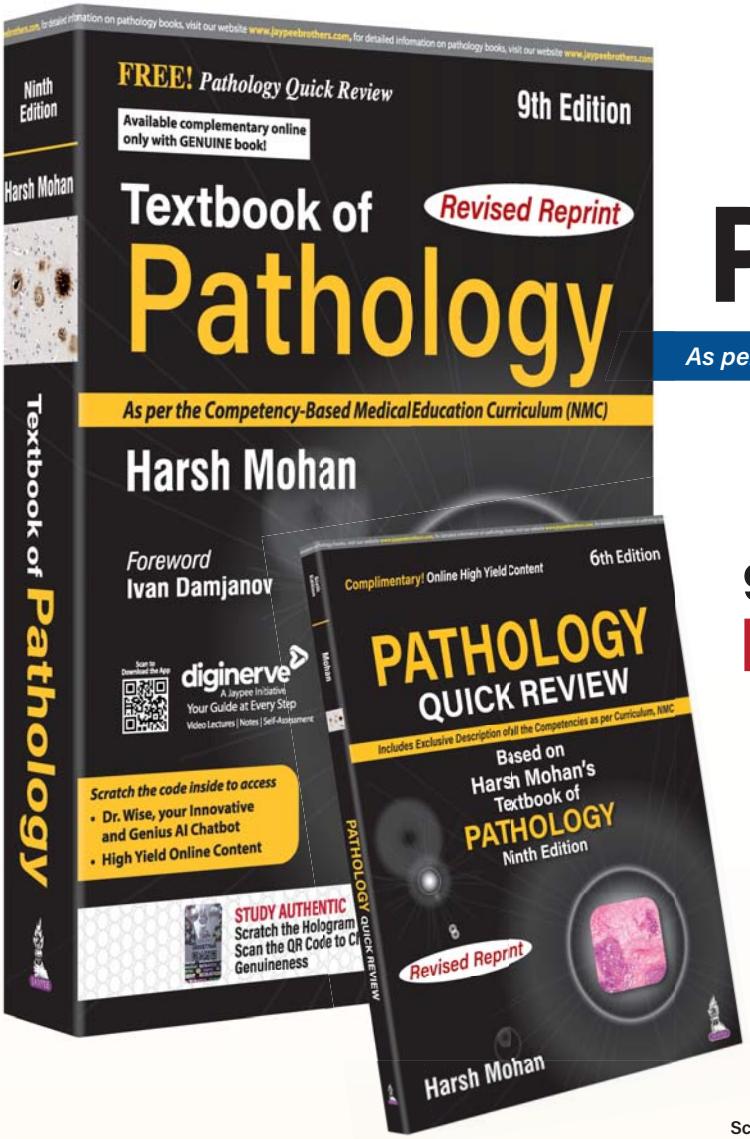

Textbook of PATHOLOGY

As per the Competency-Based Medical Education Curriculum (NMC)

Harsh Mohan

9th Edition

Revised Reprint

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Key Features

- Competency-based curricular changes recommended by NMC incorporated as core/must-know, and non-core/desirable-to-know and their corresponding new assessment methods included.
- Updated text in academic and diagnostic pathology includes current definitions, additional causes, contemporary mechanisms, up-to-date international classifications of diseases (including WHO classifications of neoplasms, 5th edition) and modern approach to diagnosis and prognosis, in a simple, lucid, easily understandable and reproducible, user-friendly format.
- Newer content includes a new chapter on 'Blood Banking and Transfusion' and another on 'Clinical Pathology' incorporated in Appendix I along with Basic Diagnostic Cytology. Pathology of COVID-19 and its impact on different organ-systems incorporated in different chapters.
- Labelled gross and photomicrographs of high quality and better resolution added and amended, many of these were generously contributed by Prof Ivan Damjanov in previous edition, several schematic illustrations updated and amended.
- New tables added at several places for enhanced learning.
- Key points as gist at the end of every topic in bulleted points for a rapid revision of the subject in an ultra-short time.
- Chapter orientation paragraph given at the beginning of every chapter to peek through the entire chapter and meant to aid the user in systematic and organised learning.
- Revised companion book prepared exclusively on the pattern of competencies/outcomes as recommended by the NMC is given for free to the user of the textbook for a quick review of the subject.
- Complimentary online learning resource includes high-yield questions (long-answer type, short-answer type, MCQs, clinical scenario-based and image-based) and clinical cases along with key and explanatory notes.
- Cheers! No overburdening!! With all these additions and insertions, girth of the book remains unaltered, care having been taken not to overburden the already stressed students.

SAMPLE PAGES

Chapter Orientation are provided at the beginning of each chapter

Key points are provided to highlight important information

CHAPTER 7

Neoplasia*

Chapter Orientation

The term 'neoplasia' literally means new growth; the mass of new growth produced is called 'neoplasm' or 'tumour'. However, all 'new growths' are not neoplasms. For instance, in the processes of embryogenesis, tissue repair, hyperplasia and hormonal stimulation, there is growth of new cells but this proliferation follows normal law of growth, and cells stop proliferating after a useful purpose has been served. On the other hand, neoplasia is 'pathological disturbance in growth' which has distinct clinical, morphologic (gross and microscopic), molecular and genetic features. While the causes and mechanisms of most of the usual cell proliferations such as in inflammation, tissue repair or hormonally-induced cell growths are known, knowledge of definite etiology and pathogenesis in neoplasia has yet been ever-expanding. However, due to high mortality associated with cancer, revolutionary advances have been continuing in its diagnosis and treatment by coordinated research in various branches of medical and allied sciences. In short, learning of neoplasia is not static, but has still been ongoing process in forward direction.

In this chapter, the subject of neoplasia is presented under following major headings: 1) nomenclature and classification, 2) characteristics of tumours, 3) epidemiology of cancer, 4) molecular basis of cancer (theories, fundamentals, molecular hallmarks), 5) etiology and pathogenesis: carcinogens and carcinogenesis (physical, chemical, and biologic agents), and 6) clinical aspects of neoplasia (clinical effects on host, grading and staging, diagnosis, and precision oncology and liquid biopsy).

KEY POINTS Nomenclature and Classification

- A neoplasm or tumour is a mass of tissue formed as a result of abnormal, excessive, uncoordinated, autonomous and purposeless proliferation of cells, even after removal of growth stimulus which caused it.
- Neoplasms may be 'benign' when they are slow-growing and localised without causing much difficulty to the host, or 'malignant' when they proliferate rapidly, spread throughout the body and may eventually cause death of the host.
- All tumours have 2 basic components: *parenchyma* comprised by proliferating tumour cells, and *supportive stroma* composed of fibrous connective tissue and blood vessels.
- The tumours are named with suffix '-oma' to denote benign tumours. Malignant tumours of epithelial origin are called *carcinomas*, while malignant mesenchymal tumours are named *sarcomas*.
- A few examples of combination of tumours are mixed tumours, teratoma, blastoma, hamartoma, and choristoma.

Figures are used to highlight various diagnostic understanding

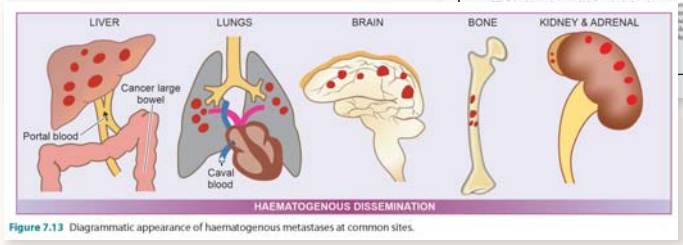


Figure 7.13 Diagrammatic appearance of haematogenous metastases at common sites.

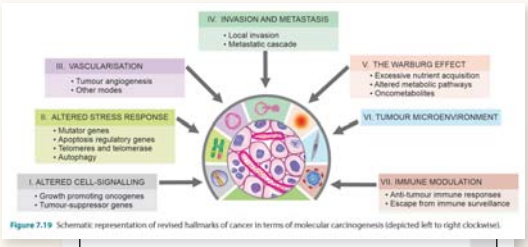


Figure 7.19 Schematic representation of revised hallmarks of cancer in terms of molecular carcinogenesis (depicted left to right clockwise).

TABLE 7.5 Important oncogenes, their mechanism of activation and associated human tumours.

	TYPE AND ONCOGENE	PROTO-ONCOGENE	ACTIVATION MODE	ASSOCIATED HUMAN TUMOURS
1.	GROWTH FACTORS			
	i) *PDGF-β chain*	*SIS (PDGF-β)*	Overexpression	Gliomas, sarcoma
	ii) *TGF-α*	*RAS (TGF-α)*	Overexpression	Gliomas, carcinomas
	iii) FGF	*HST-1*	Overexpression	Osteosarcoma
		FGF3	Amplification	Breast cancer, stomach cancer
	iv) c-MET	*HGF*	Overexpression	Follicular carcinoma thyroid, hepatocellular Ca
2.	GROWTH FACTOR RECEPTORS			
	i) *EGF* receptors	*ERB B1 (HER-1, EGFR)*	Various mutations	Adenocarcinoma lung
		ERB B2 (HER-2/neu, EGFR)	Amplification	Ca breast, ovary
	ii) *c-KIT* receptor (steel factor)	*c-KIT*	Point mutation	Gastrointestinal stromal tumour (GIST)
	iii) *RET* receptor	*RET*	Point mutation	MEN type 2A and type 2B, familial medullary Ca thyroid
	iv) *FMS*-like tyrosine kinase receptor	*FLT-3* gene	Point mutation	Acute myeloid leukaemia
	v) *PDGF* receptor	*PDGFR-β*	Overexpression, translocation	Gliomas, leukaemias
	vi) *ALK* receptor	*ALKR*	Translocation Point mutation	Adenocarcinoma lung, lymphomas Neuroblastoma
3.	CYTOPLASMIC SIGNAL TRANSDUCTION PROTEINS			
	i) GTP-bound (G) proteins	*RAS (several types)*	Point mutation	Common in 1/3rd human tumours, Ca lung, colon, pancreas
	ii) Non-receptor tyrosine kinase	*ABL-BCR*	Translocation Point mutation	Chronic myeloid leukemia (CML) Acute leukaemias
	iii) *JAK/STAT* signal transduction	*JAK2*	Translocation	Myeloproliferative disorders, ALL
4.	NUCLEAR TRANSCRIPTION FACTORS			
	i) *C-MYC*	*MYC*	Translocation	Burkitt lymphoma
	ii) *N-MYC*	*MYC*	Amplification	Neuroblastoma, small cell Ca lung
	iii) *L-MYC*	*MYC*	Amplification	Small cell Ca lung
5.	CELL CYCLE REGULATORY PROTEINS			
	i) Cyclins	*Cyclin D*	Translocation	Ca breast, myeloma, mantle cell lymphoma
		Cyclin E	Overexpression	Ca breast
	ii) CDKs	*CDK4*	Amplification	Glioblastoma, melanoma, sarcomas

Tables are provided to simplify complex topics in structured parts

PATHOLOGY ***Grossly***, the affected part is dry, shrunken and dark black, resembling the mummified foot. It is black due to liberation of haemoglobin from haemolysed red blood cells which is acted upon by hydrogen disulfide (H_2S) produced by bacteria resulting in formation of black iron sulfide. The line of separation usually brings about complete separation with eventual falling off of the gangrenous tissue if it is not removed surgically (i.e. spontaneous amputation) **(Fig. 3.24)**.

Histologically, there is necrosis with smudging of the tissue. The line of separation consists of inflammatory granulation tissue **(Fig. 3.25)**.

Important Points are Highlighted in the Green Boxes

PATHOLOGY ***Grossly***, the affected part is soft, swollen, putrid, rotten and dark. The classic example is gangrene of the bowel that may occur due to strangulated hernia, volvulus or intussusception. The part is stained dark black due to the same mechanism as in dry gangrene **(Fig. 3.26)**.

Histologically, there is coagulative necrosis with stuffing of affected part with blood. The mucosa is ulcerated and sloughed. Lumen of the bowel contains mucus and blood. Affected tissue area intense acute inflammatory exudates and thrombosed vessels. The line of demarcation between gangrenous segment and viable bowel is generally not clear-cut **(Fig. 3.27)**.

Flow Charts are used to break down broad concepts

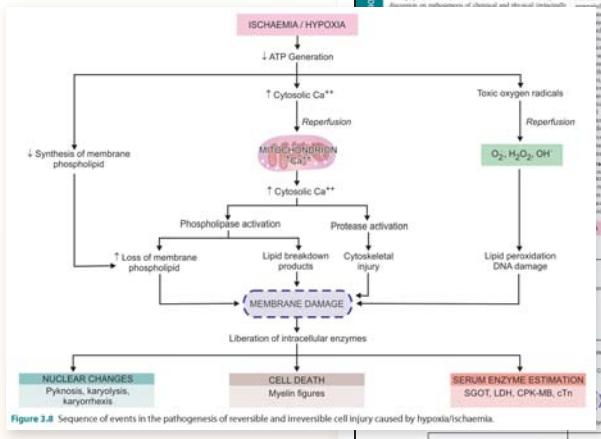


Figure 3.8 Sequence of events in the pathogenesis of reversible and irreversible cell injury caused by hypoxia/ischaemia.

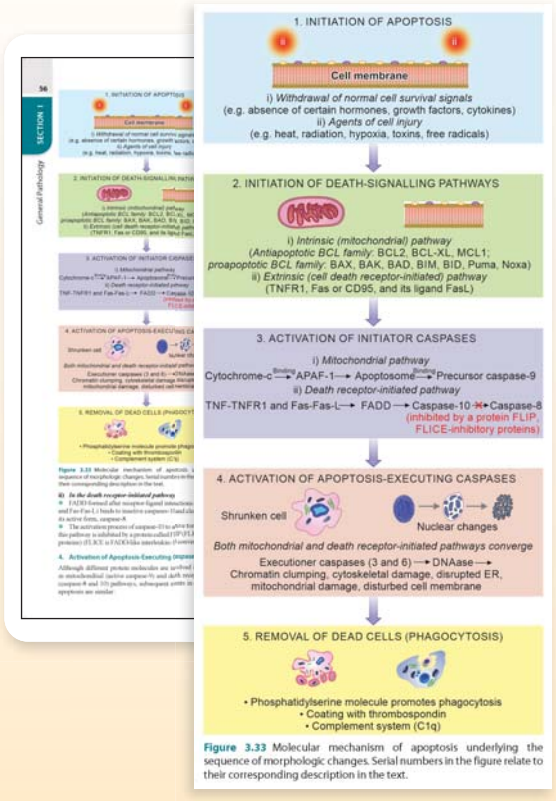
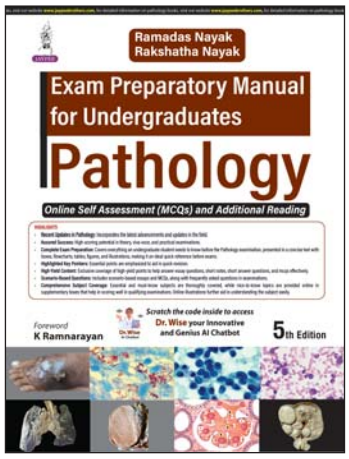
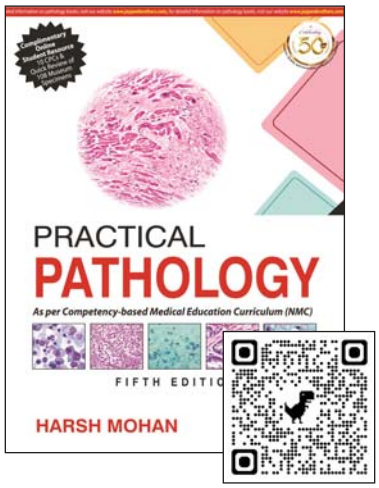


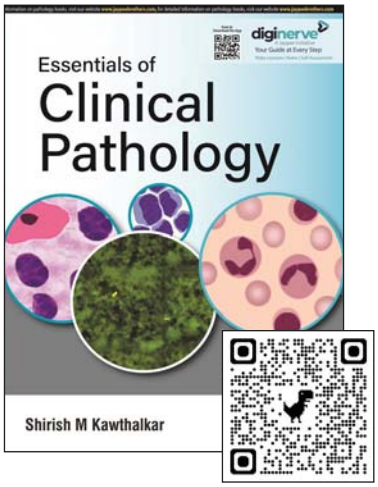
Figure 3.33 Molecular mechanism of apoptosis underlying the sequence of morphologic changes. Serial numbers in the figure relate to their corresponding description in the text.

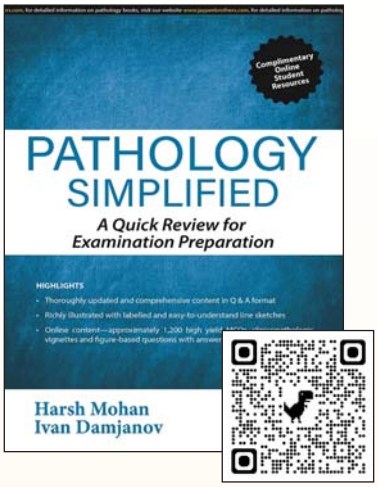
Step Wise Diagrams are given to illustrate complex topics in structured parts

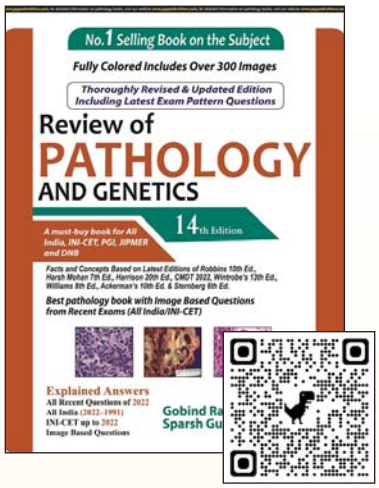
Other Essential Titles

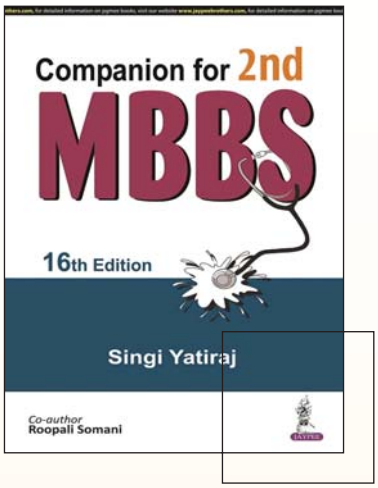


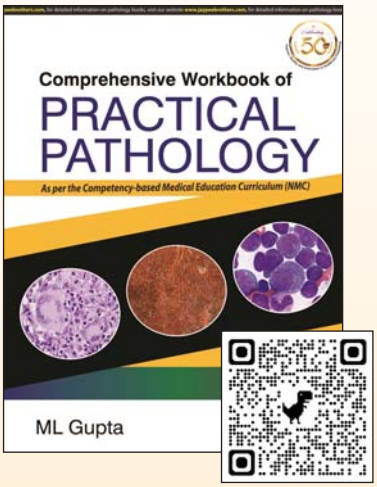












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