PNEUMOCONIOSIS

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Historical

In medical writings from the 'Epidemics of Hippocrates' (460-370 B.C.) to the 'Treatise of the Diseases of Tradesmen' by Ramazzini (1633-1714), the historian may discover references to the occurrence of lung diseases among workmen engaged in dusty occupations, but the significance of these passages is largely speculative. By contrast the allusions contained in the texts (Meiklejohn, 1947) of Benson's Patents, 1726 and 1732, for grinding flints are precise and comprehensive:—

'Any person ever so healthfull or strong working in that business cannot probably survive above two years, occasioned by the dust sucked into his body by the air he breaths, which being of a ponderous nature, fixes there so closely that it is now very difficult to find persons which will engage in the business, to the great obstruction and detriment of the said trade.'

These statements appear authentic as it is recorded that calcined flint was introduced for use in earthenware manufacture in 1720 (Shaw, 1829).

Thackrah of Leeds (1832) discussed at length the health of 'operatives subjected to dust' and ever since physicians have recorded from their own local experience the melancholy tale of dust, consumption and premature death. Knight (1830) a practitioner of Sheffield relates that, 'The fork grinders who use a dry grindstone die at the ages of 28 and 32' and among the preventive measures which he recommends is that this occupation should be confined to criminals.

The disease then, as now, was incurable, and so the demand was for prevention and control. There is scarcely a single modern measure or device which is not recorded and discussed by the older writers. Thackrah (1832) mentions substitution of harmful by less noxious substances, exhaust ventilation, wet processes in place of dry, face masks, alternation of employment and reduced hours of labour. In a very interesting passage he describes the benefits of natural water infusion through fissures in the rock in lead mines:—

'The particles, on the contrary, are laid as they are formed, by the continuous oozing, dropping and splashing of the insinuating water.'

At a Scottish cotton mill we learn from Ritchie

(1828) about the complete enclosure of carding and spinning machines and how from the picking room alone 828 lb. of cotton dust was collected in the exhaust chamber during a single year. Calvert Holland (1843) discussed the advantages of down draught exhaust ventilation with flues under the floor as against overhead hoods and ducts.

On the clinical side, early detection of the disease by regular medical supervision, chest measurement, estimation of pulmonary vital capacity and inhalation therapy, all are noted.

Lack of co-operation by the workmen then as now was a problem: 'We remark with regret the men's inattention to health, their indifference to the prevention of disease. They think nothing of injurious agents till their health is destroyed and the time for prevention is past.' Employers have responsibilities and duties: 'It is especially incumbent on masters to regard the health of the persons they employ; to examine the effects of injurious agents, to invent and provide remedies and to enforce their application.' But this cannot be achieved voluntarily and so the need of Factory Acts is foreseen: 'Whatever improvement may be effected without a legislative enactment . . . this improvement will be but temporary.' Finally, for the physician, there is wise counsel: 'He must not be content with the loose statements of thoughtless and prejudiced workmen' (Thackrah, 1832), and nowadays I, personally, would venture to add employers and trade union officials.

In support of their arguments these early authors provide extensive statistical tables revealing the dreadful toll of fibroid phthisis in the 18th and 19th centuries, but repetition here would serve little purpose; indeed by false comparisons might lead to a wholly unjustified complacency about the tragic present.

The historical development of opinion on the etiology and pathology of lung diseases in tradesmen requires brief notice, but before this it is necessary to examine the evolution of the nomenclature of the disease.

Nomenclature and its Significance

During the 18th century the dust diseases of the lungs were identified by terms descriptive of the clinical features, asthma, rot, phthisis or con-

sumption, to which was prefixed the name of the associated trade, as in knife-grinders' asthma and potters' rot. The next phase in nomenclature was related to the study of morbid anatomical changes in the lungs observed at necropsy. In 1838 Stratton proposed the term anthracosis to describe the appearances which he had observed in a coal-miner's lungs. Later, about 1866, Zenker recorded the presence of red iron oxide in the lungs of women engaged in handling this powder. This condition he named siderosis and for the various affections of the lungs produced by the inhalation of dust-like particles he proposed the generic title pneumonokoniosis (πνεύμων, lung and κόνις, dust). Following this practice Visconti, in 1870, introduced the term silicosis for those cases in which the noxious dust was composed of siliceous or flint, and Merkel added chalicosis when fine gravel or stone was the offending agent. Byssinosis was the form associated with the inhalation of cotton, flax and linen dusts. Thus a stage of finality was reached whereby pneumonokoniosis was recognized as the general title signifying a condition of dust disease of the lung. Under this heading certain varieties were identified and named according to the causative dust, anthracosis, siderosis, silicosis, chalicosis and byssinosis. Collectively these constituted the pneumonokonioses or dust diseases of the lungs. At present there is considerable confusion in the use of these terms as exemplified by this definition in the current Compensation Act (1946) in South Africa: "Silicosis" means any form of pneumoconiosis due to the inhalation of mineral dust.

So in effect silicosis may mean asbestosis. This reflects the fact that the original generic term has proved awkward and pedantic in regular use. In practice it has become shortened to pneumonokoniosis; an attempt to abbreviate it still further to koniosis failed. Two forms of spelling, pneumonokoniosis and pneumoconiosis, are in regular use, but the latter receives most favour throughout the world and is the only spelling recorded in the 'New Oxford Dictionary' (1909 edition). Classical scholars agree that both spellings are defensible, but omission of the central 'n' is wrong, rendering the term etymologically meaningless. Usage and convenience, however, have established the shorter form; the need is to adopt a uniform spelling.

With the advance in knowledge of pathology of dust affections of the lungs, a more serious difficulty has arisen, namely, interpretation of the term pneumoconiosis. Does it signify simply the accumulation of dust in the lungs, a dust-ridden state, or does it involve the concept of disease, a departure from health? That there is no agreement is reflected in such distinctions as are im-

plied in 'active' and 'inert' dusts and 'benign pneumoconiosis' (Prendergrass and Leopold, 1945). The problem is still further complicated by attempts to embrace certain legal and administrative considerations in relation to workmen's compensation (Fletcher, 1948). And again how are we to regard byssinosis, bagassosis, berylliosis, aluminosis, manganese pneumonitis, bronchomycoses and a host of others? The confusion is world-wide and has become so great that it almost appears as if the original terminology should be abandoned and that some authoritative body such as the International Labour Office should endeavour to formulate and establish a classification and nomenclature for universal use.

At present in this country the practical everyday consideration is the legal definition for purposes of workmen's compensation under the National Insurance (Industrial Injuries) Act, 1946.

"Pneumoconiosis" means fibrosis of the lungs due to silica dust, asbestos dust or other dust, and includes the condition of the lungs known as dust reticulation.

This is fully discussed in the contribution by Dr. Sutherland.

Pathology and Etiology

The earliest references to the noxious effects of substances inhaled into the lungs are to the clinical manifestations in certain occupations, notably metalliferous mining. Thus Hippocrates writes of 'the wan complexion and the difficulty of breathing of the metal diggers' and Georgius Agricola (1557) remarks that 'when the dust is corrosive it ulcerates the lungs and produces consumption.'

Apart from recording the same illness in an ever increasing number of trades and occupations, no advance occurred until the 19th century when study of the morbid anatomical changes at necropsy permitted correlation with the clinical manifestations. Diemerbroek (1832) relates that 'in dissecting the bodies of masons, dead from asthmatic affections, he found heaps of sand in their lungs, and in dividing the pulmonary substance, he seemed to be cutting a sandy body.' Gregory (1831) describes the lungs of a miner as being infiltrated with black matter resembling, if not identical with, coal.

Calvert Holland (1843) is among the earliest observers to suggest that in cutlery grinders' asthma the 'structural modifications in the lungs are of two distinct types and so admit of an easy classification.'

In the one case, they (the symptoms) were often accompanied with considerable constitutional vigour, and though harassing in the extreme, interfered comparatively little with the appetite

FIG. 1.—Male aged 40 years. Radiographic appearances natural for age.

or the digestive functions; and in connection with these symptoms, the chest was observed to be prominent and well-developed. In the other case, the thorax was remarked to be flat and contracted, the powers of life feeble, and the animal frame exhibiting indications of gradual emaciation.'

The same author gives an equally comprehensive account of the marked structural changes in the lungs, among which he particularly notes the hard, black, gritty enlargement of the bronchial glands immediately at the bifurcation of the trachea and:

'In some instances the lungs have presented an appearance as if black currants had been distributed throughout the whole substance of them.'

This is still an accurate description of 'classical silicosis.' These quotations reveal that the physicians were already beginning to distinguish between simple dust fibrosis and this condition complicated by tuberculosis. Hall (1857) asserts this quite clearly:

'There is no necessary connexion between the Sheffield grinders' disease and thoracic consumption, although both affections may be present in the same individual.'

The absolute distinction was not possible until Koch's discovery of the tubercle bacillus in 1882.

Since then pathologists have added greatly to our knowledge of the minute histology of the

lesions and associated changes in the lungs. Considerable controversy, however, has ranged over the separate and combined effects of the dust and tuberculous infection, and this problem remains unresolved.

From the outset dust was generally recognized as the noxious agent though Laënnec (1818) rejected the idea that dust could be retained in the lungs and so induce disease. Calvert Holland (1843) dismisses this opinion as lack of experience of manufacturing processes. Summing up the chapter of his book on dust diseases, Thackrah (1832) records:

'Dust is the great bane of manufacture, and whether it be farina, animal or vegetable fibre, or evolved from minerals, stone, lime, coal or metal, it injures the respiratory organs, in proportion to the mechanical irritation it induces on the bronchial membrane.'

During the ensuing years the occurrence of the disease was reported from practically every country in which metalliferous mining was carried on. Presenting evidence before a Royal Commission, Haldane (1914) made this significant declaration:

'I believe the disease is the same all over the world, and the end comes through tubercular infection; and

'We ought to know what the qualities are in

FIG. 2.—Male aged 58 years; china biscuit placer (pottery; pure flint risk), 45 years. Generalized fine silicosis; died 4 months later; myocardial degeneration; silicosis without tuberculosis; confirmed post-mortem.

the dust which makes it dangerous, so that we may be able to say with regard to any dust off-hand whether that dust is dangerous or not.'

In the following year Collis (1915) provided part of the answer when, as a result of comprehensive analytical studies in various industries, he concluded:

'While most dusts have an injurious influence, silica in the form of free silicon dioxide alone stimulates the formation of fibrous tissue.'

So just as Haldane had proclaimed the essential unity of the pathological process, Collis had defined the essential unity of the causative agent. The disease was silicosis and the problem was to discover the mode of action of the silica particles. Thackrah and Calvert Holland had attributed it to mechanical irritation. McCrae (1913) had demonstrated that 'the largest particles which gained access and became embedded in the lungs did not exceed 10 microns in diameter and 70 per cent. of all particles were less than 1 micron.' This enabled Gye and Purdy (1922(a) and 1922(b)) to determine: (1) 'that particles lose their sharpness when embedded in the phagocytic dust cells,' and (2) 'that the reaction (fibrosis) is noted at points remote from the presence of silica.'

They advanced a chemical hypothesis of action:

'Finely divided silica in the lungs slowly forms a silica sol, which acts as a direct cell poison.'

While this has proved generally acceptable some authors (Heffernan and Green, 1928, and Heffernan, 1946) dissent. Reviewing this aspect of the subject in 1945 King concludes:

'Successful as the solubility theory may be in explaining the observed facts of silicosis and in indicating new lines of approach, it must be recognized that it does not always appear to work in practice.'

For a time (Middleton, 1936) it was thought, but now rejected (I.L.O., 1938), that alkalis could accelerate the action of silica, while Haldane (1917) had suggested that coal dust had an inhibitory action, a quality which Heffernan and Green (1928) had likewise attributed to 'clay substance, carbon and shale dust,' and Kettle (1935) to oxide of iron. This idea has been advanced still further in relation to powdered metallic aluminium (Denny, Robson and Irwin, 1937, 1939(a) and 1939(b)).

Gardner (1920) in America represented another viewpoint, namely, that 'the lung lesion is due to a combined irritant action of the tubercle bacillus and the silica, which action neither of them alone can produce.' This reflects the other characteristic of the disease which remained unexplained,

namely, the excessively high incidence of tuberculosis as a complication of silicosis. Following extensive laboratory observations Gye and Kettle (1922) concluded that 'silica caused a coagulation necrosis in which all of the tubercle bacilli were protected from phagocytosis and enabled to multiply rapidly with impunity,' a process further facilitated by disorganization of the lymphatic drainage of the lung.

Just when it appeared that free silica had been established as the single causative noxious agent, a new dust disease was reported and confirmed. This was asbestosis, fibrosis of the lungs due to the inhalation of asbestos dust. Asbestos is a complex mineral silicate, so the whole question of the etiology was re-opened in relation to this and other silicates (combined silica as opposed to free silica) and for a time the term 'silicatosis' was current. Then Jones (1933 and 1934), following analyses of the ash of silicotic lungs, disputed the free silica hypothesis by submitting that while free silica might be the causal agent in isolated cases, its action was insignificant compared with that of a fibrous mineral silicate, known to geologists as sericite. This hypothesis, in turn, has not withstood the critical judgment of experts and has not been confirmed by animal tests.

Finally the alarming incidence of chronic pulmonary disease among coal-miners, particularly in South Wales, focused attention. The occurrence of silicosis among the stone workers in the mines was well known, but these cases accounted for a very small number of the total. A special inquiry was instituted by which Hart and Aslett (1942) established that among underground workers and certain others engaged in handling coal, there exists another form of pneumoconiosis, to which they applied the name 'dust reticulation,' a term descriptive of the radiographic and pathological changes. Belt and Ferris (1942) originally described the pathological changes and Gough (1940, 1947(a) and 1947(b)) has since added considerably to this knowledge. As yet the causative dust constituent has not been defined and is proving a very difficult problem as 'coal' or 'mine' dust is a very complex mixture of mineral dusts, including not only carbon but free and combined silica, silicon dioxide and silicates. In addition the workmen are exposed to various gases derived from explosives and to volatile emanations resulting from the natural decomposition of coal. Moreover, the possible role of associated tuberculous infection in the production and course of the disease remains undiscovered.

Social Aspects

Throughout our industrial history the pneumoconioses have created serious social problems as

revealed in the classical statistical surveys of Farr (1875) and Ogle (1885). Today, as never before, they constitute a major political problem.

Commencing in 1919 the various forms of pneumoconiosis have been brought within the scope of workmen's compensation under which certification of the disease is solely the responsibility of the Pneumoconiosis Medical Board of the Ministry of National Insurance. As a result comprehensive authoritative statistics are available and the present position may be concisely represented in the following table derived from this source.

CERTIFICATES OF TOTAL OR PARTIAL DISABLEMENT BY REASON OF THE DISEASE* (1939-1947)

Industry or Process	No. of certificates	Yearly average (nearest whole number)
Refractories Industries ..	153	17
Sandstone Industry ..	306	34
Pottery Industry ..	474	53
Asbestos Industry ..	103	11
Metal Grinding Industries ..	35	4
Coal Mining Industry ..	19,166	2,130
Masons and Stone Dressers ..	220	24
Sandblasting and Steel Dressing ..	218	24
Metalliferous Mining ..	108	12
Haematite Mining Industry ..	81	9
Slate Mining Industry ..	157	17
Other Industries ..	210	23
All	20,885	2,321

* The disease means silicosis, asbestosis or pneumoconiosis or any of these diseases accompanied by tuberculosis.

The absolute numbers are impressive, but their relative significance involves consideration of many factors. Thus in the pottery industry 53 cases in a year may not seem a large number unless it is appreciated that they accrue from a population of 6,000 workmen (Meiklejohn, 1949). This is even more cogent in relation to sand-blasters and steel dressers (Merewether, 1936). By contrast the population at risk underground in coal mines (550,000) helps account for the large total of cases but does not reveal that the certified cases are almost entirely derived from South Wales (65,000 underground workmen) (Fletcher, 1948). Between 1919 and 1927 the average annual incidence of cases in the metal grinding industries in Sheffield alone was 30 as against 4 at present for the whole of Great Britain. This reflects the replacement of sandstone grindstones by non-siliceous wheels, which has taken place since 1928.

The related social problems are alarming and

admit of no complacency; workmen's compensation is no solution. Morris (1947) and Meiklejohn (1947, 1949) have dealt with these in coal-miners and pottery workers respectively. Coal-miners may be taken as an example. It is estimated that every single certified case involves compensation payments to the workman and his dependants of at least £1,000. This means that the coal mining industry since 1939 has acquired an average annual liability of £2,000,000. Even more important is the fact that the men involved are the most highly skilled face workers at the height of their production. Every certified case represents the loss of 10 to 20 years' man-output. Many are only partially disabled and require re-settlement in alternative employment, a peculiarly difficult problem in areas so long restricted to heavy industry. Most serious of all is the drift of population from the mines and the mining areas; wastage of man-power exceeds recruitment. In varying degree this picture is true of all dangerous dusty trades. Over one hundred years ago Calvert Holland (1843) wrote this of Sheffield grinders' asthma:

'It will also be obvious, from the various facts brought under consideration, that the evils must in various ways be felt by every portion of the community, vice, ignorance and destitution, exert a widely pervading influence. The inability of the artisan to continue his occupation from disease, throws him and, perhaps, a numerous family, upon the parish for support, not in a few solitary cases, but in hundreds of instances; and it is scarcely necessary to observe that on his death the same dependence, if not indeed in an aggravated form, becomes indispensable for years. When the parish is not burthened in this stage of suffering, the injury to society, from the inadequate struggles of the afflicted artisan to live, is scarcely in any degree mitigated. The children, without having received any education at all, are put to work at seven, eight and nine years old, from which circumstances, and the fruitful consequences which flow from them, in a later stage, additional claims upon the parish funds are created, or ignorance and immorality extend, maintaining low and depraved habits of feeling among the labouring classes.'

Social legislation may have advanced but effective practice still lags far behind knowledge.

Silicosis

Silicosis is the specific variety of pneumoconiosis due to the inhalation of silica dust. Its occurrence is world-wide, affecting all races, males and females alike. In relation to the pathological lesion, Haldane's (1914) assertion that 'the disease is the same all over the world, and the end comes

through tubercular infection' is fundamentally true. The incidence and severity of the disease and the accompanying clinical manifestations in particular communities, however, by reason of different industrial practices, associated dusts, racial constitution and habits and climatic conditions, vary considerably from one country to another. This is equally true within a single geographical area as exemplified by the disease in Staffordshire potters, Cornish tin miners and Aberdeen granite masons. Likewise there are differences within an industry as observed in foundry workers and even among workers in the same occupation. These significant matters are all too often overlooked both by writers and readers. Nevertheless certain facts are of general application and provide the background against which the problem in particular industries or processes may be studied.

Silica is widely distributed in nature, forming 70 per cent. of the earth's crust. It is the chief constituent of many rocks and also occurs as flint and natural quartzose sands. In addition to being abundant, readily available and comparatively cheap, it has certain qualities peculiarly valuable in industry. Thus it is insoluble in acids except hydrofluoric (pottery manufacture and glass making), highly abrasive (metal grinding and sand blasting) and refractory to very high temperature (brickmaking for furnace linings and moulding in foundries).

All processes which involve the manipulation of silica, especially in the dry state, in such a way that it is broken up into minute particles and inhaled, are liable to produce silicosis. Thus in Great Britain the disease is associated with coal and metalliferous mining in siliceous strata, dressing and cutting of sandstone, grinding of metals on sandstone, pottery manufacture, making of silica bricks (the refractories industries) and in foundries, the cleaning and freeing of castings from adherent sand. In these industries large numbers of men and women are at risk, but there are many other operations such as flint crushing, packing of scouring powders, tunnelling and the cleaning of the stonework of sandstone buildings in which the exposure may be very intense though involving only a small number of workmen. As will appear later the purpose of the occupational history of a patient suspected to be suffering from pneumoconiosis is to define the period, intensity and character of the dust hazard. In the production of the disease it is generally accepted that:

1. The silica must exist in the crystalline form as free silicon dioxide.
2. To gain entrance to the lungs and establish effective occupation, the particle size must be under 10 microns.

FIG. 3.—Male aged 43 years; tunneller drilling in silica rock, $3\frac{1}{2}$ years. Generalized coarse silicosis.

3. The dust particles are most active when under 2 microns in diameter and freshly fractured mechanically or by heat at very high temperatures; this ensures the maximum superficial area relative to size.

4. The intensity and period of exposure are inversely proportional so that if the dust cloud is very intense the period necessary for development of the disease is correspondingly short and vice versa.

5. The action and reaction are intensified by infection, notably tuberculosis, and the growth of the tubercle bacillus is favoured.

Under present conditions in Great Britain, silicosis in a diagnosable stage seldom occurs under 15 to 20 years' exposure to the risk, and so is rarely seen under the age of 40 years. If adequate precautions to control the dust cloud are not taken or effectively maintained then the disease may occur in flint crushers, rock tunnellers, sand blasters and scouring powder packers after two to five years' exposure. The writer once observed a group of cases in flint crushers after less than twelve months in the process. While in very intensive risks it is almost certain that every workman will be affected, in the ordinary run of industry it is not inevitable that every workman should contract the disease in the course of a working lifetime. There is a personal constitutional factor involved in the development and

progression of the disease. This factor has not been defined, though infection, especially tuberculosis, is suspect.

Pathology and Course of Silicosis

With every breath the workman inhales the dust, sometimes the concentration is high, sometimes low. The coarse particles are arrested and rejected by the natural defences of the respiratory tract, while the finest particles gain access to the alveoli from which they are transferred by scavenger cells and aggregated in the lymph nodes within the lung parenchyma under the pleura and at the hilum. Over a period of years the attack goes on and the occupation of the lungs by the dust advances. All the while a reaction is occurring in the lymph nodes, which is finally revealed by the appearance of tiny spherical nodules of fibrosis. Slowly these nodules increase in diameter and others appear elsewhere. Individual nodules achieve a size up to 5 mm.; contiguous nodules become confluent and in the most advanced stages large 'cricket ball' masses result. These fibrous areas diminish the amount of functional lung so the healthy areas try to compensate. The lung between the fibrous areas becomes emphysematous and bullae appear along the free margins. The bed of the pulmonary circulation becomes greatly extended and narrowed and this is reflected by hypertrophy of the right ventricle.

FIG. 4.—Male aged 72 years; coal-miner (Scotland), 56 years. Bilateral massive silicosis with bullous emphysema at apices.

(Reproduced by courtesy of Drs. Sutherland and Tyrrell, Pneumoconiosis Medical Board)

FIG. 5.—Male aged 50 years; colliery fireman 21 years, then steel-dresser 12 years. Massive silicosis and dust reticulation.

(Reproduced by courtesy of Drs. Sutherland and Tyrrell)

An indolent pleurisy anchors the lungs to the chest wall, diaphragm, pericardium and mediastinum. At first the changes are localized in the root zone or the upper lobe of one lung, usually the right, but slowly and inevitably the disease appears in the contralateral lung and so it advances in both. But it is not necessarily an orderly advance, indeed it rarely is; from the stage of localized nodulation massing may result even before the nodulation has extended elsewhere. This is usually suggestive of an infective process. At any stage overt tuberculosis may appear and lead to a fatal outcome. In the absence of tuberculosis the advance is slow but death may be precipitated by acute intercurrent disease such as influenza or pneumonia, while in older patients death may occur from cardiac failure.

The foregoing description indicates a slowly developing continuous process and some may wonder why so long a period elapses before the disease can be diagnosed. The explanation would appear to be that a substantial period is necessary to complete the silica reaction. Moreover, in the beginning the individual nodules are very minute and the fibrous tissue immature. The actual identification of the disease during life depends on the appearance of abnormal radiographic opacities in the lung fields and presumably these must be

of a certain minimum size and radio-opacity (maturity of fibrous tissue) before they can be distinguished from the normal structures of the lungs. These are personal opinions but are supported by the fact that occasionally at necropsy the extent of the disease is considerably greater than is revealed by a recent radiograph, and indeed histological examination may establish the presence of the disease when the radiograph is considered normal. This is particularly true in acute risks such as sand-blasters. The fact that pathological examination is more precise than radiographic examination does not invalidate the fact that during life the diagnosis of the disease must include demonstrable X-ray changes in the lungs.

Diagnosis

In life the diagnosis depends on the following triad, and while any of these may lead one to suspect the presence of the disease, it must ultimately be firmly based on all three. Any anomaly should raise doubt in the mind of the physician, indicating the need for further investigation and even for a visit to the place of work to investigate the actual environmental circumstances of the particular case.

1. Clinical history and examination with special reference to symptomatology.

FIG. 6.—Male aged 39 years; flint crusherman for 14 months; no other dust risk. Massive silicosis; died 4 years later of congestive heart failure; diagnosis of silicosis without tuberculosis confirmed post-mortem.

2. The history of the occupational risk to define precisely the period and intensity of the silica risk.

3. Radiographic examination of the chest.

Clinical and Related Aspects

For a period of years, save in very serious dust hazards, the workman, though inhaling silica particles regularly at his job, is unaware of any injury to health; the pathological process is latent, the onset of the disease insidious. During this phase radiographic examination of the chest may disclose definite silicotic changes but the patient denies any inconvenience. This, however, is not strictly true for close questioning will usually reveal that he is troubled by a cough and slight breathlessness on exertion. He is fully aware of these symptoms but has dismissed them as due to smoking or his 'age.' The cough is dry and harsh, occurring in bouts first thing in the morning or last thing at night. The shortness of breath is reflected by the fact that he now prefers to ride where he had previously walked, or if he does walk his pace is slower and he is aware of the hills. Then suddenly and indirectly the disease in the lungs is discovered. The workman suffers from a 'cold' or an attack of influenza; recovery is delayed while the cough and breathlessness are aggravated. Even so his general condition is

good; he is not sick, only distressed. At this juncture he becomes the victim of a host of extraneous aggravating circumstances. Convalescence is prolonged and sick benefits inadequate; the family begins to run into debt. The doctor advises an X-ray examination of the chest and the diagnosis of early silicosis is confirmed. Anxious as the workman may be to return to work, this must be postponed for the doctors and his trade union have advised a claim for compensation. This involves more delay during which his domestic problems increase and his mind is focused on his chest. All the while the work habit is slowly being lost. He is sick with fear and worry; he knows the doctors are not mistaken because 'it is on the X-ray.' Somewhere and to someone this tragedy happens every day, a tragedy in which the practice of the art of medicine and the well-being of an individual and his family are sacrificed on the altars of scientific medicine and the welfare state. Facts triumph over judgment, life becomes more important than living.

The change to an easy cough with mucopurulent sputum, occasionally blood-stained, loss of appetite, weakness and wasting indicate associated tuberculosis. The patient is now obviously a sick man and thenceforth deterioration is rapid.

In simple silicosis even when the changes in

FIG. 7.—Female aged 32 years; china polisher (pottery pure flint risk), 19 years. No specific appearances of silicosis.

FIG. 8.—Same case 8 years later; had not worked interval; at home nursing husband suffering from tuberculosis of lungs. Post-mortem revealed massive silicosis both lungs with active tuberculosis left; note cervical rib (R) and influence of tuberculous infection.

the lungs and pleura are gross, clinical examination of the chest yields remarkably little information; it is a silent disease. Inspection properly carried out as a definite technique is probably superior to all other methods of examination, but unfortunately so few doctors nowadays take time to look at the patient. The excerpt quoted from Calvert Holland (see page 600) is a salutary reminder of how much we have sacrificed to instrumental aids. Important points to note are the general condition and facies, response to the effort of undressing, configuration and movement of the chest, line of the trachea and, if visible, the position and extent of the cardiac impulse. Cyanosis occurs only in advanced disease and then only if associated with cardiac embarrassment; clubbing of the fingers is very exceptional. Areas of fibrosis cannot be mapped out by percussion but when massing has occurred near the apex of the upper lobe dullness may be noted posteriorly with resonance, due to emphysema, anteriorly. On auscultation one is impressed by the patchy diminution or absence of breath sounds and adventitia due to bronchitis and pleurisy are rarely audible. The heart is seldom displaced or enlarged; obvious enlargement usually signifies con-

comitant disease of the cardiovascular system or kidneys.

In the case of silicosis complicated by tuberculosis the signs and accompanying changes are those usually associated with tuberculosis of the lungs. As a useful generalization it may be said that in simple silicosis the patient is fit but distressed, in silicosis accompanied by tuberculosis he is sick and in declining health.

Occupational History

The purpose of the occupational history is to define the period and intensity of the silica dust hazard. This is only to be achieved by recording the periods of employment in chronological order, noting the nature of the work, the materials involved, the tools, the environmental conditions and any protective measures taken to eliminate or diminish the risk. It is supremely important to note that the disease may not necessarily have been acquired in the latest employment but in some antecedent one. Thus a bricklayer's labourer may have worked previously as a sandstone mason or a cutlery grinder may now be using emery wheels but during his early years in the trade worked on sandstone grindstones.

Radiographic Examination of the Chest

It is generally accepted that the radiographic findings in silicosis afford the most reliable *single* piece of evidence in establishing the existence and actual stage of the disease in any particular case. This, however, is only true if the radiograph is technically satisfactory, properly exposed and carefully processed. The radiographic appearances are represented by changes which reflect the characteristic pathological lesion. In the early stages they comprise small opacities, variously described as nodulation or mottling, at first localized and later in classical silicosis involving the whole lung fields, thus presenting a 'snow-storm' appearance. When the disease is confluent, massive shadows appear usually in the root or sub-apical zone; it is very rare for an isolated opacity to occur in the basal area. When active tuberculosis supervenes the whole picture is modified and continues to alter with the advance of the tuberculous process.

Frequently when the patient is first seen the whole clinical and radiographic picture is that of extensive tuberculosis of the lungs, but chiefly for reasons related to workmen's compensation the clinician must decide whether or not silicosis is present. It is useless to believe that in such cases one can distinguish radiographically between silicotic and tuberculous opacities. In such circumstances a presumptive diagnosis only is possible based almost entirely upon a critical assessment of the occupational risk.

Differential Diagnosis

If the case is investigated as outlined above, then almost absolute accuracy can be achieved in the recognition of silicosis and its complications. Difficulty only arises in the earliest stage in an attempt to anticipate the disease radiographically and in the determination of associated tuberculosis. It is usual, however, to record that the disease must be differentiated from miliary tuberculosis of the lungs (acute and chronic), carcinoma of the lung especially miliary carcinomatosis, haemosiderosis in mitral disease, sarcoidosis, bronchomycoses, virus infection of the lungs, tropical eosinophilia, bilharziasis, ascariasis and retained radio-opaque dust. While it is true that these conditions may on occasion closely simulate the radiographic appearances, X-ray examination of the chest, absolutely indispensable as it is, is only a single factor in clinical diagnosis and must be recognized as such. While it is true to say that radiology has contributed more than any other method to the accurate recognition of the disease, it is no less true that the dramatic radiographic appearances have warped clinical

judgment and obsessed trade union officials often to the detriment of the individual patient.

Prognosis, Management and Treatment

Simple silicosis, even of advanced degree, is compatible with many years of life and considerable capacity for work in the patient's own skilled job. There is no reliable evidence that silicotic patients are more liable to acute respiratory infections than their fellows and even if they are, the danger of contracting these is no greater at work than in their own homes or at the street corner. If they do suffer such an illness recovery is often delayed and bronchopneumonia may be fatal. The important issue which arises is what advice should be given to a patient about his job. On first principles he should not again expose himself to any dusty work. This is the advice which is regularly given, but it altogether ignores a whole host of equally important social factors. He must work, not for economic reasons alone, but paradoxically because work is the only treatment of any real value for him and, strangely enough, for his wife and family. If under the age of 40 years, an age at which he is still adaptable and has young dependants, he should seek alternative employment, but this is supremely important, he should not give up his work until he is assured that the alternative job is available, secure and suited to his capacity. Over 40, unless he is fortunate in finding a suitable alternative job, then he should continue in his own skilled work. It is the job he knows and can accomplish with the minimum effort. If the hours of work can be adjusted for him so much the better. Craftsmanship, self-respect and happy living are inseparable and all too often to sacrifice one's craft is to lose all. It is not disputed that there is danger in continuing to work in the dust, but the danger is relative and must be assessed accordingly. The craftsman is intelligent and if left to make the choice prefers to wear rather than rust out.

Active tuberculosis may become manifest at any stage so it is important that the silicotic workman should be under regular periodical medical supervision. As a means of detecting the transition from simple to complicated silicosis E.S.R. (erythrocyte sedimentation rate) tests have been advocated but, in this matter, there is nothing superior to the shrewd clinical observation and judgment of the experienced physician.

In silicosis accompanied by tuberculosis the workman must give up his employment not only to safeguard himself but his fellows. As a general rule this is no undue hardship as he is or soon will be totally unfit for work and present rates of compensation and additional benefits approximate to his previous earnings.

Treatment is almost entirely restricted to relief of symptoms. In the case of the partially disabled workman emphasis has already been made on the importance of work. Therapy by means of inhalations of powdered metallic aluminium is at present under trial in Canada and in this country, but the method is still in the experimental stage.

Prevention and Control

This is the only satisfactory approach to the problem and the principles are reasonably well-defined. Wherever possible non-injurious materials should be substituted for silica; thus in cutlery grinding, carborundum and alundum have replaced sandstone grindstones, in china manufacture alumina has replaced flint and in sand blasting non-siliceous abrasives have been substituted for quartzose sand. It should be noted that due to the chronic nature of the disease the beneficial results of these measures are not immediately reflected in morbidity and mortality statistics; one must wait the passing of the old generation. Mechanization of processes is another valuable method whereby the number of men at risk is substantially reduced. The intensity of the noxious dust cloud can be reduced by effective

exhaust ventilation and the use of wet methods in place of dry. Respirators are of limited value either because they fail to arrest the most dangerous minute particles or because they are uncomfortable for continuous wear during heavy work. Other aids are comprised in good house-keeping in the factory. Inhalation of powdered aluminium metal for therapeutic purposes has already been mentioned. The same method is being applied as a prophylactic measure, but due to the nature and course of the disease many years must elapse before any opinion as to its value can be pronounced.

Conclusion

The foregoing account has necessarily been restricted to general consideration of the subject. Particular occupations and centres of industry have individual characteristics and social problems. Some of these will appear in the contributions which follow.

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