A changing industrial environment in the western world during the past decades has resulted in a considerable change in the spectrum of occupational and environmental lung disorders that we observe nowadays in medical practice. Modern radiological imaging is an important diagnostic tool for the correct management of these diseases.

This volume, unique in its concept, not only covers in a comprehensive way the imaging features of the well-known coal worker’s pneumoconiosis and the severe forms of silicosis and asbestosis, but also deals extensively with the effects of new organic and inorganic materials, used in the modern chemical industry as well as with the noxious effects of cigarette smoking.

The eminently readable text is complemented by superb illustrations.

The editors, P.A. Gevenois and P. De Vuyst are well-known experts in the field. The authors of the individual chapters are outstanding specialists in the epidemiology, etiology, immunology, pathology, pathophysiology, and radiology of dust inhalation diseases. I would like to thank and to congratulate most sincerely the editors and the authors for their top-level contributions.

This superb book will be of great value for general and chest radiologists but also for pneumologists and all those active in occupational and environmental medicine. It provides them with the latest information on a very interesting medical field with an important medicosocial impact.

I am confident that it will meet the same success with the readers as many previous volumes in this series.

Leuven

Albert L. Baert
The spectrum of classical pneumoconiosis has changed during the past decades in industrialized countries: as a result of better control of air dust levels and reduction of workforce in large sectors of industry, the incidence of severe forms of silicosis, coal worker’s pneumoconiosis and asbestosis (lung fibrosis), has decreased over time. Most incident cases of disabling pneumoconiosis result from exposures dating back several decades ago.

Parallel to the reduction of exposures in industries using natural minerals, the development of the chemical industry has led to the production of large numbers of organic and inorganic materials, including metallic alloys. More and more of these substances, including manmade organic particles, are reported as causes of interstitial lung disease in groups of exposed workers. Many different patterns of interstitial lung diseases have been reported: lung fibrosis, lung granulomatosis, giant cell pneumonitis, non specific interstitial pneumonia, chronic organizing pneumonia. Without a careful occupational history-taking and/or inquiry, all these forms may mislead the etiological diagnosis towards their respective idiopathic forms.

The major tools for diagnosis of pulmonary and pleural diseases are the imaging techniques to which this book is dedicated. Chest film reading and scoring according to the Classification established by the International Labour Office (ILO) is a standardized and wide-world used method and represents a common language between people working in this field, including in developing countries. This system facilitates for example the interpretation and comprehension of the epidemiological literature on pneumoconiosis. Many compensation systems rely on the presence of grade 1/1 or 1/0 small opacities on a plain chest film. This is however not the most sensitive to detect early changes and not the most specific one to diagnose pneumoconiosis in individuals. Indeed, the prevalence of small irregular opacities in an adult smoking population is high and the interpretation of films is subject to important inter and intra-observer variability in boundary grades, which are crucial in the acceptance of pneumoconiosis. This issue is even more important since incident cases of obvious pneumoconiosis with large opacities or high ILO grades have become exceptional. Computed tomography (CT) has largely been reported as more sensitive and more specific than chest radiograph and is thus now widely used for the diagnosis and compensation of pneumoconiosis. One of the consequences is the detection of abnormalities consistent with pneumoconiosis in patients without symptoms or lung function alterations. On the other hand, CT studies have been invaluable in the description and the distinction of the various forms of dust-related lesions, such as rounded atelectasis, diffuse pleural thickening and pleural plaques.

The majority of workers who are exposed to asbestos nowadays are end-users, in contact with asbestos still in place in buildings such as electricians, plumbers, demolition workers, asbestos removers… Most of them are self-standing workers, without any medical control or surveillance and often working without protective devices. The currently diagnosed cases are principally non malignant pleural lesions with little or no effect on lung function and the main cause of asbestos-related deaths among them is malignant mesothelioma rather than respiratory failure due to lung fibrosis. Since the incident cases of true asbestosis are rare, the development of lung fibrosis in a person with low cumulative exposure and/or low concentrations of asbestos bodies and fibers in bronchoalveolar lavage raise the possibility of idiopathic pulmonary fibrosis (IPF). Unusual exposures may however still be at the origin of severe
diseases. Environmental exposures to tremolite asbestos have been documented in Turkish residents and migrants with burdens of fibers equal to those seen in industrial settings and subsequent asbestos-related diseases, including asbestosis.

Occupational lung diseases have also extended their links and limits. There is a clear association between silicosis or silica exposure and connective tissue disorders such as rheumatoid arthritis, systemic sclerosis and ANCA-positive vasculitides. Even “IPF” has been shown in several epidemiological studies associated with occupational exposures to metals or wood dusts. These studies beside exclude patients with asbestos or silica exposures of any importance, considering them respectively as having asbestosis or silicosis, irrespective of low cumulative exposures and/or radiological abnormalities not consistent with pneumoconiosis. The proportion of « IPF » cases with exposure to exogenous dust may be more important than previously thought. In this regard, it is interesting to note that coal miners may develop an IPF-like disease with a honeycomb pattern on thin-section CT rather than a classic coal worker’s pneumoconiosis, and that these cases are now compensated in France.

Occupational agents may interact with other agents, infectious or not. Drugs can induce or trigger interstitial and/or pleural disease and this may induce confusion if these changes develop in persons with prior occupational exposure. This is particularly true for asbestos, and several cases have been reported of patients with prior asbestos exposure, who developed rapidly progressive pleural thickening or effusion, while being treated with bromocriptine for Parkinson’s disease.

There is still important clinical and basic research work in occupational and environmental diseases. The research areas concern the description of new diseases due to organic and inorganic materials, nanotoxicology, mineralogical studies on the lung, genetical and immunological susceptibility to pneumoconiosis...There are indeed differences in the individual susceptibility to the adverse effects of chemicals and metals. Berylliosis and hard metal disease may be observed after exposure to low-doses, and are known to affect only a minority of exposed workers. Important advances have been made in basic research on the immunogenetic basis of berylliosis. A human HLA class II mutated gene was found to be strongly associated with clinical berylliosis and probably with hard metal disease.

Imaging, even by CT, is crucial, but only a part of the diagnosis of occupational disease. Many patterns of interstitial lung disease, can be either idiopathic or due to an exogenous cause. Other diagnostic tools are essential. They include a careful occupational and environmental history taking, which may necessitate a visit of the house or of the workplace. Some diagnoses, especially in the field of hypersensitivity pneumonitis require sagacity worthy of Sherlock Holmes himself! They also may require mineralogical studies on bronchoalveolar lavage, immunological tests (serum precipitating antibodies, lymphocyte transformation tests...) and in some cases lung biopsy.

The diagnostic work-up in occupational and environmental lung and pleural diseases needs sometimes more than a simple chest plain film with lung function tests. In clinical practice, this can not be accepted as sufficiently accurate to confirm or refute the diagnosis of pneumoconiosis in a dust-exposed worker with interstitial lung disease. Advances in imaging, mineralogical, pathological and immunological techniques have been instrumental in describing new patterns of disease and they allow a comprehensive approach to occupational and environmental disorders. This is crucial for making a correct clinical diagnosis and for not missing treatable diseases, in the description of new patterns of diseases, and for making scientifically based expertise of difficult or litigious cases. The changing spectrum of environmental and occupational diseases makes thus essential very close collaboration between radiologists, pneumologists, occupational physicians, environmental hygiene specialists, immunologists, mineralogists, and pathologists.
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