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quire our school authorities, local provincial and Dominion governing bodies; to provide adequate clinics for the reception and treatment of the deafened child, and monies to further the work so ably started by doctors in the larger medical centres.[4]

It is the opinion of all otologists that the care of the deafened must be commenced early in life. It is also recognized that the doctors are anxious and willing to carry out the treatment and re-establishment of those who have serviceable hearing, but it is recognized that before any work can be done on a provincial or Dominion-wide scale, the Departments of Health and Education must provide buildings, equipment and lay teachers, to re-habilitate those deafened children who require speech training, voice correction and re-habilitation in their lives. We know little has been done on any concerted large scale. It would therefore seem necessary that we, from the several provinces of this Dominion, should return buoyant in the hope that work would commence as soon as possible on a program for the diagnosis, treatment and education of the hard of hearing child. This was stressed by the President of the Canadian Otolaryngological Society in his address before that organization.

SUMMARY

There are children born deaf and there are others who become deaf between birth and two years of age. Ordinarily, these will not be able to learn to speak.

Heredity is responsible for only 25% of deafness in children. Early diseases of childhood cause 75%.

Early training of the hard of hearing child is necessary.

Surgical removal of infected and hypertrophied tonsils and adenoids, the patency of the Eustachian tube, is one of the first surgical procedures in treatment.

Radium applicators in the fossa of Rosenmueller may be necessary.

Children in elementary schools are group-tested and if found with a hearing loss of more than twenty decibels, are referred for further attention. The audiometer is being used more and more with greater satisfaction to the otologist.

Institutionalizing of children should be avoided if possible.

School authorities and local provincial and Dominion governing bodies will find it necessary to take greater interest and provide more facilities for the examination and training of the hard-of-hearing child.

REFERENCES

1. FISHER, G. E.: *Eye, Ear, Nose & Throat Monthly*, 5: 222, 1949.
2. MACFARLAN, D.: *J. A. M. A.*, 137: 774, 1948.
3. FEE, G. A.: *Nat. Soc. Deaf.*, 1948.
4. NEWHART, H.: *Trans. Oph. & Oto.*, March, 1943.

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THE PNEUMOKONIOSES: DEVELOPMENTS, DOUBTS, DIFFICULTIES*

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PREVIOUSLY in the history of the pneumokonioses there have been times when the path of progress has disappeared in a forest of data and the traveller was beset with doubts. It seems that we have reached just such a point now, when we must stop and take our bearings. Firstly, what really is the present position? In England we have some 800 deaths a year from pneumokoniosis and there are over 16,000 cases registered as "disabled persons". These numbers may not appear striking compared with the 4,000 deaths a year of adults from road accidents and the 20,000 deaths from respiratory tuberculosis. But, when we stop to try to estimate the relative risks of death from these causes in relation to the numbers of people exposed to them, then the risk from pneumokoniosis appears fantastically high. On this basis, the figure of 800 deaths a year from pneumokoniosis would have to be multiplied by an unknown but large factor to view this risk in correct perspective. It can be said with certainty, however, that a few months' exposure to silica or asbestos dust in gross or, to use Leroy Gardner's happy word, "insulting" concentrations will inevitably cause death. However, when we go further and think, not of one small country, but of the world and consider the toll of life throughout the ages from these diseases from all the mining, quarrying, fabricating and construction work carried out, we may well

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regard them as one of the major scourges of humanity.

Again, when we think of the achievements of medicine in preventing and curing diseases like diphtheria, we cannot help but ask, with something of shame, why the mortality from these dust diseases remains so high, seeing that in theory at any rate, they are wholly preventable. It must be confessed that understanding of the problem and of its urgency, by the professions, managements, workers, and the public generally, is inadequate and ill-diffused and we cannot hope for real advances until this is rectified. This comes about because the pneumokonioses have no spectacular impact on the community, like accidents or dramatic epidemic diseases which make news headlines: silently and insidiously they creep, like old age, upon their victims distributed over many industries, who fade away with little but local disturbances. They are difficult to diagnose and often masquerade as other diseases. The medical teaching schools and textbooks pay little attention to them and nothing to the industrial processes which cause them—without a knowledge of which, diagnosis is but guess work, and also, the terminology used is vague and confusing. What is needed is national and international team-work like that applied to the study of atomic energy and then a very large measure of control of these diseases would be assured. The first steps are to agree, internationally, on a standard terminology, standard criteria of diagnosis, standard x-ray technique, and standard type films, minimum standards of preventive measures, of compensation and rehabilitation procedures, strictly enforced by law: priorities of aspects for immediate research on the medical, engineering and other scientific aspects and, above all, we need intensive national programs of education with the aid of the popular, professional, scientific, and trade papers. Instruction should be made compulsory in relevant degree and diploma courses, in technical institutes and trade schools, and for workers, managerial, and supervisory staffs engaged in work which involve risk of these diseases.

Let us look, very briefly, at some of these points. Consider first the confusing terminology used with these diseases. So far, I have had to use the words "these diseases" with the equally vague alternative "pneumokoniosis" in this discussion, for this very reason. Pneumokoniosis means "dust and the lungs" a vague generality which indicates no particular entity and implies no pathology and no disability. To medical men it means, without further definition, merely "any respiratory condition which may be ascribed to dust". Thus, it may include anything from an abnormal x-ray picture as seen in welders, or silver finishers, or iron-oxide workers, due to diffraction of the rays by minute particles of metal dust which causes no symptoms, no disease, and no disability, to gross fibrosis of the lungs with extreme shortness of breath, compensatory emphysema; pneumothorax, bronchiectasis, and gross pleural, pericardial, and diaphragmatic adhesions, as in advanced asbestosis. It may include cancer of the lung due to radioactive dusts, beryllium granulomatosis, and all the curious affections associated with workers handling grain, bagasse, mouldy cotton and hay. Clearly an expression which implies both trivialities and most serious diseases is of no practical use and is liable to mislead. Again, consider the expression "siderosis". It was coined first I think, as a respectable name for "grinders' rot"—which, at least, told us something—to describe an interstitial fibrosis of the lungs then thought to be caused by fine metal particles: a quarter of a century later, some people used it as a name for silicosis in hæmatite miners, and now, after another quarter of a century it is used to describe both the killing fibrosis of hæmatite miners and the harmless spotting of the lungs by fine metal particles, discernible only by radiography.

Then if, in desperation, we turn to the dictionaries, what do we find? Taking one at random, after stating the derivation of pneumokoniosis to be from the Greek, meaning lung plus dust, it goes on: "A lung condition due to the inhalation of minute particles. It is attended by fibroid induration and pigmentation. See aluminosis, anthracosis, asbestosis, byssinosis, chalicosis, ptilosis, siderosis, silicosis, and tabacosis." As you will observe, some of these expressions are merely alternative appellations, some may not exist and a number are not necessarily attended by, and are certainly not associated with, fibroid induration of the lungs. But, let us not give up: on turning to "ptilosis" we are told it is "a form of pneumokoniosis caused by inhaling the

dust from ostrich feathers''. What this definition really means only the bird knows. I will not weary you further except to remind you that compound names are used, as for instance, silico-anthracosis and I have seen several cases with both silicosis and asbestosis to which, fortunately, as yet no special name has been applied. This is confusion worse confounded: no wonder we mislead and are misled and when the experts collide in the fog, the courts of law flee to the dictionaries, and what a paradise for our friends the lawyers. We simple minded and, of necessity, practically disposed persons, must really avoid being hypnotised by the theoreticians and shackled by the lexicographers, and tidy up this nomenclature. It might profitably be discussed and agreed internationally, as has already been done in defining the meaning of the term ''silicosis''.

In some countries, the law has had to step in for its own urgent purposes and tell us what we mean; for industrial health is dependent not only on doctors and nurses but also on engineers, chemists, physicists, and so on and is primarily a responsibility of governments and of industrial managements, and so everyone concerned must understand exactly what is meant and required. Thus, in our own National Insurance (Industrial Injuries) Act, we find that ''the expression 'pneumokoniosis' 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 has its pitfalls especially in the use of the word ''dust reticulation'', and its limitations, in that it does not include such conditions as ''byssinosis'' (which is dealt with separately); but in spite of this it includes the great majority of related killing and disabling forms of pneumokoniosis, and thus is a practical step forward.

Now a word or two on diagnosis: diagnosis is difficult, which is one reason why both here and in England special boards have been constituted to determine finally diagnosis and degree of disability. In every suspected case, for accurate diagnosis we need to know all about the dust or dusts concerned and the length of exposure to them, the complete occupational history from first starting any work, the clinical history and findings, and the radiological appearances, and inadequate information on and faulty interpretation of any of these aspects can wholly wreck the diagnosis and cause much hardship and injustice. We need, therefore, medical, scientific, and industrial knowledge and so—team work of the best class.

You will note that the points I have mentioned—causative dusts and processes, clinical findings and x-ray appearances—as the basis of diagnosis, are also the basis for progress. Without accurate diagnosis the harmfulness of a dust and the hazard of a particular process cannot be assessed, and without these, appropriate preventive measures cannot be devised. I cannot stress too much the importance of accurate diagnosis and this, it is vital to remember, involves non-medical as well as medical considerations. This leads to the further point that while a certain amount of mysticism is not alien to the art and science of medicine, the age of witchcraft in the practice of industrial health is long since past. Apart from the fact that the patient needs a reasoned prognosis which necessarily is based on accurate diagnosis, managements cannot cope with the responsibilities laid upon them by law without skilled medical aid, and consequently may be quite knowledgeable in these matters.

Now what do we know of the relative harmfulness of dusts? Regrettably, not nearly enough. Nevertheless, we can make some useful generalizations. The first is, that all dusts whatever their composition will cause functional damage, but only some kinds have been proved to be capable of causing fibrosis of the lungs, that is, true pneumokoniosis. The latter are those dusts containing silica in the free state as SIO_2 and also out of the multitude of other mineral dusts, only the small class of fibrous silicates known as asbestos. These in their pure forms, for example as quartz or asbestos, unmixed with other dust, and in the absence of infections particularly tuberculosis, give rise to the classical forms of silicosis, a nodular fibrosis, and of an asbestosis, a cobweb fibrosis, which are closely reflected in the typical x-ray pictures.

Therefore, classical silicosis and classical asbestosis are relatively easy to diagnose, but so often we find these classical types modified by the action or mere presence of other relatively inert dusts, to which the patient has been exposed at some time or another. Infections and some of these other dusts accelerate the development of fibrosis and others are thought to inhibit it, but all tend to modify, often profoundly,

the pathology and radiographical appearance of the classical types. This is where we may run into great difficulties in diagnosis and prognosis and in the advice we give to managements. Excluding terminal broncho-pneumonias, tuberculosis is the most frequent infection. In a series of over 2,000 deaths from silicosis and a series of 232 deaths from asbestosis investigated by my department, 50 and 31% respectively were complicated by tuberculosis. This susceptibility to tuberculosis is covered by our law, which compensates the sufferer from silicosis or asbestosis whether or not the disease is accompanied by tuberculosis.

Exposure to other dusts such as in nickel refining by the Mond process (which also causes cancer of the ethmoid), in the manufacture of chromates and bichromates, and arsenic, is associated with an increased incidence of cancer of the lung, but none of these dusts also causes a true pneumokoniosis. A point of considerable interest which comes to light from our records is the possible association of asbestosis and cancer of the lungs. In the same series, 13.2% of cases of asbestosis were found on autopsy to have cancer of the lungs. This is in contrast to silicosis in which our figure is 1.32%. Little is really known of the mode of action of silica and asbestos dust and the wherefore of these variations, but they emphasize the need for most accurate data on the human cases which occur. There are obvious reservations to the direct application of animal work to humans. Scientists have propounded both mechanical and chemical theories in explanation, but as E. J. King has shown by laboratory experiment the different rates of solubility of silica from different dusts do not parallel their fibrosis producing powers. Similarly Leroy Gardner's mechanical theory as to the mode of action of asbestos dust does not wholly explain the production of asbestosis. Again, Andrew Meiklejohn and W. W. Jones, in a follow-up of pottery workers, exposed to alumina dust have been unable to find any slowing up of the disease in cases of silicosis, in spite of the promise of the laboratory experiments on the inhibition of the solution of silica by alumina. It does not follow that these theories should be discarded, but rather that other factors, including them, play a part and, maybe, in respect of the added susceptibility to tuberculosis particularly, the production of an imbalance of the intracellular enzyme system is one. Most enigmatic of all is the disease described by Shaver and Riddell, and also the mode of production of the fibrosis associated with exposure to beryllium, both of which pose questions as to the rôle of fumes in the production of pneumokoniosis.

However, as all this may be, we must, like St. Paul, throw out a few anchors in the storm and, from the practical point of view, free silica and asbestos are the inherently hazardous dusts which must be dealt with, whether encountered singly or mixed with other dusts. Although so-called individual "susceptibility", that is, the particular state of the individual's lungs plays a definite part, naturally, the greater the amount of silica or asbestos inhaled, the greater the damage to the lungs. But, an important point is that they may be present in quite low proportions in a mixture of dusts and yet cause much damage. Thus I have seen death from silicosis from exposure to an inert dust containing only 10% of added free silica and some samples of French chalk containing less than 10% of free silica and some asbestos, can cause both silicosis and asbestosis at the same time, although the pure material is inert. The reason for this is not difficult to see. A certain amount of silica or asbestos must be retained in the lungs to cause disabling disease, and the effect of overloading the lungs at the same time with large amounts of inert dust is to disorganize the normal lymphatic drainage, and so ensure the retention of the dangerous dust.

This may well be the mode of production of the coal miner's lung, the result of exposure to massive concentrations of coal dust containing very little free silica. With exposure to concentrations of dust of this order, local blockage of lymphatics caused by overloading with dust, or by infections, will prevent the clearing of adjacent choked lobules and give time for the silica, a tissue poison, to act. Thus, we get a condition of focal atelectasis with focal fibrosis and compensatory focal emphysema throughout the lungs, as is beautifully shown by Gough of Cardiff University in his whole lung sections, and this is a clearly progressive condition with continued heavy exposure.

So far I have touched generally on the problems of the true pneumokonioses, dusts in their etiological aspects and the pathological features

of these diseases, and have stressed the vital importance to progress of pooling the skill of medical men, scientists, engineers, and managements. I have referred to the many variations from the classical types of these diseases and the consequential difficulties in diagnosis and prognosis and in assessment of the hazard of the causative work. I have indicated that the doctor's approach to problems in this field is three-pointed by way of occupational history, clinical findings, and radiographical appearances. Now, a few words on the third point—the radiographical appearances. This is just as much a headache as the other two, but is vitally important and may be conclusive. The pathological basis of the disease, that is the fibrosis, is of course reflected in the x-ray picture, but the classical appearances are more often than not modified by coincident tuberculosis, or by other disease, or obscured by the highly diffracting effect of retained inert dust. Moreover, different radiographical techniques and different types of films can mislead completely. Pending internationally agreed techniques and type films which are long overdue, the only practical suggestion is to collect your own library of type films from cases whose complete occupational and clinical histories with autopsy findings are available. This can best be achieved by local medical societies.

In discussing this tremendous national problem of the control of pneumokoniosis, I may have given a pessimistic outlook in saying too much about the difficulties of the position, but history teaches us that always just prior to great advances in knowledge and its application, we have wallowed in just such confusion. Can we hope for a dramatic discovery to guide us, such as that of the bacillus of tuberculosis which ended one long period of confusion as to the nature of these diseases, or that of the roentgen rays which pointed so imperiously the route to better diagnosis? Is it too much to hope that lines of research at present being pursued will lead to a discovery of equal magnitude on the preventive side, such as a perfect inhibitor or an instantaneous continuously recording dust counter, or, dream of dreams, a dust arrester which could be worn on the chest or like a miner's head lamp or both, and prevent dust reaching the mouth and nose and yet leave the face completely free from encumbrance? I hope so.

INFECTIOUS MONONUCLEOSIS

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AN analysis of the last 45 cases of mononucleosis seen on the medical service of the Vancouver General Hospital has convinced us that it is timely to take stock of present knowledge of the condition.

Infectious mononucleosis is a self-limited disease occurring in young people and characterized by fever, sore throat, a swelling of the cervical lymph nodes and frequently of other lymph nodes, an increase in the mononuclear leukocytes and a granulopænia, and a positive Paul Bunnell test. Of these symptoms and findings three are of chief diagnostic importance: (1) Cervical lymphadenopathy. (2) Increase in the mononuclear leukocytes. (3) A positive Paul Bunnell test.

Unless a patient exhibits at least two of these three findings, it is difficult to see how the diagnosis can be more than pure speculation. All the cases in our series have at least two of three criteria, and most of them have all three. An analysis is as follows:

Total cases, 45.
Cases with cervical lymphadenopathy, 36.
Cases with marked increase in mononuclear leucocytes, 44.
Cases with positive Paul Bunnell test, 36.
Of the 9 cases in which cervical adenopathy was not recorded, 5 had a marked tonsillitis.

The one case which did not have a marked increase in the mononuclear leukocytes had 33% mononuclear forms in a total of 4,650, with 35% of polymorphonuclears and 28% staff cells. This patient had enlarged cervical glands, and the Paul Bunnell test was positive in dilution 1:320. He, therefore, showed the main features of the disease.

Before proceeding with an analysis of the cases it is appropriate to discuss the Paul Bunnell test or heterophile agglutination. The serum of normal subjects contains an agglutinin (Forssman) for sheep's red blood cells which does not exceed a titre of 1:8. In most cases of infectious mononucleosis, after 7 to 10 days, the serum shows an increase in this sheep's red cell agglutinin and the titre may rise to very high figures (in this series to 1:10240). Several examinations at intervals of a few days may be necessary to determine whether the result is positive. It is generally agreed that a rise to 1:32 or more is definitely abnormal. A rise in titre has been observed in individuals who have recently received injections of horse serum, but it is rare in other conditions apart from infectious mononucleosis. It is

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