CELLULAR ADAPTATIONS

On exposure to stress for the sake of survival, the cells make adjustments with the changes in their environment (i.e.adapt) to the physiologic needs (physiologic adaptation) and to non-lethal pathologic injury (pathologic adaptation).
Such physiologic and pathologic adaptations occur by following processes,
Decreasing or increasing their size i.e. atrophy and hypertrophy respectively, or by increasing their number i.e. hyperplasia.
Changing the pathway of phenotypic differentiation of cells i.e. metaplasia and dysplasia.
In general, the adaptive responses are reversible on withdrawal of stimulus. However, if the irritant stimulus persists for long time, the cell may not be able to survive and may either die or progress further. e.g. cell death may occur in sustained atrophy; dysplasia may progress into carcinoma.

ATROPHY- Reduction of the number and size of parenchymal cells of an organ or its parts which was once normal is called atrophy.

MORPHOLOGIC FEATURES. Irrespective of the underlying cause for atrophy, the pathologic changes are similar. The organ is small, often shrunken. The cells become smaller in size but are not dead cells. Shrinkage in cell size is due to reduction in cell organelles, chiefly mitochondria, myofilaments and endoplasmic reticulum. There is often increase in the number of autophagic vacuoles containing cell debris .These autophagic vacuoles may persist to form 'residual bodies' in the cell cytoplasm e.g. lipofuscin pigment granules in Brown atrophy.

HYPERTROPHY- Hypertrophy is an increase in the size of parenchymal cells resulting in enlargement of the organ or tissue, without any change in the number of cells.

MORPHOLOGIC FEATURES. The affected organ is enlarged and heavy. For example, a hypertrophied heart of a patient with systemic hypertension may weigh 700-800 g as compared to average normal adult weight of 350 g. There is enlargement of muscle fibres as well as of nuclei.
At ultrastructural level, there is increased synthesis of DNA and RNA, increased protein synthesis and increased number of organelles like mitochondria, endoplasmic reticulum and myofibrils.

HYPERPLASIA- Hyperplasia is an increase in the number of parenchymal cells resulting in enlargement of the organ or tissue. Quite often, both hyperplasia and hypertrophy occur together.

METAPLASIA- Metaplasia is defined as a reversible change of one type of epithelial or mesenchymal adult cells to another type of adult epithelial or mesenchymal cells, usually in response to abnormal stimuli, and often reverts back to normal on removal of stimulus.
However, if the stimulus persists for a long time, epithelial metaplasia may transform into cancer.

DYSPLASIA- Dysplasia means 'disordered cellular development', often accompanied with metaplasia and hyperplasia, it is therefore also referred to as atypical hyperplasia. Dysplasia occurs most often in epithelial cells.

CELL SWELLING

Reversible injury
Hydropic change (cloudy swelling / vacuolar degeneration):

- It means accumulation of water within the cytoplasm of the cell.
- It is the commonest and earliest form of cell injury from almost all causes.
- The causes of it include acute and subacute cell injury.
- Impaired regulation of Na and K at the level of cell membrane
- Intracellular accumulation of Na and escape of K
- Rapid flow of water into the cell (to maintain the iso-osmotic condition)
- Cellular swelling

APOPTOSIS
Apoptosis is a form of 'coordinated and internally programmed cell death' having significance in a variety of
physiologic and pathologic conditions .

NECROSIS
Necrosis is defined as a localised area of death of tissue followed by degradation of tissue by hydrolytic enzymes
liberated from dead cells; it is invariably accompanied by inflammatory reaction.

Types of Necrosis
Morphologically, there are five types of necrosis: coagulative, liquefaction (colliquative), caseous, fat, and fibrinoid necrosis.
1. COAGULATIVE NECROSIS.
This is the most common type of necrosis caused by irreversible focal injury, mostly
from sudden cessation of blood flow (ischaemia), and less often from bacterial and chemical agents. The organs
commonly affected are the heart, kidney, and spleen.

2. LIQUEFACTION (COLLIQUATIVE) NECROSIS.

Liquefaction or colliquative necrosis occurs commonly due to ischaemic injury and bacterial or fungal infections.
It occurs due to degradation of tissue by the action of powerful hydrolytic enzymes. The common examples are infarct brain and abscess cavity.

3. CASEOUS NECROSIS.
Caseous necrosis is found in the centre of foci of tuberculous infections. It combines features of both coagulative and liquefactive necrosis.

4. FAT NECROSIS.
Fat necrosis is a special form of cell death occurring at two anatomically different locations but morphologically similar lesions. These are: following acute pancreatic necrosis, and traumatic fat necrosis commonly in breast.

5. FIBRINOID NECROSIS.
Fibrinoid necrosis is characterised by deposition of fibrin-like material which has the staining properties of fibrin.
It is encountered in various examples of immunologic tissue injury (e.g. in immune complex vasculitis, autoimmune diseases, Arthus reaction etc), arterioles in hypertension, peptic ulcer etc.