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3rd Edition

Textbook of PATHOLOGY & GENETICS for NURSES

Semester-III & IV



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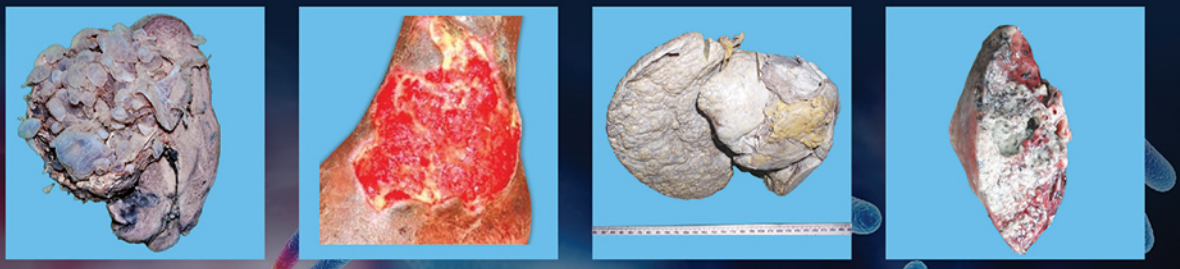

Ramadas Nayak
Sharada Rai
Rakshatha Nayak

Foreword
AR Raghupathy



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CHAPTER

2 Cellular Adaptations

CHAPTER OUTLINE

- Hypertrophy
- Hyperplasia
- Atrophy
- Metaplasia
- Dysplasia

Cells constantly adjust to maintain their health and function, especially when faced with changes or stressors. This adaptation process enables cells to survive in challenging conditions, balancing between a normal and damaged state. However, adaptations have limits; if the stress becomes too severe or prolonged, cells can eventually become injured.

Cellular adaptations: When exposed to unusual or potentially harmful conditions, cells can change to survive. This new steady state, or adaptation, allows them to keep working even in an abnormal environment. These adaptations are reversible changes that affect a cell's size, number, type, activity, or function. Essentially, these changes are the cell's way of coping with ongoing stress.

Types of Cellular Stress

- **Physiologic stress:** Happens with normal body processes. For example, hormone changes during pregnancy cause the uterus and breasts to enlarge.
- **Pathologic stress:** Occurs when cells need to adapt to avoid damage, often with some loss of normal function. An example is the changes in the bronchial lining of a smoker, which are an effort to protect the tissue.

Types of cellular adaptations (Fig. 2.1): Cells adapt to stress in four ways, each with a different mechanism. These are (i) hypertrophy, (ii) hyperplasia, (iii) atrophy, and (iv) metaplasia.

HYPERTROPHY

Definition: Hypertrophy is **the increase in an organ or tissue size due to the increase in individual parenchymal cells' size (enlargement)**. This adaptation does not produce new cells but makes existing cells larger.

Causes of hypertrophy: Hypertrophy is caused by two main factors:

1. **Increased workload or demand:** When cells need to work harder, they enlarge to handle the extra load.
2. **Hormonal stimulation:** Hormones can stimulate cells to increase in size to perform specific functions.

If the underlying cause of hypertrophy is not resolved, this growth can eventually lead to cell injury, especially in organs like the heart.

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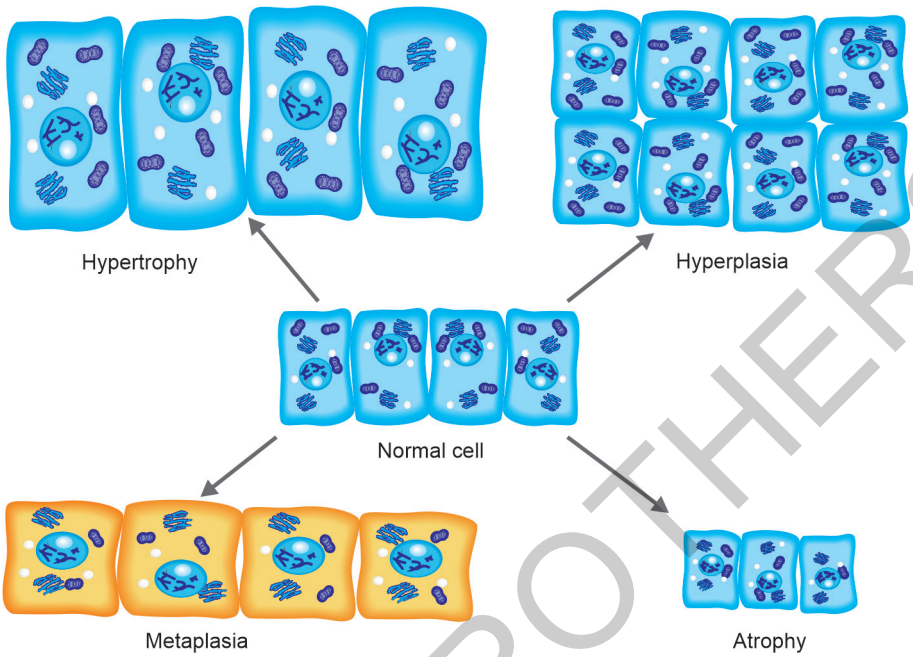

Fig. 2.1: Different types of cellular adaptations.

Types and Examples of Hypertrophy

Hypertrophy develops in organs composed of nondividing cells, such as muscles, such as cardiac and skeletal muscles. Hyperplasia can be physiological or pathological.

Physiological Hypertrophy

This is a normal, healthy response to increased functional demand or hormonal influence. Examples are as follows: -

- **Hypertrophy of skeletal muscle:** Bulging of skeletal muscles of bodybuilders, athletes, or manual laborers (e.g., engaged in "pumping iron"). The muscles grow larger in response to physical training or heavy work.
- **Hypertrophy of smooth muscle:** The smooth muscles of the **uterus grow during pregnancy in response to a hormone called estrogen.**

Pathological Hypertrophy

This occurs in response to disease or abnormal stress, often leading to issues over time. Examples are:

- **Cardiac (heart) hypertrophy:** The heart muscle, especially the left ventricle, enlarges (left **ventricular hypertrophy)** to pump against high blood pressure or a damaged heart valve (aortic stenosis, mitral incompetence). Although the heart initially adapts to improve blood flow, this extra strain can eventually lead to heart failure.
- **Bladder muscle hypertrophy:** This occurs when the bladder muscle thickens in response to an obstruction, such as an enlarged prostate that restricts urine flow from the urinary bladder.
- **Stomach muscle hypertrophy:** The stomach's muscular layer may thicken in response to a narrowed pylorus (pyloric stenosis).

Morphological Changes

- **Gross appearance (Figs. 2.2A and B):** The affected organ appears larger than usual.
- **Microscopic appearance:** Cells and their nuclei are visibly larger than normal (**Fig. 2.1**), indicating increased cellular growth.

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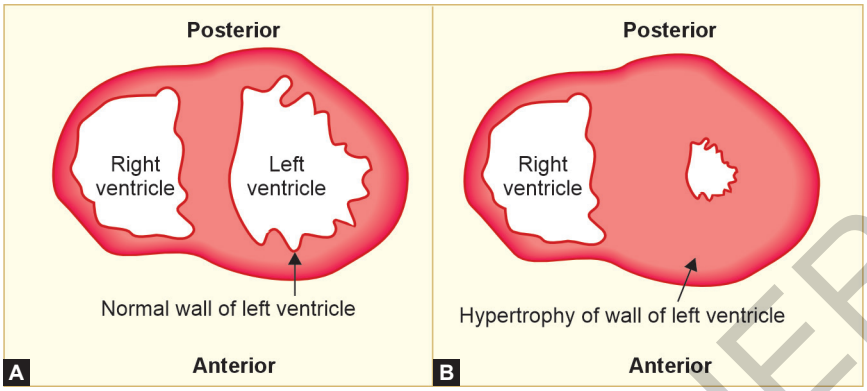

Figs. 2.2A and B: (A) Transverse section of normal heart; (B) Transverse section of heart with hypertrophy of left ventricle characterized by the thickening of the left ventricle wall.

Consequences: Hypertrophy is a critical cellular adaptation that allows tissues to handle increased demand or respond to hormones. However, in situations of chronic stress (like high blood pressure), hypertrophy can progress to tissue damage.

HYPERPLASIA

Definition: Hyperplasia is an **increase in the number of functional (parenchymal) cells** in a tissue or organ, **causing the organ or tissue to increase in size**.

Causes of hyperplasia: Not all cells can undergo hyperplasia. Only cells that have the ability to divide and regenerate, such as:

- **Labile cells** (e.g., skin cells, cells in the lining of mucous membranes, bone marrow cells, and lymph node cells).
- **Stable cells** (e.g., liver, and thyroid cells) can undergo hyperplasia.

However, **permanent cells** like **neurons** and **cardiac muscle cells** lack this capability and cannot multiply.

Hyperplasia will only continue as long as the stimulus causing it remains. Unlike **neoplasia** (uncontrolled, abnormal cell growth), hyperplasia is a regulated process and stops when the stimulus is removed.

Types and Examples of Hyperplasia

Hyperplasia can be physiological or pathological. It can be caused by hormonal stimulation or a compensatory process.

Physiological Hyperplasia

This is a normal, healthy response to hormonal or physical needs:

- **Hormone-induced hyperplasia:** Examples are as follows:
  - **During puberty, pregnancy, and lactation**, the glandular cells of the **breast** increase in number.
  - During pregnancy, the **uterus** grows larger from the hormone estrogen, which also involves hypertrophy (increase in cell size).
- **Compensatory hyperplasia:** This occurs to compensate for lost tissue. For example, when part of the liver is removed for a liver transplant, the remaining liver cells multiply to restore the organ's size.
- **Demand-induced hyperplasia:** The body can create more cells to meet increased physiological needs:

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- At high altitudes, where oxygen is low, bone marrow produces more red blood cell precursors, increasing red blood cells (a condition called secondary polycythemia).
- In cases of chronic blood loss, like excessive menstrual bleeding, the bone marrow increases red blood cell production.

❖ **Immune response hyperplasia:** This occurs as a response to infections. For example, swollen tonsils and lymph nodes in a throat infection (streptococcal pharyngitis) due to an increase in immune cells

Pathological Hyperplasia

This occurs in response to abnormal stimuli, often from excessive hormone production or chronic irritation:

❖ **Excess hormone stimulation:** Examples are as follows:
- **Endometrial hyperplasia:** Excess estrogen can cause an abnormal thickening of the endometrium (lining of the uterus), which, if persistent, can increase the risk of endometrial cancer.
- **Benign prostatic hyperplasia (BPH) (Figs. 2.3 and B):** Androgens (male hormones) can cause an enlarged prostate in men.
- **Gynecomastia:** Excess estrogen in men (due to estrogen therapy for prostate cancer or liver disease) can cause breast enlargement.

❖ **Chronic irritation or injury:** Long-term irritation or inflammation can trigger hyperplasia, especially in areas like the skin or oral mucosa.

❖ **Viral infections:** Certain viruses, like the human papillomavirus (HPV), can cause hyperplasia of skin or mucous membranes, leading to warts or other lesions.

Note: Pathological hyperplasia can increase the risk of cancer development over time, as with endometrial hyperplasia leading to endometrial cancer if untreated.

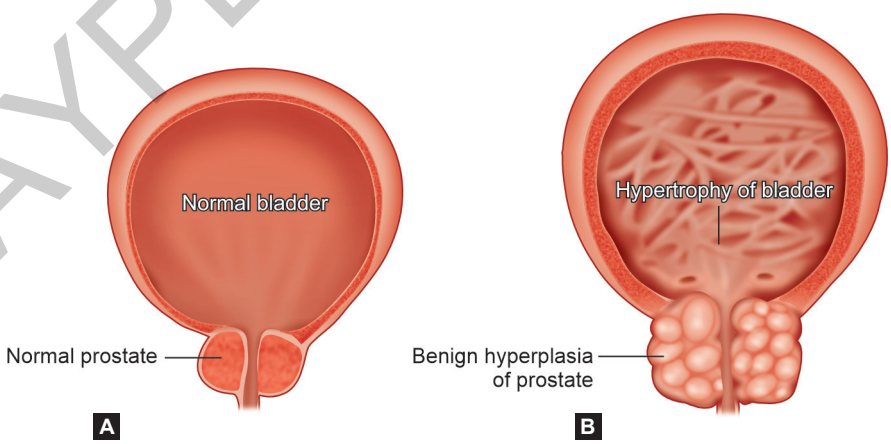
Morphology of Hyperplasia

❖ **Gross appearance:** The affected organ or tissue appears larger than usual.

❖ **Microscopic appearance:** There is a noticeable increase in the number of cells (**Fig. 2.1**), often with more cells in the process of dividing (mitosis).

ATROPHY

Definition: Atrophy is the **reduction in an organ or tissue size** due to a decrease in both the number and size of its functional



Figs. 2.3A and B: Cut a section of the prostate along with the urinary bladder. (A) Normal prostate; (B) Benign hyperplasia of the prostate leading to enlargement of the prostate.

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(parenchymal) cells. This can cause the affected tissue or organ to shrink or lose mass.

Causes of Atrophy

Atrophy can be physiological (normal in certain stages of life) or pathological (resulting from illness or injury).

Physiological Atrophy

Occurs naturally during development or aging:

- **During fetal development:** Certain embryonic structures, like the thyroglossal duct, atrophy (shrink and disappear) as the fetus grows.
- **During adult Life:** Some organs shrink over time due to hormonal changes or aging. Examples:
  - **Thymus gland:** Shrinks after childhood.
  - **Brain, heart, and reproductive organs:** May undergo gradual atrophy as part of aging.
  - **Uterus:** Shrinks after menopause due to reduced hormone levels.

Pathological Atrophy

This can be local (affecting a specific area) or generalized (affecting the whole body).

Local causes of pathological atrophy

- **Disuse atrophy (decreased workload):** Caused by reduced use or inactivity, such as muscle shrinking when a limb is cast or after prolonged bed rest.
- **Denervation (loss of innervations) atrophy: Loss of nerve supply leads to muscle atrophy. An example is muscle shrinkage** in conditions like **poliomyelitis (polio)**.
- **Ischemic (diminished blood supply) atrophy:** Reduced blood supply causes tissue to shrink. Example: **Brain atrophy** due to reduced blood flow from narrowed arteries.
- **Pressure atrophy:** Persistent pressure on tissues can lead to atrophy. An example is kidney atrophy due to pressure from a blockage (hydronephrosis).
- **Loss of hormone signals:** Tissues that depend on hormones shrink if the hormone signal is lost. Examples: Atrophy of breast and uterine tissue after menopause.

Generalized causes of pathological atrophy

Starvation (inadequate nutrition) **or** protein-calorie **malnutrition:** Severe lack of nutrition leads to atrophy of muscles and fat tissue, resulting in wasting.

Morphological Changes in Atrophy

- **Gross changes:** The affected organ appears smaller and often shrunken.
- **Microscopic changes:** Cells are smaller (**Fig. 2.1**) because of the reduction in cell organelles (e.g., mitochondria, myofilaments, and endoplasmic reticulum).

Related Terms

- **Hypoplasia:** A condition where an organ or tissue is underdeveloped from birth, smaller than normal.
- **Aplasia:** A severe developmental failure where an organ or tissue does not fully develop, leaving only rudimentary or minimal tissue.

METAPLASIA

Definition: Metaplasia is a reversible change in which **one type of mature (adult) cell in a tissue or organ** is **replaced by another adult cell type**.

Causes of Metaplasia

This change is usually an adaptive response to long-term irritation or stress. If the irritating factor is removed, the cells may revert to their original type. For example, if a person quits smoking, the metaplastic cells in their airways can eventually return to their original state. Metaplasia generally occurs as a protective mechanism. Some cell types are better

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at handling certain stresses or irritants than others. When exposed to chronic irritation, tissues may replace sensitive cells with a cell type that can better tolerate the new environment.

Types of Metaplasia

Metaplasia is classified into two main types: (i) epithelial and (ii) connective tissue metaplasia.

Epithelial Metaplasia

Epithelial metaplasia is a change in lining cells, such as skin or mucous membranes. It is the most common type of metaplasia. There are two types: **squamous** and **columnar** metaplasia.

Squamous metaplasia

In this type, glandular or specialized epithelium (lining) cells are replaced by squamous (flat) cells. This often occurs due to chronic irritation. Examples of squamous metaplasia are:

- **Respiratory tract:** The normal ciliated columnar cells in the airways can turn into squamous cells **(Fig. 2.4)** due to irritants like smoking or vitamin A deficiency. While squamous cells are tougher, they lack the protective functions of the original cells (like mucus production), which can lead to infection risks.
- **Cervix:** Chronic infection in the cervical lining (columnar cells) can lead to squamous metaplasia.

Columnar metaplasia

In this type, columnar cells are replaced by squamous cells, which may be better suited to an acidic or otherwise hostile environment. Examples of columnar metaplasia**:**

- **Barrett esophagus:** Chronic acid reflux can cause the squamous cells of the esophagus to change into columnar cells, which are more resistant to stomach acid.
- **Intestinal metaplasia:** Sometimes, the stomach lining cells may change to resemble cells found in the intestine. This can be a response to chronic irritation.

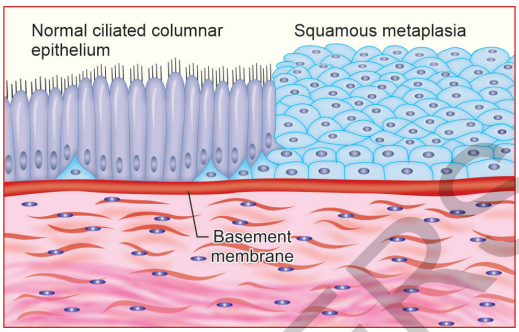

Fig. 2.4: Squamous metaplasia in which columnar epithelium (left) is replaced by squamous epithelium (right).

Significance of epithelial metaplasia: While metaplasia is a protective response, prolonged irritation can lead to dysplasia (abnormal cell growth) and may increase the risk of cancer. For example, Barrett's esophagus can progress to esophageal adenocarcinoma if the reflux issue is not treated.

Mesenchymal (Connective Tissue) Metaplasia

In this type, one kind of connective tissue changes into another. This type of metaplasia is less common than epithelial metaplasia.

- **Osseous (bone) metaplasia:** Formation of bone tissue in places it does not normally exist, such as in muscle or scar tissue. Example: myositis ossificans, where bone forms in muscle after intramuscular bleeding following an injury or trauma.

DYSPLASIA

Definition: Dysplasia is an abnormal and disorganized growth of cells.

Common Sites of Dysplasia

- **Squamous epithelium**: This is common in areas like the uterine cervix, respiratory tract, and skin, especially where there is frequent irritation (like in smokers).

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- **Columnar epithelium:** Found in the lining of organs like the colon (in ulcerative colitis) and esophagus (in Barrett esophagus).

Causes: Chronic irritation or inflammation, like smoking or infection with HPV (human papillomavirus), can lead to dysplasia. Removing the cause, such as quitting smoking, may allow dysplasia to regress and disappear.

Significance: Dysplasia involves irregular cell development and can sometimes be a warning sign for cancer. However, dysplastic cells are not fully cancerous and can return to normal if the irritant causing the abnormal growth is removed.

Features of Dysplasia (Fig. 2.5)

The **cells that show cytological features of malignancy** are called dysplastic cells, and the term dysplasia is used to describe these changes. It literally means disordered growth. The changes of dysplasia include:

- **Loss of uniformity:** Cells lose their usual shape and size, a condition known as *pleomorphism*.
- **Abnormal nuclei:** The nuclei (cell centers) become **larger, darker (hyperchromatic),** and more variable in size.
- **Increased nuclear-to-cytoplasmic ratio:** The nucleus is larger than that in normal cells. This increases the nuclear-cytoplasmic ratio.
- **High mitotic activity:** There are more cell divisions than normal, and these divisions can occur at all layers of the tissue, not just the basal layer.
- **Loss of polarity:** Cells are arranged haphazardly, lacking the usual organized layer structure.

Classification (Types) and Progression

Depending on the thickness of epithelium involved by the dysplastic cell, it may be classified as:

- **Mild-to-moderate dysplasia:** Involves part of the tissue thickness and can often be reversed if the irritant is removed. Thus, **dysplasia need not progress to cancer**. It may act as a precursor to cancer but does not always lead to it.
- **Severe dysplasia:** When dysplastic changes affect the entire tissue thickness, it is known as *carcinoma in situ*, a preinvasive stage of cancer.
- **Once the tumor cells breach the basement membrane,** the **tumor is said to be invasive,** and carcinoma in situ may take years to become invasive. Most in situ tumors, with time, penetrate the basement membrane and invade the subepithelial stroma.

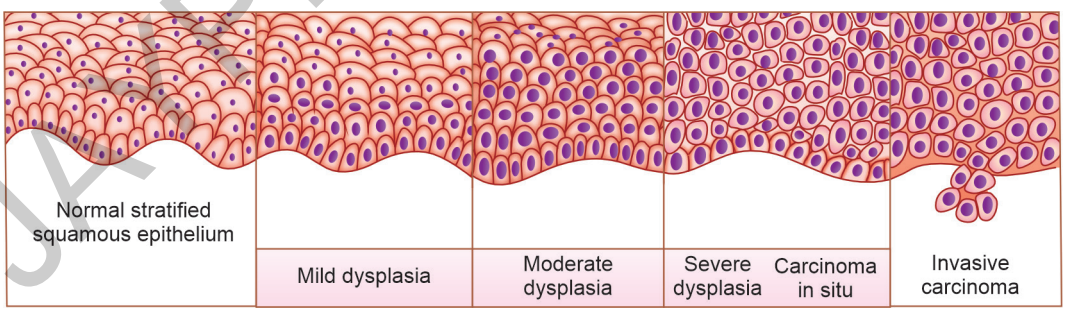

Fig. 2.5: Diagrammatic appearance of dysplasia and carcinoma in situ.

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SELF-ASSESSMENT EXERCISES

Short Notes

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

Multiple Choice Questions

1. **An increase in cell size refers to:**
   a. Hyperplasia b. Hypertrophy
   c. Atrophy d. Dystrophy
2. **The organ which can show both hyperplasia and hypertrophy is:**
   a. Liver b. Kidney
   c. Uterus d. Heart
3. **An example of connective tissue metaplasia is:**
   a. Myositis ossificans
   b. Keloid
   c. Hypertrophied scar
   d. Intestinal metaplasia
4. **Barrett esophagus is an example for:**
   a. Metaplasia b. Atrophy
   c. Hyperplasia d. Hypertrophy
5. **Replacement of one adult cell type by another adult cell type is known as:**
   a. Hypertrophy
   b. Hyperplasia
   c. Metaplasia
   d. Dysplasia
6. **An increase in the number of cells in an organ or tissue is termed as:**
   a. Hypertrophy b. Hyperplasia
   c. Metaplasia d. Anaplasia
7. **Which of the following is an example of metaplasia?**
   a. Enlargement of the uterus during pregnancy.
   b. Replacement of normal lung epithelium with squamous epithelium in smokers.
   c. Shrinkage of brain tissue in Alzheimer's disease.
   d. Increase in the number of red blood cells at high altitudes.
8. **Which cellular adaptation is characterized by decreased cell size due to reduced workload or adverse conditions?**
   a. Hyperplasia b. Hypertrophy
   c. Atrophy d. Dysplasia
9. **Dysplasia is often considered a precursor to which of the following conditions?**
   a. Hypertrophy
   b. Cancer
   c. Atrophy
   d. Apoptosis
10. **All the following are types of cellular adaptation, *except*:**
    a. Hyperplasia b. Hypertrophy
    c. Metaplasia d. Dysplasia
11. **Which type of cellular adaptation is typically reversible if the stressor is removed?**
    a. Atrophy b. Hypertrophy
    c. Hyperplasia d. All the above
12. **Which of the following best describes cellular metaplasia?**
    a. Reversible change in which another adult cell type replaces one adult cell type.
    b. Permanent increase in the number of cells in a tissue.
    c. Decrease in cell size due to a reduction in workload.
    d. An abnormal and potentially precancerous growth in cells.
13. **The nature of the lesion in Barrett esophagus is:**
    a. Congenital anomaly
    b. Inflammatory disease
    c. Metaplastic process
    d. Neoplastic lesion

ANSWERS

1. b	2. c	3. a	4. a	5. c	6. b	7. b	8. c	9. b	10. d
11. d	12. a	13. c							

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- Divided into six sections as per the syllabus for III and IV semesters, namely General Pathology (Chapters 1 to 6), Systemic Pathology—Part I (Chapters 7 to 12), Clinical Pathology—Hematology (Chapters 13 to 16), Systemic Pathology—Part II (Chapters 17 to 21), Clinical Pathology—Laboratory Analysis (Chapters 22 to 25), and Genetics (Chapters 26 to 32)
- Includes a chapter outline at the beginning of each chapter
- Concise text in bullet for easy review and recollection
- Highlights essential points to be remembered during the examination in bold letters
- Provides self-assessment exercises at the end of each chapter: Essay questions, Short answer questions, and Multiple Choice Questions (MCQs)
- Glossary for ready reference

Ramadas Nayak MBBS MD graduated from Mysore Medical College, Mysuru, Karnataka, India, and completed his postgraduate training in Pathology at Kasturba Medical College, Mangaluru, Karnataka, India. He currently serves as a Professor in the Department of Pathology at Srinivas Institute of Medical Sciences & Research, Mukka, Surathkal, Karnataka, India.

Over an illustrious teaching career spanning 45 years, he has held key academic leadership roles, including Head of the Department of Pathology at Kasturba Medical College (Manipal Academy of Higher Education, Manipal, Karnataka, India) and at Yenepoya Medical College, Mangaluru. He has authored 84 scientific papers in national and international journals and has served as an examiner for undergraduate and postgraduate pathology examinations at several universities. He has also contributed substantially to major research initiatives, notably serving as Project Officer for the Atlas of Cancer in India, a project supported by the Indian Council of Medical Research (ICMR) and the World Health Organization (WHO). Additionally, he served on the Editorial Committee for the South Asia Edition of the *British Medical Journal*. He is the author of 10 notable books, including *Essentials in Hematology and Clinical Pathology* (2nd edition), *Rapid Review of Hematology*, *Exam Preparatory Manual for Undergraduates: Pathology for Dental Students*, *Textbook of Pathology for BPT Students*, *Textbook of Pathology for Allied Health Sciences*, *Histopathology Techniques and their Management*, *Manual of Transfusion Medicine, Review of Postgraduate Pathology-General Pathology*, *Review of Postgraduate Pathology-Systemic Pathology* and *Rapid Review, MCQs, AETCOM, and OSPE in Pathology*. His excellence in teaching has been recognized with multiple awards, including the *Good Teacher Award* twice and the *Best Audio-visual Award* eight times during his tenure at Kasturba Medical College. He also received the *Teacher of the Year Award 2022* at Yenepoya Medical College.

Sharada Rai MBBS MD is a Professor in the Department of Pathology at Kasturba Medical College, Mangaluru, affiliated to Manipal Academy of Higher Education, Manipal, Karnataka, India. She completed her postgraduate studies at Kasturba Medical College. She is a popular hematologist with a vast experience in clinical pathology. She has been teaching undergraduate and postgraduate students for the past 28 years. She has many publications in national and international journals. She has guided several short-term projects funded by the Indian Council of Medical Research (ICMR). She is an examiner for both undergraduate and postgraduate examinations in many universities. She is also a PSG-FAIMER fellow.

Rakshatha Nayak MBBS MD DNB is an accomplished professional who graduated from KS Hegde Medical Academy, Constituent of Nitte (Deemed to be University), Mangaluru, Karnataka, India. She pursued her postgraduate studies in Pathology at Yenepoya Medical College, Yenepoya (Deemed to be University), Mangaluru, Karnataka, India. Currently, she is an Associate Professor in the Department of Pathology at Kasturba Medical College, Mangaluru, under Manipal Academy of Higher Education, Karnataka, India. Rakshatha's educational journey includes serving as a tutor in the Department of Forensic Medicine at Kasturba Medical College, Manipal Academy of Higher Education (Manipal), Mangaluru, from 2014 to 2015. She also served as a tutor in the Department of Pathology at Yenepoya Medical College, Mangaluru. Her commitment to continuous learning is evident through her completion of the ISN-ANIO Clinical Nephropathology Certificate Programme, organized by the International Society of Nephropathology in 2018. Furthermore, she successfully passed the Diplomate of the National Board examination in Pathology that same year. Rakshatha's contributions to academia and research are notable. She has authored and presented several scientific papers in esteemed national and international journals and conferences. Her dedication to excellence has been recognized through awards, including the Short-Term Studentship (STS) awarded by the Indian Council of Medical Research (ICMR) in 2011. Throughout her academic journey, Rakshatha has demonstrated outstanding performance. She secured the highest score in Pathology at KS Hegde Medical Academy. She ranked 3rd in the Phase-II MBBS (Pathology) Examination held in 2010 by the Rajiv Gandhi University of Health Sciences (RGUHS), Bengaluru. Additionally, she ranked first in General Medicine at KS Medical Academy, Mangaluru, during the final-year MBBS examination conducted by RGUHS, Bengaluru, in 2012. With 6 years of valuable teaching experience, Rakshatha has taught students in Medical, Dental, and Allied Health Sciences. She has contributed as a co-author to the *Manual of Transfusion Medicine, Review of Postgraduate Pathology-General Pathology, Review of Postgraduate Pathology-Systemic Pathology, and Rapid Review, MCQs, AETCOM, and OSPE in Pathology*. Her dedication to education was further recognized when she secured second place in the *Best Audio-visual Award* three times at Kasturba Medical College, Mangaluru, in 2022, 2023, and 2024.

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