THE RADIOLOGY OF ASBESTOSIS*

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Asbestos can damage the lung and may cause pulmonary fibrosis, pleurisy with the formation of thick plaques and mesothelioma of the pleura and peritoneum, and carcinoma of the lung. Forty-three cases of deceased asbestos workers who had X-rays of their chests taken during their working lifetime are reviewed. Chest X-ray comments are based on uncomplicated cases of asbestosis, and the impressions gained are documented.

Selikoff[1] determined the presence or absence of pleural calcification, giving due regard to X-ray technique. In Selikoff's[1] cases, films of both anterior oblique positions were also taken, with the object of demonstrating pleural calcification. Some cases of calcification and pleural thickening will be missed if routine oblique films are not taken. The pleural location of plaques can be more clearly determined when seen tangentially in the oblique films. In the postero-anterior views the plaques may appear broadside on, with an irregular, indeterminate outline. It is to be anticipated that a smaller number of plaques will be found in a survey which fails to use both higher kilovoltage and oblique position techniques.

Plaques of soft-tissue density are probably more common than calcified plaques. In addition pleural lesions are more frequent in asbestosis than radiologically discernible fibrotic changes in the lungs.[2] Calcification is a typical late manifestation, and, although of great diagnostic aid, the detection of the earlier non-calcified plaque of soft-tissue density would be more helpful in early assessment of asbestosis. Pleural effusions in early asbestosis are infrequent. Asbestos bodies have only occasionally been found in the pleura. The sudden appearance of a unilateral or bilateral pleural effusion after many years of exposure is more suspicious of a sensitivity response than of a direct local toxic reaction.[3] Collins[4] mentions 2 cases of benign asbestos pleurisy (unproved) with clinical evidence of lung disease—presumably asbestosis—before the episodes of pleural effusion. These 2 cases, unlike the case referred to by Eisenstadt,[3] had significant tuberculin reactions. The review presented revealed 2 possible cases of pleural effusion. The 2 cases may well be a manifestation of uncomplicated asbestosis.

Eisenstadt[3] suggests that pleurisy may be acute, subacute or chronic. The history of prolonged exposure to asbestos dust is of greatest diagnostic significance. The appearance of an effusion on one side, followed sooner or later by a similar one on the other, seems to be most characteristic of asbestosis. The effusion is usually self

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limiting and disappears within a period of weeks or months. Pleural calcification is frequently absent at the time of active pleura parenchymal pulmonary disease.[3]

NON-CALCIFIED RADIOLOGICAL PLEURAL CHANGES

Pleural changes are sometimes the only radiological evidence of pulmonary asbestosis. Pleural changes, of a minor nature, include costophrenic blunting in the cases observed. Soft-tissue thickening of the pleura is usually noted along the axillary pleural line. A narrow or emphatic linear or lamellar shadow along the axillary margin, usually greater than 2 - 3 mm. in thickness and of variable length, gives less diagnostic difficulty than the more centrally disposed faint opacities of soft-tissue densities described by Anton.[2] A linear outline of calcification, when present, makes these soft-tissue density plaques more obvious. The lamellar pleural plaques are commonly detected at the lung bases. In some cases they are associated with obliteration and distortion of the corresponding costophrenic sulcus. The triad of exposure, lung fibrosis and peripheral plaques of soft-tissue density (lamellar thickening of the axillary pleural line) suggests the radiological diagnosis of asbestosis.

Subpleural oedema may produce a similar lamellar-like change. This may occur in cases associated with left ventricular, left atrial, or congestive cardiac failure. Hypoproteinaemic states or tuberculous pleural involvement can also emphasize the pleural line.

More extensive uni- or bilateral pleural involvement may obscure underlying parenchymal pathology or distort the cardiac outline. Opacification of either hemithorax due to massive pleural change can occur with asbestosis. Basal pleural reaction is not uncommon; however, the inverse state may occur, leaving a translucent lower lung field. A shaggy cardiac outline is not a noticeable feature in the X-rays reviewed. It is possible that this particular change is a feature of the industrial exposure to asbestos rather than the group exposed in the mining of the material.

Galatius-Jensen and Halkier[6] reported 2 cases of well-circumscribed pleural densities. Histology revealed a chronic inflammatory pleural response. They refer to these lesions as pleural hyaloserositis and indicate that unnecessary thoracotomies may be prevented if this differential diagnostic possibility is kept in mind. Tivenius[5] described 10 similar cases of benign pleural lesions simulating tumours.

CALCIFIED PLEURAL PLAQUES

It would appear there is a dose-response relationship with regard to the development of pleural plaques and their detection. The most extensive lesions are radiologically detectable and the disease asbestosis is also present, despite the absence of any other radiological abnormality.[8] The presence of plaques can be taken as evidence of exposure. Focal pleural calcification may be noted radiologically in asbestosis,[2,9] but may also be seen in those who work with asbestos or other silicates without evidence of pulmonary disease.[10]

There is strong evidence from Finland[11] that environmental (i.e. non-industrial) exposure to asbestos leads to the relatively common finding of focal pleural calcification (calcified pleural plaques) in a community.[12] The deposit of calcium on the pleural surfaces of the lungs and chest cavity is recognized as an occasional sequel of pleurisy, tuberculous or otherwise, and of injuries followed by haemothorax. In a study of calcified pleural plaques at Sea View Hospital, where 80% of those examined had tuberculosis, Taylor and Schwartz[13] reported an incidence of plaque formation of 0·1% among 10,000 cases examined in 3 years.

Smith[10] examined the radiographs of 8,779 workers and found evidence of pleural calcification in 0·4%. The highest incidence of calcification occurred in workers associated with the dust from tremolite (6·3%). Calcimire (a complex of mica, tremolite and asbestos) had an incidence of 1·7%. Mica had an incidence of 1·6% and bakelite 1·5%. Pleural calcification is a reliable and accurate index of long-standing previous asbestos exposure.[9]

No portion of the pleura is immune to calcification. The common site of calcific plaque formation is diaphragmatic. It may be seen along the lateral chest wall and paravertebral gutters. Various portions of the mediastinal pleura may be involved. The pericardial surface, particularly the left heart border, is not infrequently calcified.

The concentration and distribution of calcium in different parts of the pleural plaques are irregular. The accumulation of calcium seems to be a secondary phenomenon dependent on age. Calcified plaques of the parietal pleura are often observed on gross autopsy inspection. They are seldom seen on routine X-ray examination of the chest, obviously owing to the low calcium content of the plaques.[14] Irregular opacifications of non-calcified plaque formation may be noted in conjunction with pleural calcification. Associated pulmonary fibrosis is not an uncommon radiological accompaniment to pleural calcification, in the cases examined.

An early pattern of pleural calcification may remain unchanged over many years although the calcification becomes more dense. Radiographs of a father and son are available over a period of 7 and 10 years, respectively, to confirm this. The type of calcification noted varied in size from a single linear deposit of a few centimetres in length in the region of the diaphragm, to massive deposits, bizarre in shape, extending over a large part of the lung fields. No evidence of hilar lymph gland calcification was seen. Uni- or bilateral calcification varying from fine slivers and short, thick plaques to extensive, well-defined areas was noted in both symmetrical and irregular patterns. Multiple calcific plaques are common. Mediastinal pleural

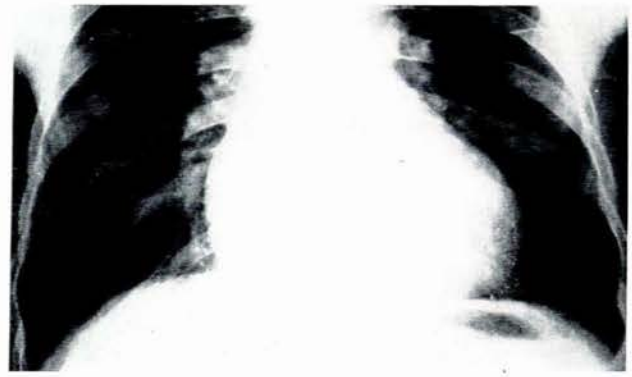

Fig. 1. Accentuation of fissures.

involvement may be extensive when seen in lateral profile. The diaphragmatic calcifications are often dense and follow the contours of the muscle, producing thick or thin curvi-linear plaques. Other changes noted basally as well as on the upper lung fields were patterns of leaf-like contours with spiked edges, large shield-like calcifications and radiolucent centres in areas of calcification giving a button-like appearance to the region. Evidence available indicates that the pleural calcifications seen with asbestos involve the parietal pleura. Calcification or accentuation of interlobar fissures in isolated instances suggests visceral involvement. Accentuation of interlobar fissures is a feature in some of the cases in this series (Fig. 1).

Extensive areas of calcification may obscure the pulmonary parenchyma. Tomographic studies may reveal linear areas of calcification within thick areas of pleural fibrosis. It is likely that asbestos exposure is the most common cause of pleural calcification.

The findings of calcification described here generally indicate asbestos exposure more than 20 years before.[3]

LUNG CHANGES IN ASBESTOSIS

Comments in this article are based on postero-anterior films on asbestos miners who, to the best of my knowledge, had not been engaged in other forms of mining. Care has been exercised in excluding complicating factors such as associated tuberculosis, neoplasms, or any other pathology. The X-rays reviewed are concerned with uncomplicated asbestosis. All the cases reviewed had worked in crocidolite or amosite mines. It is important to realize that these minerals occur in banded iron-stone which contains up to 40% free silica, and that mill dust may contain up to 20% free silica. The country rock, and to some extent the ore, contain important amounts of the iron minerals magnetite, haematite and ferric hydrate (40% or more).[15]

Definite pulmonary disability due to asbestosis has been reported in the absence of an abnormal radiographic pattern.[16] A definite relationship between macroscopic presence of asbestosis and linear markings on the X-rays could be demonstrated.[17] Leathart,[18] reviewing both the literature and his own series of cases, recorded the following parenchymal changes:

1. Pin-point mottling.
2. Ground-glass homogeneous veiling of the middle third of the lung fields.
3. Accentuation of the lung markings which criss-cross to give the appearance of a wide-mesh net.
4. Enlargement and shagginess of the hilar shadows.
5. A shaggy and indefinite outline of the heart.
6. Translucency of one or both upper lung fields.

The radiological changes in the South African asbestos worker differ from Leathart's description. A not unimportant reason for this difference may be the high silica and iron content of the South African mined asbestos rock. In asbestosis, pleural plaque formation, as well as calcified changes and pleural reactions, may occur in conjunction with a mixed pulmonary fibrotic reaction and lend difficulty to radiographic interpretation (Fig. 2 and 3). Disturbance of the normal vascular pattern with the basal pulmonary arterial tree assuming a bead-like disruption may be a suggestive early change in asbestosis.

Homogeneous clouding of the radiographs, or the ground-glass homogeneous veiling of middle and lower lung fields is the change usually found when fine fibrosis predominates. Peripheral basal straight-line fibrosis at this stage may simulate Kerley B lines. This diffuse, fine fibrotic change preserves the anatomical contours of the lung. In the radiographs reviewed the change was not commonly observed on its own. Punctiform and nodular changes often accompanied fine linear fibrosis.

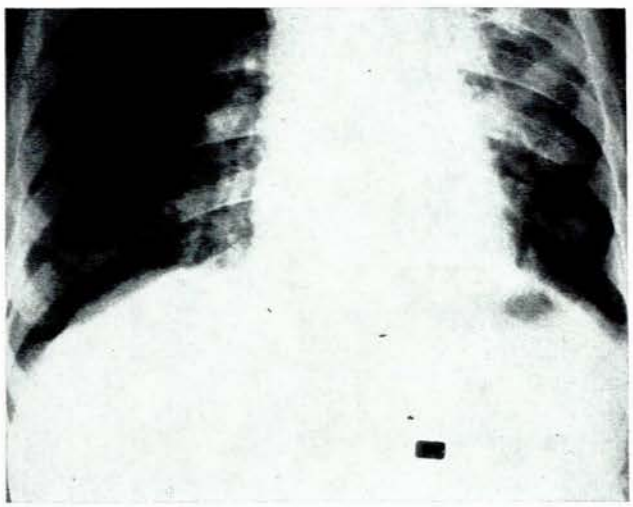

Fig. 2. Soft-tissue plaques, axillary pleural thickening and coarse fibrosis.

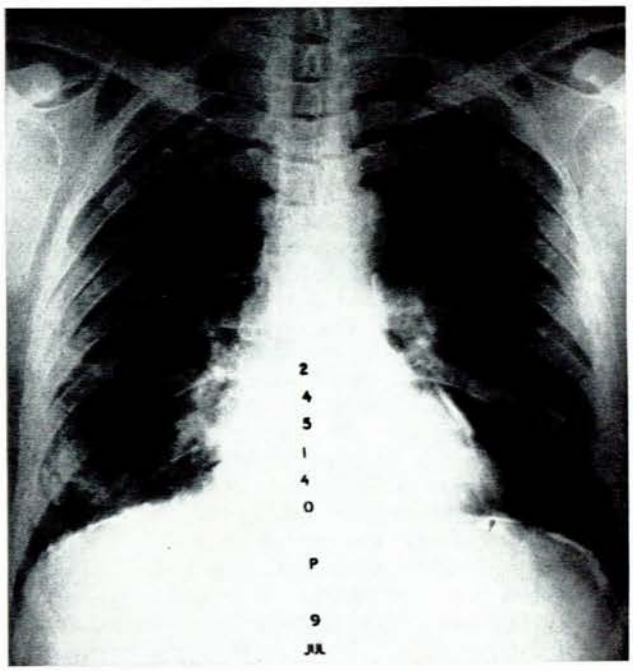

Fig. 3. Calcified pleural changes.

In a good quality radiograph, mottled or nodular changes, rarely exceeding 5 mm. in diameter, seem to be composed of individual rounded nodulations of very

variable size with moderately poorly-defined margins. Pronounced nodular changes are common; they may involve the whole lung field and be the predominant pattern (Fig. 4).

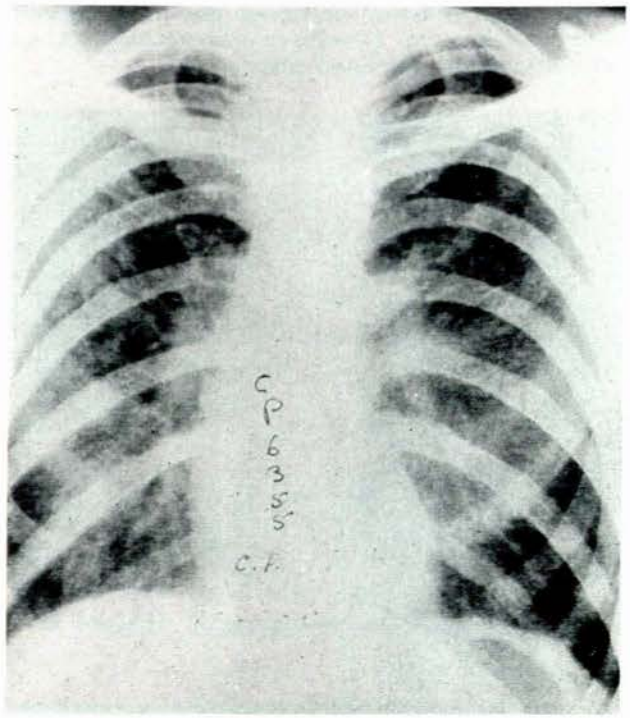

Fig. 4. Nodular lung changes.

Confluence of nodulations producing massive fibrotic lesions was not confined to any particular segment of the lungs. These may occur in one or both lungs, and may be single or numerous. As many as three massive lesions were noted in the right apical region in one of the cases under observation. It is suggested that basally distributed massive opacifications of unusual nature might be questionably neoplastic in view of the high incidence of basal carcinoma in asbestosis.[19] The massive fibrotic lesions may be peripheral or centrally disposed. The iron content of massive fibrotic lesions does not appear to be significantly increased on routine staining of histological sections for iron.[20] The possibility of these lesions being complicating tuberculosis should be considered.[20] Massive fibrotic lesions were seen in at least 6 cases under review. The areas of opacification varied from 2 to 10 cm. The background pattern, radiologically, was predominantly nodular in the cases with confluent opacifications (Fig. 5). Coarse fibrotic lung changes were also present in 3 cases. No area of necrotic cavitation or calcification was detected in the areas of massive fibrosis. No suggestion of hilar calcification or adenopathy was detected in any of the X-rays reviewed. (Massive fibrosis in silicosis, associated with gold-mining, is infrequent in comparison with massive fibrosis in asbestosis. Basally distributed massive fibrosis in gold-miners is, in fact, almost non-existent.) (Fig. 6.)

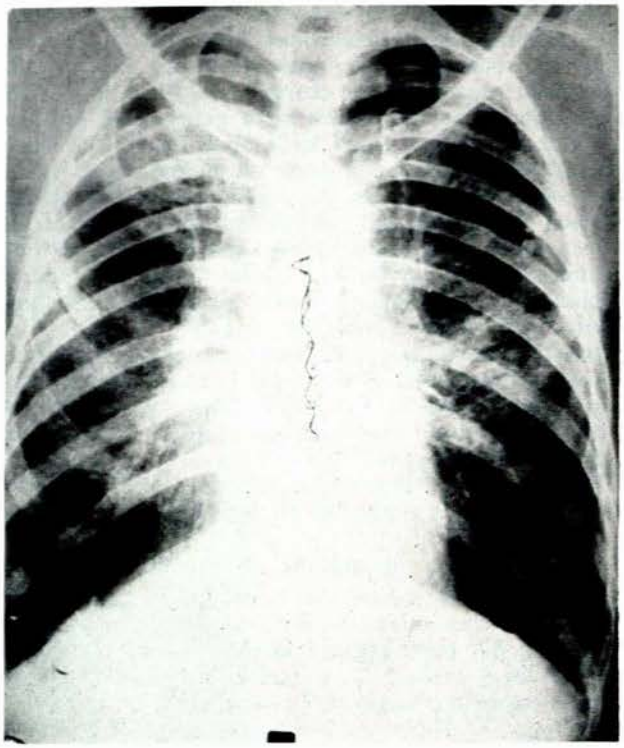

Fig. 5. Pleural calcification, right apical massive fibrosis, nodulation and coarse basal fibrotic lung changes.

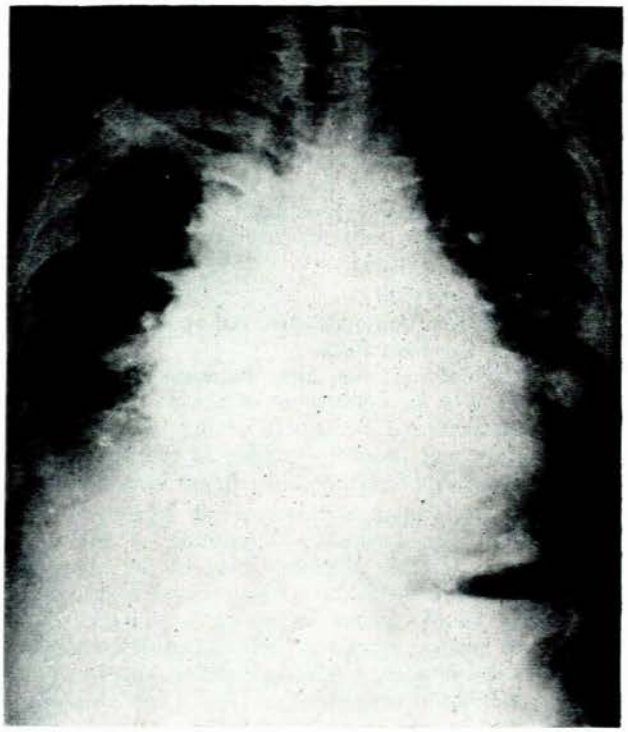

Fig. 6. Massive right basal fibrosis with extensive bilateral pleural changes.

The edges of the massive fibrosis were not clearly outlined, and in the smaller lesions the appearances suggested coalescence of nodular lesions. The radiological patterns did not always correlate with postmortem changes in the lungs. Established coarse fibrosis produced distortion of vascular patterns. Strand-like broad linear bands with associated areas of translucency were not confined to any particular region of the lung. These coarse fibrotic changes were not related to the areas of massive fibrosis. They were seen with accompanying nodular or pneumonitic changes. Massive fibrosis in one lung field associated with coarse fibrosis of the opposite side was not unusual. Solid fibrosis of a lobar or segmental region produced a homogeneous opacification on X-ray (Fig. 7). These areas of pneumonitic fibrosis occur as the sole lesion, or in conjunction with linear punctiform nodular or massive fibrosis. One or both lung fields can be involved. No significant difference of the lung patterns on X-ray could be attributed to exposure to amosite or crocidolite.

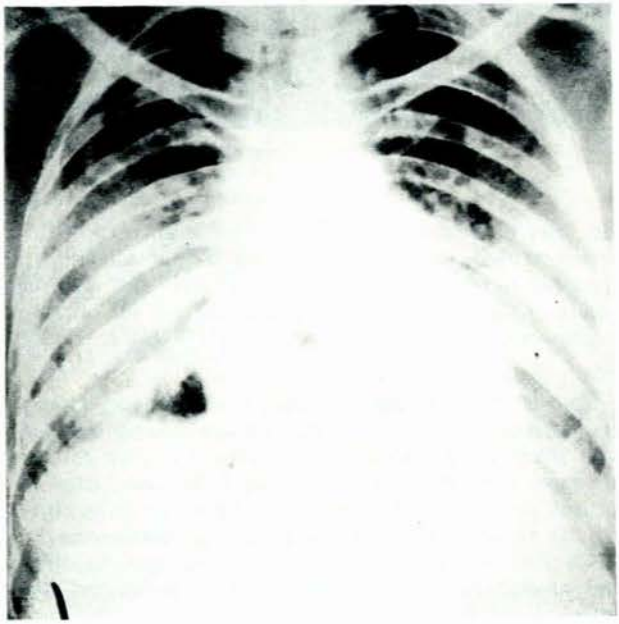

Fig. 7. Bilateral pneumonitic fibrosis.

SUMMARY

Pleural effusions, non-specific pleural reaction, accentuation of the fissures, lamellar pleural thickening and non-calcified pleural plaques should be regarded as significant changes in patients who give a history of asbestos exposure. These changes may occur with minor histological evidence of asbestotic fibrosis in the lungs. Calcification of the pleura is a late manifestation of asbestos exposure.

Combined pleural and parenchymal radiological lesions are generally rare in the uncomplicated pneumoconioses. In asbestosis combined lesions occur commonly and should alert the clinician to the diagnosis. There is absence of lymph gland involvement.

In South Africa progressive massive fibrotic lesions occur more commonly in asbestosis than in the gold- and coalworkers' pneumoconioses. Massive fibrosis is not uncommon as a basal lesion in asbestosis; in the pneumoconioses associated with gold- and coalmining, a basal massive fibrotic lesion is an unusual manifestation. Pleural changes associated with a basally-disposed massive fibrotic change are almost diagnostic of asbestosis.

Nodular, pneumonitic and massive fibrotic changes often occur in combination. Fine and coarse linear fibrosis usually accompany punctiform and nodular patterns. The radiological changes bear a direct relationship to the degree of fibrosis of the lung determined histologically. The radiological pattern, however, does not always correlate with the histological changes.

The iron content of massive fibrotic lesions does not appear to be significantly increased on routine staining of histological sections for iron.[20]

REFERENCES

1. Selikoff, I. J. (1965): Ann. N.Y. Acad. Sci., **132**, 351.
2. Hurwitz, M. (1961): Amer. J. Roentgenol., **85**, 256.
3. Eisenstadt, H. B. (1965): J. Amer. Med. Assoc., **192**, 419.
4. Collins, T. F. B. (1967): S. Afr. Med. J., **41**, 639.
5. Anton, H. C. (1967): Brit. J. Radiol., **40**, 685.
6. Galatius-Jensen, F. and Halkier, E. (1965): *Ibid.*, **38**, 944.
7. Tivenius, L. (1963): Thorax, **18**, 39.
8. Hourihane, D. O'B., Lessof, L. and Richardson, P. C. (1966): Brit. Med. J., **1**, 1069.
9. Lawson, J. P. (1963): Clin. Radiol., **14**, 414.
10. Smith, A. R. (1952): Amer. J. Roentgenol., **67**, 375.
11. Kiviluoto, R. (1960): Acta radiol. (Stockh.), suppl. 194, 1.
12. Oosthuizen, S. F., Theron, C. P. and Sluis-Cremer, G. K. (1964): Med. Proc., **10**, 496.
13. Taylor, H. K. and Schwartz, S. (1937): Quart. Bull. Sea View Hosp., **3**, 65.
14. Meurman, L. (1965): Acta path. microbiol. scand., suppl. 181.
15. Sluis-Cremer, G. K. and Wagner, J. C. (1964): In *XIV International Congress of Occupational Health, Madrid, 16 - 21 September 1963*, vol. 2, p. 608. Amsterdam: Excerpta Medica Foundation.
16. Milby, T. H. *in* Gafafer, W. M., ed. (1964): *Occupational Diseases: A Guide to Their Recognition*. PHS Publication No. 1097. Washington, DC: US Department of Health, Education and Welfare.
17. Caplan, A., Gilson, J. C., Hinson, K. F. W., McVittie, J. C. and Wagner, J. C. (1965): Ann. N.Y. Acad. Sci., **132**, 379.
18. Leathart, C. L. (1960): Brit. J. Industr. Med., **17**, 213.
19. Bohlig, G. and Jacob, G. (1956): Dtsch. med. Wschr., **81**, 231.
20. Webster, I. (1968): Personal communication.