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THE ASBESTOS CONNECTION

J. Kumar

Asbestos is being increasingly linked with a number of serious diseases. Studies show some workers in the asbestos industry in India are suffering from asbestosis

It's unique characteristics like strength, wear-resistance and flexibility have found asbestos useful in over three thousand commercial and industrial applications, but it has one serious drawback—it can cause severe health hazards. Inhalation of asbestos and its dispersion in the lungs can lead to asbestosis (a lung disease), lung cancer and cancers of the membranes lining the lungs and the abdomen (mesothelioma of the pleura and the peritoneum).

The Factories Act, 1948, stipulates stringent preventive control measures against asbestos exposure, protective equipment for workers and their periodical examination. Because of the serious nature of asbestosis, it has been made a notifiable disease under section 89 of the Factories Act, and also a compensable disease under the Workmen's Compensation Act. Though there are no Indian standards, international standards set limits to asbestos exposure to workers, ranging from two fibres per cubic centimetre (2 million fibres per cubic metre) to 0.2 fibre per cc for different varieties of asbestos fibres. But the Asbestos Working Group in the US reported in 1980 that there is no safe exposure limit for asbestos, that all commercial and several non-commercial forms of asbestos cause disease and recommended a new standard of 0.1 fibre per cc as a maximum workplace exposure limit. This is the smallest quantity that can be measured by techniques currently available.

Despite this awareness and the statutory provisions, workers in India are still exposed to much higher levels of asbestos, and some suffer from asbestosis as revealed by studies carried out so far. In a study done in an asbestos cement factory last year by this author and Dr. V. P. Gupta of the Central Labour Institute, Bombay, we found that 6.5 per cent of the 320 workers examined suffered from asbestosis, environmental measurements showed asbestos concentrations above the threshold levels in two sections within the factory. And in April this year, Drs S. P. Shah and S. R. Kamat, of the Department of Chest Medicine, G. S. Medical College and K. E. M. Hospital, Bombay, reported 24 cases of asbestosis at a conference on occupational health.

No case of cancer due to asbestos has, however, been notified yet. This can be due to the fact that asbestos workers are not followed-up after they retire and occupational histories of cancer patients are not recorded. In cross-sectional studies of workers, it will be difficult to find a person who is still working while suffering from lung cancer or mesothelioma of pleura.

The asbestos industry in India employs about 7,000 workers in 19 units spread in Andhra Pradesh, Gujarat, Haryana, Maharashtra and Tamil Nadu. Much of the asbestos used here is imported, and only a small quantity, about 20,000 tonnes, is mined in Andhra Pradesh, Bihar and Rajasthan. Various studies and reports in the press have recently highlighted asbestos health risks, and this has caused an increased awareness among the management and workers, resulting in improved working conditions. But only a continuous follow-up will indicate whether the hazards are sufficiently controlled or not.

Unlike in most other cases of occupational diseases, asbestosis has no cure and continues even if the afflicted worker is removed from the source of exposure; even if conditions are better in a factory today, the history of past exposure becomes a key factor, since asbestosis develops slowly and appears after 15 to 35 years. As for mesothelioma, even a short exposure can cause it, and it is difficult to prevent. All this has prompted a ban on the use of asbestos and a search for substitutes in some countries. It is also statutory in western countries to use a warning label on asbestos products.

Asbestos and its uses

A naturally occuring mineral, consisting mainly of silicates in the fibrous form, asbestos derives its name from a Greek word meaning "unquenchable". It comes in two varieties — the soft and coiled serpentine variety occurring as chrysotile fibres, and the straight amphibole variety occurring as amosite, crocidolite, anthophyllite, tremolite, actinolite, etc. Ninety per cent of the 6 million tonnes of asbestos mined in the world is chrysotile and about 7 per cent crocidolite. The physical properties of the fibres play a role in causing diseases, as we shall see later. For instance, the soft and the coiled variety causes asbestosis while the harsh and the stiff varieties can penetrate the lungs and reach the pleura to cause mesothelioma of the pleura.

The mineral fibres are processed or mixed into various products — floor tilings, gaskets and packings, building materials as asbestos cement sheets, friction products like brake and clutch linings, paints, coating and sealants, asbestos reinforced plastics for electric motor components, asbestos cement pipes for chemical process pipings, water supply pipes and electric wire conduits, asbestos textiles for heat and fire-protective clothing and draperies, asbestos paper for beverage filters, gas vapour ducts for corrosive compounds, electric wire insulation, etc. Nearly 80 per cent of the production in India goes to make asbestos cement.

In several of these processes, asbestos fibres and dust are released and inhaled by workers. Particularly dusty are mixing of fibres, dismantling of old lagging, spraying asbestos fibres on walls and ceilings, carding, weaving and spinning of fibres, twisting asbestos around welding rods, use of asbestos for rope, either dry or wet, to grout expansion gaps between refractory bricks in furnaces and kilns and clearing and maintenance of exhaust ventilation ducts and disposal units. The risk of getting asbestosis is the highest in the textile manufacturing and insulation processes where the fibres are finely divided and is the least in asbestos mining where airborne fibres are very few. It has been estimated that asbestos insulation workers are eight times more likely to develop lung cancer than the general population; for smokers, the risk is ninety-two times higher.

The size and type of the fibres and how they are dispersed are important in causing the hazards. Harsh and straight fibres are more likely to reach the periphery of the lungs and the pleura; crocidolite, which is a straight fibre, is more likely to cause mesothelioma than other fibres. Soft and coiled fibres, like chrysotile and anthophyllite, are probably involved more in asbestosis. Fibres longer than 200 micrometres and wider than 3

micrometres are intercepted in the nose and cleared by the cilia. The decreasing diameter of the pheripheral airways in the respiratory tract also helps block the larger fibres. Dispersion is another important factor; wide dispersion allows dust particles to penetrate deeper into the respiratory system and in this way the manufacture of asbestos textiles, where fibres are widely dispersed, is more dangerous.

That asbestosis is a serious affliction among asbestos textile workers was established beyond doubt by the 1930s (the first case was reported in the UK in 1900). In asbestosis, the inhaled fibres damage the airsacs in the lungs. This impairs the process of gas exchange between the oxygen in the inhaled air and the carbon dioxide in the blood, and turns the elastic tissue between the airsacs into rigid collagenous tissue which is termed pulmonary interstitial fibrosis (Fig. 1). Consequently, the lung function is restricted; the person feels increasingly breathless on exertion and finally disabled to varying degrees. And once the disease sets in, it progresses even after the worker is prevented from further exposure to asbestos, and there is no specific treatment. Asbestosis, however, occurs only in occupational asbestos workers, and depends mainly on cumulative exposure to asbestos dust. Smoking increases the risk.

How is asbestosis diagnosed? This is based on the history of exposure to asbestos and symptoms of breathlessness on exertion, end inspiratory crackles (crackling sound heard at the end of the inspiratory phase of the breath sound because of the forced opening of the smaller airways which are closed during inspiration due to interstitial fibrosis), reduction of lung function such as transfer factor and vital capacity, finger-clubbing or the thickening of the nail beds (which can occur in a variety of respiratory, cardiovascular and other diseases, or may even be hereditary and not specific to asbestosis) and radiological findings of parenchymal fibrosis. According to the Pneumoconioses Medical Board, UK, a worker who has been exposed to asbestos and shows any two of the above symptoms and disability is certified to suffer from asbestosis and one with the symptoms but without disability as "possible asbestosis".

Using this criteria, we conducted a study, selecting 320 workers at random out of the 960 employees in an asbestos factory in the middle of 1981.

Fig. 2. Asbestos bodies appear as elongated, golden brown structures, 20 to 100 micrometres long and 3 to 5 micrometres wide, with dumb-bell-shaped ends

The workers were subjected to thorough medical examination, including lung function tests and chest X-ray full-size. The results revealed that 20 workers suffered from asbestosis, and showed "restrictive-type" reduced lung function. The workers were exposed to both crocidolite (blue fibre) and chrysotile fibres, and fibre concentrations were higher than the permissible levels in the fibre grinding and loading, and pipe-finishing departments. But the 20 workers suffering from asbestosis were scattered in all departments. This was probably due to high exposure in the past (for which there was no record) in all departments and interdepartmental mixing of workers. Dust control measures in the factory had been introduced only two years before the study while the afflicted workers had been exposed for an average of 12 years. And for the same length of exposure, asbestosis incidence was higher in smokers than in non-smokers. Measures to control exposure in the two departments and effective medical monitoring of all employees were suggested to the management.

There is a point to remember here. A person who inhales asbestos fibres may show asbestos bodies in the sputum, and the presence of these bodies in the sputum is generally mistaken to indicate asbestosis. This is not so. Asbestos fibres, on reaching the airsac, causes hemolysis because of

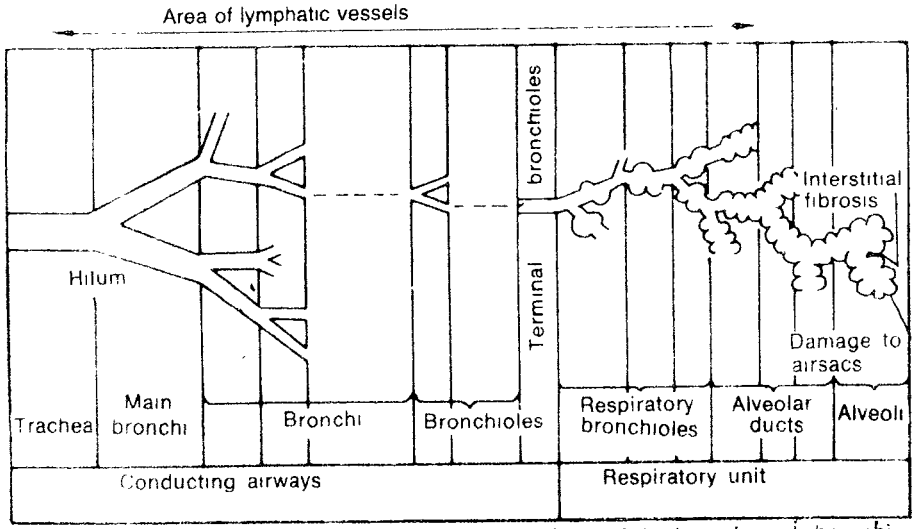


Fig. 1. Conducting airways and the respiratory unit. Sizes of the bronchi and bronchioles have been shown shorter and the respiratory unit much larger

irritation. This liberates hemosiderin (iron-containing component in the hemoglobin) which is taken up by the alveolar macrophage along with the asbestos fibre. The macrophage condenses on the asbestos fibre to give it a coating of hemosiderin and protein of its cytoplasm, and the macrophage cell wall ruptures, giving rise to asbestos bodies. This is basically a defence mechanism. Under light microscopy, using a wet smear from a centrifuged sputum specimen, asbestos bodies appear golden yellow, rectilinear and dumb-bell shaped at the ends (Fig. 2).

In general, asbestosis appears 15 to 35 years after first exposure to asbestos, and the occurrence is time-and dose-related: this means that the disease showing up today is *not necessarily the result of present-day work conditions*. The risk of contracting asbestosis is minimal below a certain exposure, and smokers carry a higher risk than non-smokers. Asbestos workers are at no excess risk of contracting tuberculosis, but asbestosis patients are at high risk of developing lung cancer.

Cancer-causing capacity

Much concern is now being caused by the cancer-inducing capacity of asbestos. Lung cancer as an asbestos health hazard was first reported by Merewether in the UK in 1947 based on a study of 235 death certificates recording asbestosis. Later, other studies confirmed this. Asbestos lung cancer does not differ from other lung cancers, except that it is more common in the lower lobes. And though, unlike asbestosis, it is not dose-related, significantly greater risk has been observed in the highest exposure group. Again, smokers run a higher risk.

Mesothelioma of pleura and the peritoneum is a rare form of cancer occuring in the general population, and is solely attributed to asbestos exposure. Even an exposure as short as a month can cause this cancer after 20 to 40 years. And unlike in other cases, smoking plays no role. This type of cancer has been reported among relatives of asbestos workers, who had been exposed to asbestos from work clothes carried home, among populations near asbestos factories and mines and among people who had non-occupational asbestos exposure. This particularly stresses the need for regulating asbestos waste disposal, maintenance and repair of asbestos products and provision of work clothes and lockers in factories so that the workers do not go home in work clothes.

keeping the exposure to asbestos within the prescribed threshold limit, mesothelioma of pleura and peritoneum is difficult to prevent. Measures are, therefore, being adopted in other countries to find harmless substitutes for asbestos. Besides, these countries have also made it statutory to use warning labels on all products containing asbestos.

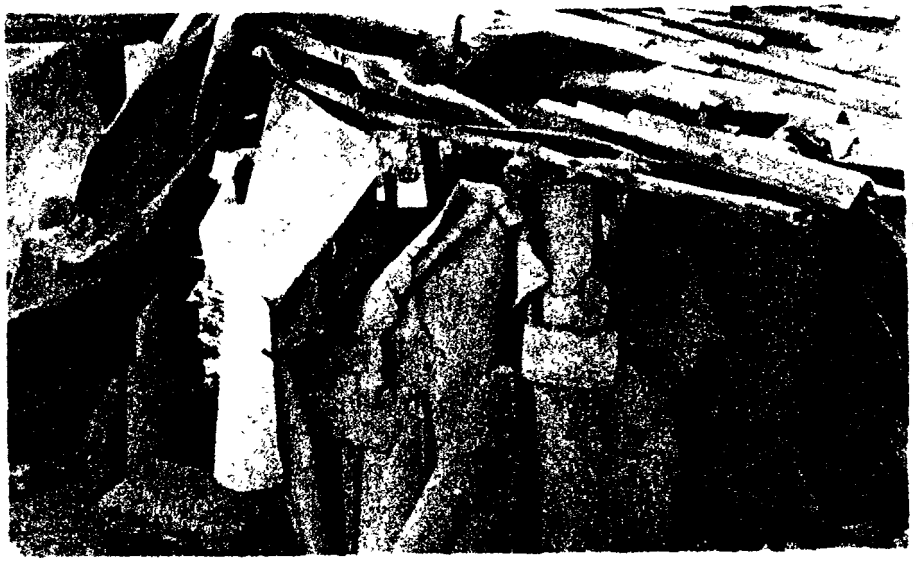
How exactly asbestos causes these disease is not still clearly known. One of the theories put forth earlier incriminated the fibrous nature of asbestos because fibres smaller than 5 micrometres in length or finely powdered asbestos caused negligible effect. But considering there are other fibres, including man-made fibres, which do not pose a similar threat, asbestos hazards were attributed to its fibrous nature and the chemical properties — fibre dissolution in biological fluids and liberation of toxic elements like magnesium, nickel and chromium.

Threshold limit value

As fresh evidence accumulated about the harmful effects, particularly of mesothelioma, the asbestos exposure limit has been continuously revised. Cumulative asbestos exposure is expressed as fibre/years per cc and is obtained by multiplying the average concentration of fibres during work hours and the length in years of a worker's exposure. It was earlier found that less than one per cent of the workers developed asbestosis at an exposure of 100 fibre/years per cc. Based on this, the National Institute of Occupational Safety and Health (NIOSH), USA, had suggested a threshold limit value for exposure of two fibres per cc and a peak concentration (sampling time of 15 minutes, mainly to prevent mesothelioma). In 1976, this was recommended to be reduced to 0.1 fibre per cc with a peak concentration not exceeding 0.5 fibre per cc. In 1980, following the Asbestos Working Group's (established by the NIOSH and the Occupational Safety and Health Association, OSHA, USA) finding that there was indeed no safe exposure limit, OSHA promulgated a maximum workplace exposure limit of 0.1 fibre per cc. Debates are also going on among the European countries about much reduced and stringent standards. Meanwhile, occupational health and environmental agencies have proposed a ban on all non-essential uses of asbestos.

To prevent asbestos health hazards, the simple and most effective way is to eliminate asbestos by using other harmless fibres. A good deal of research is going on elsewhere to find these substitutes. And one such is polypropylene wire mesh which can be used in place of asbestos for reinforcing cement. Because of its wide variety of uses, asbestos cannot, however, be easily eliminated. Till such time as asbestos is in use, all measures must be taken to prevent harm to workers, their families and those who use asbestos products.

Within the factory, therefore workers must be first educated about the health hazards and correct work practices. The factory must maintain good house-keeping, general ventilation and local exhaust ventilation. fibres should be mixed in a wet condition and floors swept and scrubbed with wet mops to keep airborne fibre concentrations within prescribed limits, workplaces should be moni-



Cancer-causing Chemicals by N. Irving Sax

A labourer's hut made almost entirely from asbestos scraps

Monitoring airborne asbestos fibres

THE casual connection between the inhalation of asbestos fibres and the production of diseases such as lung fibrosis or asbestosis, bronchial carcinoma and mesothelioma is now fully established. Though the increased mortality from the incidence of lung cancer has been linked with occupational exposure to asbestos, the association between general airborne exposures and increased lung cancer mortality has not yet been established. This is primarily because of the low level exposure to asbestos, especially when other known and suspected carcinogens are present in the atmosphere. The problem is compounded by the lack of a reliable method for the quantitative analysis of airborne asbestos fibres when they are present in much lower concentrations than other particles. The method now recommended is applicable for occupational settings and depends on visual discrimination of fibres in the presence of a background of non-fibrous particles.

The membrane filter technique in which the number of asbestos fibres greater than 5 micrometres in length are counted was possibly first used by the British Occupational Hygiene Society (BOHS) in the study in an asbestos textile factory. It is now agreed that asbestos fibres are the etiological contaminant of concern; hence the currently recommended safe air concentrations and sampling procedures are defined in terms of fibre concentration and not otherwise. Particles with the following geometric dimensions are called as fibres: length greater than 5 micrometres, diameter less than 3 micrometres, and a length-to-diameter ratio greater than 3:1. Only such fibres collected on the membrane filter are counted at 400 to 450 times magnification (4 mm objective) with phase contrast illumination.

The main disadvantage of this method is that it does not differentiate asbestos from other fibres. Airborne fibres smaller than 5 micrometres in length may also be present in the environment and will be drawn on the membrane filter; however, these cannot to be counted using the currently recommended membrane filter technique with phase contrast microscopy. Electron microscopy can be used for identification and evaluation of such fibres. The use of this technique may not be practical for routine air monitoring and the results cannot be directly compared to the current permissible limits. Besides, the significance of such small fibres is not yet definitely known. There is a general consensus among experts that mechanical and physical effects of respirable fibres greater than 5 micrometres in length are responsible for the fibrosis appearing in asbestotic lung disease. However, there is no such agreement with regard to the carcinogenic nature of asbestos fibre. Nevertheless, the working group of the International Agency for Research on Cancer in 1977 concluded that fibres greater than 5 micrometres in length are more active in producing tumours. Thus the diameter of the fibre, its length and morphology are the important parameters in the etiology of lung diseases associated with exposure to asbestos fibres.

The setting of various standards for asbestos for the protection of workers' health was based on this theory and the concept that exposure up to certain levels can be tolerated without undue risks. In 1980, both the US National Institute of Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA) concluded that: (1) there is no safe exposure limit for asbestos; (2) all commercial and several non-commercial forms of asbestos cause disease; (3) a new standard of 0.1 fibre per cc should be used as a maximum workplace exposure limit; and (4) optical phase contrast microscopy is the most reliable and economically feasible method for determining airborne levels of asbestos, with the lowest reliable detection limit of 0.1 fibre per cc.

Various techniques have been suggested for evaluation of airborne asbestos fibres while the airborne samples are collected on a membrane filter and counted with the aid of phase contrast microscopy. The current standardised membrane filter methods of fibre-counting developed by the BOHS, NIOSH, the American Conference of Governmental Industrial Hygienists and the Asbestos International Association (AIA) are more or less similar. Any method can be used. The AIA method is, however, used as a reference so that results from different countries can be compared.

Other methods which have been used for identifying and characterising the fibres are: infrared spectroscopy, polarised light microscopy and X-ray diffraction analysis. The methods cannot be used for evaluation of airborne asbestos fibres. The membrane filter method, though considered to be still the standard technique for monitoring asbestos in the occupational environment, is time-consuming and tedious, besides open to considerable experimental error and arbitrariness in the number of fibres counted. Hence there is a need for evaluating asbestos exposure by a faster and more feasible technique. One instrument which is capable of giving rapid fibrous concentrations in the air is the Fibrous Aerosol Monitor, Model FAM-1, developed by the GCA Corporation, USA, under contract to NIOSH. The instrument has been evaluated against the NIOSH-approved membrane filter sampling technique and the results have shown a strong correlation. The instrument is now available in the market.

S. CHAKRAVORTI

Mr. Chakravorti is Deputy Director (Chemical), Central Labour Institute, Bombay.

check if the control measures are adequate or not.

Workers' health should be monitored through an effective industrial health programme. This programme should include sputum examination for exfoliated malignant cells, lung function tests and full-size chest X-rays. This will help in early diagnosis of asbestosis and lung cancer and inform about the adequacy of the safety measures used. Smokers should be discouraged from working in asbestos factories, and a "quit smoking programme" should persuade smoking workers to give up smoking. Workers' condition should also be followed up after they are discharged from work on medical grounds or on retirement. It is also important to protect a worker's job even after he is paid any compensation for asbestosis or lung cancer due to asbestos. And all workers should be provided with work uniforms and lockers, and proper arrangements for washing and bath so that they do not expose their family members to asbestos.

To protect the population in the vicinity of a factory, it is essential to pack and dispose the wastes carefully. Other countries use highly trained personnel for this job. All asbestos-containing products should carry a warning label so that users, like garage workers who repair brakes or ship breakers (asbestos is used for insulation in ships), take adequate precaution and use protective devices while handling asbestos.

Dr Kumar was till recently Deputy Director (Medical), at the Central Labour Institute, Bombay. Earlier, he was a medical officer at Coal India Ltd. After graduating in medicine from Bangalore University, Dr Kumar specialised in industrial health at the All-India Institute of Public Health, Calcutta. He is now an industrial physician at the Ciba-Geigy of India, Ltd, Bombay.