

nevertheless that a considerable number of men who develop complicated pneumoconiosis when aged 30 to 40 are a very bad risk. Tuberculosis was present in 36 per cent. of all pneumoconiotic deaths, in 16 per cent. of all deaths due to simple pneumoconiosis, and in 39 per cent. of all deaths due to complicated pneumoconiosis. The figure (36 per cent.) of the extent of complicating tuberculosis in coal miners' pneumoconiosis is higher

than the figure given by Gooding and others, but it is considerably lower than the figure for other industries, especially for slate mining in North Wales where tuberculosis is present in 75 per cent. of pneumoconiotic deaths.

Finally, one is impressed by the apparently increased susceptibility of coal miners with simple pneumoconiosis to pneumonia and acute respiratory infection.

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ASBESTOSIS

By H. WYERS, M.A., M.D., D.I.H.

Although certain difficulties remain to be explained, the theory that silica is a causative agent in pulmonary fibrosis is widely accepted. When, however, the fibrogenic potentialities of silicates are considered, these difficulties are very greatly increased. Olivine, for example, has been shown to provoke only a foreign body reaction and has been suggested for foundry work, whereas asbestos is notorious. Studies in mineralogical composition of atmospheric dusts and ashed lung tissue are progressing and to the data found theories of solubility and crystal form are being applied. If the solubility is high, as in the case of silicic acid gels and sols, the material is eliminated too fast to produce fibrosis. A low solubility, such as in mixtures of quartz and a minimum, equally retards fibrosis (King, 1938). There appears also to be an optimum size of particle with a surface area which will release silica in sufficient quantity for a sufficient length of time.

In animal experiments this was found to be about 0.1 micron (Febbens *et al.*, 1945). In the case of asbestos fibres, it has been shown that

whereas there is an excess of chrysotile over hornblende varieties in dusts, ashed lung tissues contain only hornblende (Sundius, 1938; Kuhn, 1941). It is concluded that it is the chrysotile in solution which is the fibrogenic agent. Gardner (1938) demonstrated by animal experiments that longer fibres produced a fibrotic reaction which did not occur with shorter fibres and concluded that asbestosis is, at any rate in part, a mechanically conditioned complex of lung changes. In support of this mechanical theory, Johnstone (1948) records the clinical observation that workers at the Thetford Mills, exposed to high concentrations of extremely fine asbestos dust, did not suffer from asbestosis.

Structurally silica shows continuous three-dimensional arrangements of SiO_4 tetrahedra and to these may be related such a surface as to produce the characteristic biological effects. Those silicates, therefore, which most nearly resemble quartz in having a three-dimensional network of silicon-oxygen tetrahedra are likely, theoretically, to produce similar biological phenomena. These

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are grouped in diminishing order of similarity by Nagelschmidt (1949) as alkali feldspars, sheet structures such as micas and kaolin, double and single chain structures like amphiboles and pyroxenes and, finally, the orthosilicates such as olivine. Animal experiments tend to confirm this, but it is pointed out that the type of binding of the silicon-oxygen tetrahedra is of biological importance and aluminium silicates, such as sericite and kaolin, are not likely to present a major silicosis hazard.

The diagnosis of pathological states of the lungs, due to dusts, also presents many clinical and radiological difficulties. A high incidence of pulmonary disease among those exposed to pure silica is presumptive evidence of an etiological relationship, but the rare case of talc pneumoconiosis is difficult to establish. High atomic weights and consequent radio-opacity produce shadows on the X-ray film from inhaled dusts which are not necessarily pathological, as, for example, in the siderosis of arc welders (Doig and McLaughlin, 1936).

Asbestos, its History, Uses and Mineralogy

Whatever doubts there may be as to the fibrogenic potentialities of many silicates, the processing of asbestos unquestionably exposes the worker to the risk of lung disease. It has been used on a small scale since very early times, being known to the Greeks as $\delta\alpha\sigma\beta\epsilon\alpha\sigma\tau\epsilon\varsigma$, signifying unquenchable. Used as a substantive, $\delta\alpha\sigma\beta\epsilon\alpha\sigma\tau\epsilon\varsigma$ was unslaked lime, whilst the masculine gender, $\delta\alpha\sigma\beta\epsilon\alpha\sigma\tau\epsilon\varsigma$ was used to denote asbestos. The word $\alpha\sigma\beta\epsilon\alpha\sigma\tau\iota\varsigma$ which in modern English speech signifies the disease asbestosis, was to the Greeks a plastering or stuccoing. This last word was originally suggested by Cooke (1924) when he also drew attention to a passage in Herodotus describing a cremation cloth made of asbestos. The Romans knew it under the name of amianthus, a word of Greek origin signifying absence of miasma or pollution. They obtained it from the Italian Alps and the Ural. It seemed appropriate that the Vestal Virgins, guardians of the Sacred Fire, symbol of the deepest emotions and instincts in Roman family life, the last rite of paganism to be extinguished, should have used asbestos lamp wicks. Another fibrous mineral resembling Italian amianthus was spun and woven into a cloth in Siberia during the 13th century. A factory for the manufacture of asbestos articles was opened in Russia in 1760. The re-discovery of asbestos in 1876 contributed greatly to modern industrial expansion.

Today asbestos is used for a wide variety of purposes. Textiles enter into the manufacture of fire-fighting suits, safety curtains and boiler

mattresses. Asbestos enters into the composition of lagging material for steam pipes, jointing for steam pipes, panelling of rooms, tiles, the lining of chemical pans, the coating of bulkheads of ships and marine piers and perhaps most significantly into the brake linings and clutch rings of motor cars.

The properties which commend asbestos to modern industrialists are its resistance to heat, acids, electricity and sea water and its fibrous structure which enables it to be spun. Unlike vegetable fibre, it can be split in a longitudinal direction to microscopic size without discovering an ultimate element and without loss of identity. The shortest fibre recorded is Grand Canon chrysotile and is of the order of 0.00075 mm. in length, whilst the longest is exhibited in the Maritzburg Museum and is 43 in. in length. This latter fibre came from Umsinga, Natal, and is also probably chrysotile. The best length of fibre for spinning is 14 in. (3.75 cm.).

The asbestiform minerals form a group of silicates which differ widely in chemical composition, but resemble each other in being fibrous and in their resistance to heat, acids and sea water. Legally 'asbestos means any fibrous silicate mineral, and any admixture containing any such mineral, whether crude, crushed or opened' (Asbestos Industry Regulations, 1931). Mineralogists classify them as follows:—

1. Serpentine Group, $2H_2O, 3MgO, 2SiO_2$. Characterized by a high percentage of magnesia and water, generally white in colour, naturally lubricant and therefore suitable for stuffing boxes and glands. Examples, chrysotile, picrotite.

2. Rhombic Amphiboles, $(MgFe)O, SiO_2$. Soda confers a brown colour when present. Like the third group the members of this division contain high percentages of ferric oxide and have a low water content. The texture is harsh and elastic, enclosing a maximum amount of air in a minimum of incombustible matter. They are particularly efficient as heat insulators. Examples, anthophyllite, amosite.

3. Monoclinic Amphiboles. Tremolite, $CaO, 3MgO, 4SiO_2$. Actinolite, $CaO, 3(MgFe)O, 4SiO_2$. Crocidolite, $NaFe, (SiO_3)_2, FeSiO_3$.

The presence of soda in this group confers a lavender blue colour (Hall, 1918). The ferric oxide content of crocidolite encourages slag formation when it is employed as a wrapping for electrodes. The countries of origin are chiefly Canada (white chrysotile), South Africa (blue crocidolite and the brown amosite), Australia and the U.S.S.R. It is found in the banded ironstones, the deposits being classed as cross fibre, slip fibre and mass fibre according to the direction of cleavage relative to the orientation of the vein.



FIG. 1.—Asbestosis bodies in a section of fibrotic lung. From the case shown in Fig. 4. Photomicrograph x 400.

Chemical analysis explains some of the varying properties of the three groups.

	Canadian Chrysotile	Blue Crocidolite	Amosite
	Per cent.	Per cent.	Per cent.
Silica ..	41	50	47
Alumina ..	3	1	6
Ferric Oxide ..	2	35	37
Magnesia ..	40	2	6
Alkalis ..	—	8	1
Water ..	14	4	3
	100	100	100

Manufacture

The mineral is obtained by open mining, akin to quarrying. Outside the mine adit the crude asbestos is reduced in size and pieces of iron are detached manually ('cobbing') or by mechanical means. It is then transported to the factory in paper-lined sacks where it is crushed in a large edge runner or a small pan mill, foreign matter removed and the fibres opened in a Crighton machine. The ancient process of carding follows,

by which the fibres are made to lie parallel in a continuous 'sliver.' Essentially this machine consists of toothed drums and condenser rollers. The derivation of the word 'card' from the Latin *carduus*, a teasel or thistle, doubtless indicates the means by which this operation was carried out on other fibres in primitive times. Slivers are twisted together to form strong yarn on spinning frames, by ring or flyer, as in other textiles. The material is then plaited into rope or it may be woven into cloth on the conventional power loom. To the medical observer, perhaps, the loom as a special significance for the weaver's beam has given its name to living tissues. Occupying a horizontal position in modern looms, its historical position was vertical. The Greeks called it *tarax*, as they did a ship's mast or indeed anything which was set upright. The word came to be applied to the cloth itself and eventually to the histological structure of living organisms.

History of Asbestosis

In 1906 the Departmental Committee on Compensation for Industrial Diseases (1907) heard evidence from various authorities arising out of



FIG. 2.—L.E.S., female, aet. 35. Spinning dept., five years. P.M. section of lung showing blue-black polygonal areas of asbestosis and thickened pleura. At the lower pole a squamous carcinoma surrounds an abscess cavity.

the inclusion of certain industrial diseases in the Third Schedule of the Workmen's Compensation Act, 1906. At that enquiry Montague Murray reported the case of an asbestos worker who had died in the Charing Cross Hospital in 1900 of 'typical fibroid phthisis.' This man, a card room worker aged 34, had stated that nine of his workmates had died round about the age of 30. Whilst agreeing that fibroid phthisis was a clinical entity and specific to certain occupations, the Committee felt they could not recommend its inclusion in the list of diseases under the Third Schedule owing to difficulties chiefly of certification. Cases of asbestosis continued to be reported, notably by Fahr (1914) and by Cook (1924). It was Seiler's case (1928), however, which seemed to establish an unequivocal relationship between asbestos and pulmonary fibrosis and which precipitated

the inquiry leading to the Report of Merewether and Price (1930). In the following year appeared the Silicosis and Asbestosis (Medical Arrangements) Scheme (1931), the Asbestos Industry (Asbestosis) Scheme (1931) and the Asbestos Industry Regulations (1931). The first dealt with the constitution and functions of a Medical Board, the second with the award of compensation and the third with the control of dust. Legislation has been generally effective in controlling the disease.

Since 1931 all entrants to dusty department have been medically examined initially and again, annually by panels of experts. Use has been made of radiology and much pathological material examined. Dust exhaustion has received special attention; in one factory spinning frames are being totally enclosed in perspex with an ingenious

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device which permits easy access to bobbins. Handling has been reduced to a minimum and good housekeeping kept on a high standard. With very few exceptions, cases certified by the Board as asbestosis have industrial histories extending to periods prior to 1931.

Pathology

Whereas the heavier and smaller particles of silica find their way to the alveoli and thence to the lymphatic channels, the lighter and larger particles of asbestos block up the finer bronchioles. Chains of silicate tetrahedra with intervening cations determine the fibrous structure of asbestos and these chains in frayed bundles interlock and are arrested. Hence silicosis is a fibrosis of the lymph nodes and asbestosis a fibrous cuffing of the bronchioles. Dangerous fibres measure about 20 microns and a noxious concentration is said to occur when the dust count exceeds five million particles per cubic foot of air (Page, 1937).

There is no local necrosis or leucocytic reaction to the trapped fibre such as would occur if a protoplasmic poison like colloidal silica hydroxide were liberated. It seems significant that if the fibrous character of chrysotile asbestos is absent, as in its chemical equivalent serpentine, the dust is inert. Instead, the lining epithelial cells are desquamated as a result of friction and make attempts to engulf fibres which proving too long, are sometimes observed projecting from these 'dust' cells. Asbestosis giant cells are also seen, due to small aggregations of dust cells. They are pigmented, stippled and multinuclear. Fibroblasts appear around bronchioles and alveoli, in interlobular septa and in the subpleural connective tissue. Asbestosis bodies and pigment are also present. Finally there is an invasion by collagenous fibres to form the characteristic reticular pattern around the distal arborizations of the bronchial tree. The alveoli, capillaries and venules are crushed by the fibrosis.

Asbestosis bodies can be demonstrated in the lungs, pleura and sputum. The length of time taken for them to develop is unknown. They cannot be found in the mineral itself. It has been shown by staining reactions with sodium nitro-russide and ammonium sulphide and by dissolving the outer portion with concentrated sulphuric acid that they consist of a central fibre of asbestos (Fig. 1) with an iron-containing, segmented covering which is thought to have been deposited by a colloidal reaction between the fibre and the blood proteins (Gloyne, 1929). Their presence is held to be evidence of exposure to asbestos and not of the disease itself.

Dense, sessile adhesions of the pleura are common and the interlobar fissure obliterated.

The pleura has a ground glass appearance with yellow, horn-like plaques 2 or 3 in. in diameter besides patches of recent pleurisy. The cut surface of the lung exhibits the typical blue-black polygonal areas, roughly lobular in distribution, $\frac{1}{4}$ in. to $\frac{1}{2}$ in. (3 mm. to 6 mm.) in diameter on a red background of bronchopneumonia. The areas tend to confluence at the bases and exhibit the 'pleural drift' common to the pneumoconioses. Confluence is becoming rarer as the dust cloud becomes less concentrated (Fig. 2). Compensatory emphysema, on the other hand, seems to be occurring more frequently as cases live longer. It occurs chiefly along the margins of the lungs, sometimes giving rise to bullae but not so commonly as in silicosis. One case in the present series of 115 fatal cases died of a spontaneous pneumothorax consequent on rupture of such a bulla. The right heart is generally enlarged and there are the usual signs of back pressure in the liver.

Clinical Syndrome

In the writer's series the average exposure for males was 16.2 years, for females 7.3 years and for all cases 10.4 years. The duration of the disease was 6.4 years for both sexes. The mean age at death was 46.5 years in the case of men, 37.5 years in the case of women and 40.8 years for all.

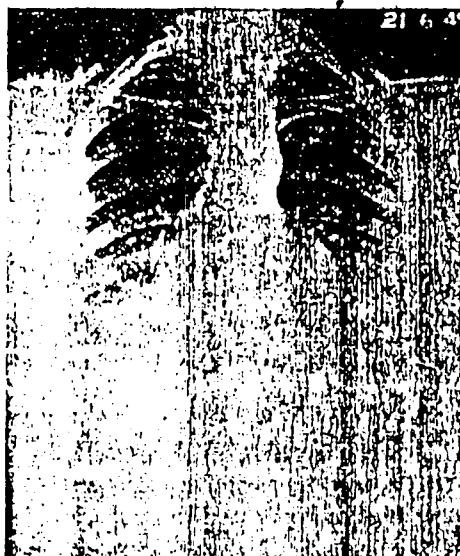


FIG. 3.—J.N., male, aet. 45. Sectional dept., 18 years. X-ray of lung showing fine mottling. Fingers show gross clubbing. Advanced asbestosis.

cases. Dyspnoea is a universal complaint: Burton Wood regarded asbestosis as a mono-symptomatic disease. Dry cough is noticeable on rising in the morning and during changes of temperature. A paroxysm usually terminates with the production of some thick mucoid sputum, very rarely flecked with blood and in the later stages containing minims of pus. An increase in the quantity of sputum indicates a complicating factor. Anorexia, loss of weight, fatigue and chest pains are occasional symptoms.

The cardiovascular signs of the disease are important, because once established they are permanent, whereas adventitious sounds in the chest tend to be evanescent. An earthy cyanosis has been described. Of 53 consecutive cases of asbestosis examined by the writer, 29 showed clubbing of the fingers, the toes being similarly affected in eight of the cases, an incidence of 54.7 per cent. In the same series the second pulmonic sound was found to be accentuated in 33 patients, that is to say 62.2 per cent. of the cases. These signs appeared separately, together and quite often not at all even in advanced disease. The liver was never palpable in an ambulatory patient nor was there oedema of the dependent parts.

The chest is flattened bilaterally. The respiratory excursion rarely exceeds 1 in. (2.5 cm.). Percussion often reveals a sensation of resistance to the finger ('feeling') which may be general or localized. Probably this is a reflection of the dense character of the fibrotic lung or of the pleural plaques which are often seen post mortem. Hyper-resonance is occasionally noted. Dry, crackling sounds can generally be heard along the emphysematous margins but often become generalized or disappear altogether in the same individual. An odd sonorous rhonchus may be heard. There is deficient air entry at the bases and expiration over the anterior aspect of the chest is harsh.

Radiological Appearances

Whereas the roentgenogram may show considerable involvement in silicosis with few, if any, symptoms, the reverse is apt to be found in asbestosis (Sayers, 1938). This is the opinion of all with experience of the disease. The typical appearance of the lung fields is a diffuse haze or pin-point mottling in the lower halves or two-thirds (Fig. 3). Emphysema may be seen in the upper zones and, of later years, a granular pattern in the lower zones somewhat resembling cotton wool which has been teased out (Fig. 4). The domes of the diaphragm are flattened and may show coarse striae running up into the lower lung fields. The vertical diameter of the chest is

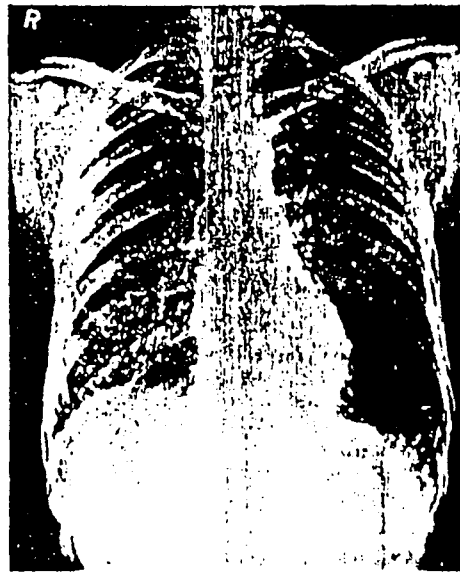


FIG. 4. T.M.R., aet. 45. Sectional-dept., 18 years. Asbestosis bodies shown in Fig. 2. X-ray of lung shows coarse fibrosis and emphysema. Gross clubbing of fingers. Eventually died with tuberculous infection.

diminished. The costo-phrenic sulcus is frequently blunted. A shaggy left cardiac border is often seen in advanced cases and sometimes the entire silhouette and diaphragmatic cupolae are blurred.

Complications

Bronchiectasis has been stated to occur frequently. Only three undoubted cases occurred in the present series and one of these was a congenital condition affecting the apex.

Pregnancy was associated with the deaths of two women suffering from asbestosis. The disease does not seem to be adversely affected by pregnancy, but the strain of labour may tip the balance in favour of right heart failure.

Bronchopneumonia is almost invariably found post mortem. The next commonest complication is tuberculosis with a much lower incidence than in silicosis and affecting a lower age group. In 115 fatal cases, 15 men and 21 women, 31.3 per cent. in all, showed also pulmonary tuberculosis. The mean age at death was 34.9 years after an average exposure to asbestos of 7.6 years. Taking the remaining cases alone, the age at death was 43.6 years, after an exposure of 11.5 years.

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An excess mortality from cancer of the lung in cases of asbestosis was recorded by Merewether (1947). Of 6,884 cases of silicosis 1.32 per cent. were found to be complicated by pulmonary cancer whereas the incidence was 13.2 per cent. in 235 cases of asbestosis. Wedler (1943) collected 92 post-mortem reports of asbestosis cases from different parts of the world and found an incidence of 16 per cent. pulmonary cancers. The development of the lesion corresponded with the lengths and intensity of the exposure (12 to 42 years). Boemke (1947) called attention to the association of asbestosis with pulmonary cancer, the latter almost always taking the form of carcinoma of the pavement epithelium. He thought it possible that, in addition to chemical processes, the purely mechanical effects of the asbestos needles may also be responsible for the development of carcinoma.

In the present series of 115 deaths from asbestosis 11 males and six females had pulmonary cancer whilst cancer of other organs was present in three males and four females (pancreas, colon, stomach, ovary). The proportion of pulmonary cancers in the series was therefore 14.8 per cent. Squamous cancers were present in nine cases, oat cell in five, columnar cell in one, endothelioma of the pleura in one and the histology unknown in one. Two of the cases were brothers. The mean age at death was 52 years, the mean exposure to asbestos dust 17.3 years and they died a mean period of 6.6 years after leaving the industry (Fig. 2).

Socio-Medical Aspects of Asbestosis

It is difficult to assess the effects of such factors as personal habits and the preference of families and those of defective physique for certain occupations. Meiklejohn (1929) has pointed out the possible effects in the past on the spread of tuberculosis among Sheffield knife grinders by the habit of spitting into the water trough through which the abrasive wheel passed. Stewart (1929) has indicated that the high incidence of tuberculosis among boot operatives is due to the attraction of disabled persons and their families to a light occupation. Similarly the family histories of those dying of asbestosis and tubercle frequently disclose the presence of tuberculosis in those of the same household who have never been exposed to asbestos.

Post-employment survival showed a sex differentiation; the mean period for men in this series was 2.3 years and for women 9.8 years. No doubt many women ceased work to marry and symptoms of asbestosis did not appear until some time later.

Of the 115 deaths only two had commenced

work subsequent to the 1931 Regulations, a man after ten years' exposure and a woman who also had tuberculosis after two years' exposure. Three other men have also developed the disease after exposures of five, four and four years respectively, the last one having been certified in 1946.

The writer has 28 cases under observation at present. The men are occupied as follows: business executive, storekeeper, painter and window cleaner, logger (in other employ), cleaning brasses, ventilation engineer, in charge of sports and social sections, laboratory handyman, retired, small printing press operator, foreman in charge of stores, foreman in charge of rubber department, light carpenter, tally clerk (week-end relief), handyman in canteen, at home recovering from tuberculosis and one relief gateman. The women are occupied as follows: light housework, in charge of works solarium, canteen work, repairing overalls in works laundry, three doing light housework and two doing sewing duties and mending duties at home.

The reduction in the intensity of the dust cloud seems to be producing a more chronic type of disease. Merewether's investigation of 1929 indicated a period of seven years' exposure for the disease to develop, whereas this series, which commenced in 1931, indicates that an exposure of ten years is necessary.

Through the kindness of Dr. Roodhouse Gloyne, the writer had access to the late Dr. Burton Wood's notes on 89 cases of asbestosis which he had collected up to 1931. Of these cases 17 showed clubbing of the fingers, i.e. 19.1 per cent., a much lower figure than the incidence of 54.7 per cent. in this series. Radiologically, the fine mottling is giving place to a coarser shadowing which seems to be due to compensatory emphysema. At post mortem, confluence of the characteristic lesion is becoming rarer and cancer incidence is rising.

Other Silicates

Of the other silicates, talc is the only one concerning which there is definite evidence of a harmful effect on the lungs, although there is some clinical and radiological evidence of pulmonary fibrosis due to mica. McLaughlin *et al.* (1949) reported the first case of talc pneumoconiosis, confirmed at post mortem, to have occurred in this country. The radiographs showed nodular shadows which, on the left side were fluffy. Histologically the lung showed greyish nodules of loosely woven fibrous tissue in association with bronchioles. There were also 'curious bodies' closely resembling asbestosis bodies. Mineralogical investigation proved that the condition was not, in fact, asbestosis but due to talc, a hydrated

magnesium silicate $Mg_3Si_2O_5(OH)_2$. Talc may in certain instances arise as an alteration product of tremolite and it may replace anthophyllite, in both cases inheriting an imperfectly fibrous form. Norwegian and Canadian talcs are platy but contain a proportion of fibres. The respiratory

passages appeared to have acted as a filter allowing the fibres to get into the lungs whilst rejecting the plates. The authors concluded that talc pneumoconiosis may be ascribed primarily to the physical form (acicular crystals) of the mineral, but that asbestos is more actively fibrogenic.

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We appeal to all members of the medical profession who do not already do so, to subscribe to the above Royal Medical Foundation. In every profession some must fall by the wayside; others must inevitably fall upon evil days.

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