双语版

中国科学院教材建设专家委员会规划教材

医学英文原版改编双语教材

（供临床、基础、预防、口腔、药学、检验、护理等专业使用）

TEXTBOOK OF DIAGNOSTICS

诊 断 学

Original Editors

- Richard F.LeBlond Richard L.DeGowin Donald D.Brown
- David R.Ferry
- Diana Nicoll Stephen J.McPhee Michael Pignone

Chief Editors of Adaptation Edition

Lü Zhuoren（吕卓人）

Lei Han（雷 寒）

科学出版社

www.sciencep.com

中国科学院教材建设专家委员会规划教材
医学英文原版改编双语教材

供临床、基础、预防、口腔、药学、检验、护理等专业使用

TEXTBOOK OF DIAGNOSTICS

诊 断 学

Original Editors

- **Richard F. LeBlond　Richard L. DeGowin**
  Donald D. Brown
- **David R. Ferry**
- **Diana Nicoll　Stephen J. McPhee**
  Michael Pignone

Chief Editors of Adaptation Edition

Lü Zhuoren（吕卓人）
Lei Han（雷　寒）

科 学 出 版 社

北 京

图字:01-2005-5903

图书在版编目(CIP)数据

诊断学 = TEXTBOOK OF DIAGNOSTICS / 吕卓人,雷寒主编. —北京:科学出版社, 2006.3
(中国科学院教材建设专家委员会规划教材,医学英文原版改编双语教材)
ISBN 7-03-016581-0

Ⅰ. 诊… Ⅱ. ①吕… ②雷… Ⅲ. 诊断学-双语教学-医学院校-教材-英、汉 Ⅳ. R44

中国版本图书馆 CIP 数据核字(2005)第 145129 号

责任编辑:夏 宇 李国红 / 责任校对:张怡君
责任印制:刘士平 / 封面设计:黄 超

科学出版社 出版
北京东黄城根北街16号
邮政编码:100717
http://www.sciencep.com
新蕾印刷厂 印刷
科学出版社发行 各地新华书店经销
*
2006 年 3 月第 一 版 开本:787×1092 1/16
2006 年 3 月第一次印刷 印张:23 1/4
印数:1—5 000 字数:764 000

定价:48.00 元

(如有印装质量问题,我社负责调换〈环伟〉)

Contributors of Adaptation Edition

Kang Xixiong	Capital University of Medical Sciences
Lei Han	Chongqing University of Medical Sciences
Li Qifu	Chongqing University of Medical Sciences
Li Yaocai	Medical Science College of China Three Gorges University
Lü Jiyuan	Shanxi Medical University
Lü Zhuoren	Medical School of Xi'an Jiaotong University
Wang Yingde	Dalian Medical University
Xiao Wei	Medical School of Shandong University
Xu Anding	Medical School of Jinan University
Xu Shaoyong	Yunyang Medical College
Xue Xiaolin	Medical School of Xi'an Jiaotong University
Yang Jiong	Medical School of Wahan University
Zhang Jun	Medical School of Xi'an Jiaotong University
Zhang Xiaoyong	Nanjing Medical University

《诊断学》改编委员会名单

主　编　吕卓人　雷　寒

副主编　张小勇　杨　炯　徐少勇

编　者　（以姓氏拼音为序）

康熙雄　首都医科大学

雷　寒　重庆医科大学

李启富　重庆医科大学

李耀彩　三峡大学医学院

吕吉元　山西医科大学

吕卓人　西安交通大学医学院

王英德　大连医科大学

肖　伟　山东大学医学院

徐安定　暨南大学医学院

徐少勇　郧阳医学院

薛小临　西安交通大学医学院

杨　炯　武汉大学医学院

张　军　西安交通大学医学院

张小勇　南京医科大学

PREFACE FOR ADAPTATION EDITION

As is pointed out in the document (Higher Education 2001-4) issued by the Ministry of Education, "education should be geared to the modernization, whole world, and future." Undergraduate education must provide conditions for teaching common courses and specialized courses in English so as to meet the challenges brought by economic globalization and scientific and technological revolution. Meanwhile, carrying out reform in bilingual teaching will definitely promote teaching reform, construction of teaching contingent, disciplinary development, and cultivation of medical undergraduates' abilities. Nevertheless, lack of suitable textbooks matching bilingual teaching is one of the obstacles to bilingual teaching.

Textbooks for bilingual teaching take the following forms. First, direct adoption of original English textbooks. The merits are language advantage and sufficient adoption of the advanced concepts in diagnostics teaching in the United States and European countries. The shortcomings are too much burden on students and great gap in style between the original textbooks and the existing planned textbooks in China. For instance, there are no textbooks in western countries that can match Diagnostics in our country, and items including laboratory diagnosis, experimental examination and common clinical diagnostic techniques are not listed in "Physical Examination" published in foreign countries. Moreover, the writing style varies greatly and original textbooks have much greater length but poorer practicability. Second, translating the Chinese-version textbooks of Diagnostics into English. One strong point is that they can match the existing textbooks, so students' burden is not heavy; it facilitates learning with Chinese textbooks referred to at the same time, especially in the case of medical students' poor ability to use English. Because Diagnostics textbooks in China have been revised several times, their scientific and practicable properties become more obvious. They are much cheaper than original English textbooks, which makes them affordable to students and thus helps promote bilingual teaching. The disadvantage lies in the fact that the quality of English is hard to guarantee. Third, combing the merits of the two forms. Adopting the style of the planned Diagnostics textbooks in China and consulting the way of expression in original textbooks. Purchasing copyright and adopting paragraph by paragraph to compile adapted English-version textbooks, integrating more foreign advanced thoughts in teaching such as communication skills and making them bilingual teaching textbooks with Chinese characteristics. We adopted the third form in this textbook, hoping to make it an English-version Diagnostics textbook with distinctive features for medical undergraduate students. Therefore, after friendly negotiation with The McGraw-Hill Companies, Science Press reached cooperation agreement with McGraw-Hill. Based on respecting intellectual property rights, we are entitled to using the textbooks or monographs (including illustrations) published by The McGraw-Hill Companies and compiling adapted Original medical textbook series suitable for medical education in China.

As one book of the adapted original medical textbook series under the cooperation be-

tween Science Press and The McGraw-Hill Companies, Diagnostic Examination follows the principles mentioned above. We selected three high-quality textbooks from the books published by McGraw-Hill as the chief source for adaptation: (1) DeGowins' Diagnostic Examination by LeBlond RF, et al.: 2004, 8th ed; (2) Pocket Guide to Diagnostic Tests by Nicoll D, et al.: 2004, 4th ed; and (3) Basic Electrocardiography in Ten Days by Ferry DR: 2001. The style refers to that of eight-year-program textbooks Clinical Diagnostics and Experimental Diagnostics in the hope to be in agreement with the reference ranges of examinations in the planned textbooks and make this textbook suitable for the actual situation of clinical practice and teaching in China.

The contents focus on "mastery" and "familiarity" required by the Syllabus. Covering about two-thirds of the Chinese-version Diagnostics, this book both retains the completeness of the original textbooks and gives prominence to key points. Every effort has been made to have a good combination of words and illustrations in the book and lively forms. To add practicability to Appendix, we have included Laboratory Reference Ranges, Case Record (English-Chinese), English-Chinese Vocabulary, and List of Figures.

Most members on the Compiling Committee come from first-rate medical universities in China. All the members have good English reading and writing abilities and rich experience in diagnostics teaching thanks to their experience of having studied in English-speaking countries. Because we are pressed for time and the practice of adapted English textbooks is in its infancy, faults or errors are inevitable in spite of our conscientiousness and efforts. We appreciate suggestions and comments from teachers and students of medical colleges and universities, which will help revision and improvement in the future republication of this book.

Zhuoren Lü

Dec. 15, 2005

改编版前言

教育部教高[2001]4 号文件中明确指出要按照“教育面向现代化、面向世界、面向未来”的要求，为适应经济全球化和科技革命的挑战，本科教育要创造条件使用英语进行公共课和专业课教学。同时，推行“双语教学”必将促进教学改革、师资队伍建设、学科发展和本科生的能力培养。然而，缺乏与“双语教学”相适应的教材是开展“双语教学”的“瓶颈”之一。关于“双语教学”教材可有几种形式：①直接采用原版教材，优点是语言上有优势，并能充分吸取欧美国家“诊断学”教学的先进理念；缺点是学生负担太重，而且原版教材编写体例与我国现有的规划教材差距很大，如西方国家没有与我国“诊断学”相对应的教材，“诊断学”教材中的“实验诊断”、“器械检查”、“临床常用诊断技术”等部分均未纳入国外的“Physical examination”之中，而且体例相差很大：原版教材篇幅较大，实用性较差。②将中文版“诊断学”规划教材翻译成英文，优点是能与现有的教材匹配，学生的负担不大，尤其目前在医学生的英语应用能力较差的情况下，有助于与中文教材对照学习；何况我国“诊断学”教材经多次修订，科学性和实用性强的优点很突出；价格明显低于原版教材，在学生经济能力有限的情况下，该教材有助于推广“双语教学”的进程；缺点是难以保证英文质量。③综合上述两种形式的优点，采用我国“诊断学”规划教材的体例，参照原版教材的表述方式，采取购买版权、整段摘用的形式，编写成“英文改编版”，融入较多的国外先进的教学思想，如交流技巧，形成具有中国特色的“双语教学”教材。本教材采用了第三种方式，以期成为具有鲜明特点的供医学本科生使用的“诊断学”英文版教材。为此，科学出版社与 McGraw-Hill 出版公司经过友好协商，达成合作协议，以尊重知识产权为前提，摘录 McGraw-Hill 公司出版的原版教材或专著，包括教材中的图，编写成适合中国医学教育所需的英文改编版系列教材。

本书作为科学出版社与 McGraw-Hill 出版公司合作出版的医学英文原版改编系列教材之一，遵循上述原则，从 McGraw-Hill 公司出版的原版书中，精选了 3 本高质量的教材或专著作为改编版的蓝本：①DeGowins' Diagnostic Examination by LeBlond RF, et al.: 2004, Ed: 8th; ②Pocket Guide to Diagnostic Tests by Nicoll D, et al.: 2004, Ed: 4th; ③Basic Electrocardiography in Ten Days by Ferry DR: 2001。写

作体例参照八年制教材“临床诊断学”和“实验诊断学”，力求与规划教材中各种检查的参考范围完全统一，使本教材符合我国临床和教学的实际情况。内容以大纲要求的“掌握”和“熟悉”为主，总体上比中文版“诊断学”大约少1/3篇幅，既保证教材的完整性，又突出重点。本教材尽可能做到图文并茂、形式活泼。“Appendix（附录）”从实用性出发，编入“Laboratory Reference Ranges（检验参考范围）”、“Case Record（English-Chinese）（双语表格病历）”、“English-Chinese Vocabulary（中英文名词及缩写对照）”和“List of Figures（插图索引）”等。

本编委会的来源学校涵盖国内多所一流的医学院校，编委均有英语国家留学经历，具有良好的英语读写能力和较丰富的“诊断学”教学经验。鉴于时间比较仓促，英文改编版的形式还是一种探索，尚缺乏经验，尽管编委们十分认真和努力，但难免存在一些疏漏、不妥或错误之处，恳请全国各医学院校的老师和同学们在使用本教材的过程中，提出宝贵的意见和建议，惠予指导，以在再版时加以修正和改进。

吕卓人

2005年12月15日

CONTENTS

Part A Common Symptoms and History Taking

Chapter 1 Common Symptoms

FEVER

Internal body temperature is maintained within a narrow range, ± 0.6 ℃, in each individual. However, the population range of this set point varies from 36.0—37℃ making it impossible to know an individual's normal temperature without a prior established baseline. Internal body temperature varies throughout the day—it's lower in the morning and higher in the late afternoon and evening. Daytime workers who sleep at night register their minimum temperature at 3:00 to 4:00 a.m., whence it rises slowly to a maximum between 8:00 and 10:00 p.m. This pattern is reversed in nightshift workers. The transition from one pattern to the other requires several days. Heat is produced by the chemical reactions of cellular metabolism, so a temperature gradient extends from a maximum in the liver to a minimum on the skin surface. Customarily, the body temperature is measured in the rectum, the mouth, or the axilla. Among these sites, the rectal temperature is about 0.3—0.5℃ higher than that of the oral reading; the axillary temperature is about 0.2—0.4℃ less than the oral value.

PATHOGENY AND SORT

The cause of a fever is very complex. Many factors can trigger fever. A fever usually means the body is responding to a viral or bacterial infection. Sometimes heat exhaustion, extreme sunburn or certain inflammatory conditions such as temporal arteritis may trigger fever as well. In rare instances, a malignant tumor or some forms of kidney cancer may cause a fever. Thus fever can be identified as infective fever and noninfective fever. The most common identified causes of fever in patients in the modern era are infectious.

Infective Fever

Bacterial, viral, rickettsial, fungal, and parasitic infections either localized or systemic (occult abscess is common) all can cause fever.

Noninfective Fever

Many factors can trigger noninfective fever. Noninfectious inflammatory diseases, and systemic lupus erythematosus, sarcoidosis, Crohn disease, polymyalgia rheumatica, vasculitis (giant cell arteritis, Wegener disease, polyarteritis nodosa), non-Hodgkin lymphoma, Hodgkin disease, leukemia, adenocarcinoma, habitual hyperthermia, subacute thyroiditis, Addison disease, drug fever and so on. (1) Vascular thrombophlebitis, tissue ischemia and infarction, vasculitis, subarachnoid hemorrhage. (2) Inflam matory systemic lupus erythematosus, acute rheumatic fever, still disease, vasculitis, serum sickness, any severe local or systemic inflammatory process (e.g., sarcoidosis, bullous dermatosis). (3) Congenital familial mediterranean fever, other familial periodic fevers, porphyrias. (4) Endocrine hyperthyroidism, pheochromocytoma. (5) Mechanical/Traumatic tissue necrosis (e.g., myocardial infarction, pulmonary infarction, stroke), exercise. (6) Metabolic/Toxic drug reactions, gout. (7) Neoplastic leukemia, lymphomas and solid tumors. (8) Neuroleptic seizures. (9) Psychosocial factitious.

OCCURRENCE MECHANISM

Internal body temperature is tightly regulated to maintain normal cellular function of vital organs, particularly the brain. Deviation of temperature by more than 4℃ above or below normal can produce life-threatening cellular dysfunction. Regulation of internal temperature is controlled by the hypothalamus which maintains a set point for temperature. The autonomic nervous system plays a key role in maintaining body temperature by regulating blood flow conducting heat from the internal organs to the skin and innervating sweat glands. Increasing flow and dilating cutaneous capillaries radiates heat away from the body and increases conductive loss while production of sweat increases evaporative heat loss. Behavioral adaptations are also important; in hot conditions people become less active and seek shade or a cooler environment when they are able. Declines in body temperature are opposed by increased heat generation in muscles by shivering and by behavioral adaptations such as putting on clothes and seeking warmer environs. Deviations of body temperature indicate changes in the set point, increased heat production, decreased heat dissipation, failure of regulatory systems, or any combination of those. Increased body temperature results from excessive production of heat or interference with heat dissipation.

Release of endogenous pyrogens, particularly interleukin (IL)-1, triggered by tissue necrosis, infection, inflammation, and some tumors, elevates the hypothalamic set point leading to an increased body temperature.

Each of these mechanisms may be physiologic (i. e., occurring as a normal response to a physiologic challenge), or pathologic (i. e., temperature elevation as a result of damage to the normal thermoregulatory pathways). Physiologic elevation of temperature results from an elevation of the hypothalamic physiologic set point for body temperature, a fever. Pathologic elevations of body temperature, hyperthermia, result from unregulated heat generation and/or impairment of the normal mechanisms of heat exchange with the environment.

Some patients are unable to mount a febrile response to infection, for example, elderly patients and those with renal failure or on high doses of corticosteroids. Fever occurring in several specific patient groups requires special consideration from the clinician. These include fever in immunocompromised hosts, HIV-infected patients and nosocomial fever.

CLINICAL OCCURRENCE

Grade of Fever

Fever can be identified as four gradients extend based on the oral reading, low-grade fever: 37.3—38℃, middle-grade fever: 38.1—39℃, high-grade fever: 39.1—41℃, hyperthermia: above 41℃.

Clinical Features of Fever

Onset of fever may be marked by a chill with shivering and cutaneous vasoconstriction as the body begins generating increased heat and decreasing heat loss; particularly severe chills are called rigors. When the new set point is reached, the skin is usually warm, moist, and flushed; but absence of these signs does not exclude fever. Occasionally, the skin temperature may be subnormal or normal while the core temperature is markedly elevated. Tachycardia usually accompanies fever, the increase in the pulse rate being proportionate to the temperature elevation. During the fever, the patient usually feels more comfortable in a warm environment. The new set point and the pattern of the fever curve depend upon the dynamics of the particular pathophysiologic process. Return of the set point to normal, either temporarily or permanently, is marked by sweat and flushing as the body dissipates the accumulated heat. Night sweats occur in many chronic infectious and inflammatory diseases, and some malignancies, particularly lymphomas. They represent an exaggeration of the normal diurnal variation in temperature, the sweat marking the decline of the fever at night.

PATTERNS OF FEVER

The pattern of temperature fluctuations may be a useful diagnostic clue. Many patterns have been defined.

Continuous Fever

A fever with a diurnal variation of 0.5—1.0℃. This pattern can be seen in typhoid and acute pneumonia.

Remittent Fever

A fever with a diurnal variation of more than 2℃ but with no normal readings. This pattern can be

seen in sapraemia, acute rheumatic feve and acute infectious endocarditis.

Intermittent Fever

Episodes of fever separated by days of normal temperature. Examples include tertian fever from Plasmodium vivax in which paroxysms of malaria are separated by an intervening normal day; quartan fever in which paroxysms from P. malariae occur with two intervening normal days; intermittent hepatic fever (Charcot hepatic fever) in which chills and fever occur at irregular intervals, marking the intermittent impaction of a gallstone and cholangitis.

Undulant Fever

The body temperature increases to 39℃ in several days and then slowly returns to the normal. And the body temperature increases and decreases in the same way again and again. This pattern can be seen in brucellosis and tumor.

Relapsing Fever

Bouts of fever occurring every 5—7 days from infection with spirochetes of the group Borrelia and Colorado tick fever.

Episodic Fever

Fever lasts for days or longer followed by prolonged periods (at least 2 weeks) without fever and with remission of clinical illness. This pattern is typical of the familial periodic fevers.

Pel-Epstein Fever

Occurring in Hodgkin disease, bouts of several days of continuous or remittent fever followed by afebrile remissions lasting an irregular number of days.

FEVER OF UNKNOWN ORIGIN

Sometimes it's not possible to identify the cause of a fever. This syndrome was initially described in 1961. To qualify as an FUO, three conditions must be met: (1) the illness must be at least 3 weeks in duration; (2) the temperature must be repeatedly 38.3℃; and (3) no diagnosis should have been reached after at least 1 week of hospitalization. Recent authors have suggested that the 1 week of hospitalization be replaced by at least 3 outpatient visits or at least 3 days in the hospital. The most common identified causes of FUO in immunocompetent patients in the modern era are noninfectious inflammatory diseases, infections, and malignancies, especially hematologic malignancies. Fever remains unexplained in almost 50% of patients, especially those with episodic fevers.

(Xu Shaoyong, Wang Bin)

HEADACHE

Headache refers to pain perceived more than momentarily in the cranial vault, the orbits, and the nape of the neck; pain elsewhere in the face is not included. An urgent intensive search for serious pathology is required if the patient describes a severe headache unlike any experienced in the past.

CLINICAL OCCURRENCE

Arteriovenous malformations, hydrocephalus; Endocrine Pheochromocytoma. Idiopathic intracranial hypertension. Inflammatory/Immune giant cell arteritis, sarcoidosis. Infectious meningitis (bacterial, viral, mycobacterial, fungal, drugs), encephalitis (viral, e. g., herpes simplex, HIV, West Nile fever, eastern and western equine encephalitis, St. Louis encephalitis, California encephalitis, La-Crosse encephalitis, and dengue), rickettsial infections, sinusitis, otitis, mastoiditis, non-CNS viral infections (e. g., influenza, cytomegalovirus, varicella), parasites (e. g., malaria, neurocysticercosis), protozoa (e. g., toxoplasmosis). Metabolic/Toxic analgesic rebound headache, hypoxia, hypercapnia, hypoglycemia, alcohol and illicit drug withdrawal, carbon monoxide poisoning, caffeine abstinence. Mechanical/Trauma: accelerated and malignant hypertension, trauma, head trauma, concussion, muscular tension, temporomandibular joint disease, increased intracranial pressure, glaucoma, and decreased intracranial pressure (CSF leak). Primary and metastatic brain tumors, tumors in the sinuses, pituitary tumors. Migraine, cluster headache, paroxysmal hemicrania, trigeminal neuralgia, occipital neuralgia. Psychosocial stress. Intracranial aneurysm, hemorrhage (intracerebral, subdural, epidural, and subarachnoid), venous sinus thrombosis, stoke, cerebral vasculitis.

PATHOPHYSIOLOGY

The head contains many pain sensitive structures. Mechanisms of headache include inflammation, infection, arterial dilation, hemorrhage, changes of pressure within closed spaces, expanding mass lesions producing traction or compression of structures, trauma, tissue ischemia, and tissue destruction. The common extracranial cause of headache is sustained contraction of the muscles of the head, neck, and shoulders.

The pain-sensitive intracranial structures are the dura and the arteries at the base of the brain, the cerebral arteries in the same region, the great venous sinuses, and certain nerves (CN-Ⅴ,-Ⅸ,-Ⅹ, and C1—3). The greater portion of the dura and cranium is insensitive. Mechanisms producing headaches from intracranial disorders include (1) traction on the superficial cerebral veins and venous sinuses, (2) traction on the middle meningeal arteries, (3) traction on the basilar arteries and their branches, (4) distention and dilatation of the intracranial arteries, (5) inflammation near any pain-sensitive region, and (6) direct pressure or traction by tumors on cranial and cervical nerves. The resulting headaches may be throbbing when arteries are involved; otherwise the pain is steady. Headaches are often intensified by movements of the head, certain postures, and rapid changes in CSF pressure.

HISTORY

Inquire carefully for the attributes of pain (PQRST): (1) Provocative and palliative factors trauma, medications, substance abuse, position of the head and body, coughing, straining, emotional tension, relief with massage, and resolution with sleep. (2) Quality whether burning, aching, deep or superficial, lancinating, throbbing, or continuous. (3) Region involved cranial, facial, orbital, unilateral, bilateral. (4) Severity. (5) Timing: when headaches began, frequency, time of day, duration, pattern of intensity.

Other inquires about family members with a headache history: Identify associated symptoms, for example, fever, stiff neck, nausea and vomiting, constipation or diarrhea, diuresis, rhinorrhea, visual disturbances (e. g., photophobia, scotomata, tearing, diplopia), cerebral symptoms (e. g., confusion, slurred speech, aura, paresthesias, anesthesias, motor paralysis, vertigo, mood, and sleep disturbances).

(Xu Anding)

EDEMA

PATHOPHYSIOLOGY

The distribution of water between blood and interstitial tissues is maintained by a net equilibrium between hydrostatic and oncotic pressures. Normally, fluid flows into the extravascular interstitial space in response to hydrostatic pressure in the precapillary arterioles and capillaries (intravascular > interstitial), which is only partially offset by the opposing oncotic pressure (intravascular > interstitial). In the postcapillary venules, the lowered intravascular hydrostatic pressure is more than compensated by the intravascular oncotic pressure, resulting in return of interstitial water to the intravascular space. Interstitial fluid, proteins, and cells are also removed from the interstitial space and, ultimately, returned to the blood through the lymphatics. Alteration of any of these forces upsets the equilibrium. An increase in the systemic venous pressure in congestive heart failure produces generalized edema; occlusion of a vein may result in localized edema. Obstruction of lymphatic channels produces lymphedema. Reduction in the plasma albumin (the plasma protein with the highest contribution to oncotic pressure) results in lowering the oncotic pressure of the plasma, permitting edema to form; this type of edema may first appear in areas of decreased tissue pressure such as the periorbital tissues. Increased capillary permeability may cause edema that is not dependent. Tissue inflammation by bacterial, chemical, thermal, or mechanical means increases capillary permeability to make localized edema. Excessive accumulation of interstitial fluid, either localized or generalized, is termed edema. When the amount of generalized edema is great, the condition is termed anasarca or dropsy. In the adult, about 4.5kg (10 lb) of fluid accumulates before it is detectable as pitting edema. To demonstrate the presence of edema, gently press your thumb into the skin against a bony surface, such as the anterior tibia, fibula, dorsum of the foot, or sacrum. When the thumb is withdrawn, an indentation persists for a

short time.

The distribution of edema should be noted; the amount of fluid is roughly proportional to its extent and thickness. Because dependent edema responds to gravity, it first appears in the feet and ankles of the walking patient or over the posterior calves or sacrum of the supine patient. As the amount of dependent fluid increases, a fluid level may be detected under the skin; seldom does dependent edema rise higher than the heart. Anasarca can be recognized at a glance by the obliteration of superficial landmarks under the skin. Chronic edema of the legs leads to fibrosis of the subcutaneous tissues and skin, so they no longer pit on pressure; this is sometimes called brawny edema (i. e., musclelike). Symmetric edema affecting both legs suggests that the problem is in the pelvis or more proximally, while edema limited to the arms and head suggests superior vena cava obstruction. Edema limited to one extremity suggests a local problem with vascular channels or local inflammation.

The processes involved in edema formation are the same whether the edema is generalized or local. To evaluate local edema, the examiner must consider the local anatomy of the arteries, veins, lymphatics and soft tissues, the presence of any local inflammatory or structural disease and then form hypotheses as to the likely mechanism and anatomic site of the local problem.

Because exclusive dependence upon clinical information may miss cardiovascular causes of bilateral leg edema, consider measurement of B-type natruretic peptide and/or echocardiographic evaluation with estimation of pulmonary artery pressure, right and left ventricular size and function and tricuspid valve function. The following approach, based upon the anatomic distribution of edema, is diagnostically useful.

CLINICAL OCCURRENCE

Localized Edema

Inflammation infection, angioedema, contact allergy; Metabolic Causes gout. Insufficiency of the Venous Valves (with or without varicosities). Venous Thrombosis postoperative, prolonged air or automobile travel. Venous or Lymphatic Compression malignancies, constricting garments. Chemical or Physical Injuries burns, irritants and corrosives, frostbite, chilblain, envenomation (insects, snakes, spiders). Congenital amniotic bands, arteriovenous fistulas, Milroy disease.

Bilateral Edema Above the Diaphragm

Superior vena cava obstruction.

Bilateral Edema Below the Diaphragm

Congestive Cardiac Failure with elevated jugular venous pressure including elevated pulmonary artery pressures caused by left heart abnormalities, intrinsic pulmonary disorders, right heart abnormalities, and constrictive pericarditis; Portal Vein Hypertension or Obstruction cirrhosis, portal vein thrombosis, schistosomiasis. Inferior Vena Cava Obstruction thrombosis, extrinsic compression, pregnancy; Loss of Venous Tone drugs (calcium channel blockers, angiotensin-converting enzyme inhibitors, other vasodilators), convalescence, lack of exercise.

Generalized Edema

Hypoalbuminemia nephrotic syndrome, cirrhosis, chronic liver disease, protein losing conditions (e. g., enteropathy, burns, fistulas). Renal Retention of Salt and Water corticosteroids, nonsteroidal antiinflammatory drugs (NSAIDs). Increased Capillary Permeability sepsis, systemic inflammatory response syndrome (SIRS), interleukin (IL)-2.

IDIOPATHIC EDEMA

Recurrent and chronic edema may be observed in women in the 3rd to 5th decades in the absence of cardiac, hepatic, or renal abnormalities or of venous or lymphatic obstruction. Affective disorders and obesity may coexist. Possible mechanisms include exaggerated capillary leakage on assuming the upright posture and inappropriate chronic diuretic administration started initially for minor degrees of peripheral edema (diuretic-induced edema). Both mechanisms probably lead to inappropriate activation of hormones involved in salt and water retention.

TROPICAL EDEMA

Pitting edema of the ankles often occurs abruptly in normal adults within 48 h after they have traveled from a temperate climate to the heat of the tropics or in temperate zones when weather changes from cool and dry to warm and humid. It spontaneously resolves in a few days of acclimatization.

ANGIOEDEMA

Subcutaneous soft-tissue edema begins abruptly and spreads to involve several centimeters of tissue with diffuse borders. Erythema is not prominent. Angioedema often involves the face, lips, or tongue, and is life-threatening when the larynx is involved. Causes include hereditary absence of C1 esterase, exposure to allergen, and angiotensin-converting enzyme inhibitors.

(Lü Jiyuan)

MUCOCUTANEOUS HEMORRHAGE

Mucocutaneous bleeding occurs with platelet abnormalities (number or function) or problems of the vessel wall; bleeding into joints or viscera is more likely related to clotting factor deficiencies or inhibitors. Extravasation of blood in the skin produces an area that is first red, then blue; in a few days, the degradation of hemoglobin changes the color to green or yellow and fades. Because the blood in the area is extravascular, the color does not blanch with pressure.

ETIOLOGY AND PATHOGENESIS

Problems of the Vessel Wall

Vascular Abnormalities eroded or traumatized large vessels, hereditary hemorrhagic telangiectasia, vasculitis, infections, et al.

Platelet Disorders

Platelets are primarily responsible for initial hemostasis by aggregation at the sites of endothelial damage. The platelet plug is then stabilized by fibrin deposition from activated coagulation.

Blood Abnormalities quantitative platelet defects (e.g., autoimmune thrombocytopenic purpura, heparin-induced thrombocytopenia, hypersplenism), qualitative platelet defects (e.g., aspirin, von Willebrand disease, Glanzmann syndrome), thrombotic thrombocytopenic purpura, bone marrow failure (e.g., aplastic anemia, leukemia, chemotherapy), meningococcemia, cryoglobulinemia, disseminated intravascular coagulation.

Coagulation Disorders

Coagulation is a complex process involving many different blood proteins, platelets, calcium, and tissue factors. There is a constant balance between initiation of coagulation and coagulation inhibitors designed to protect against inappropriate coagulation while directing clot formation to the sites of vessel injury. Disorders of blood coagulation may be congenital or acquired. Congenital abnormalities usually are deficiencies of specific factors or decreased factor function. Acquired disorders may be factor deficiencies or functional inhibition of coagulation. Deficiencies in factors Ⅰ, Ⅱ, Ⅴ, Ⅶ, or Ⅹ, liver disease, vitamin K deficiency, greatly decreased or abnormal fibrinogen. Warfarin administration, vitamin K deficiency (dietary or absorptive), or hepatic insufficiency lead to loss of the vitamin K-dependent coagulation factors (Ⅱ, Ⅶ, Ⅸ, Ⅹ) and to loss of proteins S and C.

CLINICAL OCCURRENCE

Petechia

A petechia (plural, petechiae) is a round, discrete hemorrhagic area less than 2mm in diameter. Doubtful spots can be circled with ink, their disappearance in a few days rules out angioma.

Purpura

When hemorrhages of either size occur in groups, the condition is termed purpura. Purpuric lesions may become confluent and they usually do not elevate the skin or mucosa. Spontaneous purpura from platelet or vessel defects usually occurs on the lower extremities, although slight trauma to the skin may induce it elsewhere.

Ecchymosis

A larger spot is an ecchymosis (plural, ecchymoses).

Hematoma

A hematoma is an area in which underlying hemorrhage causes elevation of the skin or mucosa; extravasated blood frequently colors the surface and dissects along tissue planes.

Epistaxis (Nosebleed)

Nosebleed can be a spontaneous and trivial occurrence or a sign of serious local or generalized disease. Local Causes: lacerations, rhinitis sicca.