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Dear Art,

I enclose my comments on your question concerning the definition and classification of terms pertaining to pneumoconiosis. I have added an extra paragraph at the end on pneumoconiosis without radiological change, which is a problem upon which I should be glad to have the views of the Panel.

If it is also suitable for the discussion, would the following question be considered by the Panel?:-

*note
confusion
note*

"Since radiography provides the single most important piece of evidence in the diagnosis of pneumoconiosis, the present lack of agreement as to the earliest characteristic radiological signs of pneumoconiosis and as to the method of classification of the more advanced stages, is an important source of national and international confusion. Does the Panel agree that if an international body, such as W.H.O. or I.L.O. were to assemble sets of radiographs derived from men working in some of the major pneumoconiosis-risk industries and were to circulate them for classification to leading experts in different countries, some measure of present agreement and disagreement would be obtained and that, in the light of these findings, a conference of the experts concerned might be able to agree upon a common system? Later circulation of further sets of films might then ensure that the system was being used in the same way in different countries."

I am not sure whether this is an appropriate topic for the Panel. I have been trying to stimulate I.L.O. to do something about this question of radiological classification for two years without success, but I think it is possible that W.H.O. might be stimulated by a resolution emerging from a Saranac Symposium.

Yours sincerely,

Charles

*Very much looking forward to my trip.
I will bring the text & my other papers with me*

DEFINITION AND CLASSIFICATION OF TERMS PERTAINING TO PNEUMOCONIOSIS

Pneumoconiosis

Strictly, pneumoconiosis means a "process in the lungs caused by dust" and is theoretically initiated when the first dust particles become lodged in the lung parenchyma and cause any reaction. For practical purposes the definition of pneumoconiosis proposed by the International Conference of the I.L.O. in Sydney in 1950 is reasonable: "Pneumoconiosis is a diagnosable disease of the lungs produced by the inhalation of dust, the term 'dust' being understood to refer to particulate matter in the solid phase but excluding living organisms". The terms "diagnosable" here implies the presence of signs or symptoms, but not always loss of function (paras. 5 and 27 of the report of the Conference). To me, the word "disease" here implies any abnormal condition of the lungs. It is always difficult to define just where normality ends and abnormality begins. No sharp distinction can be drawn between the accumulation of soot or dust in a town dweller's lung and the earliest changes of coal pneumoconiosis, or even silicosis. (1, 2). It would be reasonable in some cases to speak of "town dwellers' pneumoconiosis". In scientific medicine the diagnosis of pneumoconiosis should thus be made as soon as any characteristic change can be detected, whether this be clinical or radiological during life, or histological after death, so long as this change can be reasonably attributed to the presence of dust. In most forms of pneumoconiosis histological changes are the first to appear, and radiological abnormality precedes the appearance of clinical or functional changes by many years (although in some forms of pneumoconiosis such as asbestosis and byssinosis and, as I shall show later, in some cases of coal pneumoconiosis, the reverse is true). These radiological changes are due to dust in the lung which, for scientific and logical reasons, must be regarded as constituting pneumoconiosis.

The chief objection to applying the term pneumoconiosis to cases without disability is psychological. A man may be told that he has pneumoconiosis on the grounds of radiological evidence, and may then be disgruntled if he be not granted compensation: or, in an area such as South Wales where large numbers of men are seriously affected by the disease, he may be alarmed by the diagnosis and develop an anxiety state. It would, therefore, be advantageous to have a separate term which could be used for non-disabling pneumoconiosis, but since non-disabling and disabling pneumoconiosis are usually but different stages of a single pathological process, it would be illogical in scientific medicine to use separate terms. Moreover, the radiologist or pathologist, as he classifies radiographs or sections of lungs, cannot distinguish the disabled from the non-disabled and must apply the term pneumoconiosis to all those in whom he finds abnormality attributable to dust. We use the term tuberculosis to cover both the healed apical scar and extensive active disease; we distinguish the two by suitable prefixes, and sensible doctors do not alarm their patients by telling them that they have pulmonary tuberculosis when they discover an apical scar in a routine radiograph.

For purposes of compensation it may be necessary

by special investigators. It might be appropriate to describe pneumoconiosis of severity sufficient to merit compensation as "compensatable pneumoconiosis", and to define its limits for each form of pneumoconiosis in appropriate histological, radiological, clinical or functional terms.

In the Pneumoconiosis Research Unit we have not found it difficult to explain to a man that his radiograph "shows that he has been a miner, but does not show enough dust to do any harm" and that he cannot therefore expect compensation. In border-line cases also we have found no difficulty in explaining that doctors may disagree with one another in assessing the severity of the disease so that in such cases, while application for compensation may be worth making, the man must not be surprised if it fails despite our own diagnosis of pneumoconiosis. We have found the Welsh coal miner much more ready to accept the existence of observer error among doctors than are members of the medical profession themselves!

Active and Inactive Dusts

The pathological activity of a dust depends not only on its chemical nature but also on its physical characteristics, such as size-distribution, and upon the dose administered, so that no simple classification of dusts into active and inactive is possible. For example, a given quantity of 20 Angstrom silica may be highly active if given in a single large dose, but the same quantity may have no effect at all if administered in small concentration over a longer period.

to / It might be possible to reach a theoretically sound classification of dusts according to their activity on the basis of the relationship between the amount of material retained in the lung and the degree of pathological change. Thus, an active dust would be one in which the degree of pathological reaction and its presence in the lung is great in relation to the amount of retained material (e.g. silica and beryllium) and an inactive dust would be one in which the pathological change is relatively small (e.g. coal and iron), but here we meet the difficulty that dusts such as coal which would be classified as inactive per se may, in combination with tuberculous infection, produce gross pathological changes.

A vast amount of study would be required to achieve anything but a rough classification along these lines and it would still be of little practical use. As I hope to show in my paper on the Epidemiology of Coal Miners' Pneumoconiosis, we now have evidence that Welsh anthracite or steam coal dusts produce much more pneumoconiosis in men exposed to them than do English bituminous coal dusts in the same airborne concentration, and yet the degree of fibrosis in the lungs of anthracite and bituminous coal miners in relation to the amount of retained coal does not appear to be very different. These different effects may be due to differences in the proportion of the inhaled dust retained in the lung, the reason for which we do not understand.

Any classification of dusts into active or inactive on the basis of animal experiments may be dangerously misleading. Coal dust was for years regarded as harmless in Great Britain and is still so regarded in Germany, largely because animal lungs do not show any

Silicosis

I am unaware of any difference of the meaning of the words "disease" and "condition" in relation to silicosis. I regard any pathological change attributable to free silica as a form of silicosis. I distinguish the formation of uniformly distributed, concentric, collagenous, fibrotic nodules by the term "Classical nodular silicosis".

Terms for Specific Entities

The word silicosis should only be used when it has been clearly demonstrated that the pathological changes in the lung are attributable to the action of silica. So far as I know this can only be done where the dust exposure is to pure silica or where the reaction in the lung is of the classical type. For all other pulmonary disease due to dust the word pneumoconiosis should be used, preceded by the name of the dust or occupation by or in which the disease is caused. The term "anthracosis-silicosis" should be applied only to those cases of classical silicosis in which there are also deposits of coal in the lungs (for example, rock workers in coal mines) but should not be applied to the ordinary coal workers' pneumoconiosis in which there is no evidence that silica is responsible for the fibrosis.

Clarity is gained by using prefixes to the general term "pneumoconiosis" for the pulmonary conditions attributable to specific dusts.

Pneumoconiosis Without Radiological Change

In South Wales Professor Gough has occasionally demonstrated cases of severe focal emphysema in simple coal pneumoconiosis which must have been disabling during life but which was unassociated with any radiological abnormality. Dr. Hugh-Jones will show in his paper tomorrow that the impairment of ventilatory function in coal miners with simple pneumoconiosis is not closely related to the severity of the radiographic changes and that coal miners without any radiological abnormality appear, on the average, to have impaired ventilatory capacity when compared with non-miners of the same age. This impairment is, in some cases, associated with a tendency to bronchial spasm. Whether this disability is attributable to dust inhalation we cannot yet say. Further studies of much larger samples of miners and of men from various other dusty and non-dusty occupations will be required to establish its significance.

But these preliminary findings do suggest that we may at present pay too much attention to the radiological picture in deciding the question of compensation in simple pneumoconiosis. It might be fairer to workers in dusty occupations if we were to compensate respiratory disability regardless of the radiological picture whenever this disability falls well outside the range of the normal at the appropriate age and is not clearly attributable to other non-occupational respiratory disease. Improvements in respiratory function tests, which might enable the pattern of impaired function in pneumoconiosis to be distinguished from that of emphysema and chronic bronchitis, would greatly help in this decision.

Reference