VISUAL DIAGNOSIS

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CHRONIC OBSTRUCTIVE PULMONARY DISEASE

慢性阻塞性肺部疾病

Paul S Thomas
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《影像学诊断问答》系列

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FOREWORD

对慢性阻塞性肺部疾病(COPD)的理解及怎样对其进行治疗不断取得令人鼓舞的新进展。治疗技术的一体化不断改善着患这种衰弱性疾病的病人的生活质量,但对这种病来说,医生的首要目的是要帮助病人戒烟。这需要病人家人和朋友的共同支持,这是治疗 COPD 的基本前提。

《慢性阻塞性肺部疾病》一书的编写目的,是为了使临床医生通过回答书中所提供的,与慢性阻塞性肺部疾病和伴发疾病直接相关的病例病史和图解的有关问题,进行自我测试。通过回答从相对简单到疑难问答,读者会发现本书具有一定的挑战性、教育性和知识性,每一个病例下面所提问题的答案"以及治疗方案的选择都在下页中给出。

两位作者,Paul Thomas 博士和 Deborah Yates 博士,根据他们治疗 COPD 的丰富经验,为每一病例病史提供了实践上的探讨。一般行医人员、初级医生、住院医师、护士以及医学生将会发现,本书能作为自己日常工作中的参考书和学习指南。

前言

CONTENTS

Foreword ····· 1
Introduction ····· 1
Case 1 ····· 15
Case 2 ····· 20
Case 3 ····· 24
Case 4 ····· 29
Case 5 ····· 33
Case 6 ····· 37
Case 7 ····· 41
Case 8 ····· 45
Case 9 ····· 49
Case 10 ····· 53
Case 11 ····· 57
Case 12 ····· 61
Case 13 ····· 65
Case 14 ····· 68
Case 15 ····· 72
Case 16 ····· 75
Case 17 ····· 79
Case 18 ····· 83
Case 19 ····· 87

目录

前言 ····· 1
导言 ····· 1
病例 1 ····· 15
病例 2 ····· 20
病例 3 ····· 24
病例 4 ····· 29
病例 5 ····· 33
病例 6 ····· 37
病例 7 ····· 41
病例 8 ····· 45
病例 9 ····· 49
病例 10 ····· 53
病例 11 ····· 57
病例 12 ····· 61
病例 13 ····· 65
病例 14 ····· 68
病例 15 ····· 72
病例 16 ····· 75
病例 17 ····· 79
病例 18 ····· 83
病例 19 ····· 87

此为试读,需要完整PDF请访问: www.ertongbook.com

Case 20 …… 91
Case 21 …… 94
Case 22 …… 97
Case 23 …… 99
Case 24 …… 102
Case 25 …… 104
Case 26 …… 107
Case 27 …… 111
Case 28 …… 115
Case 29 …… 117
References …… 123

病例 20 …… 91
病例 21 …… 94
病例 22 …… 97
病例 23 …… 99
病例 24 …… 102
病例 25 …… 104
病例 26 …… 107
病例 27 …… 111
病例 28 …… 115
病例 29 …… 117
参考文献 …… 123

INTRODUCTION

导言

Historical background of Chronic Obstructive Pulmonary Disease (COPD)

While COPD is a late nineteenth century and twentieth century disease, some of the elements of the conditions which we now recognize have been present for many centuries. Hut-dwellers who are exposed to indoor smoke pollution, e.g. in Papua New Guinea, probably represent some of earliest types of disease which now might be classified as COPD. It is known that emphysema has been recognized for centuries, from the writings of Hippocrates and more recently, William Harvey in the 16th century (Whitteridge 1964), and Matthew Baillie in the 18th century (Snider 1988).

Tobacco smoking by pipe and filled reeds was discovered in the Central and South American countries in the 16th century probably having been a custom for at least a millennium. It was then introduced into Europe on the return of Spanish explorers, only to be exported again to North America with the settlement of the continent by European nations. Advocates of tobacco smoking, such as Jean Nicot, whose name became linked to the tobacco derivative, suggested that it had medicinal and aphrodisiac

慢性阻塞性肺部疾病(COPD)的历史背景

尽管 COPD 是 19 世纪晚期和 20 世纪的疾病,但我们现在认识到这一疾病的某些要素已经存在了许多世纪。居住在小茅草屋里的受烟熏火燎的人群,如像住在巴布亚新几内亚的人们,可能患有代表最早的现在被分类为 COPD 的某些类型的疾病。肺气肿已经被人们认识了好几个世纪,从希波克拉底(Hippocrates)和更近代的 16 世纪的威廉·哈维(William Harvey)(Whitteridge 1964)和 18 世纪的 Matthew Baillie(Snider 1988)的著作中就已论及到这种疾病。

16 世纪在中美洲和南美洲的一些国家就已发现人们用管子和芦苇杆抽烟,这种习俗可能存在了至少一千年。随着西班牙探险者的归来,这种烟草被传入欧洲,随着欧洲国家居民在北美洲大陆定居,烟草又出口到北美。吸烟的倡导者,像 Jean Nicot(他的名字很容易想到烟草衍化物),宣传说烟草有医疗和催欲的特性,但这种习惯所伴随发生的疾病也很快被其他人所认识到。英格兰国王詹姆士一世,如此评价吸烟:"吸烟是一种有害于眼睛,有损

qualities, while the association of the habit with the development of disease was also quickly recognized by others. King James 1 of England wrote upon smoking: 'a custom lothsome to the eye, hatefull to the nose, harmefull to the braine, dangerous to the lungs, and in the black stinking fume thereof, neerest resembling the horrible Stigian smoke of the pit that is bottomless' (Golding 1995).

于鼻子,有害于大脑,危害肺,因此这种发出臭味的黑色烟雾,简直就像可怕的 Stigian 烟,是无底的陷阱。"(Golding 1995)

Etiology

The smoking habit has fulfilled King James' promise that it is dangerous to the lungs. Apart from the toil of carcinoma of the bronchus, damage gradually occurs to the airway and the alveoli. The term 'chronic obstructive pulmonary disease' (COPD) was coined for this damage in 1964 in the USA. While emphysema and chronic bronchitis have been recognized for centuries, the concept of airway obstruction is probably more recent. Systematic efforts were made to introduce terms which had clear definitions which would aid clinical assessment and research.

病因学

吸烟的习惯对肺有危险,这应验了詹姆士国王的预言。撇开支气管肺癌的危害不谈,气道和肺泡也逐渐发生损害。"慢性阻塞性肺部疾病"(COPD)一词 在 1964 年于美国定名,以代表这种损害。尽管肺气肿和慢性支气管炎已被人们认识了几个世纪,但气道阻塞的概念可能是近期才提出的。这一系统性的成就指出了明确定义的范围,有助于临床评价和科学研究。

The Ciba Guest Symposium, in 1959, defined chronic bronchitis as persistent cough productive of sputum for three months of the year, for two consecutive years. Prior to this, chronic bronchitis was a diagnosis of exclusion, indicating chronic productive cough which was not attributable to other pulmonary diseases. Emphysema was defined as an increase in the size of the 'air

1959 年的 Ciba Guest 专题讨论会,将慢性支气管炎定义为:每年 3 个月的持续性咳嗽,产生痰,且持续两年之久。在这之前,慢性支气管炎采用的是排除性诊断,是指不能归因于其他肺部疾病性的产痰性咳嗽。肺气肿定义为"终末支气管远端肺泡体积增加,伴有肺泡壁的破坏,但没有纤维化"。

spaces distal to the terminal bronchiole accompanied by destruction of their walls and without fibrosis'.

This definition is difficult to use except in autopsy studies, and for this reason, and because there is no mention of the accompanying airway obstruction the term COPD was coined to encompass all of these points; to allow the combination of any degree of clinically apparent probable emphysema and any degree of airway obstruction, which is only poorly reversible either spontaneously or with treatment. While the term remains useful to communicate a particular broad clinical category, it may not help when trying to determine pathogenetic mechanisms. No clear definitions have evolved to indicate how asthma and COPD can be distinguished, which are important considerations for epidemiological work, but less important for clinical purposes, as many of the treatment strategies are similar.

这个定义除活检研究以外很难应用,因为这个原因,而且因为没有提到伴随的气道阻塞,所以创造了 COPD 这个术语来包括所有这些要点;这样就可以把那些有任何程度的临床上有明显可能的肺气肿和任何程度的难以逆转的、无论是自发性的还是经过治疗的气道阻塞合并成一类疾病。尽管这一术语在和特别广泛的临床分类相联系方面仍然有用,但对于确定致病机制并无帮助。没有明确的定义来说明如何区分哮喘和 COPD,这在考虑流行病学方面是很重要的,但在考虑临床目的方面就不那么重要了,因为二者有许多治疗策略是相似的。

Chronic mucus hypersecretion, which often abates upon smoking cessation, used to be considered an integral part of airflow obstruction but it would seem that this element of large airway disease, with mucous gland hyperplasia, is probably separate from that of chronic airway obstruction. In agreement with this concept, the clinical studies of Fletcher and Peto, and others which have studied those with chronic productive cough and those with a decline in FEV_1, have shown that the two disorders appear to

慢性粘液分泌过度,在戒烟后可能有所缓解,过去被认为是整个气道的一部分阻塞,但现在看来这类伴粘液腺增生的大气道疾病的要素,很可能不同于那些慢性气道阻塞性疾病。为确认这一概念,Fletcher 和 Peto 开展了临床研究,其他人也研究了慢性产痰性咳嗽以及伴有 FEV_1(1 秒钟用力呼气量)下降的疾病,结果表明,这两种疾病似乎是相互独立存在的。

be relatively independent.

In the USA, COPD has been estimated per annum to cause as many as 165,000 deaths two million episodes of hospital care and ten million out-patient visits. Estimates from population-based studies range from 4% - 8% in adult white males and 1% - 3% in adult white females. The prevalence rates in 1985 in men, was 100 per 1,000, while in females it had risen by 30% over the preceding five years, to 119 per 1,000. The incidence of COPD has been estimated upon spirometric abnormalities, and defined as a reduction in predicted FEV_1 (forced expiratory volume in one second).

在美国，据估计每年有 165 000 人死于 COPD，两百万人因 COPD 发作而住院，一千万人因 COPD 而看门诊，这是根据占总人口中的 4%～8%的成年白人男性和 1%～3%的成年白人女性的调查得出以上估计数据的。在 1985 年，男性流行率为 100/1000，而女性在过去的 5 年中流行率增加了 30%，达到 119/1000。COPD 的发生率采用呼吸量测定异常来估计，用预期的 FEV_1（一秒钟用力呼气量）下降来解释。

The survival of those with a severe reduction in FEV_1 is poor. In those with a FEV_1 of less than 30% predicted, half will die within five years. The only treatment that has been conclusively shown to improve survival is that of domiciliary oxygen for more than 15 hours per day in those with hypoxemia. Survival appears to more closely related to post-bronchodilator FEV_1. Inhaled anti-cholinergic agents have not yet shown a difference in survival, although they have clear benefits by providing additional bronchodilation in combination with β2 adrenergic agonists. Therapeutic use of glucocorticosteroids has not been studied in terms of long-term survival but several large studies are in progress to assess the role of inhaled glucocorticosteroids in COPD, both a-

那些 FEV_1 严重减少的病人的生存率很差。在那些 FEV_1 少于预期的 30%的病人，有一半将在 5 年内死亡。惟一得出结论能改善存活的治疗是，对低氧血症的病人，要每天在家吸氧 15 个小时以上。生存时间似乎与支气管扩张后的 FEV_1 有更为密切的相关性。吸入抗胆碱能药物并没有显示在生存时间上的差别，尽管这类药物与 β_2 - 肾上腺素能受体拮抗剂联用可以提供额外的支气管扩张而有明显益处。有关应用于治疗的糖皮质类固醇的远期存活率还没有研究成果，但几项大规模研究正在进行，以评价吸入糖皮质类固醇在治疗 COPD 中的作用，包括作为急性和慢性治疗。

cutely and as chronic therapy.

The role of bacteria in causing these deaths and medical episodes has remained controversial, despite much research. Acute respiratory tract infections cause symptoms, lead to morbidity and mortality and may be caused by either bacteria or viruses. Exacerbations are generally defined as an increase in sputum production over baseline, an increase in the purulence of the sputum and an increase in breathlessness. The average number of exacerbations is approximately two per annum, although there is wide variation between patients. Several studies have addressed the problem of trying to identify which micro-organism is responsible for an exacerbation, using a battery of tests which we would not usually request in the routine management of such patients. It would appear that approximately one-third of exacerbations are caused by viruses, while one-half are caused by bacteria, and the so-called atypical organisms account for a small proportion, which may vary from year to year.

尽管做了许多研究，但细菌在引起死亡和医学突发事件的作用仍存在争议。急性呼吸道感染会引发症状，导致发病和死亡，并可由细菌或病毒引起。病情加重一般定义为痰产生增加超过基线，痰的脓性增大以及屏气增多。尽管病人之间存在很大差异，但每年的平均加重次数约为两次。有几项研究采用我们常规治疗这类病人时通常不需要的一组试验，以期鉴别引起病情加重的微生物。结果显示，大约有 1/3 的病人病情加重是由病毒引起的，而 1/2 是由细菌引起的，并且所谓的非典型微生物占的比率很小，且逐年都会有所变化。

At least three respiratory pathogens can chronically colonize the respiratory tract in such patients, namely non-encapsulated *Haemophilus influenzas*, *Moraxella* (*Branhamella*) *catarrhalis* and also *Streptococcus* pneumoniae. Whether *Chlamydia* pneumonia can also play a role as a chronic colonizing organism is the subject of current research, but it is becoming apparent that

在这些病人中，至少有三种呼吸道病原体可以慢性移生到呼吸道，分别是无荚膜流感嗜血杆菌、卡他莫拉菌（卡他布兰汉球菌）及肺炎链球菌。肺炎衣原体能否作为一种慢性移生病原体而发挥作用是当前研究的课题，但很明显这种微生物在 COPD 中起着重要作用。现已证实，抗生素治疗在对 COPD 症状加重的治疗中是有益的。

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this organism may well play an important role in COPD. Antibiotic therapy has been proved to be of benefit in those with an exacerbation of COPD.

Usually an oral antibiotic will suffice and the choice is largely dictated by the sensitivities of the above organisms, and recent sputum culture. In general, the antibiotics will range from ampicillin, amoxycillin/clavulanic acid, trimethoprim/sulfamethoxazole or tetracycline, to newer agents such as second generation macrolides, quinolines and cephalosporins.

一般口服抗生素就足够了，抗生素的选择取决于上述微生物的药敏性，以及最近的痰培养结果。抗生素一般包括氨苄西林、阿莫西林/克拉维酸、甲氧苄啶/磺胺甲噁唑或四环素，以及某些新药，如第二代大环内酯类药物、喹啉及头孢菌素。

The role of bacteria in the chronic phase of the disease is much less clear. Those with chronic bronchitis have been demonstrated to have chronic colonisation of their airways by bacteria, which may persist because of the mucus hypersecretion. The bacteria may also benefit from this abnormality, since antigenic components of bacteria can by themselves generate an inflammatory response, and thus increase mucus hypersecretion.

细菌在疾病慢性阶段的作用不很清楚。患慢性支气管炎的患者气道内有细菌移生已被证实，这些细菌会因为粘液过度分泌而持续存在。细菌可以从这种异常中获得好处，因为细菌的抗原成分可以自身产生炎症性反应，从而增加了粘液分泌过度。

Tobacco smoking has been conclusively proved to cause COPD, both in terms of decline in FEV_1 and also as a dose response effect in the generation of both airway obstruction and emphysema. It is noted, however, that while few non-smokers ever develop these diseases, not all individuals who smoke will suffer from COPD. In some, inherited defects of the control of enzyme

现已结论性地证实，吸烟会引起 COPD，不但可以降低 FEV_1，还会以剂量反应效应方式造成气道阻塞和肺气肿。然而，尽管很少有不吸烟者发展成这类疾病，但并非所有的吸烟者都患 COPD。有些病人，控制酶降解的遗传缺陷可解释这种倾向，例如 α-1 抗胰蛋白酶（AAT）缺陷。AAT 缺陷的病人在肺气肿病人当中只占一小部分。剩余

degradation may account for such a tendency, e.g. alpha-1 anti-trypsin (AAT) deficiency. Those with AAT deficiency are a small minority of those with emphysema. The remainder, who comprise the vast majority, may have additional, as yet undescribed defects in their ability to neutralize degradative enzymes, but none has become apparent despite much research. Other researchers have considered the balance of oxidative and anti-oxidative forces to be important, but again there is little that clearly indicates the rasons why some individuals are nore prone to lung damage from cigarettes than others.

的大多数肺气肿病人可能有另外一些还未被描述过的中和降解酶能力的缺陷,尽管进行了很多研究,但还不是很清楚。其他研究者认为氧化和抗氧化能力的平衡很重要,但同样,这也无法解释清楚为什么有些人较另一些人更容易患由于吸烟所造成的肺损害。

The recognized group of urban pollutants that include oxides of nitrogen, sulfur dioxide, carbon monoxide and particulate material, are undoubtedly harmful to those with pre-existing respiratory and cardiac disease. Their role in the development of COPD is small in comparison with cigarette smoking. Indoor pollutants also appear to predispose to an increase in respiratory symptoms, while the use of solid fuel without adequate ventilation has been described, as aiding the development of COPD in communities in India and Papua New Guinea.

公认的一组城市污染物,包括氮的氧化物、二氧化硫、一氧化碳和粒子物质,无疑对有潜在呼吸系统和心脏疾病的人是有害的。它们在 COPD 发病过程当中所起的作用与吸烟相比是很小的。室内污染物看起来也会增加呼吸系统症状,而在印度和巴布亚新几内亚的一些社区里,人们认为正是在通风不良的情况下使用固体燃料促进了 COPD 的发病。

It has been very difficult to assess the location of workplace pollutant in the development of COPD. Many workers in these dusty and polluted environments have been heavy smokers themselves. Over the past years, however, it has become clear that

想要评估工作地点污染物对 COPD 发病中的作用是非常困难的。因为许多在这些充满灰尘和污染的环境中工作的工人本身又是重度吸烟者。然而,在过去的几年中,越来越清楚地发现煤矿和金矿工人、纺织工人、农民、

there is an additive effect above that of cigarette smoke from the dusts inhaled by coal and gold miners, cotton workers, farmers grain handlers and those in the cement industry. The exact place of noxious gases and fumes in the development of COPD is less clear. Oxides of nitrogen, ammonia and sulfur dioxide have been suggested as causing COPD, although the evidence is not yet completely convincing. It also appears that cadmium workers are at an increased risk of developing emphysema.

谷物搬运者以及那些从事水泥工业的工人吸入灰尘对吸烟有加合作用。有毒气体和烟雾对 COPD 发病的确切作用还不太清楚。尽管还没有证据完全证实，但氮的氧化物、氨和二氧化硫被认为是引起 COPD 的气体。制镉工人看来也属于肺气肿发病的高危人群。

COPD, airway hyper-responsiveness and allergy

In the early 1960s, Orie et al, suggested that the airway obstruction in COPD may be caused by the association of smoking with an asthmatic or allergic tendency. This became known as the 'Dutch hypothesis'. Since that time, much effort has gone into confirming or refuting this idea. It has become clear that those who smoke have a tendency towards higher levels of lgE and blood eosinophilia. There have been no longitudinal studies delineating atopy and asthmatic features in the young before they take up smoking or not, and then studying them again in later life. This might shed further light both on the risk of developing COPD in later life and the reason for the increase in IgE and peripheral blood eosinophils in the smokers.

COPD，气道高反应性和过敏症

在 60 年代早期，Orie 等人就提出，COPD 的气道阻塞可能是由哮喘或过敏倾向与吸烟联合作用造成的。这是所谓"荷兰假说"。从这以后，人们做了很多努力来证实或驳斥这个观点。现在已经很清楚，吸烟的人有 IgE 水平升高和血液中嗜酸性粒细胞增多的倾向。没有做纵向研究，描述吸烟之前或不吸烟的人在年轻时的特异性变态反应及哮喘特征，并在其生命晚期再进行研究。这可以进一步阐明生命晚期发展成 COPD 的危险性以及吸烟者的 IgE 升高和外周血嗜酸性粒细胞增多的原因。

Those who smoke have a greater degree of airway hyper-responsiveness. Some studies have indicated that this may correlate

吸烟的人，气道高反应的程度会更高。有些研究表明，这可能与气道的炎症程度有关，但气道壁涉及的炎症

with the degree of airway inflammation, but the inflammatory cells involved in the airway wall tend to be neutrophils rather than the eosinophils, lymphocytes, and mast cells seen in the mucosae of the asthmatic subjects. Part of the hypothesis implies that those with both asthma and smoking, should have the most rapid decline in lung function, but data on this area are lacking. What does appear to be clear from the Lung Health Study, is that those smokers with airway hyper-responsiveness (as assessed by methacholine challenge), had the greatest decline in subsequent lung function as measured by FEV_1. This does not prove or disprove the original hypothesis.

细胞为中性粒细胞，而不是哮喘病人粘膜中所见的嗜酸性粒细胞、淋巴细胞以及肥大细胞。此假说的一部分暗示，吸烟并且哮喘的人肺功能应该下降得最快，但缺乏这方面的资料。肺健康研究所的研究清楚地表明，伴气道高反应性（用醋甲胆碱诱发试验进行评价）的吸烟者，随后的肺功能（通过 FEV_1 测量）下降得最快。这个结果既未证实也未否定最初的假说。

Mortality rates from COPD in the USA appear to be higher in whites, and are inversely associated with socio-economic class. Even in the absence of AAT deficiency, there can be a familial tendency to develop COPD.

在美国，COPD 的病死率在白种人中较高，并且与社会经济地位成反比。即使是没有 AAT 缺乏症，患 COPD 也会有家族倾向。

Pathophysiology

In the normal person, the small peripheral airways offer very little resistance to the expiratory flow of gas from the lungs. These airways measuring approximately less than 3 mm, are probably the major site of disease in COPD. Since they normally are a low-resistance system, a large amount of disease has to occur before gas exchange is compromised. In addition, the features of emphysema may also arise in association with the destructive

病理生理学

在正常人中，小的周围气道对于从肺中呼出的气流有非常小的阻力。这些气道，直径测量大约小于 3mm，可能是 COPD 的主要病变部位。因为它们在正常时是低阻力系统，在气体交换受损害之前一定发生了大量的病变。此外，肺气肿的特征可能也随着吸烟对终末呼吸性支气管、肺泡管和肺泡壁的破坏而出现。因此，早期肺气肿很难发现，并且是无症状的。有些敏感性但非特异性的试

effects of cigarette smoking upon the terminal respiratory bronchioles, alveolar ducts and alveolar walls. Early emphysema is therefore difficult to detect and a symptomatic. There are some sensitive, but non-specific tests which may detect such early changes such as the single breath nitrogen test, and some complex lung function tests of compliance and resistance. The most widely used method in clinical practice is the flow-volume curve and the maximum expiratory flow (MEF). Since the latter part of this curve is considered to represent the late expiratory effort to which small airways contribute the most, then a reduction in this flow at the end of an expiratory effort should demonstrate the maximum abnormality in COPD. Unfortunately, this part of the expiratory manoeuvre is often the least reproducible and can lead to difficulties in interpretation and detection of COPD.

验可以检测到这种早期变化，如单次氮气呼吸试验，以及一些检查顺应性和抵抗性的复杂肺功能试验。在临床实践中，应用最广泛的方法是流量-体积曲线和最大呼气量(MEF)。因为曲线的后面部分被认为是代表小气道在其中最起作用的晚期用力呼气，所以用力呼气终末部分的流量减少应该证明 COPD 的最大异常。不幸的是，这一部分的呼气操作方法常常难以重复，这可能给解释和发现 COPD 带来一定困难。

At an early stage of COPD, there are few symptoms and there are few indications of large airway dysfunction. The larger airways are usually only affected later as the disease progresses, when the patient breathes out by forced expiration and dynamic compression of these large airways occurs.

在 COPD 早期，很少有症状和大气道功能障碍的指征。更大的气道通常只有在疾病过程的晚期才受累及，此时病人只能靠用力呼气呼吸，并且这些大气道出现了动力性压迫。

Smokers can often be demonstrated to have abnormal lung function by the age of approximately 40 years, and from this point can be demonstrated to have a faster decline in lung function. By the time breathlessness upon exertion supervenes the basic

常可证实吸烟者在大约 40 岁时即出现肺功能异常，并且从这一时刻起，可证实其肺功能下降很快。在用力时屏息状况下进行了基础呼吸量测定，表明 FEV_1 和最大肺活量(FVC)的比例是异常的。如果有一定程度的肺气

spirometric tests, indicating FEV_1 and its ratio to forced vital capacity (FVC), are abnormal if there is a degree of emphysema then the total lung capacity (TLC) will be increased and there may be a loss of static lung compliance with a reduction in the elastic recoil of the lung. The combination of inhomogenous ventilation, combined with gas trapping in association with emphysema, leads to a reduction in effective gas exchange which is the mechanism causing hypoxemia.

肿,那么肺总容量(TLC)将会增加,可能有肺性静态顺应性下降,伴肺的弹性回缩力下降。结合不均匀性的换气,结合伴肺气肿的气体俘获,造成了有效气体交换减少,这是造成低氧血症的机制。

There is often elevated pulmonary arterial pressure in those with COPD, mainly secondary to the functional effect of hypoxemic vascular constriction. In addition, the hypoxia leads to structural changes which includes vascular muscular hyperplasia and intimal fibrosis. As pulmonary hypertension gradually progresses, cardiac output is preserved initially by a smaller stroke volume and an increased heart rate. Later, as pulmonary arterial pressure continues to rise, some patients progress to develop right heart failure with sodium retention. decreased renal perfusion, and activation of the renin-angiotensin pathway.

患 COPD 的那些病人常常有肺动脉压升高,主要继发于低氧血症血管收缩的功能效应。此外,缺氧造成了结构的改变,包括血管肌肉增生和内膜纤维化。随着肺高压的逐渐进展,开始通过弱小的心搏排血量和心率增加来维持心输出量。以后,随着肺动脉压继续上升,有些病人演变发展成右心衰,伴钠潴留、肾灌注量下降及肾素-血管紧张素路径的激活。

Diagnosis

This book intends to bring forth the definitions and treatment of COPD in a question and answer format, but the essentials of history taking, with description of the disability, estimation of smoking exposure, and relative contributions of lung destruction

诊断

本书的意图是以问答的形式阐明 COPD 的定义和治疗,但也考虑病史收集、伤残性的描述、暴露于吸烟的评估以及肺破坏和气道阻塞的相对成因等基本要素。临床病史需要包括其他常见疾病,特别是吸烟所伴发的那些

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