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of elaborate X-ray plant, is needless and uneconomic and is one of the ways in which industrial medical services can be adversely criticised both by the employer and the medical profession as a whole.

Voluntary Consultations

This particular aspect of the factory doctor's work has been intentionally left to the last. It is not infrequently the case that his most effective work is carried out in the privacy of his consulting room. The worker who is worried about his health, who may be emotionally upset because his immediate superior is a bully, or who wants information on one of a dozen other problems, should be able to find in the medical officer an adviser who can guide him through his difficulties, either by adjusting the position within the factory or by advising him to consult his own private medical practitioner and at the same time offering his help in collaboration. There is nothing worse than a feeling of disappointment and frustration in one's work. Discontent is a major cause of industrial unrest and strikes which have affected many thousands of otherwise happy and contented workers have arisen as a result of the petty failure of one person.

In modern psychotherapy, the scientific way of dealing with a man is also the humane and understanding way and humanity and understanding are wanted badly not only in industry but throughout the world to-day. The scope for this type of medical work in industry is great and can play a part of fundamental importance in the future development of the health services of this country.

And now one word in conclusion. We have discussed very briefly some of the measures carried out by the industrial medical officer and have shown how they bear on the work of the factory clinic. At the present day, however, there are probably not more than two or three hundred doctors engaged in this type of work in the country and it is estimated that of these only some fifty or sixty are employed in a whole-time capacity. It is obvious therefore that only a small percentage of industrial workers—and very often those employed in large and enlightened firms—have such facilities offered to them. If it is conceded that the work which has just been described is of importance in the general health schemes of the nation then the problem within industry becomes one of considerable urgency. Whether the work should be undertaken by far-sighted employers now or whether it should be left to the State to develop and control must be a matter of opinion and of politics. The important thing is that the work is there waiting to be done.

There are those who firmly believe that the future prosperity of the nation depends on the health and happiness of the individual workman. If this is so how can we as doctors take our part? The problem, I believe, is primarily one for the medical profession, and in part therefore for the industrial medical officer. What can be learned in the factory clinic to-day will most surely become the accepted practice throughout industry in the future and if properly developed cannot fail to contribute successfully to the problem of national fitness which is being so much discussed at the present time.

DUSTS AND THE LUNGS 1938

WITH PARTICULAR REFERENCE TO SILICOSIS AND ASBESTOSIS

By F. R. A. MERIWETHER, M.D., M.R.C.P., Barrister-at-Law
One of H.M. Medical Inspectors of Factories

Since the birth of the human race dust has exerted its baneful effects on civilisation. Some of our prehistoric ancestors died because of it, and countless are the deaths which have occurred since. To-day, the effects of dust on the human mechanism alone is one of the major problems of the age. Two questions at once obtrude themselves. Why, after so long, is it still a major problem, and what is the present position? To the first we can only excuse ourselves by pointing out that dusts are legion in variety. They act in many ways on different organs and different individuals. Their actions are often subtle and insidious, and irretrievable damage is done before the person most concerned even appreciates the attack. Scientific appreciation of the potentialities of the different dusts and mixtures of dusts, assessment of their effects, determination of appropriate preventive measures and the means of carrying them out, have all had to wait upon advances in many collateral sciences before even the full scope of the problem could be appreciated. Chemistry, physics,

pathology, bacteriology, clinical medicine, radiology, petrology, microscopy, sociology and general education have all had to bear a part before the foundations could be laid.

What then has been achieved? Not much, we may say, if man has been busy at this problem for 2,000 years or more, but when one realises that the general state of human knowledge has been such that it was not possible until within the last 100 years to advance beyond the appreciation that dust was harmful, then that the foundations have been truly laid is not an inconsiderable achievement.

It is not possible within the limits of this paper to survey all the pioneer work which has contributed to this in the past 50 years, even if the writer was competent to do so. The complications and ramifications of the subject and the labyrinths into which inquirers are forced to penetrate, make a critical survey a matter for a team of experts.

It is possible, however, to consider the problem from the point of view of any one of us who wishes to know what principles emerging from all this work will help him to do the best for his patients, and to advise inquirers of the risks which may be associated with exposure to dust.

With some diffidence, therefore, an attempt will be made to set out some rough practical generalisation which may be of help in this way.

We are constantly exposed to dust, but generally it only causes temporary inconvenience and is quickly disposed of.

The effects on the human body depend on the nature of the dust and, if inhaled, also on its concentration in the air breathed, the median size of the particles, and the existing condition of the chest and lungs.

No simple and accurate classification of dusts has yet been evolved, but certain broad distinctions can be made which are useful. Thus we have :—

(1) Dusts which are systemic poisons, as lead, arsenic, manganese, trinitrotoluene and many others.

(2) Irritant or corrosive dusts, as lime, arsenic, chromic acid, the bichromates and many others in varying degrees.

(3) Allergic dusts, exposure to which may result in the development of a hypersensitive state, and cause on further minimal exposure asthma, rhinitis, urticaria, etc. Usually these are due to foreign proteins in the dust, but not always, as, for example, workers with chromic acid and the bichromates who may become hypersensitive to these chemicals.

(4) Carcinogenic or cancer-producing dusts, as the dust of pitch, of radioactive materials and of certain ores.

(5) Dusts which carry infections such as anthrax, monilia, actinomyces and other fungi.

(6) Non-toxic inorganic dusts, the dusts we commonly think of as "dust."

Although dusts belonging to all these groups, except the first, may affect the lungs, in some cases seriously, it is the last group which is the most important on this account, because in it are included all those dusts which exert deleterious effects on the essential pulmonary tissues.

Included in this group are a great variety of dusts differing in their physical and chemical characteristics, and these characteristics play an important rôle in determining the nature of the local effects produced when inhaled into the lungs. Although much work has been done on this aspect, however, it is not yet possible to classify the members of this group so that the effects of any particular dust may be prognosticated from its physical and chemical characteristics.

Of chemical characteristics, solubility or insolubility and the presence or not of the silica radicle, and of physical characteristics, specific gravity, fragility and whether of a fibrous character or not, are all believed to have an important influence on the action of the dust when inhaled.

In spite of the many difficulties resulting from lack of knowledge, certain generalisations can be made which are helpful in practice, and that, after all, is what is required, if knowledge is to be of practical value as well as of academic interest.

In the first place, that healthy lungs can cope effectively with appreciable quantities of dust, but that all inorganic dusts, if inhaled in concentration over a period of time, are harmful, is such an obvious proposition that its importance is liable to be overlooked.

The lungs, in common with other essential organs of the body, have a large reserve, to permit of effective reaction to the demands of disease or of advancing age or of any emergency, and it is only when this reserve is seriously encroached upon that clinical signs of embarrassment appear. Moreover, the filtering action of the nasal vibrisse and passages, aided by the moist and ciliated mucous membrane of the upper respiratory tract, together with the indefatigable efforts of the endothelial cells (" dust cells ") in seizing upon be intrusive dust and transporting it into the lymphatic drainage system, are more than adequate to cope with the normal average of air borne dust and with intermittent abnormal concentrations.

It stands to reason, however, that the mass effect of daily exposure to a heavy concentration of even a totally inert dust will wear out even the most well dispositioned and responsive mechanism; dust will collect in the nasal

ges and sinuses, ciliated epithelium
ed and replaced progressively with less
differentiated and less efficient cells,
of alveoli will become choked and
out of action, intermittent and uncon-
inflammatory conditions may interfere
the lymphatic drainage and cause further
tion of dust, and over-action of less
ed areas may result in localised emphy-

Ultimately, therefore, such heavy and
nous exposure to a dust which with less
ure is inert, will, however, result in per-
nent changes and loss of efficiency, which
e manifest in course of time as a chronic
lthic tendency. It is not easy to deter-
ne to what extent heavy and continuous ex-
e to such inert dusts as ▮ shorten life,
at some intermittent disability does result
be gainsaid. Thus, the generality of
ners has an excess liability to chronic
bitis and other affections of the upper
atory tract, cement and basic slag
rs have an excess incidence of upper
atory tract affections, and the latter also
ess liability to pneumonia.

s mechanical choking up effect of ex-
over a period to heavy concentrations of
organic dust can be demonstrated clinic-
young persons with ease after ex-
for a month or two, i.e., before it is
le for permanent changes to have
ed. A very slight, but appreciable, dif-
pairment of the percussion note—less
but associated with diffuse fibrosis—will
ed, and on auscultation sticky râles will
ed, particularly when the amplitude of
thing is increased.

se effects are not specific to particular
but are common to all inorganic dusts of
oup. Neither are they due to any specific
on of the dust on the lung parenchyma;
re simply mechanical mass effects of the
al, given the chance, the lungs will dis-
of harmlessly in most cases the greater
tion of the dust, leaving no permanent
effect.

usion, therefore, to the useful generali-
that inorganic dusts which exert no
action on the lung parenchyma are
al to the lungs only because of the mass
of considerable exposure to heavy con-
ations. This is important because there
insuperable difficulties in preventing
conditions, and the Factories Act, 1937,
ognisance of it in enacting (Section 47)
here "any substantial quantity of dust
kind" is given off all practicable
res must be taken to protect those em-

though all inorganic dusts will exert
effects under these conditions, it is com-
knowledge that many dusts are much
aggressive; we know that the clinic
engaged in the trivial operation of
removing the minute burrs from china by
means of a toy size abrasive wheel and a paste
of flint and water, may die in middle life of
an intense diffuse fibrosis of the lungs due
to the flint dust.

What is the distinguishing characteristic of
a dust so potent? The researches of many able
men all point to the same factor—the presence
of the silica radicle in the dust. Silica may be
present, of course, either "free" as silicon
dioxide, SiO_2, or "combined" with many bases
as silicates.

Proof has been piled upon proof incriminat-
ing the free silica in so many dusts encoun-
tered industrially, such as quartz, flint, sand-
stone, ganister, etc.; and this, together with
evidence of the relatively innocuous character
of some common silicate dusts, such as clay,
have combined to establish the unhappy pre-
eminence of silica in the free state in this con-
nection, and to overshadow the possibility that
some silicate dusts may be harmful.

Until a few years ago, therefore, the im-
pression was widespread that dusts containing
free silica were dangerous, and dusts contain-
ing silica only in the combined state were
harmless. Since, however, there is now con-
clusive proof that one group of silicates, to
wit asbestos, are equally as dangerous as free
silica, the above view is not strictly accurate,
and we are led to the following generalisa-
tions: all dusts containing free silica are
dangerous; dusts containing silica only in the
combined form are relatively harmless, with
the exception of the asbestos group and
possibly also other light silicate dusts in which
the particle form is fibrous or plate-like and
comparatively large.

The asbestos group of minerals are mainly
silicates of magnesium and iron, and to a less
extent calcium; the particles are elongated and
fibrous, and can split up longitudinally and
fray out to an indefinite extent, thus aiding
their retention in the bronchioles. It seems
likely that this physical characteristic of
asbestos, which tends to immobilise the par-
ticles in the lungs, may point to one dis-
tinguishing attribute of the dangerous sili-
cates: there seems to be no other feature
which will explain, for example, why asbestos
has disabling and lethal potentialities, whereas
talc has not, yet chrysotile asbestos and talc are
both magnesium silicates.

For practical purposes, therefore, so far as
present knowledge goes, the dusts which cause
serious local effects on the lungs and which
may and often do cause disablement and
death, are those containing free silica and
asbestos; other dusts are harmless in this
respect unless, as has been said so happily,
"inhaled in insulting concentrations."*

* Air Hygiene Foundation of America, Inc.: Silicosis and Allied Diseases. History & Industrial Importance. Medical Series, Bulletin No. 1, p. 4. Pittsburgh, 1937.

The siliceous dusts and asbestos also differ from other dusts in that they cause a diffuse fibrosis of the lungs, silicosis and asbestosis respectively; that is to say that they irritate and destroy the essential lung tissues and excite the formation and the proliferation of ordinary scar or fibrous tissue, due, it is generally believed, to the very slow solution of the silica. This scar tissue is not only useless lumber but also, if there is much of it, exerts secondary harmful effects by blockage of the lymphatic drainage system, interference with the proper aeration of the blood, production of strain on the right side of the heart, blocking and distortion of bronchioles by contraction of the fibrous tissue, production of localised emphysema, and definitely acts as a bait for the tubercle bacillus.

The dust particles must be small, very small, to be dangerous, since they must be small enough to float in the air and small enough to get past the outer defences of the lungs, at least as far as the smaller bronchioles. Therefore, an industrial process which does not project dust particles into the air in sufficient number and of such size and weight that they will remain floating for a considerable time cannot be dangerous in this way.

With the silica dusts the dangerous particle size range is up to 10 microns, with the lighter asbestos dust it is much greater, extending even up to 200 microns. The majority of the particles, however, which get into and stay in the lungs are much smaller in each case—up to 5 microns in the case of silica dust and up to about 50 microns in the case of asbestos. That is to say, that the dust particles which are invisible to the naked eye are the important ones, this leads us to the practical point that if a silica or an asbestos process produces visible dust in the air, then the invisible dust is certainly in dangerous concentration.

The silicotic fibrosis is laid down in nodules, whereas that of asbestos is laid down as a cobwebs. This distinction is important since it is reflected in the typical radiographic appearances. The explanation lies in the different physical and chemical characteristics of the dusts. The smaller and more stimulating silica particles are taken up by "dust cells" and hurried away from the alveoli into the lymphatics and towards the many minute lymph nodes at the junctions of these passages, and from these to the large ones at root of the lung. Unfortunately, many phagocytes succumb and drop their hostile passengers on the way for others to collect the debris, until ultimately, with continued exposure to the dust, the traffic along the lymphatics becomes very congested and at the cross-roads complete stoppages occur; here we get accumulations of particles of silica and dead phagocytes, and slowly the silica dissolves and, in course of time, nodules of fibrous tissue appear. This explains many things; for example, that healthy lungs can dispose of quite a lot of dust, even silica, for so long as the traffic can be kept moving no serious effects will result; again, it explains the ill-effects of any antecedent illness which has damaged the lungs permanently, even if locally; the ill-effects of a coincident infection, for that congests the traffic in the lymphatics still more; and the danger of a late infection when silicosis has developed generally and the lymphatic system is already grossly damaged. Also clear are the causal factors underlying the production of massive silicosis, in which a mass of fibrous tissue, the size of a hen's egg, or larger, and consisting of innumerable small nodules tightly packed together, appears. Moreover, the obstruction of the lymphatic drainage by the silicotic nodules accounts also for the great retention of ordinary carbon and other dust in the silicotic lung.

In asbestosis, the course of affairs is different. With the longer, awkward and often frayed-out asbestos fibres, transportation into the lymphatics is impossible, and Gardner and Cummings have shown that the fibrosis commences around the smaller bronchioles where the asbestos particles felt up and become immobilised. In some way or another, the silica is dissociated and dissolved out and diffuses into the neighbouring tissues, and the fibrous tissue is formed in radiating strands. The lymphatic system does not, therefore, bear the first brunt of the attack, and it may be that the longer patency of the system aids the cobweb-like formation of the fibrous tissue. Within a few weeks of the lodgment of the fibres, the curious beaded and clubbed asbestosis bodies, which are altered asbestos fibres, begin to appear.

Since the fibrous tissue formed is the result of the solution of the silica, the extent of the fibrosis, which will develop in any given case, is limited by the amount of silica immobilised and retained in the lungs; therefore, on post-mortem examination one sees all grades of simple silicosis from a few scattered nodules up to massive silicosis occupying over half of the lungs.

Knowledge of the pathology of these two diseases, together with inquiry into the length of exposure to the dust and the dustiness of the process concerned in any given case, gives one such an appreciation of the symptoms, signs and clinical course of these diseases that the alleged difficulties in diagnosis mostly vanish.

Needless to say, fibrous tissue takes time to develop, and also the less the concentration of the dust in the air breathed, the longer exposure to the dust will be required before

...is develops to such an extent as to cause ...ects appreciable by the patient.

...is true that cases of so-called "acute" ...sis and similar cases of asbestosis, due to ...sure to intense concentrations of dust, ...occurred with a fatal outcome in less ...five years, but usually 15 or more years ...required for the disease to develop, de...ing on the concentration of dust in the air. ...is country the average duration of em...ment of a series of 511 cases who died of ...sis without tuberculosis was 35.1 years, ...the average in the different industries ...ed from 30.2 years in the pottery indus... 9.2 years in the manufacture of stone-...awlers. As the effects of additional pre...ve measures become apparent, which also ...matter of years, the average duration of ...loyment in fatal cases increases *pari passu*.

...nce these diseases are not infections, they ..."dry" diseases during most of their ...se, and since the earliest symptom is ...tness of breath on slight extra exertion, ...to the mechanical interference of the ...sis with the proper aeration of the blood, ...e diseases are slow, silent and insidious. ...gh, such as there is during the develop...t of the disease, is a slight, dry, morning ...gh, which may well be ascribed by the ...ent to too much smoking. The shortness ...breath, occurring in the early 40's, may also ...ut down to getting old too soon and little ...ntion paid to it, except perhaps to move ...m an upper to a ground floor flat because ...nting the stairs becomes inconvenient.

...We have, therefore, as symptoms a little dry ...rning cough, a little shortness of breath on ...ra exertion, a little "tightness of the chest" ...l, with asbestosis cases more than silicosis ...es, slight pallor with a tinge of cyanosis of ...lips. The finer, more diffuse, fibrosis of ...osis is much more effective in causing ...ective aeration of the blood and ultimately ...reased strain on the right side of the heart, ...n silicosis. As the disease progresses, the ...ortness of breath increases until it is present ...n at rest and in the late stages is very ...ressing; "colds go to the chest" and there ...e intermittent attacks of bronchitis; there is ...n from adhesions and attacks of dry ...urisy; emphysematous bulke may burst, ...using partial pneumothorax, which is not ...common in both diseases. In advanced ...osis, bronchiectasis due to dragging and ...striction by the fibrous tissue is quite a com...on concomitant, and also the pericardium ...ay be so anchored and dragged upon as to ...crease materially the dyspnœa.

...Occasionally (more commonly with asbes...sis) death may occur from heart failure with ...dropsy and a nutmeg liver, owing to back ...ressure on the heart from the extreme ...rous.

As a rule, however, cases are carried off by some infection which the remaining sound lung tissue is quite insufficient to cope with; not uncommonly this is a low grade broncho-pneumonia, but influenza or a lobar pneumonia may precipitate death, and the commonest of all intercurrent and terminal infections is tuberculosis. Some 60 per cent. of silicotics die from silicosis with tuberculosis and about 30 per cent. of fatal cases of asbestosis are also associated with tuberculosis. It is not known exactly why this should be, but a super-added tubercular infection is the supreme risk for silicotics and is an added risk for cases of asbestosis.

Tuberculosis may appear at any stage, and then the symptoms and signs of that disease tend to mask those of the pneumoconiosis. Wasting, fever and hæmoptysis are not signs of uncomplicated silicosis and asbestosis, although loss of weight does occur very late in both diseases.

The signs of uncomplicated silicosis and asbestosis are by no means obtrusive, and most clinicians form their own conceptions of them. As would be expected from the pathology, there is a very slight diffuse impairment of the percussion note, which some clinicians recognise more as a slight diffuse resistance on percussion rather than impairment of note.

The uniform bilateral character of this change is misleading since it is easy to miss it entirely, there being no normal area for comparison. It must be admitted that it is somewhat of a strain attempting to detect this alteration except in advanced cases: it must be detected quickly or appreciation soon dulls and doubt occurs; rapid light percussion, however, usually reveals it, and comparison with a normal chest of similar build will restore one's appreciation when dulled, as is likely to happen if two or three cases appear consecutively amongst a group of workers for examination. Naturally, also, the chest movement is restricted, and the breath sounds more distant—usually but not always—and the inspiratory murmur thin and higher pitched.

As already mentioned, these are silent diseases, and therefore the presence of adventitious sounds are indications of temporary superadded intercurrent infections, except in cases with concurrent tuberculosis and those with the bilateral and mainly basal bronchiectasis seen in very advanced asbestosis.

The presence of tuberculosis gives the usual indications, but may make recognition of the dust fibrosis more difficult. Usually, however, one can determine what may be described as the oasis of tuberculosis in the desert of the diffuse fibrosis. Silicosis and asbestosis may, of course, develop in lungs already diseased, and their occurrence in emphysematous chests is perhaps the most confusing. The writer

has seen one case in which asbestosis existed conjointly with silicosis.

Radiographic examination should never be omitted and, as that great pioneer Watkins-Pitchford laid down a number of years ago, radiograms must be "technically satisfactory," or doubt and confusion is certain. The value of the radiogram is in confirming the results of occupational and clinical investigation and in estimating the stage of the disease and presence of complications.

Both diseases result in the production of abnormal radiographic shadows, and these shadows reflect the underlying pathological process to a sufficient extent as to make it worth while considering them from this angle.

Thus, following exposure to silica but before the formation of nodules of fibrous tissue, all that is seen radiographically is accentuation of the linear markings and increased root shadows; these are not characteristic of silicosis since they occur with exposure to many harmless dusts, and also irrespective of any special exposure to dust at all; they are within normal limits in the absence of other signs of intrathoracic disease, and no diagnosis of silicosis or of adverse effects of exposure to dust can be made on these appearances. This stage is followed by the appearance of nodular shadows, discrete and fairly uniform in appearance, diffusely scattered over both lung fields; this reflects the underlying pathological condition in silicosis and therefore is strong contributory evidence in the diagnosis of that condition. In between the discrete mottling the lung lights up well, the white and dark areas being nicely contrasted. This is important as this contrast is not evident in a typical radiograph of asbestosis.

As the silicosis progresses many changes in the picture may be seen, the nodules become larger, may aggregate into large masses, and areas of increased translucency indicating associated local emphysema may appear. Increased density of the discrete nodular shadows may even be so pronounced as to suggest calcification and also, of course, quite commonly indications of an active tuberculosis process may show.

The co-existence of a tubercular infection at any stage modifies the picture in many ways and may obscure the diagnosis especially if, when first seen, the tubercular infection is well developed and the associated silicosis not so well developed. The general effect is to soften, agglomerate and blur over the silicotic mottling, even remote from the original focus.

The more evident this softened, blotted and blurred appearance is, the more certain one can be—provided the original mottling is associated with silicosis—of a co-existent tubercular infection, even if the sputum is repeatedly negative.

The radiographical appearances of silicosis are classified in different ways, but the essential point in the radiographic diagnosis of silicosis is the presence of typical nodular mottling.

To form a sound opinion on the radiographical appearances of the lungs of an industrial worker, it will be agreed, is often a matter of extreme difficulty or impossible. Apart from shadows caused by diseases affecting equally the non-industrial population which may cause confusion, such as diffuse carcinomatosis, tuberculosis—chronic miliary or other manifestations—etc., the inhalation of mixed dusts produces variations in the picture thus, silicosis in the potter tends to vary radiographically from silicosis in the sandblaster which again differs from silicosis in the granite worker; the mixer of boiler insulating materials exposed to asbestos fibre, ground up pitchers (fired earthenware body), Kieselguhr, ground up bricks and other organic and inorganic dusts, shows a radiographical picture which can often be recognised as intermediate between those of silicosis and asbestosis.

Again, exposure to fine particulate metal spray (e.g., in zinc metallisation), iron oxide fine particles projected during electric welding, fine metal particles from shot blasting, and gross exposure to talc, or cement dust in the absence of added flint—causes diffuse mottling in the radiographic pictures in some variety which is very confusing as in none of these examples does the picture indicate the existence of a true diffuse fibrosis in the lung parenchyma. A point of some help is to regard with grave suspicion a picture in which a diffuse fish net appearance instead of a diffuse discrete nodulation can be identified since considerable exposure to some harmless dusts produces this appearance.

In asbestosis, also, the underlying pathological process suggests the typical radiographical appearances.

The diffuse fine cobweb fibrosis in its developed stage results in the production of a diffuse fine cobweb mottling, which at the ordinary distance from the viewing box looks like a fine pinhead mottling; the result of this is that the whole film lights up very badly and compared with a typical film of silicosis the absence of the dark areas between the shadows is striking. This has been aptly described by Burton Wood as the "ground glass appearance." Typically it is very uniformly distributed and denser towards the bases. As the fibrosis increases, becomes denser and contracts, the typical pinhead mottling tends to disappear at the bases, but there is no difficulty then in interpreting the shadows as the result of gross and massive fibrosis. The obscuration of the pericardial and diaphragmatic outlines with the "shaggy

heart are outstanding at this stage. No distinctive gradations in the radiographic appearances of the asbestos fibrosis have been generally accepted as yet, and there is extreme difficulty in evaluating such stages.

The effect of a superadded tubercular infection is, as with silicosis, to obscure the typical mottling, and to render the identification of the asbestosis element more difficult, more so than in the case of silicosis.

The main features of the radiographic appearances of asbestosis are, therefore: (1) general lack of translucency; (2) diffuse bilateral pinhead mottling, denser at the bases —the ground-glass appearance; and (3) progressive obscuration of the diaphragmatic and pericardial outlines—with the "shaggy heart." The latter is rare in radiograms of simple silicosis.

It will be agreed that distinctive radiographic appearances are very strong contributory evidence in diagnosis, but that many borderline and difficult cases will be sent for opinion. This being so, a personal collection of radiographs of cases, in which the occupational and case histories together with the post-mortem findings are known, is of the utmost value for comparison.

In diagnosis, weight should always be given to all three aspects: (1) occupational history —nature of, concentration of, and length of exposure to the dust, remembering that the dusty job may have been many years previously; (2) clinical signs and symptoms; (3) radiographic appearances.

With regard to disability in uncomplicated silicosis or asbestosis: the amount of disability produced obviously depends on the extent of the fibrosis, and the nature of the man's work. Usually the disability is remarkably slight until the disease is advanced, and there is no long period of invalidism; the advent of tuberculosis or of some other acute infection quickly closes the scene, since the margin of unaffected and fully functioning lung tissue is too small to cope with added strains of this nature.

Treatment is obviously symptomatic and preventive of further infections; if superadded infections are avoided, life in fair comfort may continue for many years, even in developed cases. The fibrosis does not develop further after the retained silica has exerted all its potential action, but in asbestosis the secondary mechanical and other sequelæ which occur in cases with dense basal fibrosis are an added danger.

In this slight survey only some of the more practical points which appeal to the writer have been stressed. The literature on the subject of the pneumoconioses is now very extensive, and no reference has been made to much invaluable work. A few recent publications only are arbitrarily selected and recommended below as being encyclopædic in range and eminently readable, and by acknowledged authorities.

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OCCUPATIONAL CANCER AND DERMATITIS

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THE consideration of cancer of the skin that has an occupational origin cannot be a discussion of the causing cause of cancer, for cancer of the skin occurs both in employed and amongst the unemployed. It occurs in those engaged in modern industry and in those engaged in domestic occupations, but in some industries its occurrence is so regular and so frequent that one can properly say that, but for the patient's occupation in this industry, the cancer would not have occurred, so that it may be said to be produced by the skin hazard of the trade and the more abstract question of the causing cause is left for further research.

The registration of deaths was put on an organised basis in this country in 1837, but, as regards industrial medicine, death certificates provide no statistics of the necessary facts for coming to any conclusion on this matter. They do not give us the requisite information as to the victim's former employment. Death certificates supply no clue as to the technical process on which a man may have been antecedently engaged. There is no cross-reference either to cases in his family, grandparents, parents, brothers or sisters.