THE METHOD OF EXPRESSING THE SILICA CONTENT OF THE LUNG

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WITH increasing knowledge of the effects of inhaled dusts upon the lungs has come an increase in the number of cases of Workmen's Compensation in which pulmonary disease is attributed to exposure to this form of risk during employment. Where a post-mortem examination has been made, and the opinion of a pathologist is sought, no difficulty is encountered in those cases of silicosis in which the disease is well developed, since the lesions are characteristic and easily recognised. The position, however, is by no means so easy where lesser degrees of pulmonary fibrosis are present and, in particular, where it becomes necessary to try to assess the bearing which these may have upon the susceptibility to, and the progress of, intercurrent pulmonary disease—for example lobar pneumonia. In these difficult circumstances recourse is frequently had to the chemical estimation of the quantity of silica recoverable from samples of lung. Certain standards have been set up, and the presence of silica in quantities in excess of the "normal" forms valuable corroborative evidence that a pulmonary fibrosis is attributable to its influence, where the subject in question has been exposed to this risk in his employment, and on the other hand the absence of an excess of silica is evidence that any pathological condition in the lung is due to other causes.

Such a mathematical expression has not merely an attractive simplicity but it is unequivocal, and in lay or legal eyes is likely to be preferred to a personal opinion involving the complexities of a histological examination. We do not think it necessary to labour the fact that such information cannot be rightly assessed unless it be considered in conjunction with the histological picture which alone indicates the degree of lung damage. What we wish to do, in this short paper, is to point out certain pitfalls into which the mere expression of silica content in a certain form may lead, and to suggest the desirability of the adoption of a standard method of expression. Whilst limiting ourselves to silicosis, the points we raise would apply equally to other pneumokonioses.

The more obvious ways of expressing the silica content of the lung are:

- (a) As a percentage of the moist weight of the organ.
- (b) As a percentage of the dry weight.
- (c) As a percentage of the mineral ash.
- (d) As total silica.

The information which is desired is the amount of silica available for unit quantity of the organ. The whole difficulty is that the estimate of quantity in the lung is not at all easy, since the organ is very frequently altered by pathological conditions (some of which are incident to the disease and some to the dying state), which result in the destruction of lung tissue and its infiltration with a variety of extraneous substances all of which are capable of disturbing the weight relationships. It is to the extent and reality of these disturbances which we wish to direct attention.

(a) *Silica as a percentage of the wet weight of samples of the lung.* This method of expression is not used and would be of little value on account of the very wide alterations in lung weight which common pathological conditions such as oedema, hypostasis, or terminal pneumonia, involve.

(b) *Silica as a percentage of the dry weight.* This is a method which is widely used. Kettle and Archer (1933) and McNally (1933) state that the ordinary dried lung does not contain more than 0.2 per cent. of silica. Although an improvement upon the first-mentioned method of expression, this is not free from fallacies. The dry weight of a lung, or any part of it, will vary considerably with any pathological condition which may be present. A couple of examples will illustrate this.

Case 1. A man aged 53, who had worked for 12 years as a stonemason, died of lobar pneumonia. There was no naked-eye evidence of silicosis but microscopically small scattered foci of fibrous change were present and a few definite silicotic nodules were found in the bronchial lymph glands. The percentage of silica in the dried lung on the consolidated side was 0.139, and in the aerated, non-pneumonic, side 0.276. The difference of course is due to the great increase in ratio of organic to mineral matter produced by the pneumonia.

Case 2. That of a workman exposed to the same risks who also died from pneumonia. The following results were obtained:

	Pneumonic lung (right)	Non-pneumonic lung (left)
Weight of whole organ	1000 g.	450 g.
Dry weight	131 g.	62.6 g.
Total ash	6.81 g.	3.64 g.
Silica (as percentage of dry weight)	0.528	1.016
Silica (as percentage of ash)	10.17	17.41
Total silica in lung	0.69 g.	0.64 g.

The above examples show that pneumonic consolidation, by greatly increasing the proportion of organic matter, can have the effect of halving the percentage of silica in the affected lung when expressed in this way. We do not put this gross form of disease forward as a pitfall likely to entrap any but the most unwary; but we do wish to emphasise the degree of the error which can arise from such change. In view of the frequent occurrence of foci of consolidation from tuberculosis, terminal pneumonia or other cause, in these cases it is clear that if the silica content is to be expressed in this form widely differing figures may be obtained from different samples of lung, whilst if a comprehensive sample of the whole organ be used the percentage figure for the

silica content will be lowered in proportion to the extent of adventitious pathological change.

(c) *Silica as a percentage of the mineral ash.* This method of expression might at first appear less objectionable than any of the foregoing, since all organic material is destroyed. It is, however, open to precisely the same fallacies and for the same reasons. The presence of other pathological products will result in an increase in the quantity of ash (as exemplified in case 2 above, in which the ash in the pneumonic lung was almost twice that in the healthy lung). There are also conditions in which a special increase in certain inorganic substances can take place. We have encountered one remarkable example of this which we shall quote.

Case 3. A stonemason working chiefly, and latterly exclusively, in limestone but who at various periods had also been exposed to silica dust. The lungs exhibited a most extraordinary degree of calcareous infiltration, containing hard stony masses and being covered by an extensively calcified pleura. Analyses showed the presence of large quantities of inorganic ash, amounting to about 23 per cent. of the dried tissue. Histological preparations showed very extensive fibrosis of the lung with heavy calcium deposits and, in the less disorganised portions, the whorled nodules of silicosis. With polarised light, scanty, colourless, bright, doubly refractile particles were detectable in the affected zones. The average results obtained by a number of analyses of ash are as follows:

	%
Carbonate (as calcium carbonate)	6.4
Phosphate (as calcium phosphate)	37.2
Iron (as phosphate)	7.0
Calcium unaccounted for (as hydroxide)	19.2
Silica (as SiO ₂)	2.4

In the samples from the most affected portions of the organ, which included overlying calcified pleura, the ash represented 35 per cent. of the dry tissue, its silica content being from 1.2 to 1.8 per cent. In the less extensively mineralised lung the ash was only 7 per cent. of the dry weight with a silica content correspondingly raised to 4.24 per cent. In this case a chemist, to whom a portion of the lung was submitted for analysis and who had not the assistance of the microscopic findings, arrived at the conclusion, on the basis of finding 1.1 per cent. of silica in the ash, that there was no evidence of silicosis.

Since we did not obtain the whole lung we are unable to give the total quantity of silica which it contained, but assuming a normal weight of about 650 g. we can calculate that this would come out at between 2 and 4 g. per lung. Actually the lungs were much over normal weight, on account of the large inorganic content and extensive fibrosis, and the total silica must have been considerably in excess of this.

In this connection we would draw attention to Stewart and Faulds' findings in the sidero-silicosis of haematite miners, and those of Cummins and Sladden in the silicosis of South Wales colliers. The average figures obtained from their papers can be tabulated as follows:

	Percentage of dry lung		Silica content of ash %
	Ash	Silica	
Stewart and Faulds	18.5	1.7	9.2
Cummins and Sladden:			
Group A	8.8	2.4	27.1
Group B	7.6	1.2	16.3
Group C	5.4	0.13	2.4
Group A, advanced silicotics; Group B, early and moderate silicotics; Group C, controls.			

It is clear that if judged only on the basis of their silica content percentage there might be reason to consider the degree of silicosis in Stewart's cases as only moderate. But if proper allowance is made for the effect of the much greater content of adventitious inorganic matter (on an average the lungs held 8.3 per cent. of iron oxide), we find that the proportion of silica to lung is almost as high as in Cummins' and Sladden's group A.

Cummins and Sladden have suggested the ratio of

$$\frac{\text{Organic tissue-solids (combustibles)}}{\text{Silica}}$$

as one which will give a fair indication of the degree of silicosis, and find ratios of 38 : 1 in advanced silicotics and 720 : 1 in controls. This would undoubtedly be the case if the combustibles represented normal lung—but this is far from the case, and the objections we have offered against the dry weight also obtain here. Thus in a pneumonic case (case 1) the ratio for the healthy lung is 93 : 1, whilst for the consolidated lung it is 180 : 1.

(d) *Total silica.* For reasons set out in the preceding paragraphs we have come to the conclusion that the method of expressing siliceous infiltration in the lungs least liable to errors and most valuable for purposes of comparison is as the total silica content of the organs. If, however, a single lung is used, it must be remembered that the organs are of slightly different size, the right being the heavier and, as the findings of Cummins and Sladden show, often containing a considerably larger total quantity of silica than the left. It might therefore be preferable as a routine to use only one side—*e.g.* the right—for analysis. If this be done, the only fallacy which remains is the differences in lung size between individuals: a person with a large lung and corresponding vital capacity would presumably accumulate more silica than a small one with a smaller vital capacity. Whilst we have no figures to show us what differences arise in this way, it does not seem likely that these would ever be great.

In a problem in which we are looking for a measure of the departure from the normal, the method of assessing this which shows the largest range of change in correlation with the degree of pathological alteration is the most desirable. It is of interest, therefore, to test the figure we have advocated against others which are in use. For this purpose we can again utilise the results of Cummins and Sladden in the groups of Welsh miners already mentioned. Thus:

	Total silica	Silica percentage of dry lung	Silica percentage of ash
Group A (silicotics)	9.4	2.36	27.1
Group B (exposed to risk)	4.4	1.24	16.3
Group C (controls)	0.29	0.13	2.4

Reduced to simple ratios the silica contents as expressed by these three methods are found to be: total silica 1 : 15 : 32; as a percentage of dry lung 1 : 9.5 : 18; and as a percentage of ash 1 : 7 : 11. It therefore appears, from this

as from other considerations, that the method of expression which best emphasises the pathological condition is the total silica.

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