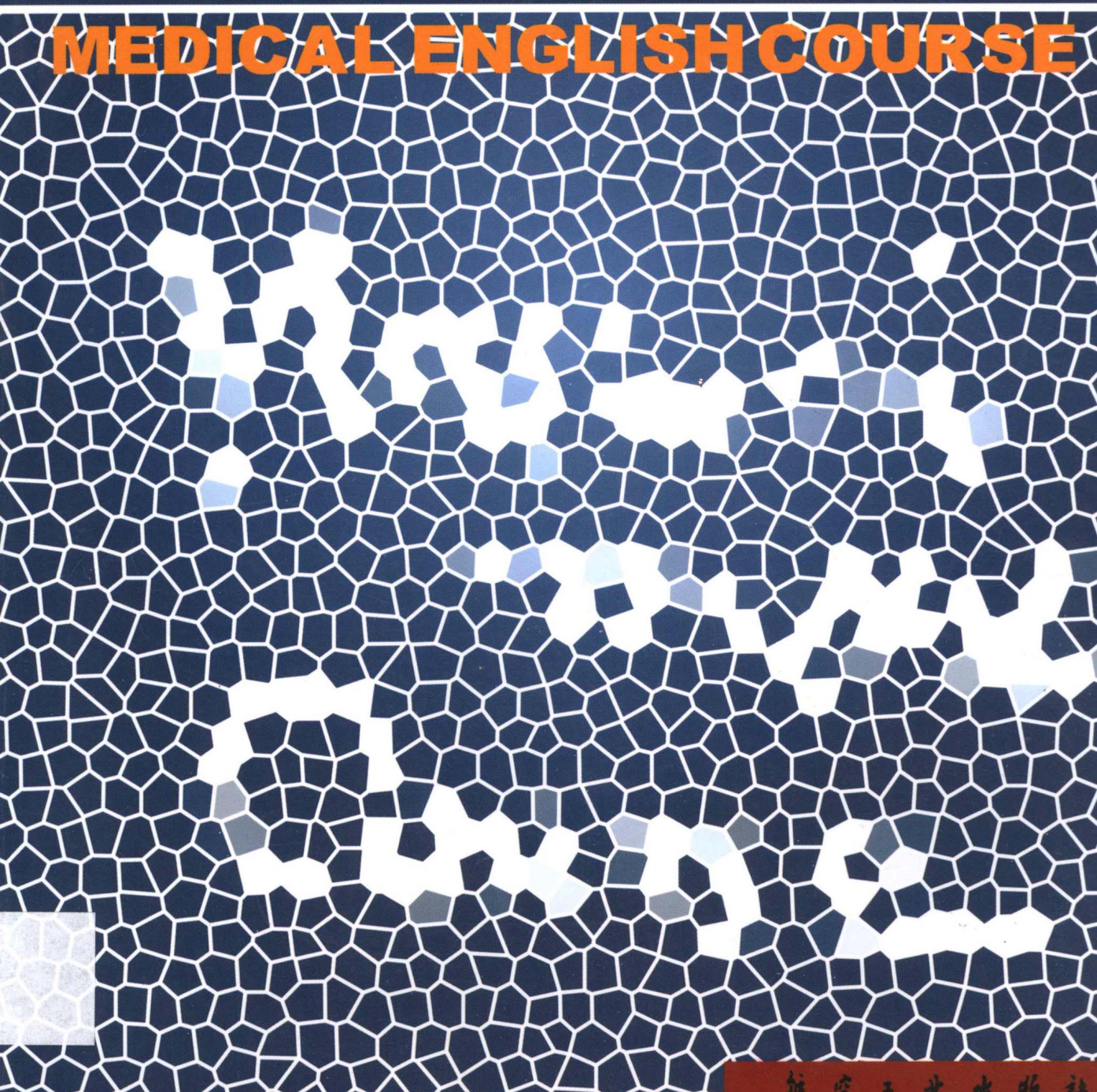
高等医学院校专业阅读教材

# 医学英语教程

主编：张宝军 肖世娥

MEDICAL ENGLISH COURSE



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# 前　言

《医学英语教程》是根据《大学英语教学大纲》"专业英语"的具体要求，结合一般医学院校的实际情况，由承德医学院和张家口医学院两校共同设计编写的一套供高年级学生阅读使用的教材。其宗旨是指导学生阅读有关医学专业的英语书刊和文选，进一步提高阅读英语科技资料的能力，并能以英语为工具，获取医学专业所需要的信息。

《医学英语教程》所选文章，均以语言规范，文章难易适中，表达方式多变为首选条件；其内容包括医学科普、卫生保健、医学基础、临床医学及最新医学知识等。

本书共32课，每课分为课文、词汇、注释、补充读物和医学会话。书后的附录包括：医学英语常用词缀和构词成分、处方、药物说明书、证明信和便条等应用文实例。

限于编者水平，书中缺点和错误在所难免，欢迎批评指正。

编　者

# CONTENTS

# LESSON ONE

## TEXT

### Disease and Its Manifestations

Disease is a structural or functional change within the body judged to be abnormal. Minor changes that are of no importance may be judged to be variations of the normal state rather than disease. Pathology not only includes the study of basic structural and functional changes associated with a disease, but also includes the study of causes that lead to the structural and functional changes and the manifestations that result from them. Furthermore, pathology is concerned with the sequence of events that leads from cause to structural and functional abnormalities and finally to manifestations. This sequence is referred to as the *pathogenesis* of disease. The term *etiology* means the study of causes. It is commonly misused as a synonym for cause.

*Diagnosis* is the process of assigning a name to a patient's condition. The name applied (the noun) is also called the *diagnosis*. If possible, a diagnosis should be the name of the disease that the patient has, e. g., multiple sclerosis. Assigning this name implies that the illness will follow a course similar to that of other patients with the same disease. A diagnosis is a generalization (oversimplification) used for convenience of communication and thinking. Sometimes the findings cannot be expressed in terms of a disease, e. g., paralysis of unknown cause. In such cases the clinical problem is used as the diagnosis until the patient's disease becomes evident. Some clusters of findings commonly encountered with more than one disease have been named (assigned a diagnostic term). These clusters of findings are called *syndromes*. For example, leakage of protein into the urine, low serum protein, and edema are a common set of findings in long-standing diseases of the renal glomerulus. This constellation is called the nephrotic syndrome because it is not one disease, but a set of findings common to several diseases. Therefore, the patient may be diagnosed as having the nephrotic syndrome until the specific disease is known.

We will use the term *manifestations* to refer to the data that can be gathered about an individual patient, namely, symptoms, signs, and laboratory abnormalities. *Symptoms* are evidence of disease perceived by the patient, such as pain, a lump, or diarrhea. The written description of symptoms in the patient's record is referred to as the *history*. *Signs* are physical observations made by the person who examines the patient, such as tenderness, a mass, or abnormal heart sounds. Signs are recorded under *physical examination*. *Laboratory abnormalities* in the broad sense refers to the observations made by tests or special procedures, such as X-rays, blood counts, or biopsies.

Structural diseases are those diseases characterized by structural changes within the body as the most basic abnormality. They are also referred to as *organic diseases*. Structural changes are called *lesions* and may be biochemical or morphologic (visible). The term *lesion* is most often used in reference to morphologic change whether it be at the gross (naked eye), microscopic, or electron microscopic

level[①]. Three broad categories suffice to classify most structural diseases, although some diseases fall into more than one category and some are difficult to classify.

*Genetic and developmental diseases* are defined as diseases that are caused by abnormalities in the genetic makeup of the individual (genes or chromosomes) or abnormalities due to changes in uterus (during embryonic and fetal development). The range of abnormalities in this category is very broad, extending from deformities present at birth (congenital anomalies) to biochemical changes caused by genes but influenced by environment so they appear later in life, e.g., diabetes mellitus. Due to overlap in classification schemes many genetic and developmental diseases can also be classified as injuries, inflammations, proliferations, or even neoplasms.

*Acquired injuries and inflammatory diseases* are diseases due to internal or external forces or agents that destroy cells or intercellular substances, deposit abnormal substances (foreign bodies or materials produced by the body), or cause the body to injure itself by means of the inflammatory process. External agents of injury include physical and chemical substances and microbes. The major internal mechanisms of injury are vascular insufficiency, immunologic reactions, and metabolic disturbances. The direct effects of injury are referred to as *necrosis* if cells are killed in the injured area and *sublethal cell injury* if the injured cells are capable of recovery. Sublethal cell injury may also be called *degeneration*, but this term has many other vague connotations. The few types of necrosis have myriads of causes. Acute sublethal cell injury tends to appear similar regardless of cause or cells involved, but chronic sublethal cell injury has many variations. There are two general reactions to injury: inflammation and repair. *Inflammation* is a vascular and cellular reaction that attempts to localize the injury, destroy the offending agent, and remove damaged cells and other materials. *Repair* is the replacement of damaged tissue by new tissue of the same type and/or by fibrous connective tissue. Inflammation is a stereotyped response with several important variations. Unlike necrosis and inflammation, repair is greatly influenced by the type of tissue or organ that has been injured.

*Hyperplasias and neoplasms* is a category used to describe diseases characterized by increases in cell populations[②]. Repair also may involve increases in cell populations, but the purpose is obviously to replace that which has been lost. In hyperplasia and neoplasia, the cell increase is beyond normal. *Hyperplasia* is a proliferative reaction to a prolonged external stimulus and will usually regress when the stimulus is removed. *Neoplasia* is presumed to result from a genetic change producing a single population of new (neoplastic) cells, which can proliferate beyond the degree allowed by the mechanisms that normally govern cell proliferation. Neoplasms are divided into two groups, benign and malignant, based on whether they will remain localized (benign) or will continue to grow and spread (malignant). *Cancer* is synonymous with malignant neoplasm. The situation is made more complicated by certain types of hyperplasias that slowly evolve, presumably through a series of genetic changes induced by external agents, into malignant neoplasms.

Functional diseases are those diseases in which the onset begins without the presence of any lesions (biochemical or morphologic). The basic change is a physiologic or functional change and is referred to as a *pathophysiologic change*. Some long-standing functional diseases may, however, lead to secondary structural changes. Either organic or functional diseases can have manifestations that are either structural or functional in nature.

Many mental illnesses are considered functional disorders, although some may have a genetic or other organic basis. The more mental illnesses are investigated, the more it is appreciated that there is likely to be an organic basis (on a biochemical level) to many of them. The most common functional disorders are tension headache and functional bowel syndrome, disorders that probably are due to unconscious stimulation of the autonomic nervous system.

## WORDS AND EXPRESSIONS

manifestation [ˌmænifesˈteiʃən] *n*. 表现,显示,现象
furthermore [ˈfəːðəˈmɔː] *ad*. 而且,此外
pathogenesis [ˌpæθəˈdʒenisis] *n*. 发病机理,发病机制
synonym [ˈsinənim] *n*. 同义词
diagnosis [ˌdaiəgˈnəusis] *n*. 诊断
sclerosis [skliəˈrəusis] *n*. 硬化(症)
imply [imˈplai] *vt*. 意指,暗示
generalization [ˌdʒenərəlaiˈzeiʃən] *n*. 概括,普遍化
oversimplification [ˈəuvəˌsimplifiˈkeiʃən] *n*. 过分简单化
paralysis [pəˈrælisis] *n*. 麻痹,瘫痪
leakage [ˈliːkidʒ] *n*. 漏,泄漏
edema [iˈdiːmə] *n*. 浮肿,水肿
renal [ˈriːnəl] *a*. 肾脏的,肾的
glomerulus [gləuˈmerjuləs] *n*. 小球,血管小球
constellation [ˌkɔnstəˈleiʃən] *n*. 群集,集体作用因素
nephrotic [neˈfrɔtik] *a*. 肾病的
diarrhea [ˌdaiəˈriə] *n*. 腹泻
tenderness [ˈtendənis] *n*. 压痛,触痛
morphologic [ˌmɔːfəˈlɔdʒik] *a*. 形态学的
gross [grəus] *a*. 肉眼能看到的
suffice [səˈfais] *vi*. 足够
embryonic [ˌembriˈɔnik] *a*. 胚胎的
fetal[ˈfiːtl] *a*. 胎儿的
deformity [diˈfɔːmiti] *n*. 畸形,残废
anomaly [əˈnɔməli] *n*. 异常,反常,畸形物
overlap [ˌəuvəˈlæp] *vt*. & *vi*. 部分重叠
scheme [skiːm] *n*. 系统,体制
proliferation [prəuˌlifəˈreiʃən] *n*. 增生,增殖
intercellular [ˌintəˈseljulə] *n*. 细胞间的
deposit [diˈpɔzit] *vt*. 贮存,沉淀
microbe [ˈmaikrəub] *n*. 微生物,细菌
insufficiency [ˌinsəˈfiʃənsi] *n*. 不足

disturbance [dis'tə:bəns] *n*. 障碍,失调
necrosis [nɛ'krəusis] *n*. 坏死
sublethal [sʌb'li:θəl] *a*. 尚不致命的
connotation [ˌkɔnəu'teiʃən] *n*. 涵义,内涵
myriad ['miriəd] *n*. 无数,极大数量 *a*. 无数的
regardless [ri'gɑ:dlis] *a*. 不留心的,不注意的
~of 不顾,不管
hyperplasia [ˌhaipə'pleizjə] *n*. 增生,增殖,数量性肥大
neoplasia [ˌniəu'pleizjə] *n*. 瘤形成
proliferative [prəu'lifəreitiv] *a*. 增生的,增殖的
regress [ri:'gres] *vi*. 退回,倒退,回归
presume [pri'zju:m] *vt*. 假定,认定,推测
neoplastic [ˌniəu'plæstik] *a*. 赘生物的,瘤的
synonymous [si'nɔniməs] *a*. 同义的
presumably [pri'zju:məbli] *ad*. 推测起来,大概
pathophysiologic [pæθɔˌfiziə'lɔdʒik] *a*. 病理生理学的

## NOTES TO THE TEXT

1. The term *lesions* is most often used in reference to morphologic change whether it be at the gross (naked eye), microscopic, or electron microscopic level.

   "病变"这一术语最常用于表示形态学上的变化,不论是肉眼可见的、显微镜下或电子显微镜下观察到的。

   词组 in reference to 意思为"关于"。从属连词 whether 在本句中引起让步状语从句,从句中出现虚拟语气,省略情态动词 should,表示假定的情况。

2. Hyperplasia and neoplasms is a category used to describe diseases characterized by increases in cell populations.

   增生和肿瘤是一类用以描述以细胞数量增多为特征的疾病。

   过去分词短语 characterized by 是医学文献中常用的短语,意思是"其特征为…",还可用 marked by...来表示。

## SUPPLEMENTARY READING

### Medicine as a Science

Medicine in part is a branch of applied biology. The substance of biologic science underlies most of the medical progress of the past half century which has so remarkably advanced the ability of the physician to intervene in illness. Much of this progress has been in fundamental or "basic" science, conducted in the pursuit of truth for its own sake. Significant progress has also resulted from research conducted by physician-scientists with a specified clinical goal in mind — for example, the explanation of a dis-

ease mechanism. Advances in medicine also continue to occur simply by careful clinical observations concerning patients and their illnesses, but these are now the exceptions.

The present bioscientific character of medical practice is a relatively recent development. Throughout most of recorded history medicine was anything but scientific. Diagnoses were inexact, causes of diseases poorly understood, and therapies often ineffective.

Signs of change emerged slowly in the early nineteenth century, as new principles of physics and chemistry were applied to medicine. Physiologists stressed functions of organs and tissues. Pathologists, led by Virchow (1821-1902), stressed the critical study of normal and abnormal tissues and the correlation of features of disease with precise anatomic observations. Bacteriologists, with Pasteur (1822-1895) and Koch (1843-1910) in the vanguard, began to identify the microorganisms and to implicate specific organisms in specific diseases. The groundwork for future therapies was being laid by these great scientists.

Slowly, specific therapies or specific immunizations appeared. But it was not until the decade 1935-1945 that the entry of sulfonamides and penicillin into clinical medicine made curable a large number of previously lethal and untreatable diseases. It is customary to date the beginnings of modern medicine from these relatively recent events.

The language of contemporary biologic science has become increasingly biochemical. The compositions of organs, tissues, cells, and membranes have been defined. The regulation of body processes has been described at progressively finer levels, and in chemical language. Many pharmacologic agents are now understood in terms of specific loci and mechanisms of action. The expansion of new knowledge continues at a pace that is bewildering to all but experts in a given field. Current advances are particularly rapid in immunology, molecular biology, and peptide research.

We have entered a molecular age of basic biologic science, and molecular biology is now a recognized discipline. The molecular influence pervades all the traditional disciplines underlying clinical medicine.

Medicine is not only a branch of applied biology, however. It also includes many aspects of psychology, sociology, anthropology, and economics. These disciplines, too long neglected, are now increasingly recognized as closely related to medicine as a discipline and the practice of medicine as a profession.

## MEDICAL CONVERSATION

| | |
|---|---|
| 你叫什么名字? | What is your name? |
| 我叫王明。 | My name is Wang Ming. |
| 你住在什么地方? | Where do you live? |
| 我住在香港。 | I live in Hong Kong. |
| 你多大岁数了? | How old are you? |
| 我 40 岁了。 | I am 40 years old. |
| 你是做什么的? | What is your occupation? |
| 我是工人。 | I am a worker. |

你有什么不舒服?
我咳嗽,咽喉发炎。
你病多长时间了?
我从昨天开始病的。

What trouble do you have?
I have a cough and a sore throat.
How long have you been sick?
I have been sick since yesterday.

# LESSON TWO

## TEXT

### Exercise and the Immune System

At the recent Olympic Games, some of the world's best athletes were unable to compete due to illness or injury through over-training, for example, the respiratory infection that struck UK Olympic gold medallist Sebastian Coe during the UK Olympic trials in August so affected his running that he failed even to qualify for the team to go to Seoul. The immune system may be a limiting factor in human performance. While some individuals can withstand rigorous training and competition schedules without missing a day, others are very susceptible to colds and infections. A robust immune system will allow an athlete to stay free of infection in the same way that a robust physique will allow him to remain free of injury.

Over-training is blamed by top athletes and their coaches for the illnesses that afflict them with increasing frequency as the competitive season progresses. These illnesses range from frequent persistent colds, sore throats and flu-like illnesses to severely debilitating states resembling post-viral fatigue syndrome, which can cause the athlete to miss an entire season, or even force him to give up competitive sports completely. Athletes are often reported to be suffering from glandular fever. But this is a convenient term to fit vague clinical symptoms, and only rarely is the diagnosis of infectious mononucleosis confirmed. For example, Sebastian Coe's so-called glandular fever was eventually diagnosed as a toxoplasma infection some five years ago. Earlier this year the UK 800-metre runner Diane Edwards was also diagnosed as having toxoplasmosis. The incidence of opportunistic and other infections among top athletes raises the question: can too much exercise have a damaging effect on the immune system?

Although moderate exercise appears to stimulate the immune system, there is good evidence that intense exercise can cause immune deficiency. We still do not really know why.

### Moderate Exercise Boosts the Immune System

Many recreational runners claim a state of physical well-being in which they are less susceptible to respiratory infection consequently, so some studies have set out to examine the possibility that regular exercise augments some aspects of host immunity.

None of the research in man has monitored immune function before and after a regular exercise programme, but two such studies in laboratory mice show clear evidence of immune enhancement. But laboratory mice are chronically under-exercised compared with wild mice. Perhaps being unfit impairs the immune system and the moderate exercise schedules boost their immune responses. If human volunteers who take little or no regular exercise were monitored during an exercise programme, there might also be evidence of immune enhancement.

## Immune Deficiency in Elite Athletes

Studies on elite endurance athletes suggest that regular intense training can have longer-term damaging effects on the immune system.

Members of the US national cross country ski team were found to have significantly lower resting salivary IgA levels than control subjects, and these decreased further after a 50 kilometre race (2-3 hours of exhaustive exercise). To determine whether this decrease in IgA levels might be due to the exercise itself, or might be due to the effects of cold, to the stress of competition, or to a combination of factors[①], Tomasi and his colleagues conducted a study of competitive cyclists. No differences were found between resting salivary IgA levels in cyclists and controls. However, a 70% decrease in salivary IgA levels was observed immediately after intense endurance exercise. This was not due to the effects of cold, nor to the stress of competition, since they cycled for two hours in the laboratory, in a controlled temperature and a non-competitive setting. There was also a significant reduction in NK cell activity. The reduction in IgA and NK cell activity was transitory, returning to pre-exercise levels 24 hours after this single bout of severe exercise. The authors conclude that prolonged intense exercise alters parameters of mucosal and natural immunity, and suggest that severe exercise may itself be a form of stress associated with changes in immune reactivity, since the changes occurred in the absence of cold or competition. The authors comment that the suppressive effects of intense exercise may be cumulative, which would explain the low resting IgA levels in their study of skiers who were tested at the end of the competitive season, yet normal levels in the cyclists tested earlier in the season. Low resting serum IgA levels in elite ultra-distance runners tested at the end of the season have also been found.

Other defects noted in marathon runners at rest include low total lymphocyte counts (less than 1,500 per cubic mm) particularly in those with marathon times of less than 2 hours 25 minutes.

Non-specific immunity is also suppressed by regular intense exercise. Twenty conditioned competitive cyclists and 19 untrained control subjects exercised to exhaustion in the laboratory and even at rest neutrophil adherence was lower in sportsmen than in controls, and maximal exercise revealed further differences between the two groups. Adherence of neutrophils and monocytes, and neutrophil bactericidal activity, all decreased significantly in the sportsmen following exercise, but did not change significantly in untrained men. Neutrophil phagocytic activity did not change in the sportsmen following exercise, but was significantly increased in untrained men. The authors conclude that long-term physical exercise depresses non-specific immunity, possibly rendering sportsmen more susceptible to infections.

The studies on elite sportsmen have, therefore, all revealed some immune abnormalities in the resting state, any or all of which could account for the increased susceptibility to infection seen among these athletes. No studies have specifically tested elite athletes before and after specific training schedules, even though it is those who push themselves to the limits necessary to compete at top level who are most at risk of exercise-induced immune suppression[②].

## Possible Mechanisms

A variety of hormones and chemicals are released during exercise. Cortisol and cate-cholamines are