

Editorial

Benign asbestosis: words and thoughts

My words fly up,
My thoughts remain below
Words without thoughts
Never to heaven go.
W Shakespeare, *Hamlet*, III iii, 97.

I am deeply aware of the heretical enterprise that I am venturing on, or even of the risk of causing stress in some readers by the first two words of my title. It might deserve to be called a contemptuous misnomer were it not to be used in the United Kingdom, the respected motherland of all the basic achievements in the field of that utterly bad condition called asbestosis. Nevertheless, with all due respect towards British occupational medicine, and the country which long ago directed my first steps in that branch of medicine, I dare to propose and use the term "benign asbestosis," bearing in mind all the dissent and resistance, and also all the advantages, of its possible *laissez-passer*.

There are several reasons for this notion but the most important is the irrefutable fact that many cases—perhaps up to 80% or more—of "asbestos related diseases" do have a benign course and this alone would be a sufficiently good reason to call them benign asbestosis. To refer to the common pleural affections, the circumlocution asbestos related diseases was coined apparently to avoid even the slightest connotation of the ghastly pneumoconiosis asbestosis, although the term does not sound more euphemistic than "tubercle bacillus related disease" might have done had it been used instead of "tuberculosis." Yet, since the term may also encompass the neoplastic manifestations, it was necessary to add to the common pleural manifestations the reassuring adjective "benign" and thus the poor syntagma "benign asbestos related disease of the pleura" was born, instead of allowing the term "pleural asbestosis" to come into use as one of the most typical examples of benign asbestosis.

The crucial issue, however, has not been one of semantics. Thoughts on asbestosis have changed as asbestosis itself has been changing its clinical, epidemiological, and, particularly, its prognostic pattern. This changing pattern is repeatedly calling for some revision of our attitude but also for our compliance. In one of his early studies Selikoff pointed out that "asbestosis seen by . . . [old authors] . . . was in some respects not the same asbestosis seen today": rapidly disabling cases of extensive pulmonary fibrosis

due to heavy exposure have become less frequent. This trend has continued and more recently Selikoff stated: "Today, with better controlled exposure levels, the prevalence and extent of parenchymal changes is decreasing . . . and the pleural component of asbestosis is far more commonly seen."² Selikoff's statements from 1965 and 1978 require to be updated again and may also require further modification in the future. The switch from the parenchymal to the pleural asbestos related disease is due not only to the better control of exposure but also to improved radiological technique (normal size films, lateral and especially oblique views, and CT verification). Another contributing factor is the universal experience and awareness of pleural changes as being by far the most frequent seen to follow exposure to asbestos. Increasingly important are the criteria used for the diagnosing of both pulmonary (parenchymal) and pleural asbestosis. Whereas the clinical detection of pleural pathology is almost exclusively dependent on radiology, pulmonary pathology is not. Hence the development of more and more clinical criteria for the diagnosis of pulmonary (parenchymal) asbestosis. Nevertheless, undue weight given to more or less reliable criteria (crackles, rales, clubbing, degrees of profusion of opacities in radiographs or deficits in lung function) often (too often?) leads to the diagnosis of asbestosis.

For example, in a recent paper, for a diagnosis of asbestosis "at least two of the following criteria: (i) pathological chest radiograph—that is, 1/0 or more, (ii) findings in the lung physiology in agreement with fibrosis, or (iii) inspiratory rales on auscultation" were required.³ The diagnosis of asbestosis could be established whichever of the two criteria were present. Seven of the 24 thoroughly examined subjects from a large group presented in the paper were lucky enough to have met only one of the three criteria; having only a "profusion 1/0 or more [of irregular small opacities]; no diagnosis of asbestosis was made" and there was, therefore, no need for them to change their jobs. Had they also had, for example, inspiratory rales on auscultation, however, they would have had the

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misfortune to attract a diagnosis of asbestosis, but obviously "not the same as seen by old authors," but still with all the medicolegal consequences or rights that might arise from the certified disease. This by no means implies that one should question or deprecate the validity of this or any other set of criteria that have been offered from time to time during the past decade. On the contrary, they could undoubtedly be of great help in ascertaining those initial or early, mild or discrete manifestations of asbestosis whose *presence* might be questioned but whose *existence* cannot be denied.

Another mosaic of thoughts. The life long persistence of (calcified) pleural plaques found in thousands of people in many endemic areas is indisputably a reliable biological sign and strong evidence of long lasting exposure to asbestos, both manmade and natural. Pleural plaques—as Becklake⁴ would say—have been dubbed the visiting cards of asbestos. Interestingly enough, however, in the great majority of affected people there seems to be no gross morbidity and no registered mortality from (pulmonary) asbestosis. Nevertheless, are we absolutely sure that discrete non-progressive changes do not occur? Would it not be hard to believe that asbestos fibres, as they travel through the lung parenchyma to reach the pleura, cause the damage to pleura but do not at the same time do any harm to the parenchyma? Are they really so carefully ferried by the macrophages that they are kept only to appear finally at the *parietal* pleural layer "even in the absence of adhesions"?⁷ Or, perhaps, they do not travel via the parenchyma at all?

Further information might give the natural course of asbestosis. As in many other human fibroblastic diseases it apparently depends on the unpredictable behaviour of the fibroblasts. Their inactivity, their activity, or their overactivity is reflected in corresponding degrees of the progression that directs the sequence of events and the outcome of asbestosis. The fate of the patient is thus governed by the dynamics of progression and it is the progression itself that has become the main feature and the main hazard of asbestosis.

Discussing the development and progression of asbestosis in his classic work on occupational lung disorders, Parkes states that "among men with similar exposure some subsequently have severe asbestosis, some mild and others apparently none."⁶ Moreover, "in some cases the disease is apparently arrested and no further progression occurs." It might be noteworthy that such experience stems from the time of heavy exposures. Similar data are given subsequently. Gregor *et al* showed that 30 of 39 cases (77%) with a radiographic category greater than 1/1, followed up for a minimum of three and up to seven years, showed no progression.⁷ "Even within the group of patients

who had severe asbestosis (2/2 and greater) and who were followed over this time, the majority (13/16) displayed no progression." In a nine year follow up study of men who were heavily exposed before 1966 Sheers found (only) six deaths directly attributable to pulmonary fibrosis in a total of 36 cases.⁸

In other human fibrosing disorders we have been trying to learn how to maintain the inactivity of the fibroblasts, how to suppress their activity, or how to fight their overactivity.

In asbestosis we know much about the mortality, we know that—at present—every fifth man may die of carcinoma of the lung, and we know that some will die of mesothelioma even after relatively low exposure to asbestos. We know little, however, about the great majority of those who survive and who might enjoy life without fear and who have the right to know that they have non-progressive asbestosis; whose ailment may therefore, despite all dissent be simply called *benign asbestosis* at least so long as they show no signs of progression.

We can have no doubts that asbestos may cause severe disease with much disability and none about its carcinogenicity, but we should not sentence people with asbestosis of any "profusion scores" or in any location to life long fear. The word asbestosis has to be deprived of its macabre meaning and its grim implications since we cannot ignore those millions and millions of people who retain asbestos *bodies* in their lungs as evidence of the action of asbestos (not of the mere presence of *fibres*) or those thousands and thousands of people with pleural fibrosis as evidence of benign asbestos *disease* (not merely the sign of *exposure*).

One might disregard the thoughts but not ignore the facts! The rest is research.

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