

<<病理学>>

图书基本信息

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前言

为使医学教育和国际逐步接轨，双语教学在我国医学院校已推行多年，但至今仍然缺乏被广泛认可的教科书。

自编的教材受英语水平的限制，语言表达上往往不尽人意。

Robbins Basic Pathology，是在全世界深受欢迎并被广泛采用的权威性教学用书，其立足前沿的理论知识、独特精致的编写风格、严谨规范的专业术语、图文交融的编排方式，无一不受到广大医学生和病理学工作者的推崇和青睐。

是美国医学生学习病理学的首选教材，病理医师资格考试的必读用书，也是我国中文病理教材编写的主要参考书。

Robbins Basic Pathology原版的国际版和影印版已在国内销售，但由于价格依然偏高，某些内容与中国的教学习惯不完全吻合，尚难作为病理学教科书广泛应用。

基于该书在国际上的影响力及我国的医学教育现状，北京大学医学出版社决定与ELSEVIER公司联合对该书进行改编。

其目的是在不改变原书风格和基本内容的前提下，通过改编、精编和缩编使其内容和编排顺序符合中国的教学习惯。

贴近前沿、贴近临床、贴近我国的教学实际是本书改编的主要宗旨。

本书依据Robbins Basic Pathology的最新版第8版进行改编。

在改编过程中，对本书的内容进行了删节、调整和适当补充，个别章节有较大的更新和改动。

同时，在内容上兼顾了临床医学及其他相关专业和不同学制的需求。

因此本书可用作临床医学、口腔、公共卫生专业的五年制、七年制、八年制和留学生的双语教学用书，也可作为病理医生和进修生的重要参考书，以及作为执业医师资格考试的复习用书。

本书编委均来自教学第一线，在双语教学和教材编写方面均具有丰富的经验。

在繁重的病理教学、科研和诊断工作的同时，大家辛勤劳作，不遗余力地完成了初稿的编写；另有几位美国的病理学同道也参与了本书的编写。

最后，又经美国的病理学专家翟启辉教授修改润色，反复斟酌，力求行文准确、简明易懂，体现原书的学术水平和语言风格。

本书的编委会秘书陈方杰女士和山东大学病理学教研室的吴晓娟医师做了大量卓有成效的工作，山东大学病理学教研室的李丽、吴晓娟、项磊、张晓芳和桂婷参与了部分章节的二校，出版社责任编辑也付出了辛勤劳动，在此一并表示感谢。

另外，本书原版主编Kumar教授对本书的改编给予热情支持，并在百忙之中欣然作序，我们在享受其学术成果的同时，在此谨致以衷心的感谢。

改编是双语教材编写的尝试和探索，疏漏和错误在所难免，愿广大病理学同道和学生在使用中不断提出宝贵意见，以期再版时不断完善。

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内容概要

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章节摘录

插图： Intracellular Accumulations Under some circumstances cells may accumulate abnormal amounts of various substances, which may be harmless or associated with varying degrees of injury. The substance may be located in the cytoplasm, within organelles (typically lysosomes), or in the nucleus, and it may be synthesized by the affected cells or may be produced elsewhere. There are three main pathways of abnormal intracellular accumulations: A normal substance is produced at a normal or an increased rate, but the metabolic rate is inadequate to remove it. An example of this type of process is fatty change in the liver. A normal or an abnormal endogenous substance accumulates because of genetic or acquired defects in its folding, packaging, transport, or secretion. Mutations that cause defective folding and transport may lead to accumulation of proteins (e.g., I-antitrypsin deficiency). An abnormal exogenous substance is deposited and accumulates because the cell has neither the enzymatic machinery to degrade the substance nor the ability to transport it to other sites. Accumulations of carbon or silica particles are examples of this type of alteration. **Fatty Change (Steatosis)**. Fatty change refers to any abnormal accumulation of triglycerides within parenchymal cells. It is most often seen in the liver, since this is the major organ involved in fat metabolism, but it may also occur in heart, skeletal muscle, kidney, and other organs. Hepatic steatosis may be caused by toxins, protein malnutrition, diabetes mellitus, obesity, and anoxia. Alcohol abuse and diabetes associated with obesity are the most common causes of fatty change in the liver (fatty liver) in industrialized nations. Free fatty acids from adipose tissue or ingested food are normally transported into hepatocytes. Excess accumulation of triglycerides may result from defects at any step from fatty acid entry to lipoprotein exit, thus accounting for the occurrence of fatty liver after diverse hepatic insults. Hepatotoxins (e.g., alcohol) alter mitochondrial and SER function and thus inhibit fatty acid oxidation; CCl₄ and protein malnutrition decrease the synthesis of apoproteins; anoxia inhibits fatty acid oxidation; and starvation increases fatty acid mobilization from peripheral stores. Fatty change is reversible. In these severe form, fatty change may precede cell death, and may be an early lesion in a serious liver disease called nonalcoholic steatohepatitis. In any site, fatty accumulation appears as clear vacuoles within parenchymal cells. Special staining techniques are required to distinguish fat from intracellular water or glycogen, which can also produce clear vacuoles but have a different significance. To identify fat microscopically, tissues must be processed for sectioning without the organic solvents typically used in sample preparation. Usually, frozen sections are used; the fat is then identified by staining with Sudan III or oil red O (these stain fat orange-red). Glycogen may be identified by staining for polysaccharides using the periodic acid-Schiff stain (which stains glycogen red-violet). If vacuoles do not stain for either fat or glycogen, they are presumed to be composed mostly of water. Mild fatty change in the liver may not affect the gross appearance. With increasing accumulation, the organ enlarges and becomes progressively yellow, soft, and greasy. Early fatty change is seen by light microscopy as small fat vacuoles in the cytoplasm around the nucleus. In later stages, the vacuoles coalesce to create cleared spaces that displace the nucleus to the cell periphery (Fig. 1-12). Occasionally contiguous cells rupture, and the enclosed fat globules unite to produce so-called fatty cysts. In the heart, lipid is found in the form of small droplets, occurring in one of two patterns. Prolonged moderate hypoxia (as in profound anemia) results in focal intracellular fat deposits, creating grossly apparent bands of yellowed myocardium alternating with bands of darker, red-brown, uninvolved heart ("tigered effect").

编辑推荐

《病理学(第8版)(英文改编版)》是专门为中国医学生打造的病理学双语教材。由国内20家医学院校的病理学教授联合美国华裔病理学者依据Robbins Basic Pathology的最新版(第8版)进行改编。改编以国内教学大纲为依据,对原版内容进行了删节、调整和适当补充。改编版教材既保留了原版精华,保证教材权威性,又能够最大限度地适应国内双语教学需求。供医学各专业本科生、留学生、长学制、研究生双语教学使用。

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