

高等医学英语系列

总主编 王茹



English Course of
College Medicine
大学医学英语
综合教程

主编 高元暹 秦兆霞 张淑卿



华中科技大学出版社

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前 言

国家教育部新颁布的全国《大学英语教学大纲》(修订本)明确指出：“(本科)学生在完成基础阶段的学习任务，达到四级或六级后，都必须修读专业英语。”因此，高等医学院校外语教学正面临改革。本教材将对医学专业英语教学模式的转型进行探索。转型后的教学模式将把医学生的技能与专业、认知与情感等方面逐步进行融合。

哈尔滨医科大学英语教研室的教师就是本着这一宗旨，编写了“高等医学英语系列”，针对医学实际需要，重点培养学生的语言实用技能。作为一名医学生和未来的医务工作者，必须要掌握好医学英语，这样才能与国外同行交流，彼此相互学习先进的科学技术和实践经验，才能洞察医学领域的最新发展，勇攀世界医学高峰，最终成为一名合格的、有一定影响的中英知名的医务工作者。

本丛书在体例、内容和编排等方面都与传统的医学英语教材有所不同，“高等医学英语系列”由《大学医学英语综合教程》、《大学医学英语翻译与写作教程》和《大学医学英语词汇》组成，真正实现了在精读、泛读、快读、翻译、写作和词汇等多方面呈现全方位的、多层次的医学英语语言培养体系。

《大学医学英语综合教程》由快速阅读、精读和泛读三个部分组成，每部分为一篇文章。快速阅读部分选自医学科普文章，目的是进一步巩固和加强学生的快速阅读能力。精读和泛读部分选自英美原文，包括病理、内科、外科等内容，与基础医学教学衔接。除各配有生词表外，在精读部分还配备了一些练习，特别是构词练习，目的是要学生通过构词方法，在较短的时间里能够掌握较多词汇。为减轻学生负担，便于自学，书后配有练习答案和参考译文。

在编写过程中，我们得到了哈尔滨医科大学外语部的宁薇、徐亚楠、刘玉书、李玫、田玉军等老师的帮助。同时，华中科技大学出版社的梅欣君编辑也为本套教程的编写和出版做了大量的工作。我们在此一并向他们表示诚挚的谢意。

在编写这套医学英语系列教程的过程中，编者倾注了大量的心血和的努力。由于笔者水平有限，教材难免存在不当和疏漏之处，恳请广大读者批评指正，以便及时改进和修订。

编 者
2011.4

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Unit One

Fast Reading: Alzheimer's Disease

Mental deterioration is by no means an inevitable consequence of aging, but it is common enough to be a significant public-health problem. Experts estimate that 15 percent of persons over age 65 have some mental impairment; of those, about 5 percent—more than a million—are so seriously affected that they can no longer care for themselves. Approximately half of the 15 percent have Alzheimer's disease(老年性痴呆), a disorder that is marked by progressive deterioration of memory, learning and judgment. No one knows its underlying cause, but some tantalizing(引起好奇心的) clues have been found.

For example, the brains of Alzheimer's disease patients may contain 10 to 30 times the normal amounts of aluminum(铝), which can be poisonous to nerve cells. And brain cells that normally produce acetylcholine(乙酰胆碱), a chemical released during transmission of nerve signals, seem not to do it properly.

None of those clues have yet led to any accepted explanation or cure for the disease. But until recently, many specialists considered the problem hopeless. Today scientists are pursuing each new clue and a national organization, the Alzheimer's Disease and Related Disorders Association Inc. has been set up to promote research, educate, and exchange information and advice among families of sufferers. Its address is 360 North Michigan Ave, Suite 601, Chicago, Ill. 60601.

Comprehension Exercise

Choose the best answers to the following questions.

1. Approximately how many persons over 65 in the United States are suffering from mental deterioration?

- | | |
|-------------------------|----------------------------------|
| A. More than a million. | B. Definitely over 3 million. |
| C. About 2.5 million. | D. The passage doesn't say that. |

2. According to the experts, how many of the older people have Alzheimer's disease?

- | | |
|---------------------------|-----------------------|
| A. 3 million. | B. 2 million. |
| C. Less than one million. | D. About 1.5 million. |

3. Alzheimer's disease is defined as_____.

- A. an inevitable consequence of aging
- B. a significant public-health problem
- C. a mental disorder affecting memory, learning and judgment
- D. a tantalizing clue

4. The Alzheimer's Disease and Related Disorders Association Inc. is_____.

- A. in the state of Michigan
 - B. in the city of Chicago
 - C. among families of the sufferers
 - D. on Illinois Avenue
5. We can conclude from the report that_____.
- A. scientists and specialists are divided in opinion that Alzheimer's disease is incurable
 - B. aluminum and acetylcholine are of the same class of chemicals
 - C. mental impairment is an inevitable result of aging
 - D. we are still far away from a convincing explanation and workable cure for Alzheimer's disease

Intensive Reading: Acute Inflammation

This is the immediate and early response to an injurious agent and affects the vascular and connective tissues adjacent to the injured cells. There are three major components in the process of acute inflammation: increased blood flow (vasodilation), increased vascular permeability (exudation) and egress of white blood cells into the injured tissue (emigration). These three components are coordinated and interrelated by numerous chemical mediators produced or released at the site of injury. Mediators include histamine, bradykinin, complement and many other pharmacologically active substances. The emphasis in this section is on the morphological aspects of the inflammatory process visible to the pathologist rather than on the pharmacological and biochemical events.

Vasodilation

Vasodilation occurs directly after injury. The microvasculature at the site of injury becomes dilated and filled with blood (hyperaemia). Blood flow through the dilated microvasculature is initially rapid, but soon slows because a concomitant increase in vascular permeability and loss of plasma water raises the viscosity of the blood. In the slowly moving blood the pattern of flow changes. Red cells tend to clump in the centre of the vessel lumen and leucocytes assume a more peripheral position near the vessel wall. This margination of leucocytes is an important initial step in the emigration process. If flow becomes very slow, the blood may clot and form a thrombus.

Exudation

This is the increased passage of fluid and solutes, notably proteins, through the vessel wall. The mechanisms leading to increased vascular permeability are complex and incompletely understood. They include endothelial cell contraction or damage, the effects of mediators, local haemodynamic forces and the osmotic effects of proteins escaping into interstitial tissue. The leak of proteins is roughly in proportion to their molecular size. Albumin is in the greatest amount, but if vascular permeability is extensive, large amounts of fibrinogen may leak out. Exudation is an important local defence mechanism. The increase in interstitial fluid dilutes toxins, and proteins such as globulins are effective in neutralizing agents like bacteria. This increased fluid is sometimes called inflammatory oedema or simply oedema.

Emigration

Emigration of white blood cells, principally neutrophils and monocytes, is also an important defence mechanism. These are phagocytic cells which engulf and digest foreign particulate matter such as bacteria and the debris of dead cells. The emigration of leucocytes is an active process which occurs in two stages. The cells stick to the endothelial surface (pavementing) and then actively migrate through the gaps between the endothelial cells and into the tissue spaces. The mechanism by which leucocytes stick to the endothelial cells is unknown. Emigration is an active amoeboid process and once outside the vessel, neutrophils can move as fast as 20 μm per minute. Monocytes, which differentiated into macrophages, move more slowly. Movement of leucocytes in tissue spaces is polarized in the general direction of the site of injury. This process, known as chemotaxis is mediated by various chemical attractants such as components of the complement, kinin and clotting systems. Because neutrophils are in greater number in the circulating blood and because they move faster than macrophages, the first phase of cellular infiltration into damaged tissue is dominated by neutrophils. With the passage of time, macrophage numbers increase and after 2 or 3 days macrophages outnumber neutrophils in most inflammations. In addition to leucocyte emigration, red cells may burst through the vessel wall behind an exiting white cell. This is a passive movement in contrast to energy-dependent white cell emigration and is termed diapedesis (to walk between).

Variants

Whilst the major components of acute inflammation are present in all damaged tissue, the proportions vary according to the nature, duration and extent of injury and to the nature of the injured tissue itself. Qualifying terms may be used to specify the dominant features of the inflammation. For example, serous exudates refer to the fluid blister after burns. Fibrinous exudates occur in the peritoneal cavity of rats given ulcerogenic non-steroidal anti-inflammatories, and haemorrhagic inflammation is a common local perivascular reaction to irritants injected intravenously. Excess mucous secretion in an inflamed epithelium, such as the nose in common colds is termed catarrhal inflammation.

Sequelae

Acute inflammation is the first step in a dynamic response to injury. The sequelae depend on the nature and extent of injury. Resolution means the complete restoration of normal conditions after the cause of the acute inflammation is removed. This occurs when there is minimal cell death and tissue damage, rapid elimination of the causal agent, and local conditions favouring the removal of fluid and debris by lymphatics and by phagocytosis.

New Words

1. inflammation /'ɪnflə'meɪʃn/ *n.* 发炎, 炎症
2. permeability /'pɜːmiə'biliti/ *n.* 渗透性
3. vasodilation /'veɪzədaɪ'leɪʃn/ *n.* 血管扩张

4. exudation /ˌeksjuːˈdeɪʃn/ *n.* 渗出(物)
5. egress /ˈiːɡres/ *n.* 外出; 出口
6. mediator /ˈmiːdieɪtə/ *n.* 介质
7. histamine /ˈhɪstəmiːn/ *n.* 组胺
8. bradykinin /ˌbrædiˈkɪnɪn/ *n.* 缓激肽
9. pharmacologically /ˌfɑːməkəˈlɒdʒɪkəli/ *ad.* 药理(学)地
10. morphological /ˌmɔːfəˈlɒdʒɪkəl/ *a.* 形态学的
11. pathologist /pəˈθɒlədʒɪst/ *n.* 病理学家
12. microvasculature /ˌmaɪkrəʊˈvæskjuleɪtʃə/ *n.* 微血管循环系统
13. hyperaemia /ˌhaɪpəˈriːmiə/ *n.* 充血
14. concomitant /kənˈkɒmɪtənt/ *a.* 伴随的, 相伴的
15. viscosity /ˌviːskəsɪti/ *n.* 黏稠度, 黏性
16. clump /klʌmp/ *vi.* 使(细菌)凝结成块
17. leucocyte /ˈluːkəsəɪt/ *n.* 白细胞
18. peripheral /pəˈrɪfərəl/ *a.* 周边的, 周围的
19. thrombus /ˈθrɒmbəs/ *n.* 血栓
20. endothelial /ˌendəˈθiːliəl/ *a.* 内皮的
21. haemodynamic /ˌhiːmədaɪˈnæmɪk/ *a.* 血流动力学的
22. osmotic /ɒzˈmɒtɪk/ *a.* 渗透的
23. interstitial /ˌɪntəˈstɪʃəl/ *a.* 间隙的, 间质的
24. albumin /ælˈbjuːmɪn/ *n.* 白蛋白
25. fibrinogen /faɪˈbrɪnədʒən/ *n.* 纤维蛋白原
26. globulin /ˈɡləʊbjʊlɪn/ *n.* 球蛋白
27. oedema /iˈdiːmə/ *n.* 水肿
28. neutrophil /ˈnjuːtrəfɪl/ *n. & a.* 嗜中性白细胞; 嗜中性的
29. monocyte /ˈmɒnəsəɪt/ *n.* 单核细胞
30. phagocytic /ˌfæɡəˈsɪtɪk/ *a.* 吞噬作用的
31. engulf /ɪnˈɡʌlf/ *vt.* 吞没
32. particulate /pəˈtɪkjʊlɪt/ *n.* 微粒
33. debris /deɪbriː/ *n.* 碎屑
34. paving /ˈpeɪvməntɪŋ/ *n.* 铺路; 铺筑过的地面(或路面)
35. amoeboid /əˈmiːbɔɪd/ *a.* 阿米巴样的
36. macrophage /ˌmækrəˈfeɪdʒ/ *n.* 巨噬细胞
37. polarise /ˈpəʊləraɪz/ *v.* 极化, 偏振(化)
38. chemotaxis /ˌkeməʊˈtæksɪs/ *n.* (化学)趋化性, 趋化作用
39. complement /ˈkɒmplɪmənt/ *n.* 补体
40. infiltration /ˌɪnfɪlˈtreɪʃn/ *n.* 浸润
41. diapedesis /ˌdaɪəpiˈdiːsɪs/ *n.* (复) diapedeses /ˌdaɪəpiˈdiːsɪz/ 红细胞渗出
42. variant /ˈveəriənt/ *n.* 变体, 变异
43. serous /ˈsiərəs/ *a.* 浆液的, 血清的

44. exudate /'eksjudeit/ *n.* 渗出物, 渗出液
45. blister /'blɪstə/ *n.* 水泡
46. fibrinous /'faɪbrɪnəs/ *a.* 纤维蛋白的
47. peritoneal /,perɪtə'ni:əl/ *a.* 腹膜的, 腹腔的
48. ulcerogenic /,ʌlsərəu'dʒenɪk/ *a.* 致溃疡的
49. non-steroidal /nɒn'stɜːrɔɪdl/ *a.* 非类固醇的
50. hemorrhagic /,hemə'rædʒɪk/ *a.* 出血的
51. perivascular /,peri'væskjulə/ *a.* 血管周的
52. intravenously /,ɪntrə'vi:nəsli/ *adv.* 通过静脉
53. mucous /'mju:kəs/ *a.* 黏液的, 黏的
54. secretion /si'kri:ʃn/ *n.* 分泌
55. epithelium /,epɪ'θi:liəm/ *n.* 上皮
56. catarrhal /kə'tɑ:rəl/ *a.* 卡他的, 黏膜炎的
57. sequela /si'kwelə/ *n.* (复) sequelae /si'kweli:/ 结局, 后遗症
58. resolution /,rezə'lu:ʃn/ *n.* (炎症等的)消退
59. lymphatic /lɪm'fætɪk/ *a. & n.* 淋巴管的; 淋巴管
60. phagocytosis /fægə'saɪ'təʊsɪs/ *n.* 吞噬作用

Phrases and Expressions

1. adjacent to 邻近……
2. visible to 可见……
3. rather than 而不
4. in proportion to 与……成比例
5. leak out 漏出
6. stick to 黏附于……
7. differentiate into 分化成
8. in contrast to 与……形成对比, 与……不同, 然而

Study & Practice

Part I. Word Building

1. Memorize the following roots and suffixes.

A

- | | |
|--|---|
| <ol style="list-style-type: none"> 1) vaso-/væsəu, 'veizəu/ (血) 管 3) brady-/brædi/ 慢, 徐缓 5) patho-/pæθəu/ 病 7) hyper-/haɪpə/ 过多, 超过, 亢进, 上 9) h(a)emo-/hi:məu, 'heməu/ 血液 11) neutro-/nju:trəu/ 中性, 中立 13) peritoneo-/peri'təuni:əu/ 腹膜, 腹腔 | <ol style="list-style-type: none"> 2) histo-, histio-/hist(i)əu/ 组织 4) pharmaco-/fɑ: məkəu/ 药, 药学 6) micro-/maɪkrəu/ 小, 细, 微 8) leuc(k)o-/lju:kəu/ 白, 白细胞 10) fibrino-/faɪbrɪnəu/ 纤维蛋白 12) mono-/mɒnəu/ 单, 偏, 独 |
|--|---|

B

- | | |
|------------------------------------|----------------------------|
| 1)-algia /'ældʒiə/ 痛 | 2)-blast /blæst/ 成……细胞 |
| 3)-cyte /sait/ 细胞 | 4)-emia /'i: miə/ 血症 |
| 5)-gen /dʒen/ 原 | 6)-genic /'dʒenik/ 原的 |
| 7)-genesis /'dʒeni:sis/ 生成, 生长, 发育 | 8)-graphy /'græfi/ 造影术 |
| 9)-in /in/ 素 | 10)-itis /'aitis/ 炎 |
| 11)-kinesia /kai'ni:ziə/ 运动 | 12)-lysis /'laisis/ 溶解, 水解 |
| 13)-mania /'meiniə/ 狂, 癖 | 14)-oma /'əumə/ (肿) 瘤 |
| 15)-osis /'əusis/ 病, 症, 异常增多 | 16)-scopy /'skɒpi/ 镜检 |
| 17)-pnea /pni:ə/ 呼吸 | 18)-tonia /'təuniə/ 紧张 |

2. Use the roots and suffixes above or learned before to build the following words.

- | | |
|----------------|--------------|
| 1) 血管造影术_____ | 血管紧张_____ |
| 2) 组织细胞_____ | 组织细胞瘤_____ |
| 3) 发育徐缓_____ | 呼吸徐缓_____ |
| 4) 药物记载学_____ | 药物癖_____ |
| 5) 病原体_____ | 发病机理_____ |
| 6) 微动脉造影术_____ | 微生物血症_____ |
| 7) 血钙过多_____ | 发育过度_____ |
| 8) 白细胞增多_____ | 白细胞溶解_____ |
| 9) 成血细胞瘤_____ | 溶血素_____ |
| 10) 纤维蛋白原_____ | 纤维蛋白原的_____ |
| 11) 中性白细胞_____ | 中性白细胞增多_____ |
| 12) 单肾的_____ | 单狂_____ |
| 13) 腹膜造影术_____ | 腹膜炎_____ |

3. Give the Chinese meaning of each word and then try to read them.

- | | |
|-------------------------|------------------------|
| 1) vasodilator_____ | vasoconstrictor_____ |
| 2) histoblast_____ | histiocytosis_____ |
| 3) bradycardia_____ | bradykinesia_____ |
| 4) pharmacotherapy_____ | pharmacodiagnosis_____ |
| 5) pathography_____ | patholysis_____ |
| 6) hyperkinesias_____ | hypermyotonia_____ |
| 7) leuc(k)olysin_____ | leuc(k)ogenesis_____ |
| 8) h(a)emogenic_____ | h(a)emocytology_____ |
| 9) fibrinogenemia_____ | fibrinogenolysis_____ |
| 10) peritonealgia_____ | peritoneoscopy_____ |

Part II. Translate the following into English based on the text.

1. 血管通透性_____
2. 药物活性物质_____
3. 局部血液动力学作用_____
4. 炎性水肿_____

5. 白细胞游离_____
6. 浆液性渗出_____
7. 非类固醇抗炎药物_____
8. 黏液分泌亢进_____
9. 卡他性炎症_____
10. 致炎因子_____

Part III. Fill in the blanks with the words below and then put them into Chinese with your dictionary.

distinctive	terms	granulomas	traditionally	seen in
inflammatory	resembles	tuberculoid	restricted	macrophages

Granuloma and granulomatous inflammation

These 1 are widely used with three different meanings. 2 they are used to describe a chronic 3 lesion in the form of a mass with grossly 4 a tumour, hence the suffix -oma, which is usually reserved for neoplasms.

Recently, especially in North America, granuloma has been 5 to lesions composed largely of 6 , or restricted still further to mean a collection of altered macrophages as 7 tuberculosis. To avoid confusion it is wise to indicate this usage by terms such as macrophage granuloma, epithelioid-cell granuloma or 8 granuloma.

Finally, a number of heterogeneous entities are called 9 —malignant granuloma of nose or midline granuloma, Wegener's granuloma and eosinophil granuloma of bone. These terms are 10 enough to avoid confusion.

Part IV. Translate the following sentences into English.

1. 在急性炎症的复杂环境中，补体系统可通过不同的途径激活。

2. 几乎由任何原因引起的组织坏死都可释放引起邻近活组织发生炎症的物质。

3. 内源性介子似乎可引起血管扩张和白细胞游出，似乎也可引起血管通透性增强。

4. 与缓激肽密切相关的几种肽可被类似的酶系统释放，并且也是强有力的通透性因子。

5. 有许多情况，组织的破坏或其他不良效应不是由于损害因子的作用，而是由于机体对损害所产生的这方面或那方面的反应。

Extensive Reading: Chronic Inflammation

Some types of inflammatory reaction are of much longer duration than those so far described, persisting for weeks or months after the initial injury. Any prolonged inflammation is termed chronic, the term referring solely to the duration of the inflammatory process.

There are two main types of chronic inflammation: chronic supervening on acute and chronic *ab initio*, i.e. developing slowly with no initial acute phase.

Chronic Inflammation Supervening on Acute

This is almost always suppurative in type, and presents as a persistent discharge of pus from an abscess which has ruptured or been drained surgically. There are two common causes of persistent suppuration. The first is delay in evacuation of an abscess. If an abscess remains undrained, its fibrous wall becomes progressively thicker and more rigid. When such a thick-walled abscess is opened, pressure of adjacent tissues cannot collapse the cavity, and it can only be obliterated by ingrowth of granulation tissue from its walls. If, due to inadequate drainage, pus stagnates within the abscess cavity, residual bacteria may multiply and cause re-activation of the inflammatory process. Chronic abscesses may be seen after delayed or inadequate drainage of an empyema thoracis (suppurating pleurisy) or whenever an abscess forms in bone.

The second cause of persistent suppurating infection is the presence of foreign material within the inflamed area. This may be dirt, cloth or wood, etc. driven in from outside, or a metal or plastic prosthesis introduced by the surgeon. Indigestible dead tissue may also have an effect like a foreign body. In a boil or carbuncle, substantial portions of dermal collagen may be killed by bacterial toxins: dead collagen is broken down very slowly by lysosomal enzymes and may persist in the abscess for a considerable time. A piece of dead bone (sequestrum) in osteomyelitis (infection of a bone) is another example of a persistent endogenous “foreign body”. Why a foreign body within an area of suppuration causes the infection to persist is not always clear, but it is a well-established clinical observation that suppuration will continue until the foreign material is removed.

Chronic Inflammation *ab initio*

This type of response occurs after many types of injury. The injury may be physical, chemical, such as the response to talc or asbestos, or may result from poor local circulation, as in the

ulceration of the leg often seen in association with varicose veins. Certain micro-organisms, including those that cause tuberculosis, syphilis and leprosy, characteristically induce chronic inflammation. In still other types of chronic inflammation, including rheumatoid arthritis, ulcerative colitis and Crohn's disease, the cause is not known but disturbances of immune mechanisms are believed to play an important aetiological role.

The histological appearances of chronic inflammation vary widely with different causative factors, but all types share the following morphological characteristics.

1. The reaction is usually more productive than exudative, i.e. formation of new fibrous tissue is more prominent than exudation of fluid. Tuberculosis of joints, bones and serous cavities is sometimes an exception to this generalization.

2. Destruction of tissue and resulting inflammation proceed at the same time as attempted healing.

3. The cellular reaction is pleomorphic, various cell types being present in the inflamed tissues.

4. There may be suppuration and / or necrosis.

5. The microscopic appearances in most cases are non-specific, in the sense that they give no indication of the cause of the condition.

Attempted healing occurs concurrently with tissue destruction and inflammation in all types of chronic inflammation, but the topographical relationships of inflammation and repair vary. In a chronic abscess and in many tuberculous lesions, the two processes are sharply separated. However, healing and inflammation may intermingle to form a mass composed of inflammatory cells, areas of granulation and fibrous tissue, and sometimes foci of necrosis or abscesses. Such a chronic inflammatory mass is traditionally called a granuloma.

The many different cell types which may be present in areas of chronic inflammation include neutrophil and eosinophil polymorphs, macrophages and their derivatives (epithelioid cells and giant cells), plasma cells, lymphocytes and fibroblasts. The presence of a mixture of cell types is the most characteristic histological feature of chronic inflammation.

In many cases of tuberculosis, the diagnosis can be suspected strongly from the histological appearances alone, but there are dangerous pitfalls as several other conditions may give rise to changes readily mistaken for tuberculosis. Moreover, in this regard tuberculosis is a most atypical chronic inflammation. In most instances of acute or chronic inflammation it is not possible to determine the precise cause from the histological appearances alone. It is true that suppurating inflammation is virtually always caused by bacterial infection, but the particular bacteria responsible cannot be deduced from the histological appearances. A firm diagnosis of the cause of inflammatory lesions depends upon the recognition of a specific causative agent, or on some other procedure such as serological tests for a specific infection.

New Words

1. supervene /ˌsu:pə'vi:n/ v. 意外发生, 附带发生
2. ab initio /æbi'niʃiəu/ 从开始 (拉丁语)
3. suppurative /'sʌpjʊəətɪv/ a. 生脓的, 化脓的

4. pus /pʌs/ *n.* 脓
5. abscess /'æbsɪs/ *n.* 脓肿, 溃疡
6. suppuration /sʌpjuə'reɪʃn/ *n.* 脓, 化脓
7. evacuation /i,vækju'eɪʃn/ *n.* 抽空, 排空
8. granulation /,grænju'leɪʃn/ *n.* 颗粒化, 肉芽
9. stagnate /'stæɡneɪt/ *v.* 停滞
10. residual /ri'zɪdjuəl/ *a.* 残余的
11. activation /ækti'veɪʃn/ *n.* 活化, 激活性
12. empyema /empai'i:mə/ *n.* 积脓, 脓胸
13. thoracic /θɔ:'ræsɪk/ *a.* 胸的, 胸廓的
14. pleurisy /'pluəri:si/ *n.* 肋膜炎, 胸膜炎
15. prosthesis /'prɒsθɪsɪs/ *n.* 假肢, 修补物
16. dermal /'dɜ:məl/ *a.* 皮肤的, 真皮的
17. collagen /'kɒlədʒən/ *n.* 胶质, 胶原
18. osteomyelitis /ɔstiəumaɪə'laitɪs/ *n.* 骨髓炎
19. endogenous /en'dɒdʒɪnəs/ *a.* 内源性的
20. varicose /'værikəs/ *a.* 静脉曲张的, 肿胀的
21. syphilis /'sɪfɪlɪs/ *n.* 梅毒
22. leprosy /'leprəsi/ *n.* 麻风病
23. pleomorphic /pli:ə'mɔ:fɪk/ *a.* 多性的, 多形态的
24. topographical /tɒpə'græfɪkl/ *a.* 局部解剖的
25. intermingle /,ɪntə'mɪŋɡl/ *a.* 混合, 渗入, 参杂
26. granuloma /,grænju'ləumə/ *n.* 肉芽肿
27. epithelioid /,epɪ'θɪliɔɪd/ *a.* 上皮样的
28. serological /,sɪrə'lɒdʒɪkl/ *a.* 血清学的

参考译文

慢性炎症

某些类型的炎症反应, 自开始损伤后持续数周或数月, 比上面已描述的炎症反应所持续的时间长得多, 任何延续较长时间的炎症称为慢性炎症, 慢性一词仅指炎症过程的持续。

慢性炎症有两个主要类型: 即由急性炎症演变为慢性型和一开始就是慢性, 即开始没有急性期的缓慢发展型。

由急性演变而来的慢性炎症

本型几乎均为化脓性炎症, 脓液从已溃破的或外科引流的脓肿处持续排出。持久化脓有两个常见的原因。第一, 脓肿排空延缓, 如果脓肿未引流, 其纤维性壁不断增厚、变硬, 这种厚壁脓肿切开后, 邻接组织的压力不能使脓腔塌陷, 只有从其腔壁长出肉芽组织后脓腔才能闭合。如果引流不畅, 脓液积聚于脓腔内, 残留的细菌繁殖而引起炎症过程再复燃。慢性脓肿可见于胸腔积液(化脓性胸膜炎)引流不及时或不充分, 以及骨内形成脓肿时。

持久性化脓性感染的第二个原因是在发炎区内有异物存在,这可以是外界进入的污物、布片或木片等,或由外科医生置入的金属或塑料假体,不能消化的坏死组织也可有类似异物的作用。在疔或痈处,皮肤胶原的实质部分可被细菌毒素杀死,坏死的胶原被溶酶体酶非常缓慢地分解,并可在相当长的时间内存留在脓肿中。骨髓炎(骨感染)内的一块死骨是持久性内源性“异物”的另一个例子。化脓区内的异物为何会引起顽固性感染,还不清楚,但是,异物不清除,化脓就不停止,这已为临床观察所公认。

无急性期的慢性炎症

这种炎症反应发生于许多类型损伤之后,损伤可以是物理学性的、化学性的(如对滑石粉或石棉的反应),或是局部血液循环不良所致(如静脉曲张常伴小腿溃疡)。某些微生物(包括引起结核病、梅毒和麻风的微生物)特别能引起慢性炎症。还有一些慢性炎症(包括类风湿性关节炎、溃疡性结肠炎和克隆氏病)的原因尚不明,但一般认为免疫机能紊乱起着重要的病因学作用。

慢性炎症的组织学表现随致炎因子的不同而有很大的差异,但所有的慢性炎症都具有下列形态学特征。

1. 增生性反应常常比渗出明显,即纤维组织增生比液体渗出明显。而骨、关节及浆膜腔结核病有时是这一概括的例外。
2. 组织破坏及其所引起的炎症反应同时进行,作为试图愈合的现象。
3. 细胞反应的多形性,在发炎组织内出现各种类型的细胞。
4. 可能有化脓和(或)坏死。
5. 在大多数情况下,显微镜下的表现是非常特异性的,也就是说,这些病变不能提示疾病的原因。

在所有类型的慢性炎症中,企图愈合的过程是和组织破坏及炎症同时发生,但炎症和修复的相互依存关系却因慢性炎症的类型而异。在慢性脓肿和许多结核病变中,这两个过程是截然分开的。可是愈合和炎症混合在一起而形成一个包括有炎性细胞、肉芽组织和纤维组织的肿块,有时还包括坏死或脓肿病灶。这种慢性肿块传统地称之为肉芽肿。

在慢性炎症区内可能出现的许多不同类型的细胞,包括中性粒细胞、嗜酸粒细胞、巨噬细胞及其衍化而来的细胞(上皮样细胞)、浆细胞、淋巴细胞和纤维母细胞。各种类型细胞混合存在是慢性炎症最独特的组织学特征。

许多结核病病例,单凭组织学表现即可作出高度可疑的诊断,但有误诊的危险,因为有几类别的病变很容易被误认为结核病。因此,在这一点上,结核病是一种非常不典型的慢性炎症。对大多数急性或慢性炎症都不可能单凭组织学表现来决定其确凿的病因。诚然,化脓性炎症实际上总是由细菌感染引起的,但从组织学表现却不能推断出引起炎症的细菌是哪一种。对炎症病变原因的确诊依赖于识别特殊的病原因子或依赖于其他的一些方法,如对一种特殊性感染作血清学试验。