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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 cobweb. 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 thence 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