Atrophy. Dystrophy.

II. practical training
2\textsuperscript{rd} year Dentistry

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Atrophy

Decrease in size of the cell or organ
Reduction in cell size and/or cell number, or both
Atrophic cells may have diminished function, but they are NOT death
However, atrophy may be mediated by apoptosis

**PHYSIOLOGICAL ATROPHY**
- Morphogenesis
- Physiological involution of organs (thymus)
- Ageing (brown atrophy)
PATHOLOGICAL ATROPHY

- Decreased function (muscle atrophy due to immobilisation)
- Loss of innervation
- Loss of blood supply or blood stagnation (epidermal atrophy due to tissue hypoxia)
- Pressure atrophy (decubitus)
- Lack of nutrition (cachexia)
- Lack of endocrine stimulation (menopause)
- Idiopathic (myopathies)
Hypoplasia
• Failure of the organ to attain its normal size

Agenesis (aplasia)
• Failure of development of the organ
• Renal agenesis
• Anencephaly – incompatible with life
Atrophy of the heart muscle
Atrophy of the liver
Dystrophy

Degeneration of tissue resulting from metabolic disorders and abnormal intracellular accumulation of metabolites

May be harmless or may cause varied degrees of injury
Localized in the cytoplasm, in the organelles (lysosomes), or in the nucleus

3 types of accumulated substances:
1) normal endogenous substances
2) abnormal endogenous substances
3) exogenous substances
Steatosis (fatty change)

*Abnormal intracellular accumulation of lipids*
May have or may not have effect on cellular function
Process is reversible
Most commonly seen in the liver and in the heart

**Causes:**

1) hypoxia („tiger heart“)
   LIPOMATOSIS – extracellular fat deposition
2) toxic (alcohol, mushroom, bacterial toxins)
3) lipidoses – congenital defects of enzymes of fat metabolism
   (Niemann-Pick...sphingomyelin)
4) starvation, diabetes
Fatty change of the liver

1. Fatty vacuoles in the cytoplasm
2. Nucleus of hepatocyte on periphery
3. Sinus
4. Normal hepatocytes
Lipomatosis of the heart muscle
Intracellular accumulation of proteins

**HYALINE**

- Large amount of protein is presented to the cells or the cells synthetize excessive amounts
- Usually not visible
- Microscopically, eosinophilic amorphous droplets, appears as inclusion
- In the kidneys (nephrotic syndromes), in the liver (Mallory bodies – „alcoholic hyaline“), in the brain (Lewy bodies, neurofibrillar tangles)
Lewy bodies

Neurofibrillary tangles
Extracellular accumulation of proteins

**AMYLOID**
- EXTRACELLULAR deposition of the fibrous protein aggregates
- Chemically differ but share some specific structural traits
- Grey-white colour, waxy appearance, elastic consistency
- Microscopically, eosinophilic amorphous substance
- Congo Red…stains red…shows green dichroism in polarized light
- Ultrastructurally, fibrillar bundles of the protein (β-sheets)
Types of amyloid:
1) AL amyloid (myeloma - Bence-Jones protein in the urine)
   Kidneys, vessels, myocardium, spleen, GIT, adrenal gland
2) AA amyloid (chronic inflammatory diseases, neoplasms)
   Adrenal glands, spleen, liver, lymph nodes, kidneys, bowel
3) AE amyloid (medullary carcinoma of the thyroid)
4) AS (senile) amyloid
   Myocardium
5) AD (dermal) amyloid

Clinical classification:
1) Primary amyloidosis (≈AL amyloidosis)
2) Secondary (reactive) amyloidosis (≈AA amyloidosis)
3) hemodialysis-associated amyloidosis
4) Hereditary amyloidosis - rare
Amyloidosis of the heart
Amyloidosis of the liver

1. Endothelial cells of sinusoids
2. Kupffer cells
3. Deposits of amyloid in perisinusoidal spaces
4. Atrophic hepatocytes
Amyloidosis of the liver (Congo Red)

1 Centrolobular area
2 Amyloid (Congo Red)
Follicular amyloidosis of the spleen („sago spleen“)
Follicular amyloidosis of the spleen („sago spleen“)
Amyloid nephropathy
Amyloid nephropathy (Congo Red, green dichroism)
ACCUMULATION OF MUCOID SUBSTANCES

- **Ganglion cyst** (swelling around joints and tendons in the hand or foot)
- **Myxoedema** – hyperthyroidism (pretibial myxoedema), hypothyroidism
- **Mucopolysaccharidoses** – congenital defects of enzymes

CYSTIC FIBROSIS (MUCOVISCIDOSIS)

- AR disorder. Defect of secretory process of exocrine glands
- Salty sweat
- Abnormally viscid mucus blocks the airways (secondary infections) and pancreatic ducts (malabsorption)
- Newborns: meconium ileus
**LOSS OF MUCOID SUBSTANCES**

- **Sclerosis**
  Stiffening of the tissue (atherosclerosis)

- **Fibrosis**
  Formation of excess fibrous tissue…stiffening of the tissue

 Used interchangeably.
Atherosclerosis
Intracellular accumulation of carbohydrates

**DIABETES MELLITUS**

*Impaired glucose homeostasis resulting from a relative or absolute insufficiency of insulin*

Hyperglycaemia, glycosuria
- Type I (IDDM) – juvenile, genetic predisposition
- Type II (NIDDM) – adult

**Complications:**
- Macroangiopathy – accelerated atherosclerosis, gangrene
- Microangiopathy – diabetic retinopathy, glomerulosclerosis
- Neuropathy – focal demyelination, diabetic polyneuropathy
- Increased susceptibility to infections
- Diabetic ketoacidosis
- Hyperosmolar nonketotic coma
Intracellular accumulation of carbohydrates (storage diseases)

**GLYKOGENOSIS**
- Congenital defects of enzymes of sugar metabolism
- Most frequent *Von Gierke* and *Pompe*

**FRUCTOSE INTOLERANCE**
- Liver

**GALACTOSAEMIA (MILK INTOLERANCE)**
- Liver
Renal cell carcinoma
Renal cell carcinoma