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As the book deals with basic pathology, it will be very useful for medical and dental students.

Dr. J.L. Vegad was Professor and Head, Department of Veterinary Pathology at Jawaharlal Nehru Krishi Vishwavidyalaya (JNKVV), Jabalpur. He was then Professor Emeritus of the Indian Council of Agricultural Research (ICAR), New Delhi, for two years. B.V.Sc. (Gold Medalist) from Jabalpur, Associate IVRI (1960), he obtained Ph.D. from New Zealand (1968) under a Commonwealth Scholarship. His contributions to the study of acute inflammatory response in the sheep and chicken are pioneering. He has published more than 150

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J.L. Vegad



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Preface to Second Edition

The widespread popularity of the first edition, both with students and teachers, has prompted me to bring out this Second Edition. I do hope this edition proves equally useful and is received with the same fervour.

Since the first edition was published in 1995, there have been further spectacular advances in our understanding of the molecular mechanisms involved in the pathogenesis of disease processes. This accelerating pace of knowledge necessitated revision and updating of the book. The second edition has been extensively revised and most chapters are completely rewritten. As such, it contains the latest information on molecular pathology. The changes in particular include:

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- For an easy grasp of the complicated molecular mechanisms, a large number of illustrations have been added, 79 in all, in the form of flow charts, line diagrams, and diagrammatic representations of pathological processes.
- Keeping in view the Veterinary Council of India (VCI)'s syllabus, a new chapter on 'Concretions' (Chapter 12) has been added.
- Also, in view of the VCI syllabus, a new sub-topic 'Avian Inflammation' has been added in the chapter on 'Inflammation' (Chapter 4).
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I am grateful to Mr. Suneel Gomber, Manager, International Book Distributing Co., Lucknow, for the publication of this book. I extend my sincere thanks to Dr. Madhu Swamy for going through the typescript. Dr. Priti Mishra checked the index, while Mr. Anand Parmar and Mr. Vijay Parmar of Jabalpur Graphics were most generous in extending help relating to computer and other work. I am thankful to them all. I am especially grateful to my wife Nita and eldest brother Amrit Lal Vegad for the moral support and for their faith in me and my task.

J. L. Vegad

Preface to First Edition

Recent years have witnessed explosion of knowledge in molecular biology, and consequent thereupon, in the field of molecular pathology. The study of pathology is no longer confined to morphological alterations. Molecular mechanisms involved in the pathogenesis of diseases are being continuously unraveled. No area of pathology has remained untouched by the molecular strides. Since this book deals with General Pathology, that is, study of the basic pathological processes, an attempt has been made to bring out the most recent concepts of molecular mechanisms. For, it is the conceptualization of the underlying principles that is paramount in laying the foundations of systemic pathology, and in fact, medicine as a whole.

The book is intended for both undergraduate and postgraduate students. The undergraduate may, at places, find the test a little too extensive. They are advised to skip these portions. However, should they find the new information rewarding, the idea is to make it readily available at one place. I only hope I could succeed in transmitting the excitement of the remarkable insights gained into the biomolecular origins of disease processes.

The complex mechanisms have been explained in a simplified way using line diagrams, so that they are readily understood. Also, before dealing with the pathogenetic mechanisms, physiological, biochemical, and other related aspects have been briefly reviewed, for an easy comprehension of the subject. Thus, discussion of free radical mediation of cell injury is preceded by a consideration of what free radicals are; or that of mechanisms of healing by an examination of extracellular matrix, or a discussion of thrombosis by a brief consideration of haemostatic mechanisms.

Another feature of the book is that, wherever considered appropriate, aspects of human pathology have been narrated side by side. The text will therefore act as a useful exercise in comparative pathology. As such the book may serve as a good reference for medical, dentistry, and zoology students. In fact, it should prove useful to workers in all branches of science who wish to learn about the basic disease processes.

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I am grateful to Dr. S. K. Ranjhan who encouraged me to write this book and to Shri C. M. Chawla of Vikas Publishing House for its publication. It is a pleasure to acknowledge the help of my colleagues

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- Dr. A. K. Katiyar, Dr. H. K. B. Parekh, Dr. R. G. Dhawedkar and Dr. B.C. Sarkhel - for their many contributions. Finally, my deepest gratitude to members of my joint family for their patience and understanding. I am especially grateful to my wife Nita and my eldest brother Amritlal Vegad for the moral support, and for their faith in me and my task.

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Abbreviations

	AD	=	Anno Domini
	ADCC	Ξ	Antibody-dependent cell-mediated cytotoxicity
	BC	=	Before Christ
	BCR	=	B-cell receptor
	BM	=	Basement membrane
	CD	=	Cluster of differentiation
	CTL	Ξ	Cytotoxic T- cell
	DIC	=	Disseminated intravascular coagulation
	DTH	=	Delayed-type hypersensitivity
	ECF	=	Eosinophil chemotactic factor
	ECM	=	Extracellular matrix
	EGF	=	Epidermal growth factor
	F	=	French
	FGF		Fibroblast growth factor
- Ar	G	=	Greek
	IFN	=	Interferon
	Ig	=	Immunoglobulin
	IL	=	Interleukin
	IVP	=	Increased vascular permeability
	L	Ξ	Latin
	LPS	=	Lipopolysaccharide
	LT	=	Leukotriene
	LX	=	Lipoxin
	MAC	=	Membrane attack complex
	MHC	=	Major histocompatibility complex
	mm	=	Millimetre (thousandth part of a metre)
	MPS	=	Mononuclear phagocyte system

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μm	=	Micron / micrometre (one millionth of a metre)
NCF	=	Neutrophil chemotactic factor
NK cell	=	Natural killer cell
nm	=	Nanometre (one billionth of metre)
NO	=	Nitric oxide
PAF	=	Platelet activating factor
PDGF	=	Platelet-derived growth factor
PG	=	Prostaglandin
PGI ₂	=	Prostacyclin
R	=	Receptor, e.g., IL-2R
TCR	=	T- cell receptor
TGF	=	Transforming growth factor
TNF	=	Tumour necrosis factor
TX	=	Thromboxane
VEGF	=	Vascular endothelial growth factor
vWF	=	von Willebrad factor

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Granulomatous inflammation (Chapter 4) is a distinctive form of mononuclear inflammation. It is usually produced by relatively slowly dividing infectious agents (e.g., *Mycobacterium tuberculosis*, fungi), and by agents of relatively large size (e.g., schistosome eggs). Granulomatous inflammation almost always reflects a cell-mediated immune reaction (Chapter 9).

3. Necrotizing inflammation: *Clostridium perfringens* and other organisms that secrete very strong toxins cause such rapid and severe tissue damage that cell death is the main feature. Because so few inflammatory cells are involved, these lesions resemble infarcts, with disruption or loss of basophilic nuclear staining and preservation of cellular outlines. At times, viruses also cause necrotizing inflammation when cell damage is widespread and severe.

4. Cytopathic-cytoproliferative inflammation: These reactions are usually produced by viruses, and are characterized by damage to individual cells, with little or no inflammatory response. Some viruses while replicating within cells make viral aggregates that are seen as inclusion bodies (e.g., Negri bodies in rabies) or induce cells to fuse and form polykaryons (multinucleate cells, e.g., with herpesvirus). Focal cell damage may cause epithelial cells to form blisters (e.g., chickenpox virus in humans). Viruses can also cause epithelial cells to proliferate and take unusual forms (e.g., warts in animals induced by papillomaviruses). Finally, viruses can cause dysplastic changes and cancers in epithelial cells and lymphocytes (see 'dysplasia').

5. Chronic inflammation: The final common pathway of many infections is chronic inflammation, which may lead either to complete healing or to extensive scarring (fibrosis).

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