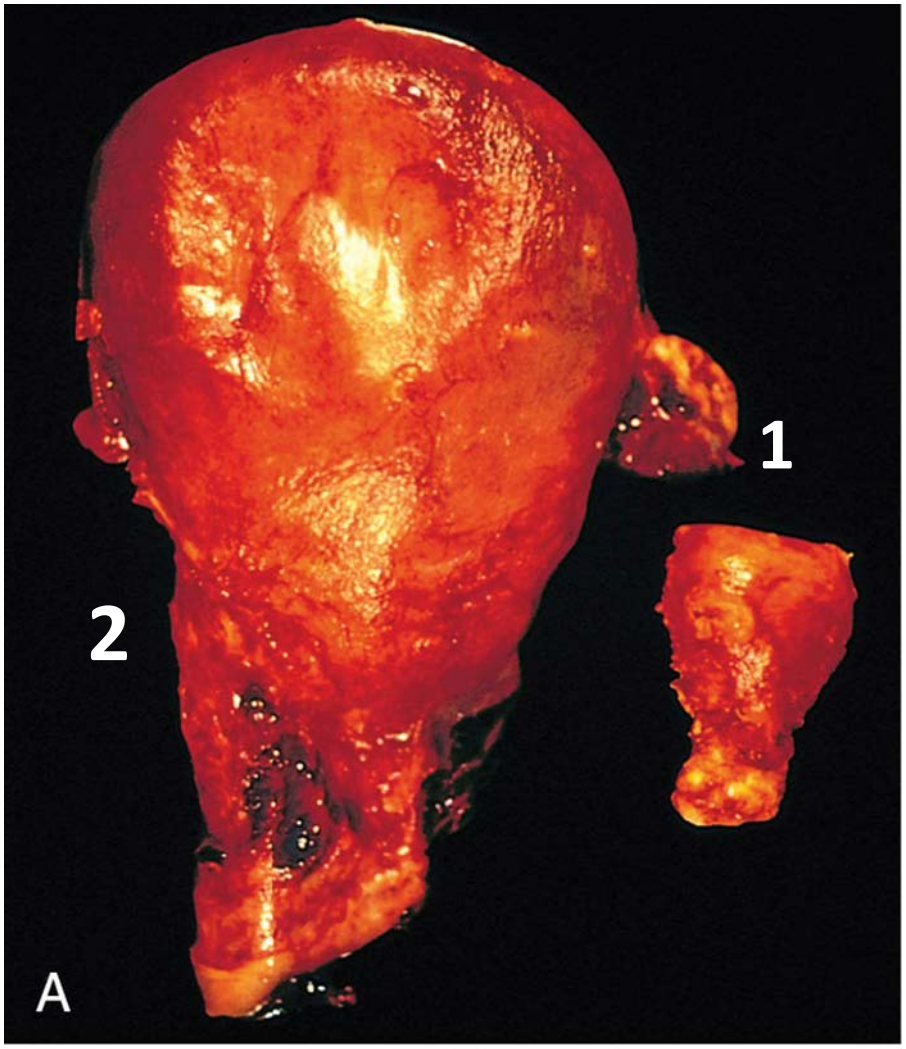
B. *Pathological* as in left ventricle cardiac muscle hypertrophy due to systemic hypertension

Hypertrophy

gross:

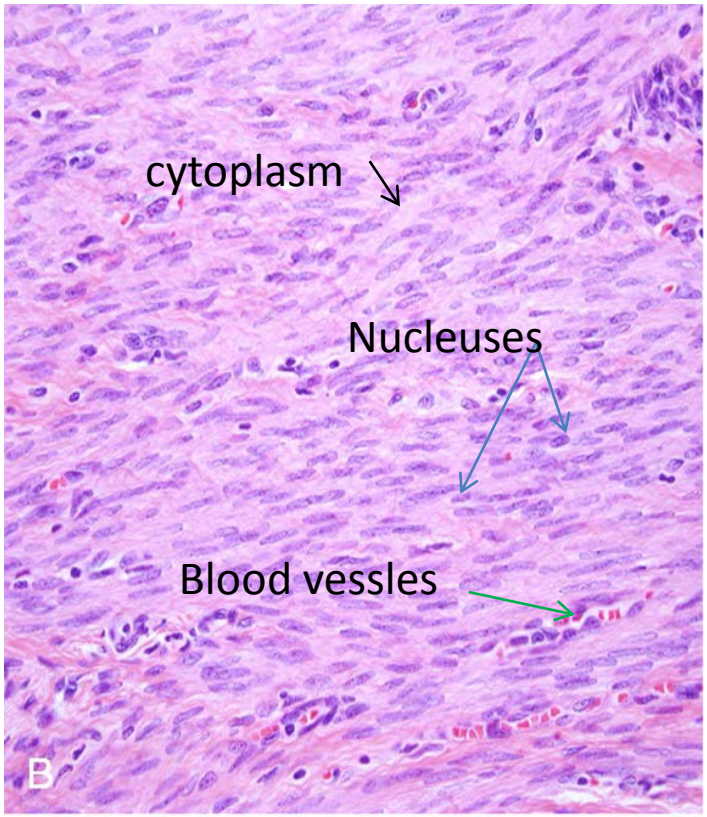
1: **normal uterine size**

2:Hypertrophy of uterus during pregnancy, increase in size of the uterus as an adaptation for pregnancy and fetal growth.

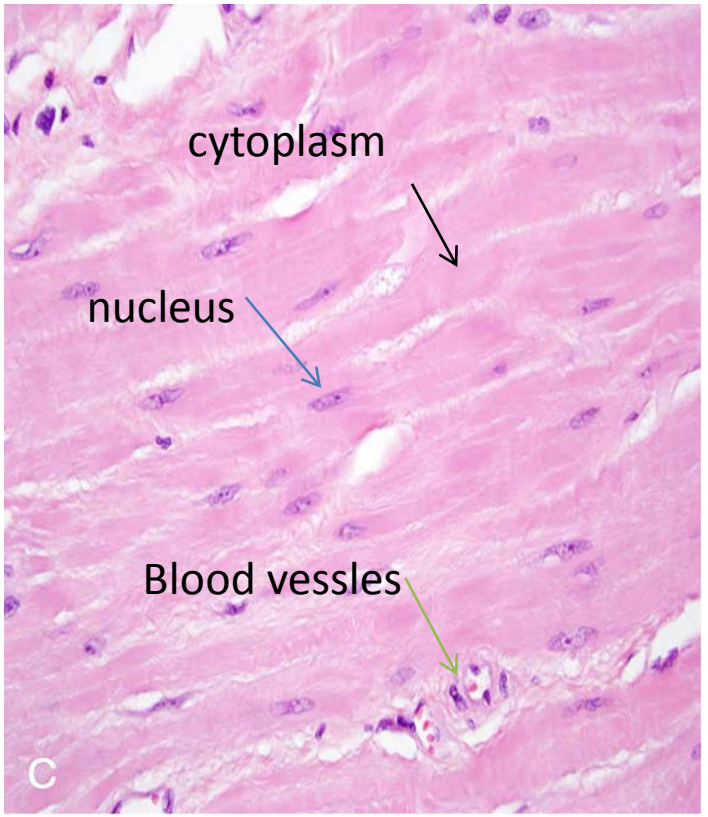


Hypertrophy: under microscope

Normal uterine smooth muscle under LM



Section showing uterine smooth muscles directly after delivery: no new cells it just increase in cell size by the increase in amount of cytoplasm



Pathological hypertrophy: LV hypertrophy

Gross:

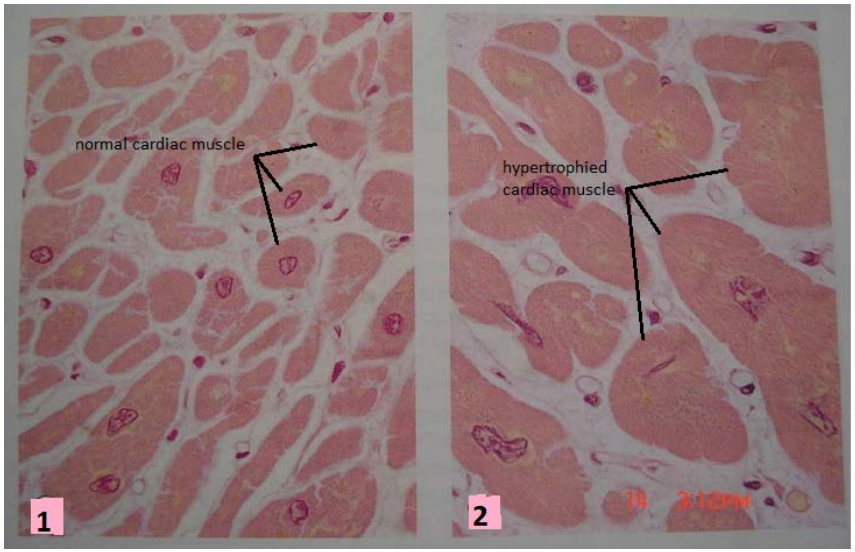
1: normal heart size

2: LV hypertrophy: increase LV size

LV hypertrophy:
under microscope

1: cross section shows normal cardiac muscle fiber

2: cross section shows hypertrophy in cardiac muscle fiber increase in nuclear and cytoplasmic size



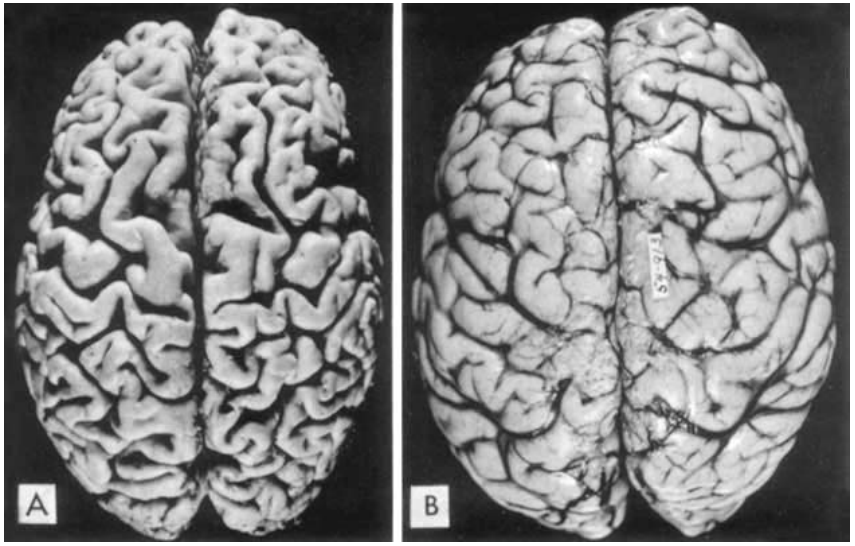
Atrophy:

Decrease in the size of a cell or organ by loss of cell substance (both size and number)

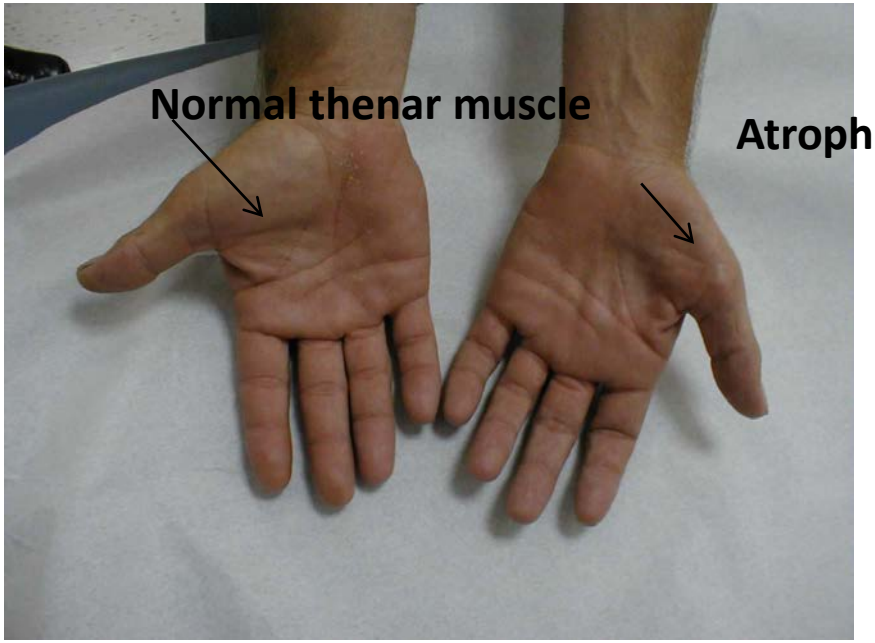
gross:

A: atrophied brain

B: normal brain



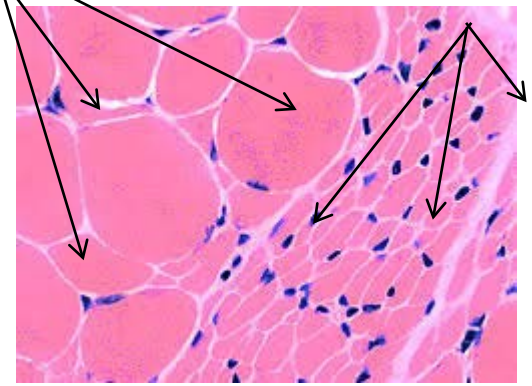
Atrophy: thenar muscle gross:



Atrophy: skeletal muscle under microscope:

Normal skeletal muscle fibers

Atrophied skeletal muscle fibers



Hyperplasia :

Increase in the number of cells in an organ which may then increase organ size.

It could be:

A:Physiologic: as in hyperplasia of glandular tissue of female breast after puberty

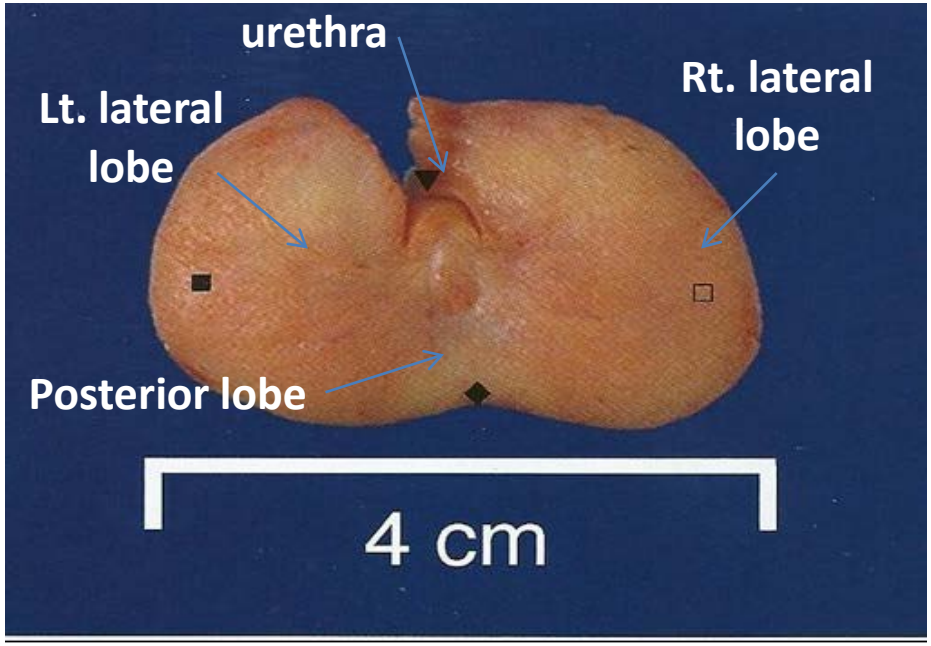
B: Pathologic: as in benign prostatic hyperplasia (BPH), endometrial hyperplasia. Pathological hyperplasia also can be divided to:

1. Typical: hyperplastic cells looks like normal cells regarding their size, nuclear morphology and orientation.

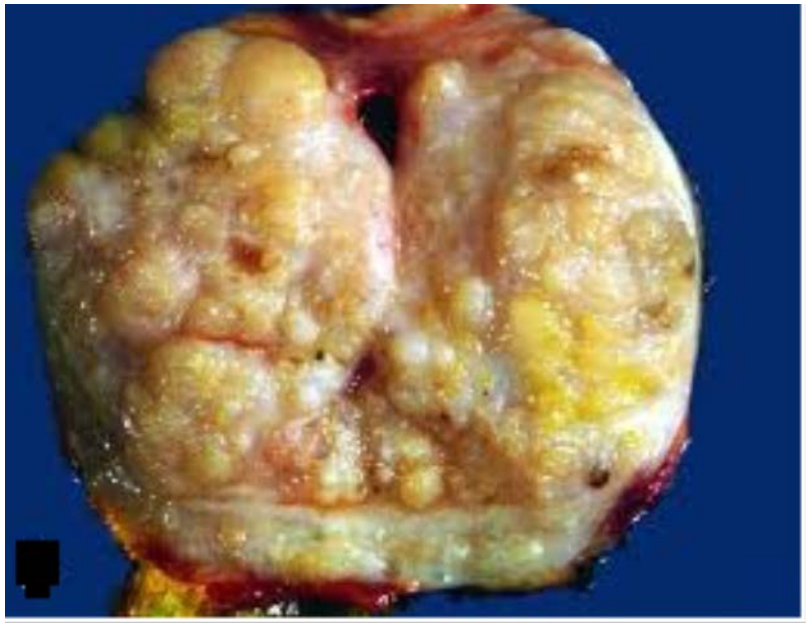
2. Atypical: hyperplastic cells clearly differ than normal cell zize, nuclear morphology and orientation

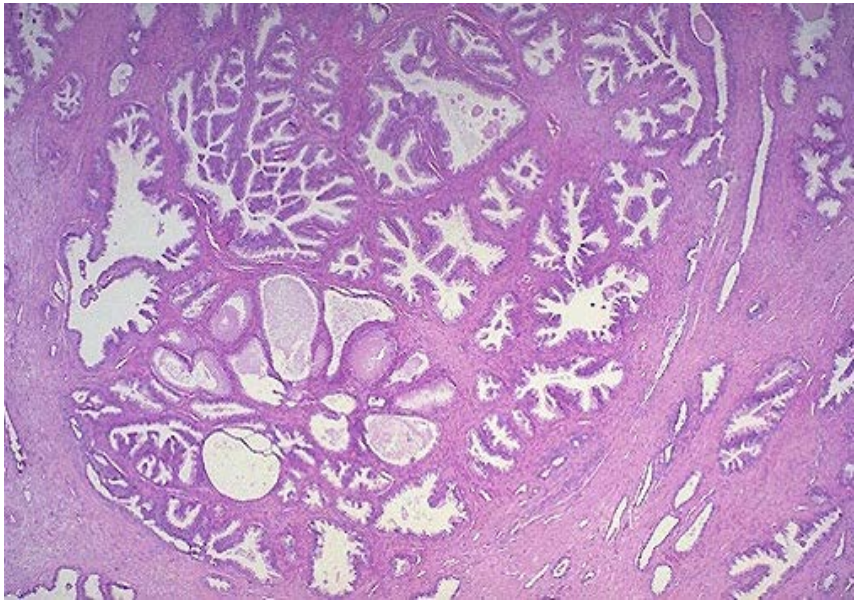
Hyperplasia normal and BPH prostate: gross

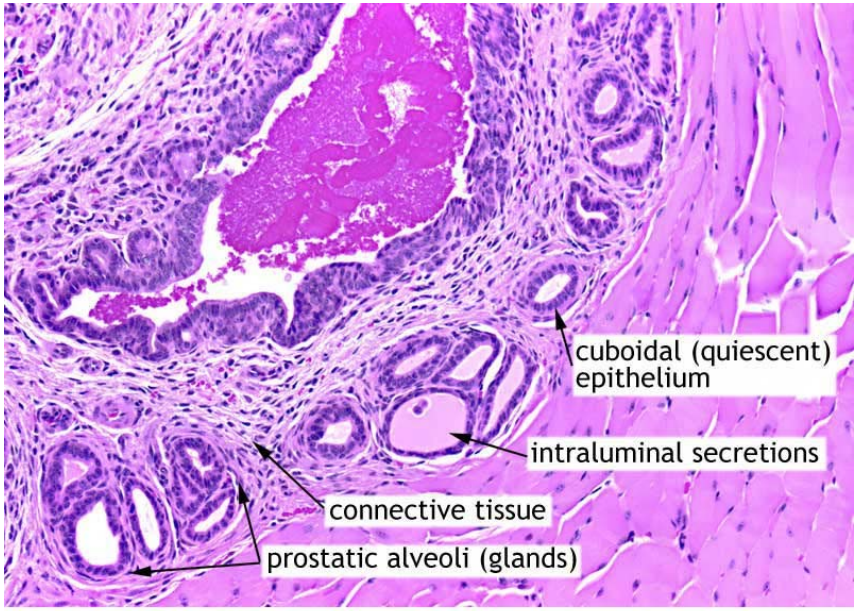
Cross section of normal prostate about 4 cm length homogenous in texture

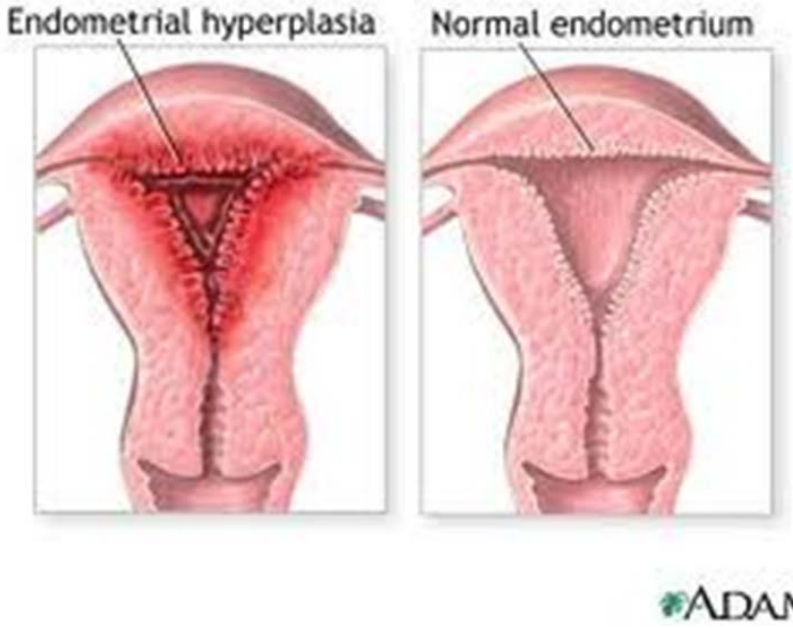


Benign prostate hyperplasia BPH: cross section shows increase size and nodular appearance





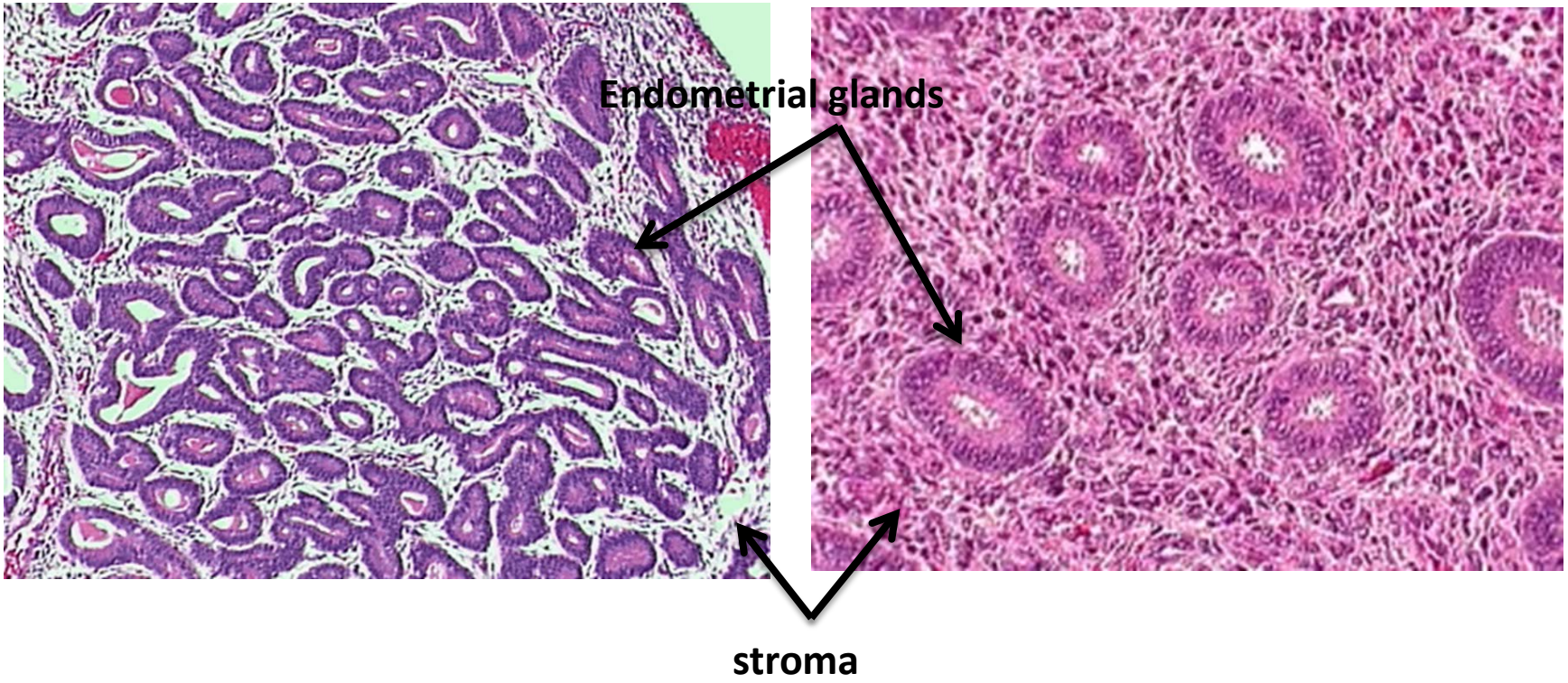



Endometrial hyperplasia
Normal endometrium
ADAM.

A: normal proliferative endometrium with discrete glands embedded in normal amount of stroma.
B: endometrial hyperplasia, clearly there is an increase in number of glands with very few amount of stroma in between, merge of some glands together with benign looking epithelia (case of typical hyperplasia).

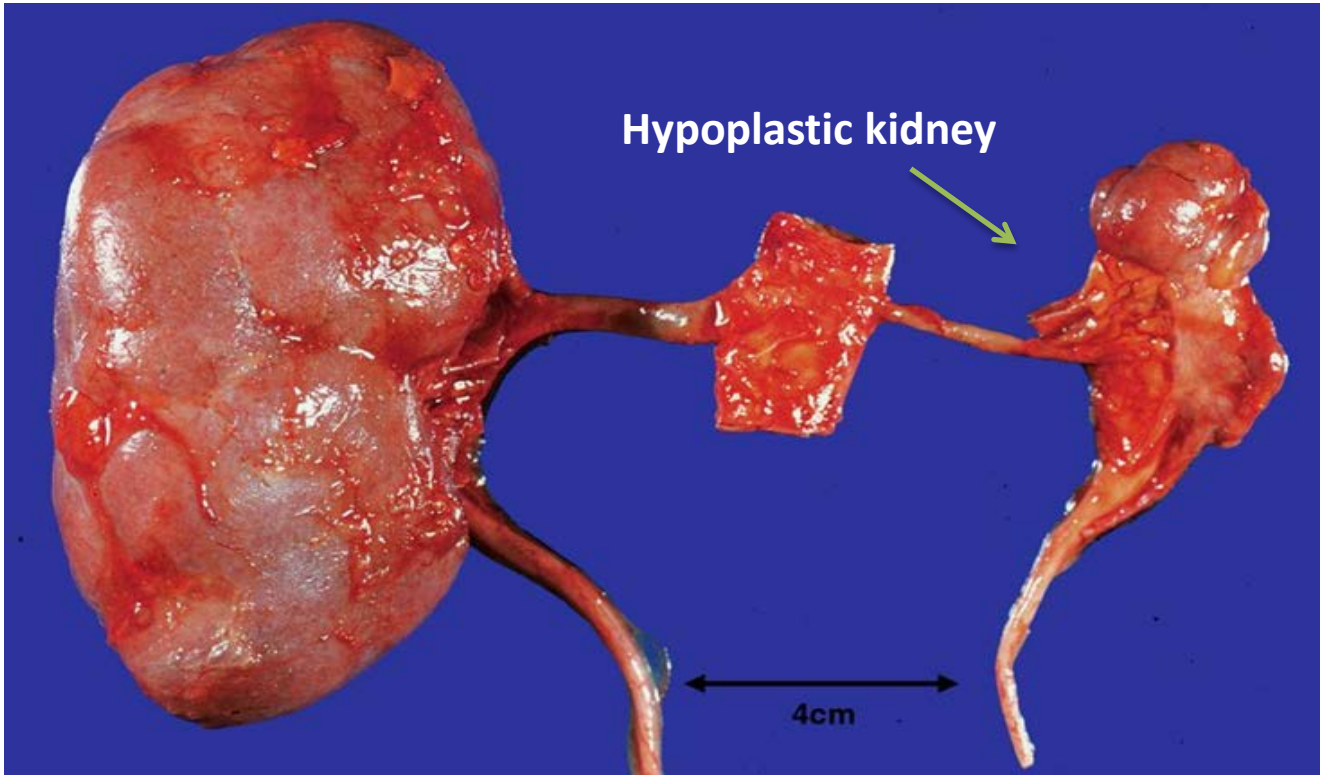
B. Endometrial hyperplasia

A. Normal endometrium



Hypoplasia:

- **Incomplete development of an organ so that it fails to reach adult size. It can occur at any organ in the body especially paired organs.**



Metaplasia

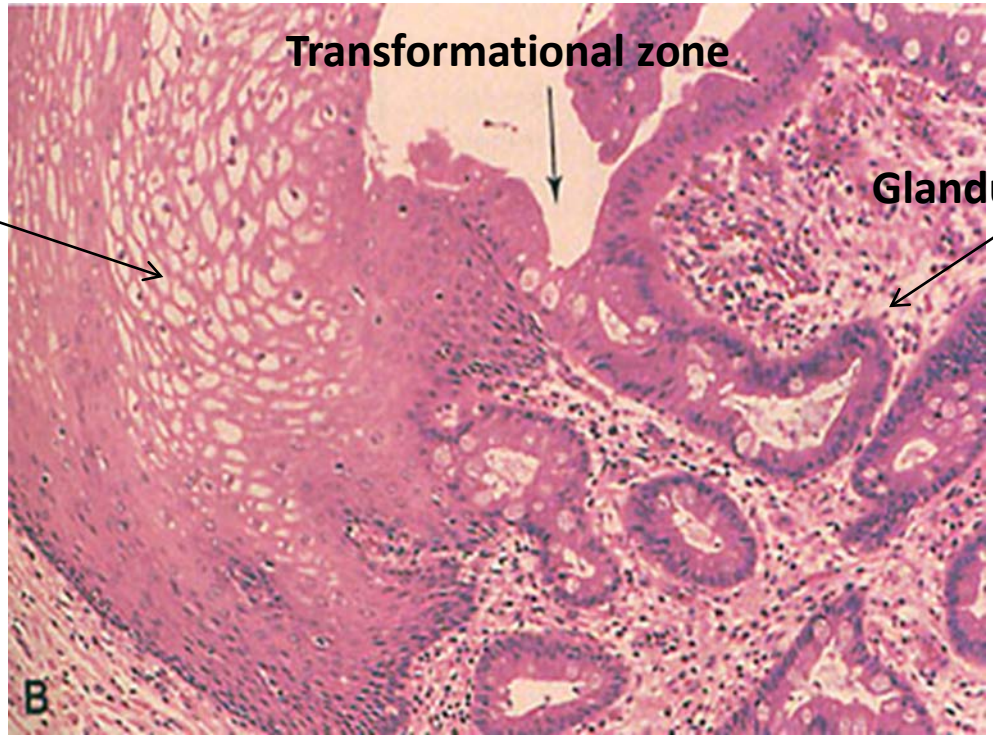
- **Change in which one ADULT cell type is replaced by another ADULT cell type**

- **Caused by: Chronic irritation (inflammation,cigarette smoke; calculi in ducts):**

- **Cervix- squamous epithelium of the endocervix replaces columnar (dysplasia and squamous CA may develop)**

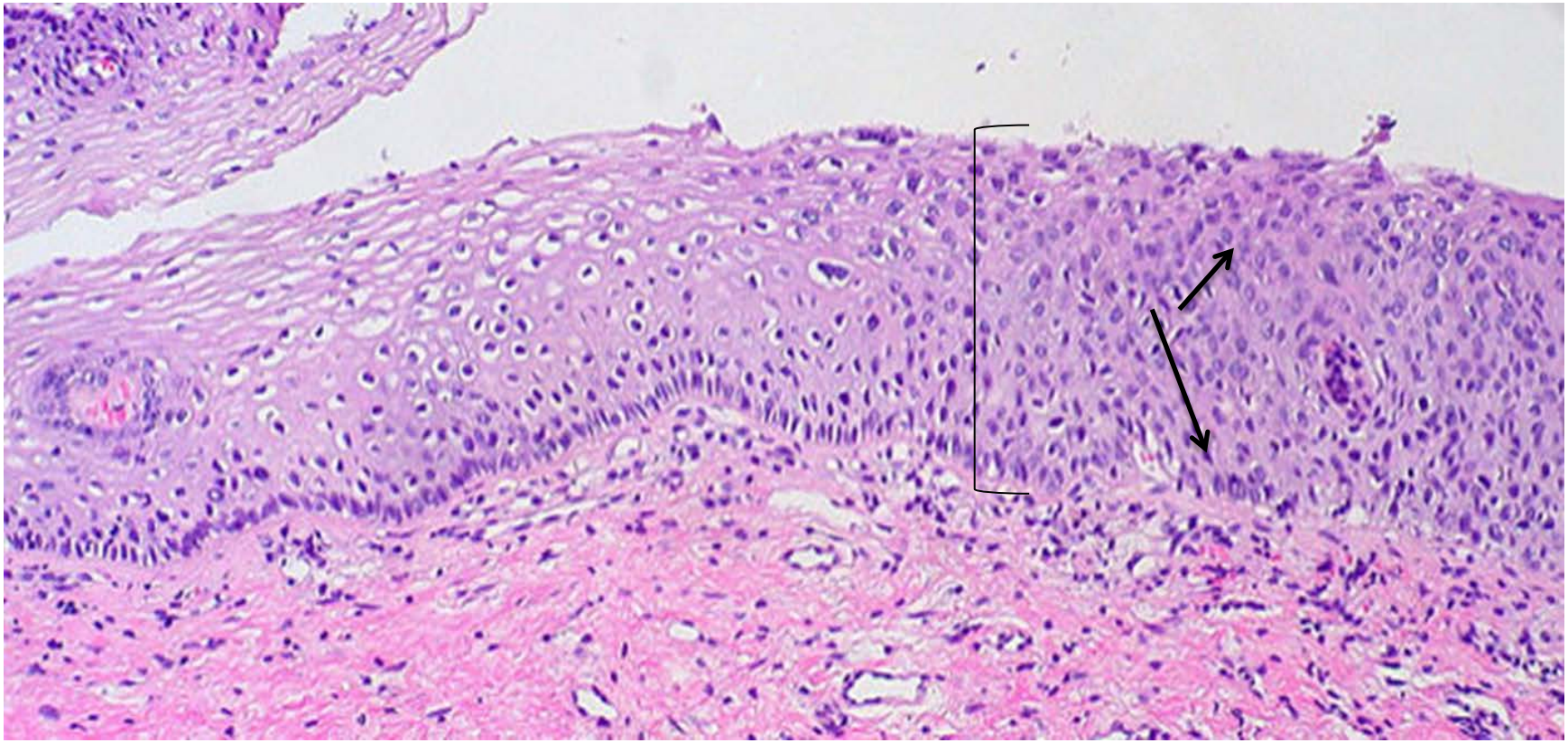
- **Barrett esophagus- gastric reflux results in columnar epithelium replacing squamous epithelium in the esophagus (dysplasia and adenocarcinoma may occur)**

Barret esophagus: **squamous epithelia replaced by glandular epithelia in the lower (abdominal) part of the esophagus due to chronic irritation by reflexed gastric juice**



Dysplasia:

- **Disturb organization and orientation of the cells**
- **Sever dysplasia equal intraepithelial neoplasia.**
- **Chronic irritation by radiation, inflammation considered the major cause.**



Cell injury

Occur in two situations:

i. **The limits of adaptive response are exceeded.**

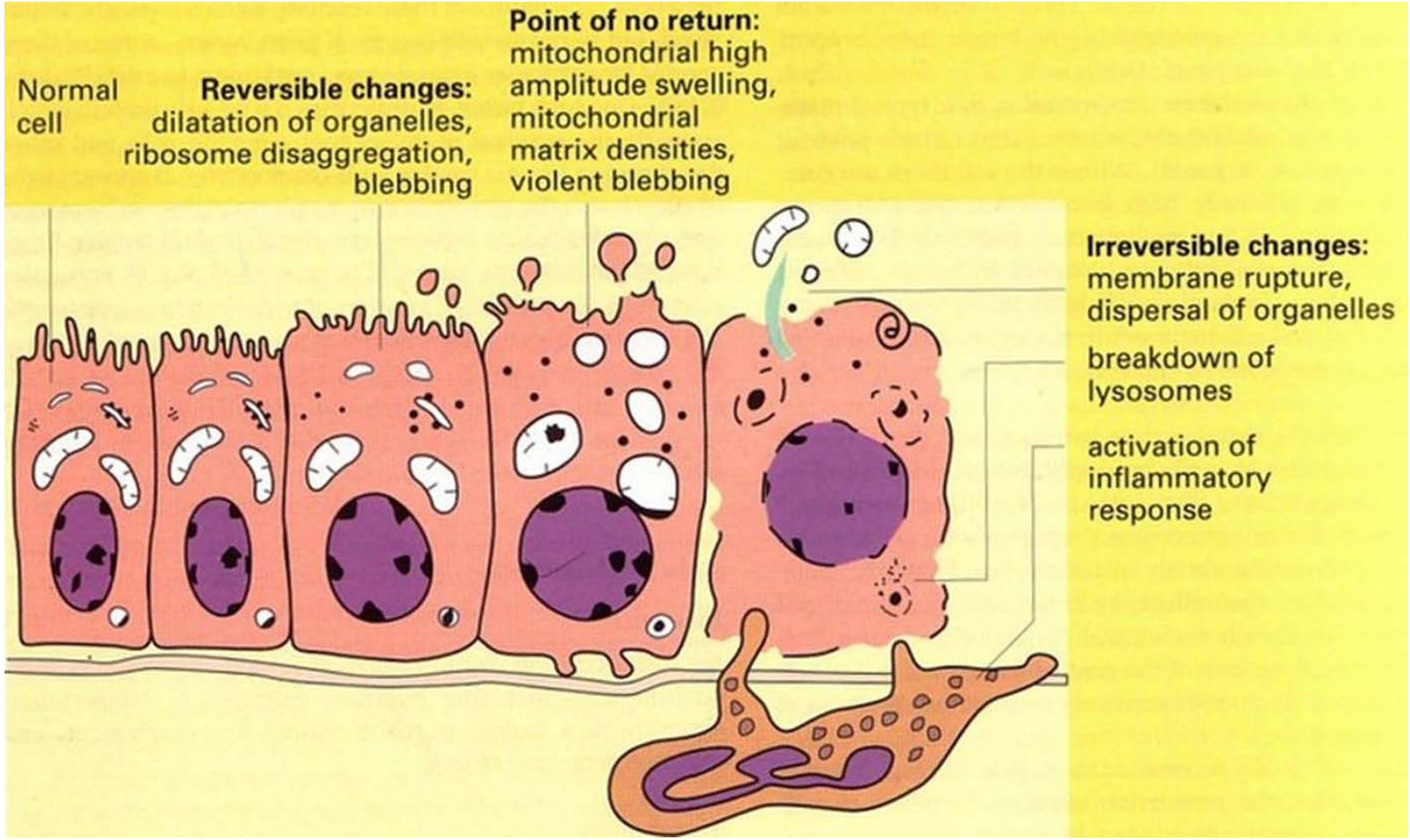
ii. **When there is no enough time for adaptive responses to take place as in sever injurious agent.**

Cell injury divided to:

1. Reversible cell injury: cellular changes will regress and disappear when the injurious agent is removed i.e. cells return to normal both morphologically and functionally e.g. cellular swelling (hydropic changes) and fatty changes.

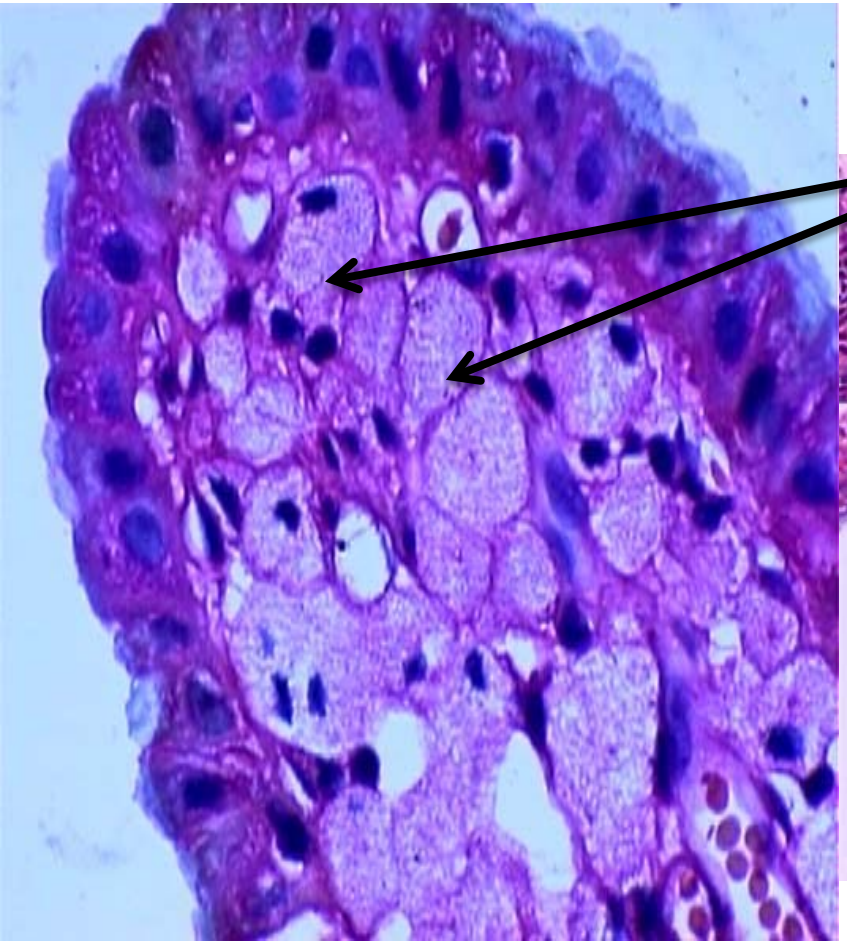
2. Irreversible cell injury (cell death): in which cell death is inevitable e.g. mitochondrial damage and autolysis by lysosomal enzymes.

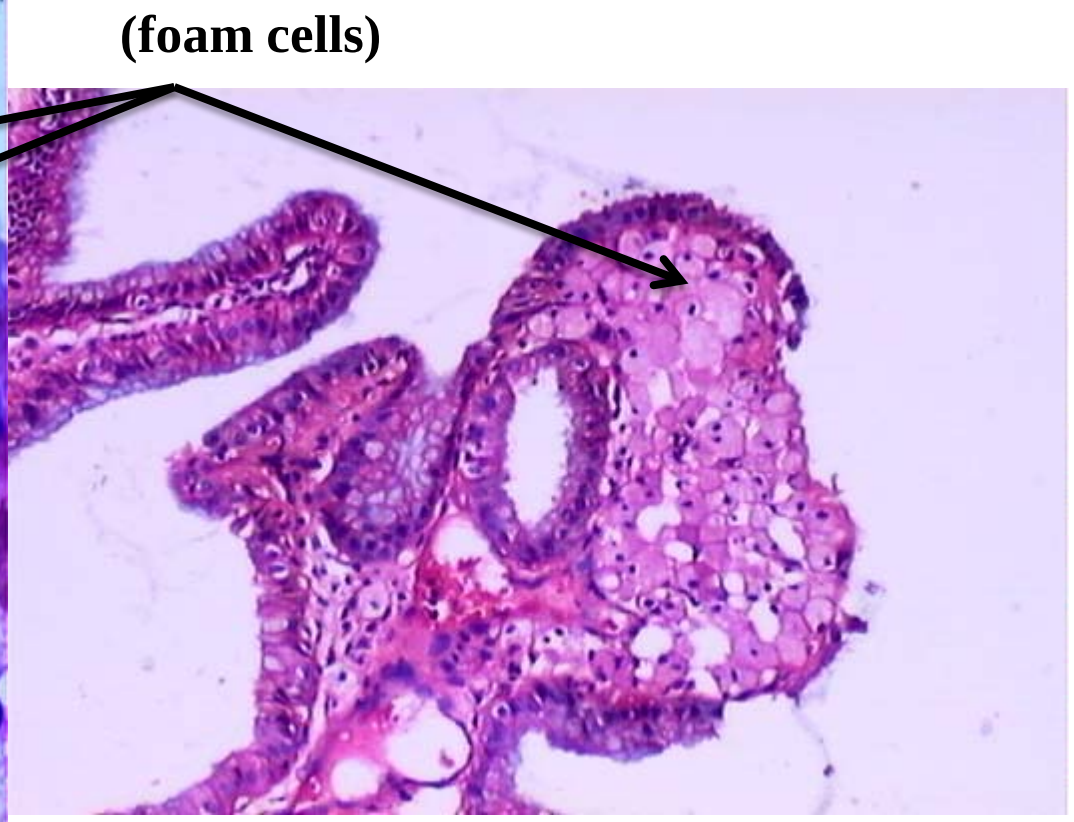
Cell injury


Normal cell
Reversible changes: dilatation of organelles, ribosome disaggregation, blebbing
Point of no return: mitochondrial high amplitude swelling, mitochondrial matrix densities, violent blebbing
Irreversible changes: membrane rupture, dispersal of organelles
breakdown of lysosomes
activation of inflammatory response

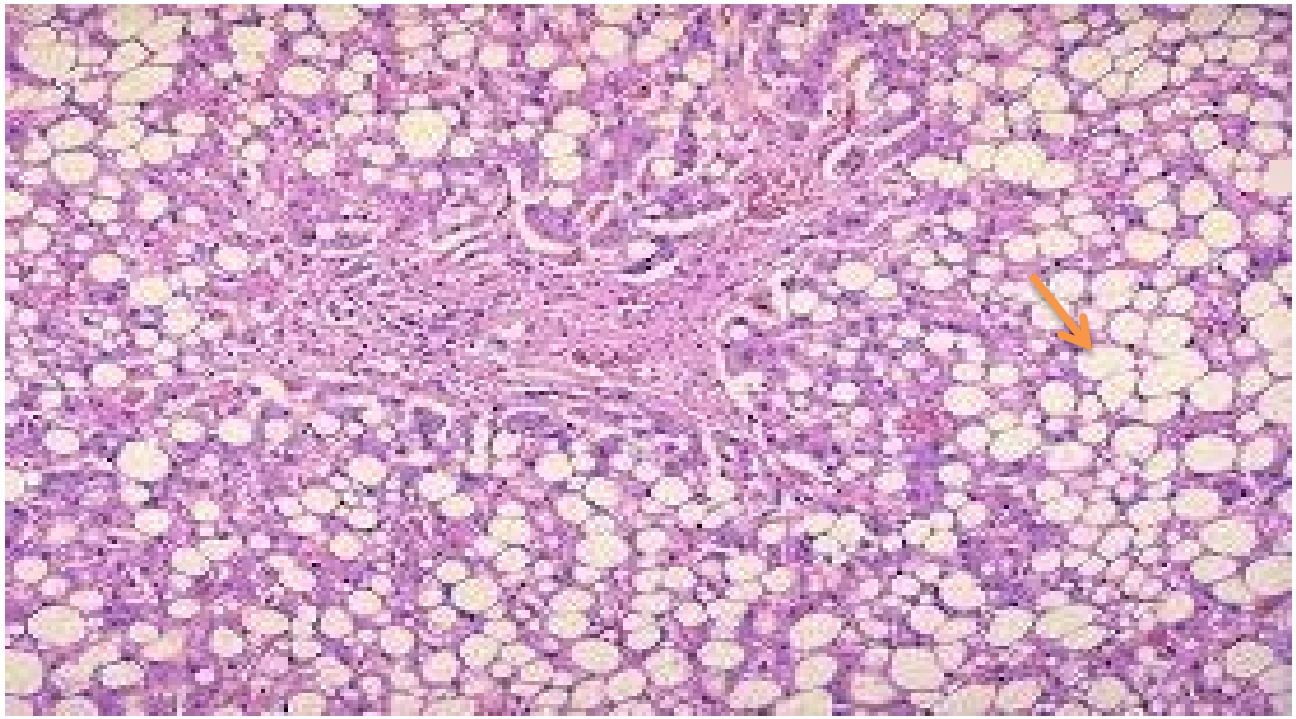
Fatty changes or accumulation

1. Cholesterol-laden macrophages (foam cells) from a focus of gallbladder cholesterolosis.





2. Liver fatty changes: Intracellular accumulations of fat in response to alcoholic hepatocellular injury. Here is fatty change of the liver due to deranged lipoprotein transport from alcoholism leads to accumulation of lipid in the cytoplasm of hepatocytes (arrow)



Cell death

1. Necrosis
2. Apoptosis


THANK YOU