

PETITIONER:
CONSUMER EDUCATION & RESEARCH CENTRE AND OTHERS

Vs.

RESPONDENT:
UNION OF INDIA & OTHERS

DATE OF JUDGMENT 27/01/1995

BENCH:
RAMASWAMY, K.
BENCH:
RAMASWAMY, K.
AHMADI A.M. (CJ)
PUNCHHI, M.M.

CITATION:
1995 AIR 922 1995 SCC (3) 42
JT 1995 (1) 636 1995 SCALE (1) 354

ACT:

HEADNOTE:

JUDGMENT:

1. Occupational accidents and diseases remain the most appalling human tragedy of modern industry and one of its most serious forms of economic waste. Occupational health hazards and diseases to the workmen employed in asbestos industries are of our concern in this writ

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petition filed under Article 32 of the Constitution by way of public interest litigation at the behest of the petitioner, an accredited Organisation. At the inception of filing the writ petition in the year 1986, though it highlighted the lacuna in diverse provisions of law applicable to the asbestos industry, due to orders of this Court passed from time to time, though wide gaps have been bridged by subordinate legislation, yet lot more need to be done. So the petitioner seeks to fill in the yearning gaps and remedial measures for the protection of the health of the workers engaged in mines and asbestos industries with adequate mechanism for and diagnosis and control of the silent killer disease " asbestosis", with amended prayers as under-

(a) Directions to all the industries and the official-respondents to maintain compulsorily and keep preserved health records of each workman for a period of 40 years from the date of beginning of the employment or 10 years after the cessation of the employment, whichever is later;

(b) To direct all the factories to adopt "THE MEMBRANE FILTER TEST";

(c) To direct all industries to compulsorily insure the employees working in their respective industries, excluding those already covered by the Employees State Insurance Act and the Workmen Compensation Act so as to

entitle the workmen to get adequate compensation for occupational hazards or diseases or death;

(d) To direct the authorities to appoint a committee of experts to determine the standard of permissible exposure limit value of 2 fibre/cc and to reduce to 1-fibre/cc for Chrysotile type of asbestos, 0.5-fibre/cc for Amosite type of asbestos and for the time being 0.2-fibre/cc for Crocidolite type of asbestos at par with the international standards;

(e) To direct the appropriate Governments to cover the workmen and to extend them Factories Act or by suitable regulatory provisions contained therein to all small scale sectors which are not covered under the Factories Act;

(f) To direct re-examination of such of those persons who are found suffering from Asbestosis by National Institute of Occupational Health but not the E.S.I. hospitals; and in particular the Inspector of factories, Gujarat, be directed to have re-examined all those workmen, examined by ESI by N.G.D.H. and to award compensation; and

(g) To direct the Central Government to appoint a committee to recommend whether dry process can be completely replaced by wet process.

2. It would appear from the record that in Karnataka, Andhra Pradesh and Rajasthan, there exists about thirty mines and the workmen employed therein are about 106 l. There are about 74 asbestos industries in nine States, namely, Haryana, Delhi, Andhra Pradesh, Karnataka, Rajasthan, Maharashtra, Kerala, Gujarat and Madhya Pradesh. It would also appear that as on August 1986 there are about 11,000 workmen employed in those industries. Basing on Biswas Committee report, the petitioner filed the writ petition. The Central Govt. accepting the said report, framed modal Rule 123A of Factories Act and on its model relevant laws and Rules were amended and are now brought into force. We are not referring to the findings and recommendations of Biswas Committee as the "Asbestos Convention, 1986" covered the whole ground.

3. In Convention 162 of the Interna-
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tional Labour Conference (ILC) held in June, 1986, it had adopted on 24th June, 1986 the Convention called "the Asbestos Convention, 1986". India is one of the signatories to the Convention and it played a commendable role suggesting suitable amendments in the preparatory conferences. It has come into force from June 16, 1989, after its ratification by the Member-States. Article 2(a) defines "asbestos" to mean the fibrous form of mineral silicates belonging to rock-forming minerals of the serpentine group, i.e. chrysotile (white asbestos), and of the amphibole group, i.e. actinolite, amosite (brown asbestos, cummingtonite-grunerite), anthophyllite, crocidolite (blue asbestos), tremolite, or any mixture containing one or more of these." "Asbestos dust" is defined as "airborne particles of asbestos or settled particles of asbestos" which may become airborne in the working environment "Respirable asbestos fibre" is defined as a particle of asbestos with a diameter of less than sum and of which the length is at least three times the diameter; "Workers" cover all employed persons; "Workplace"

covers all places where workers need to be or need to go by reason of their work and which are under the direct or indirect control of the employer;

4. Article 5(2) provides that "National laws or regulations shall provide for the necessary measures, including appropriate penalties, to ensure effective enforcement of and compliance with the provisions of the Convention.". Article 8 provides that "employers and workers or their representatives shall co-operate as closely as possible at all levels in the undertaking in the application of the measures prescribed pursuant to this Convention". Article 9 in Part III prescribes Protective and Preventive Measures, regulating that the national laws or regulations shall provide that exposure to asbestos shall be prevented or controlled by one or more of the following measures (a) making work in which exposure to asbestos may occur subject to regulations prescribing adequate engineering controls and work practices, including workplace hygiene; (b) prescribing special rules and procedures including authorisation, for the use of asbestos or of certain types of asbestos or products containing asbestos or for certain work processes. " Article 15 postulates that (1) "the competent authority shall prescribe limits for the exposure of workers to asbestos or other exposure criteria for the evaluation of the working environment (2) the exposure limits or other exposure criteria shall be fixed and periodically reviewed and updated in the light of technological progress and advances in technological and scientific knowledge, (emphasis supplied), (3) in all workplaces where workers are exposed to asbestos, the employer shall take all appropriate measures to prevent or control the release of asbestos dust into the air, to ensure that the exposure limits or other exposure criteria are complied with and also to reduce exposure to as low a level as is reasonably practicable." Clause (4) provides that on its failure to carry out the above direction to the industry to maintain and replace, as necessary, at no cost to the workers, adequate respiratory protective equipment and special protective clothing as appropriate. Respiratory protective equipment should comply with standards set by the competent authority and be used only as a supplementary, temporary, emergency or exceptional measure and not as an alternative to technical control.

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5. Article 16 mandates, that 'each employer shall be made responsible for the establishment and implementation of practical measures for the prevention and control of the exposure of the workers he employs to asbestos and for their protection against the hazards due to asbestos " (emphasis supplied). Article 17 provides demolition of plants or structures containing friable asbestos insulation etc., the details whereof are not necessary. Article 18 obligates the employer to provide clothing to the workers, maintenance, handling and cleaning thereof etc. etc. Article 19 deals with the disposal of the waste containing asbestos. Part IV consisting of Articles 20 and 21, deals with surveillance of the working environment and workers' health. Article 20 (1) provides that "where it is necessary for the protection of the health of workers, the employer shall measure the concentrations of airborne asbestos dust in workplaces, and shall monitor the exposure of workers to asbestos at intervals and using methods specified by the competent authority." Sub-Article (2) of Article 20 envisages maintenance of the records:- "the records of the monitoring of the working environment and of the exposure of workers to asbestos shall be kept for a period prescribed by the

competent authority " (emphasis supplied). Clause (3) "the workers concerned, their representatives and the inspection services shall have access to these records." Clause (4) "the workers or their representatives shall have the right to request the monitoring of the working environment and to appeal to the competent authority concerning the results of the monitoring. ". Article 21(1) envisages That "workers who are or have been exposed to asbestos shall be provided, in accordance with national law and practice, with such medical examinations as are necessary to supervise their health in relation to the occupational hazard, and to diagnose occupational diseases caused by exposure to asbestos ". Clause (2) adumbrates that such monitoring shall be free of the charge of the workers and shall take place as far as possible during the working hours. Clause (3) accords to the workers of the right to information, in that behalf, of the results of their medical examination (emphasis supplied) "shall be informed in an adequate and appropriate manner of the results of their medical examinations and receive individual advice concerning their health in relation to their work. Clause (4) is not material for the purpose of this case, hence omitted. Clause (5) postulates that the competent authority shall develop a system of notification of occupational diseases caused by asbestos.

6. Article 22, in Part V, relating to information and education is not relevant for the purpose of this case, hence omitted. In Part VI-Final Provisions, Article 24 is relevant for the purpose of this case and Clause (1) thereof states that "this Convention shall be binding only upon those Members of the International Labour Organisation whose ratifications have been registered with the Director-General". The other Articles 23, 25 to 30 are not relevant.

7. International Labour Office, Geneva, has provided the Rules regarding " safety in the use of asbestos". In Rule 1. 1.2 (Possible health consequences of exposure to asbestos dust), it is stated that there are three main health consequences associated with exposure to airborne asbestos (a) asbestosis: fibrosis (thickening and scarring) of the lung tissue; (b) lung

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cancer: cancer of the bronchial tubes; (c) mesothelioma: cancer of the pleura or peritoneum. In asbestos workers, other consequences of asbestos exposure can be the development of diffuse pleural thickening and circumscribed pleural plaques which may become calcified. These are regarded as no more than evidence of exposure to asbestos dust. Other types of cancer (e.g. of the gastrointestinal tract) have been attributed to asbestos exposure though the evidence at present is inconclusive. In Rule 1.3, definitions of asbestos, asbestos dust, respirable asbestos fibre have been defined thus :-

(a) cubestas is defined as the fibrous form of mineral silicates belonging to the serpentine and amphibole groups of rockforming minerals, including: actinolite, arnosite (brown asbestos, cummingtonite, grunerite), anthophyllite, chrysotile (white asbestos), crocidolite (blue asbestos), tremolite, or any mixture containing one or more of these;

(b) asbestos dust is defined as airborne particles of asbestos or settled particles of asbestos which may become airborne in the working environment;

(c) respirable asbestos fibre is defined as

a particle of asbestos with a diameter of less than. 3 um and of which the length is at least three times the diameter;

8. In Chapter 3, Exposure limits have been defined thus :-

3.1.1. - The concentrations of airborne asbestos in the working environment should not exceed the exposure limits approved by the competent authority after consultation with recognised scientific bodies and with the most representative organisations of the employers and workers concerned.

3.1.2. - The aim of such exposure limits should be to eliminate or to reduce, as far as practicable, hazards to the health of workers exposed to airborne asbestos fibres.

3.1.3. - The exposure level of airborne asbestos in the working environment should be established by: (a) by legislation; or (b) by collective agreement or by any other agreements drawn up between employers and workers; or (c) by any other channel approved by the competent authority after consultation with the most representative employers' and workers' organisations.

3.1.4 - it provides periodical review in the light of technological progress and advances in technical and medical knowledge concerning the health hazards associated with exposure to asbestos dust and particularly in the light of results of workplace monitoring.

9. In Chapter 4, under Monitoring in the workplace, Rule 4.4.4 is relevant for the purpose of this case which adumbrates that the measures of airborne asbestos fibres concentrations in fibres per millilitre in the workplace air should be made by the membrane filter method using phase contrast light microscopy as described in Appendix B of the Rules. All respirable fibres over 5 um in length should be counted by this method. Rule 4.4.5 provides that the measurement of airborne dust concentrations (mg/m³) in the workplace air should be made by gravimetric method as described in Appendix C to the Rules. The mass of the collected total dust should be determined and, by analysis, the of asbestos and its mass percentage.

10. Rule 4.5 Monitoring Strategy and Rule 4.6-Record keeping, have been adumbrated as under:-

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4.6.1. Record should be kept by the employer on aspects of asbestos dust exposure. Such records should be clearly marked by date, work area and plant location etc. etc.

11. In General preventive methods, in Chapter V. Rule 5.2. 1. - All appropriate and practicable measures of engineering, work practice and administrative control should be taken to eliminate or to reduce the exposure of workers to asbestos dust in the working environment to the lowest possible level. Rule 5.2.2. provides that " engineering controls should include mechanical handling, ventilation and redesign of the process to eliminate, contain or collect asbestos dust emissions by such means as (a) process separation, automation or enclosure; (b) bonding asbestos fibres with other materials to prevent dust generation; (c) general ventilation of the working areas with clean air, etc. etc.

12. Chapter VI deals with personal protection of the respiratory equipment etc., the details whereof are not necessary. Chapter VII deals with the cleaning of the

premises of the plant. Detailed instructions as to the manner in which work premises are maintained in a clean state, free of asbestos waste, have been provided and it is not necessary to enumerate all the details. Suffice it to say that every industry shall scrupulously adhere to the instructions contained in Chapter VII and IX. Chapter X deals with the supervision of the health of workers.

13. Part B deals with control of asbestos exposure in specific activities, mining and milling, asbestos cement, Textiles. In Chapter 15, Encapsulation or removal of friable thermal and acoustic insulation provides the procedure for repairs or removal of asbestos insulations. In Rule 15.10, dry stripping and Rule 15.10.1. provides that dry stripping is associated with very high levels of asbestos dust which should, therefore, be, used only (a) where wet methods cannot be used; (b) where live electrical apparatus might be made dangerous by contact with water; (c) where hot metal is to be stripped and the use of water may be damaging. Rule 15.10.2 provides that where dry stripping is employed, as effective a standard of separation as possible should be preserved between the work site and the adjacent areas to prevent the escape of asbestos dust. Rule 15.10.3 envisages that all workers within the separated area should be provided with, and should use, suitable respiratory equipment and protective clothing. All other guidelines are not necessary, hence omitted. In Rule 15.1

1, wet stripping provides procedure thus:-

" 15.1 1. 1. Areas in which wet stripping is being carried out should be separated from other work areas.

15.11.2. All workers within the separated area should use suitable respiratory protective equipment and protective clothing.

15.11.3 Electrical equipment in the area should be isolated from the entry of water.

15.11.4. At the end of the work a competent person should ensure that it is safe for the electrical supply to be restored.

15.11.5. Before removal is started, care should be taken to do: asbestos material is saturated with water. This may be made easier by the addition of a waterwetting agent.

15.11.6 (1) Where cladding has to be removed,

it should first, where practicable, be punctured and the asbestos containing material within the cladding should be thoroughly wetted.

(2) The cladding should then be removed carefully within the enclosure and all surfaces should be vacuumed or sprayed with water.

15.11.7. The water-saturated material should be removed in small sections and placed immediately in labelled containers which should then be sealed.

15.11.8. Any slurry produced should be contained and not discharged into drains without adequate filtration. etc. etc.

14. Rule 15.12 provides stripping by high-pressure water jets the details whereof are not material but suffice it to emphasise that specialised method should be carried out only by trained personnel and all precautions relevant to the operation should be taken. Special safety precautions, including those given in this section of Code, are required,

since they are very high-pressure spraying or dangerous, displaying at the proper place in addition to other stripping warning notices. Other guidelines are not relevant for the purpose of this case but suffice to state that every industry should adopt, adhere to and strictly follow the Rules provided for the safety in the use of asbestos.

15. In the "Encyclopaedia of Occupational Health and Safety", Vol-1, published by International Labour Office, Geneva, the latest 4th Edition, 1991, provides definition of asbestos as has been found hereinbefore and therefore, it is not necessary to its reiteration. Its Pathology has been stated at page 188 in Vol-1, which is as follows:-

"The retained fibres in the alveolar region are 3 um or less in diameter but may be up to 200 um long. Animal experiments strongly point to the longer fibres, 5 um and over, as being much more fibrogenic than shorter fibres. A proportion of the longer fibres, especially amphiboles, become coated with an iron Protein complex producing the drumstick appearance of asbestos bodies. All types of asbestos cause similar fibrosis. The fibrosis starts in the respiratory bronchioles with collections of macrophages containing fibres, and others lying free. These deposits organise, collagen replacing the initial reticulin web. Initially only a few respiratory bronchioles are affected, but the fibrosis spreads centrally to the terminal bronchioles and peripherally to the acinus. The areas increase in size and coalesce causing diffuse interstitial fibrosis with shrinkage. The process starts in the bases spreading upwards as the disease progresses; in advanced disease the whole lung structure is distorted and replaced by dense fibrosis, cysts, and some areas of emphysema.

The pleura, both visceral and parietal surfaces, are affected by the fibrosis and to a degree which is much greater than in other types of pneumoconiosis. The visceral surface may be sclerosed up to 1 cm thick. In the parietal pleura thickening starts as a basket-weave pattern of fibroblasts, the sheets of fibrosis lying along the line of the ribs especially in the lower thorax and posteriorly. The edges become rolled and crenated and, after many years, calcified.

The parietal thickening may be extensive and thick with little or no parenchymal fibrosis. The reasons for this are not fully understood but indicate the need to separate, if possible, parietal and visceral pleural thickening in life.

Diagnosis and types :

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Table 1 lists the types of fibrosis in the lung caused by asbestos that can be Partially or well separated clinically. Recent epidemiological research indicates that asbestosis and pleural plaque may have differing aetiologies, natural histories, and significance in terms of morbidity and mortality.

Table 1. Types of lung fibrosis caused by asbestos

Parenchymal

Pleural:

Visceral: Acute	Asbestosis
Chronic	
Parietal: Hyaline	
Calcified	Pleuralplaques

16. The Asbestosis has been signified at page 188 which is as follows:

Asbestosis The signs and symptoms of asbestosis are similar

to those caused by other diffuse interstitial fibroses of the lung. Increased breathlessness on exertion is usually the first symptom, sometimes associated with aching or transient sharp pains in the chest. A cough is not usually present except in the late stages when distressing paroxysms occur. Increased sputum is not present unless there is bronchitis, the result of smoking. The onset of symptoms (except following very heavy exposure) is usually slow and the subject may have forgotten having any contact with asbestos. Persistent dull chest pain and haemoptysis indicate the need to investigate further the diagnosis of bronchial or mesothelial cancer.

The most important physical sign is the presence of high-pitched fine crepitations (crackles) at full inspiration and persisting after coughing. They occur initially in the lower axillae and extend more widely later. Agreement between skilled observers on detecting this sign is good but it may vary from day to day in the early stages. It may also be present as an isolated sign in 2-3% of otherwise normal individuals. There are now means of recording this sign on tape. Other sounds wheezes and rhonchi are of no help in diagnosis, but indicate associated bronchitis. Clubbing of the fingers and toes was formerly regarded as an important physical sign. There is an impression that it is now less frequently seen. Its severity does not relate well to other aspects of the diagnosis. There is poor agreement between observers except when the clubbing is very pronounced. It is possible that its presence relates to the rapidity of progression of the disease.

The chest radiograph remains the most important single piece of evidence, even though the appearances are similar to other types of interstitial fibrosis. When the radiography is classified by three or more skilled readers using the ILO 1971 scheme independently, it is found that virtually all cases of asbestosis are picked up by one or more of the readers as Category 1/0 or above. The radiographic appearances are well illustrated in the set of standard films of the ILO 1980 Classification of the radiographic appearances of the pneumoconioses (see PNEUMOCONIOSES, INTERNATIONAL CLASSIFICATION OF). The classification provides a means of recording the continuum from normality to the most advanced stages on a 12-point scale of severity (profusion) and of extent (zones) affected. The earliest changes usually occur at the bases with the appearance of small irregular (linear) opacities superimposed on the normal branching architecture of the lung. As the disease advances the extent increases and the profusion of irregular opacities progressively obscures the normal structures. Shrinkage of the lung occurs, with elevation of the diaphragm. in advanced cases

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distortion of the lung with cysts (honeycomb lung) and bullae occur. The hilar glands are not enlarged or calcified unless exposure has been to mixtures of silicious dusts. This may occur, for example, in making asbestos roofing shingles or pressure pipes, and in mining. The small opacities may then be rounded rather than irregular.

The pattern of lung function provides the important third component in diagnosis. The functional changes are the result of a shrunken and non-homogeneous lung, without obstruction of the larger airways (restrictive syndrome). The total lung volume is reduced and especially the forced vital capacity (FVC), but the ventilatory capacity (FEV_{1.0}) is only reduced in proportion to the FVC, so the ratio FEV_{1.0}/ FVC is normal or even raised. The transfer factor for

carbon monoxide is reduced in later stages, but in the early stage an increase of ventilation on a standard exercise test may be the only alteration indicating impairment of gas exchange. Although the restrictive syndrome is the commonest pattern (about 40%) in about 10% of cases airway obstruction is the main feature and in the remainder a mixed pattern is seen. This is thought to be largely due to the confounding effects of cigarette smoking.

Visceral pleurisy: chronic and acute This occurs in two forms chronic and acute. The former is the commoner and is a usual accompaniment of parenchymal disease, but its severity does not run parallel with the parenchymal disease. The diagnosis is radiographic. In some cases one or both of the costophrenic angles are filled in but the more specific feature is the appearance of well defined shadow running parallel to the line of the lateral chest wall and separated from it by a narrow (1-2 nun) clear zone. This is due to the thickened pleura seen "edge on". It is illustrated in the ILO 1980 standard set of films. The thickening is best seen in the middle and lower third of the lateral chest wall, the apices are usually spared. It is common in those only lightly exposed to find this pleural thickening as the only radiographic feature. It is readily missed when present only over a short length of the wall and if the radiographic technique does not give a clear picture of the periphery of the lung. When the visceral pleura is greatly thickened it causes veiling of the lung field, obscuring both the normal structure and parenchymal changes. This probably the basis of the "shaggy heart" and the "ground glass" appearance described in the carly accounts of asbestosis. The wide recognition that small areas of pleural thickening may be the only sign of past exposure to asbestos is recent, and it seems to be a feature of the effects of low exposure to the dust. It is likely to remain an important observation for monitoring exposure to improved conditions in the future.

Acute pleurisy affecting the bases, and costophrenic angles, with effusions, sometimes blood-stained, is now a recognised sequel to asbestos dust exposure. It is associated with pain, fever, leucocytosis and a raised blood sedimentation rate. It settles in a few weeks but leaves the costophrenic angles obscured. No precipitating factors have been identified. Its recognition is important. Firstly, the cause may be missed unless and adequate occupational history is taken; secondly not all effusions in asbestos workers signify the onset of an asbestos-related cancer. A few weeks of observation may be necessary to confirm the aetiology.

Summary of diagnosis The diagnosis of asbestosis therefore depends upon

(a) a history of significant exposure to asbestos dust rarely starting less than 10 years before examination:

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(b) radiological features consistent with basal fibrosis (Category 1/0 and over, ILO 1980);

(c) characteristic bilateral crepitations;

(d) lung function changes consistent with at least some features of the restrictive syndrome.

Not all the criteria need to be met in all cases but (a) is essential, (b) should be given greater weight than (c) or (d); however, occasionally (c) may be sole sign, Other investigations are not of much help. Asbestos bodies in the sputum indicate past exposure to asbestos but are not diagnostic of asbestosis. Their absence when there is much sputum and marked radiological changes of fibrosis suggest an alternative cause for the fibrosis.

Immunological tests may be positive but do not help in consistent separation of asbestosis from other types of fibrosis. Lung function results must be assessed in relation to appropriate standards allowing for ethnic, sex and age differences and for cigarette smoking.

Asbestos corns on the fingers area of thickening skin surrounding implanted fibres are now much less common because much of the asbestos fibre is packed mechanically and gloves are worn. Corns do not lead to skin tumors and disappear on removal of the fibres.

17. Pleural plaques and sources of exposure to asbestos have been stated at page 189-191, thus :-

Pleural plaques Parietal pleural plaques alone rarely cause symptoms. They may occur alone or with asbestosis. The diagnosis in life is radiological and the appearance are more specific than in the case of parenchymal fibrosis. PA films will detect most cases, but because they are frequently thickest posteriorly their full extent is best seen using oblique views. The ILO 1980 standard film show their appearance and the scheme provides, for the first time, a separation of parietal (circumscribed) and visceral (diffuse) pleural thickening. The plaques lie along the line of the ribs, and when thick cast a well defined shadow over the lung field extending in from the lateral chest wall, where they may also be seen "edge on".

Separation from visceral thickening depends largely on a defined edge to the shadow. Both types may occur together. Dependent mostly on the length of time since first exposure, and age, patchy Calcification occurs in the edges. This produces a bizarre pattern of dense shadows likened to "glittering candle wax" or a "holly leaf". The onset of calcification reveals many small plaques not previously visible. When calcification occurs in a crater-shaped plaque on the dome of the diaphragm a diagnosis of past exposure to asbestos or related minerals can be made with confidence.

Sources of exposure to asbestos Formerly it was though easy to establish past exposure to asbestos by inquiry about work in manufacturing plants, or the application of the fibre for insulation. Now it is realised that only the most detailed history of all jobs, residences and occupations of the family will reveal possible exposures to asbestos. The reasons for this change are

- (a) the much wider use of asbestos in thousands of products especially since the Second World War (see ASBESTOS);
- (b) the recognition that significant exposure to asbestos occurred around mines and manufacturing plants in the past;
- (c) the discovery of family exposure to the dust brought home on clothing, and

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also that those working in an area where lagging is in progress may be affected, even though they are engaged in lagging;

- (d) the finding that calcified pleural plaques, indistinguishable from those occupationally exposed, also occur in the general population in localised areas in several countries (Finland, Czechoslovakia, Bulgaria, Turkey and others).

With the discovery of such diversity of sources of possible exposure, but virtually no quantitative information about its severity, and few long term follow up studies of those exposed, it is not surprised that there is controversy about the health hazards. However, some conclusions emerge which must be subject to revision in the future.

- (1) Asbestosis is primarily occupational in origin, the

result of mining , milling, manufacturing, applying, removing or transporting asbestos fibre. Exposure is much less when the fibre is bound in the product (asbestos cement and asbestos plastic and paper product). Also exposure in the past was much greater than it is today with the use of the best working practices.

(2)Asbestosis may have been caused by home exposure from dusty clothing at a time when there was no dust or hygiene control in the factories.

(3)Asbestosis does not result from the very limited exposure to which the general public is or has been subject, even though asbestos fibers are detectable in the lungs of a high proportion of adults in industrialized areas. The median numbers of fibres so detectable are two to three order of magnitude less than that found in those occupationally exposed.

(4) There are and have been important differences between countries in the use of asbestos, so that exposure for the same occupation varies widely. For example, dry wall fillers (sparkling) contain asbestos in the United States but not the United Kingdom; thus sanding of internal walls during construction and maintenance is a source of exposure in the former but not in the latter. On the other hand, spraying of crocidolite was much more widespread in the 1940s in the United Kingdom than elsewhere.

(5) Pleural plaques can arise at levels of exposure probably much lower than required to produce asbestosis. In addition it is probable that other minerals can cause plaques. For example, among chrysotile miners in Quebec calcified plaques are limited to those who have worked in two out of the eight mines. The minerals causing the plaques in general population have not been fully established. Tremolite, an amphibole often present in deposits of asbestos, may be important.

(6) Whether chrysotile and the amphiboles differ in fibrogenicity in man is uncertain, but some evidence indicates that the amphiboles may be more fibrogenic. In animals there is little difference but the amphiboles remain in the lung much longer than the chrysotile.

The relation of asbestosis to dose of dust. In only a few instances are there records of past dust sampling to relate to the prevalence or incidence of asbestosis. But the information has been exhaustively analysed for miners and millers in Quebec, a group of asbestos cement workers in the United States and asbestos textile workers in the United Kingdom, because of its relevance to setting hygiene standards. In North America the dust was measured in millions of particles/ft³, in the United Kingdom in fibres/cm³ the measurement now international used.

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All the data show a clear relation between estimated dose of dust (concentration x time of exposure) and the incidence or severity of disease, but are insufficiently precise to determine whether there is a threshold level below which asbestosis will not occur. A cautious conclusion from the North American studies is that at about 100 million particles/ft³/yr there might be a threshold or that the risk of developing asbestosis would be as low as 1% of men after 40 years' exposure could be as low as 1.1 fibres/cm³ or may have to be as low as 0.3 fibres/cm³". More precise information will only become available when the dust sampling introduced widely after the mid-1960s is related to the incidence of disease in the future.

The relation of asbestosis to lung cancer - The important questions here are: firstly, is there an excess risk of

bronchial cancer only in those who also have some degree of asbestosis? Secondly, if the dust exposures are low enough to eliminate asbestosis, will the excess lung cancer risk also be reduced to an acceptably low level? Neither question can be answered at present, and so disagreement is likely. It is known that there is a close association between asbestosis and lung cancer, about 50% of those dying from or with asbestosis have a lung cancer at post mortem. Among those knowledgeable about details of the dose-response data there would probably be agreement that dust exposures low enough to eliminate asbestosis will also reduce the excess bronchial cancer risk to a very low value. This does not extend to the risk to a very low value. This nearly so closely related to that of asbestosis (see ASBESTOS (MESOTHELIMO AND LUNG CANCER)).

PREVENTION -

This depends on successful control of dust exposure and medical surveillance to protect the individual, as far as is possible, and for the detection of health trends in the group.

Engineering control - Replacement of asbestos by other material believed to be safer has been widespread since the mid-1970s. Man-made mineral fibres and other insulating materials are rapidly replacing asbestos for heat insulation. But for other uses, for example, asbestos cement, friction material and some felts and gaskets, substitution is not at present practicable.

Dust control has been gradually improved by partial or complete enclosure of plants and the wide use of well designed local exhaust ventilation. In the textile section a completely new wet process of forming the thread has greatly reduced dust level, previously difficult to control. During maintenance work on old insulation much stricter control of exposures is possible by isolation of the working areas, and by training in the use of good working practices to reduce the dust, for example damping of the insulation before removal and the use of vacuum cleaning in place of sweeping. But removal of old insulation is likely to remain for many years a major potential source of high exposure (see also DUST CONTROL INDUSTRIAL).

Medical surveillance The insidious onset of asbestosis and the lack of highly specific features indicate the need for well recorded and systematic, initial, and periodic examinations of asbestos workers. This ensures the best chance of detecting the earliest signs. Physical examination of the chest, full-sized, high technical quality chest radiographs and test of FVC and FEV₁₋₀ are the minimum required. The interval will vary from annually up to four times yearly, with more frequent visits when there are clinical reasons. There is increasing evidence that the radiological

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features of asbestosis are in part cigarette-smoking dependent which requires the recording of smoking histories. This and the multiplicative effects of asbestos dust and cigarette smoking on the risk of bronchial cancer provide the strongest possible grounds for stopping cigarette smoking in those potentially exposed to asbestos. Personal advice on the special dangers of smoking and limiting opportunities for smoking at work are essential steps in prevention. Full personal protective equipment will be required where dust levels cannot be lowered to the hygiene standard. The system of periodic examinations also provides, if properly analysed, essential information about the effectiveness or failure of the engineering control of

the dust. Tabulation, by age and years of exposure, of the results of classifying the chest films on the ILO 1980 scheme preferably by independent readers gives early evidence of trends in the prevalence of asbestosis. This valuable information will be missed if the group findings are not examined in detail.

Treatment:-

There is no specific treatment for asbestosis. Where the rate of progression appears unusually rapid further special investigations, including lung biopsy, may be justified if it is likely to assist in the differential diagnosis, and influence treatment for example the use of steroids, but these are not of proved value. The severity of past exposure is the only factor known to influence progression rate. Thus, those with some evidence of asbestosis, if young or middle-aged should be removed from further exposure. In cases where exposure has not been heavy and asbestosis is only detected late in life,, progression may be very slow and the grounds for removal from work with asbestos, under good conditions, are less compelling. The widespread and often misleading publicity given to the hazards of exposure to asbestos may cause much anxiety to those with asbestosis, both for their own health and for that of their family. Reassurance, and the putting of the likely prognosis in true perspective, are an important Part of good treatment. The special risks of continuing cigarette smoking need emphasis. Mesotheliomas are a rare complication in those exposed only to chrysotile.

Compensation: -

The conventions on the awarding of compensation for asbestosis vary in different countries. Unusual breathlessness on exertion, as a cause of disability, may be required, even though it is not essential for a confident diagnosis of asbestosis. Compensation May be limited to those with evidence of parenchymal disease; pleural fibrosis parietal or visceral alone may not be accepted. Lung (bronchial) cancer is usually accepted as part of the disease provided there is at least some evidence of parenchymal fibrosis, but may be rejected if there is no radiological evidence of pleural or parenchymal fibrosis. There is plenty of opportunity for disagreement, especially when a factor for uncertainty of prognosis is included. It is now established did asbestos dust alone may cause lung cancer although the absolute risk is very small compared with that from the combined effects of cigarette smoking and asbestos dust. It has not been established that pleural plaques alone result in an increased risk of bronchial or mesothelial tumours, above that for similar exposures to asbestos dust without these pleural changes. The considerable uncertainty about the likely rate of Progression of the fibrosis makes assessment on first diagnosis especially difficult. Lung biopsy is not justifiable solely for compensation assessment.

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ASBESTOS (mesothelioma and lung cancer)

While pulmonary fibrosis due to exposure to asbestos (asbestosis) has been known for decades, the first reports of individual cases of asbestosis combined with pulmonary cancer which appeared from time to time in various countries were accepted more as a curiosity. They did not attract much attention until in 1947 a British Chief Inspector of Factories, E.R.A. Merewether, reported that lung cancer was found to be the cause of death in 13.2% of persons known to have asbestosis who had died and been autopsied between 1923 and 1946. A similar high proportion of cancer deaths in

asbestosis was found by other pathologists and the probability of a role of asbestos in pulmonary carcinogenesis was definitely established by an epidemiological study by Doll in 1955, and confirmed by further studies.

Soon afterwards a new surprising discovery was made in South Africa. An accumulation of cases of an otherwise very rare tumour of the pleura and peritoneum, the malignant mesothelioma, was reported by Wagner in 1959 and related to exposure to the locally mined type of asbestos, crocidolite. Soon afterwards cases were identified in non-mining occupational exposures to asbestos in England, in the United States and elsewhere. In contrast with asbestosis, and in contrast with asbestos-related pulmonary cancer, mesothelioma was found also in persons whose exposure was not necessarily occupational.

Bronchogenic carcinoma related to asbestos: -

Bronchogenic carcinoma of the lung. There is a disease very in the general population. While in many countries the total mortality from cancer slowly declines, the incidence and mortality from lung cancer increases and stands as the most frequent cause of death from cancer, particularly in cigarette smokers. It begins with transformation of the mucous membrane lining the inside of the bronchus at various level and such foci of transformation may remain at their initial spot for some time shedding at times atypical or metaplastic cells into the sputum without causing other symptoms. This is the period in which we sometimes may succeed in discovering these pre-cancerous, or the earliest cancerous, changes by sputum cytology sooner than by other diagnostic methods. Some of such early alterations of cells is reversible and may spontaneously heal when the cause disappears, e.g. when the person stops smoking. When the original focus develops definite cancer cells, the focus begins to grow, to bleed and slowly to obstruct the way, a growing malignant tumour becomes visible on the radiogram and unless it can be surgically removed as soon as confirmed, it tends to spread through growth and through dissemination by blood and by lymph and to lead eventually to death. Supporting treatment by chemotherapy and radiation successfully prolongs life and radical surgery can provide complete healing.

The various components of the bronchial lining may undergo malignant transformation and consequently the carcinoma may be composed of various cells and have various histological appearances such as adenocarcinoma or squamous, or oat-cell carcinoma.

There are no histological or other characteristics which would specify the individual lung cancer as cancer caused by asbestos.

In many cases of asbestos-linked pulmonary cancers the lungs also show pulmonary fibrosis-asbestosis

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microscopically, and often macroscopically, and on x-ray examination. Some scientists believe that so-called "asbestos lung cancer" can only develop on a pathologically changed terrain of asbestotic fibrosis. There is evidence of such a possibility in human pathology: the scar-carcinoma. Others believe that exposure to asbestos alone, particularly in a smoker, may provoke cancerous growth without also causing asbestosis. The decision between the two opinions is difficult to reach because in individual clinical cases of bronchogenic carcinoma we cannot distinguish what is an "asbestos cancer", a "cigarette cancer" or lung cancer from yet another cause. Thus, in most coun-

tries bronchogenic carcinoma is considered an occupational disease due to asbestos, e.g. for workmen's compensation, only in the presence of coexisting asbestosis. If pulmonary fibrosis were a prerequisite for development of asbestos-linked lung cancer, it would follow that lowering exposures to asbestos to levels which effectively prevent asbestosis would automatically eliminate "asbestos lung cancer".

Epidemiological data

In man the link of lung cancer with asbestos has been mainly epidemiological. While asbestosis cannot occur without exposure to asbestos and consequently every case of asbestosis must be linked with such exposure, with pulmonary cancer the situation is quite different. It is a rather common disease in the general population. The link with exposure to asbestos is based on finding whether in those exposed to asbestos lung cancer occurs more frequently than in those unexposed, i.e. whether in those exposed there is an excess incidence of lung cancers.

Since Doll's study a number of other epidemiological studies, of various levels of excellence, have been carried out which confirm that indeed there is an excess of bronchogenic carcinoma in persons exposed to asbestos, under certain circumstances, and thus that asbestos must be considered one of a number of carcinogenic substances.

What are the circumstances of a manifest risk of cancer in asbestos exposure? It has been established that smoking cigarettes greatly increases this risk. In fact the large majority of lung cancers attributed to asbestos exposure have occurred in smokers. A lung cancer in an asbestos-exposed non-smoker has been a rarity. Table 1 shows the effect of both exposures together while each of the two exposures also carries a risk by itself. A particular exposure to asbestos in the reported group of workers increased the basic risk of pulmonary cancer in nonsmokers. However, since the risk in nonsmokers was very small, its further increase still meant only very few cases, if any at all. On the other hand, when the basic risk of exposure to asbestos was combined with the 11.8 time higher risk of a smoker, this combination necessarily produced a serious risk leading to an excess of incidence of pulmonary cancer. This experience has an important practical implication: most "asbestos cancers of the lungs" could be prevented if the workers did not smoke. In fact it was found that the risk for the asbestos workers who had stopped smoking declined after 10 years to the low level existing for non-smokers.

The bronchogenic carcinoma has a long latent period, usually 20 years or more. Consequently, what excesses of incidence of pulmonary carcinoma linked with asbestos have been found to date must be linked with exposures 20 years or more development of the tumour. It is known that exposures in those days

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were generally very high. But we usually do not have any precise measurements. Thus in most existing epidemiological studies it has not been easy, and in some not possible, to establish a relation between the incidence of cancer and a certain quantitative level of exposure, other than that the exposure had been high.

Table 1

	Asbestos exposure		
	Little	Moderate	Heavy
Non-smokers	1.0	2.0	6.9
Moderate smokers	6.3	7.5	12.9

Heavy smokers 11.8 13.3 25.0
 From : McDonald, J.C. "Asbestos-related diseases: an epidemiological review" (587-601). Biological effects of mineral fibres. Wagner, J.C. (ed). IARC scientific publications No.30 (Lyons, International Agency for Research on Cancer, 1980) Vol.2.

 One quantitative measure commonly used is the duration of exposure in years. In other studies the period since first exposure and the duration of exposure. Only a few investigations have had the additional benefit of actually measured data on past levels of exposure. An example of the latter is the series of epidemiological studies of workers of the chrysotile mines of Quebec carried out by J.C. McDonald and his collaborators. This and some other studies showed a dose-response relationship, i.e. the higher was the dose, in terms of level of exposure, or of periods of exposure, or of both of them combined, the higher was the excess incidence of bronchogenic cancer. In fact the excess incidence of lung cancer and statistically significantly increased relative risk was usually found only in groups of persons most severely exposed (see Table 2)

Table 2. Relative risks of lung cancer in relation to accumulated dust or fibre exposure, before and after correction of work histories with controls matched for smoking

	Accumulated dust exposure (millions of particles per cubic foot x years)				
	<30	30 <300	300 <1000	>1000	All

Before correction					
Cases	89	73	56	27	245
Controls	108	87	42	8	245
Relative risk	1	1.02	1.62	4.10	-
After correction					
Cases	85	73	59	27	244
Controls	101	89	44	10	244
Relative risk	1	0.97	1.59	3.21	-
	Accumulated fibre exposure (fibres per ml x years)				
	<100 <1000	100 <3000	1000 >	3000	All

 After correction
 Cases 86 76 56 26 244
 Control 110 87 35 12 244
 Relative risk 1 1.12 2.05 2.77 -
 From: McDonald J.C.: Gibbs, G.W., Liddell, F.D.K.
 "Chrysotile fibre concentration and lung cancer mortality: a preliminary report" (811-817). Biological effects of mineral fibres. Wagner, J.C. (ed). IARC scientific publication No.30 (Lyons, International Agency for Research on Cancer, 1980), Vol.2.

 18. In Asbestos Medical and Legal Aspects by Barry 1. Castleman at p. 10 had stated that Dr. Merewether following the diagnosis by Homburter in his co-incidence of Primary Carcinoma at EC Lungs and Pulmonary Asbestos 1943 stated that fibrosis of the lungs as it occurs among asbestos

workers as the slow growth of fibrous tissue (scar tissue) between the air cells of the lungs wherever the inhaled dust comes to rest. While new fibrous tissue is being laid down like a spider's web, that deposited earlier gradually contracts. This fibrous tissue is not only useless as a substitute for the air cells, but with continued inhalation of the causative dust, by its invasion of new territory and consolidation of that already occupied it gradually, and literally strangles the essential tissues of the lungs. In *Malignant Mesothelioma in Norway* by Gunnar Mowe, 1986 Ed., he stated at p.8 on Aetiology of malignant mesothelioma that in 1943, Dr. Wedler reviewed malignancies in 30 asbestosis cases in Germany, and suggested a casual association between asbestosis and both bronchial and malignant mesothelioma. At p.9, he stated that in 1969, Wagner and Berry reported that

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all the main types of asbestos fibres were capable of producing mesotheliomas in rats after intrapleural or intraperitoneal installation. In the same page in para 2.2, he stated that the importance of asbestos fibre size in explaining the biological effects of asbestos was first emphasized by Timbrell in 1965. At p. 14 in para 3.2, caption lung fibre burden, he stated that lung fibre burden, which is defined as the total content of mineral fibres in the lungs, depends on external asbestos exposure. At pA 5 in Table 5, Biological effects of natural mineral fibres (asbestos related diseases), he stated that long latency time from first exposure until onset of disease is a typical feature of all the asbestos related diseases. At p. 16 in para 3.4, he stated that among 948 patients with malignant mesothelioma, 65% were pleural, 24% peritoneal and 11% pericardial. At p.21, lung fibre analysis under the caption material and methods, para 3, he stated that the lung tissue samples- for fibre analysis were obtained from twelve, pathology departments the analysis samples from 85 men and 13 women disclosed the malignant mesothelioma. At p.25, summary of his results in Paper V, he stated that the median latency time from the first year of exposure until death was 35 years (range-18-53), and the median time interval from last year of exposure until death was 14 years (range: upto 40 years). At p.32, he stated that the estimated proportion of-men with at least possible occupational asbestos exposure were 82%. At p.40, he stated that strict regulations and effective control of such work are vital in order to prevent asbestos related cancers in the future. At p. 41 in para 4, he stated that high amphibole concentration in lung tissue increases the risk of malignant mesothelioma considerably. Asbestos exposure corresponding to only one million fibres per g. of dried lung tissue is also associated with increased risk. In *Blannie S. Wilson v. Johns Manville Sales Corpn Ltd.*, 684 Federal 2nd III (1982), the United States Court of Appeal, District of Columbia Circuit, Ginsburg, J., as a Judge in the Court of Appeal deciding the question of limitation of 3 years from the date of diagnosis of mild asbestos held that the period of 3 years should be computed from the date of discovery and that asbestos, which is not a cancerous process, has a latent period of 10 to 25 years between initial exposure and apparent effect. Even longer periods of time may pass before mesothelioma manifests itself In *William T. Urie v. Guy A. Thompson*, 93 L. Ed. = 337 US 163, the Supreme Court of the United States of America laid that the limitation of three years prescribed by the statute of limitation starts from the time when the employee discovers the disease and

the cause of action accrues only when diagnosis of the disease is accomplished, and not when the employee unwittingly, contracts it nor is each inhalation of silica dust a separate torn giving rise to a fresh cause of action. 19. It would thus be clew that disease occurs wherever the exposure to the toxic or carcinogenic agent occurs, regardless of the country the type of industry, job title, job assignment, or location of exposure. The disease will follow the trail of the exposure, and extend the chain of carcinogenic risk beyond the workplace. It is the exposure and the nature of that exposure to asbestos that determines the risk and the diseases which subsequently result. The development of the carcinogenic risk due to asbestos or any other carcinogenic agent, does not require a continu-

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ous exposure. The cancer risk does not cease when the exposure to the carcinogenic agent ceases, but rather the individual carries the increased risk for the remaining years of life. The exposure to asbestos and the resultant long tragic chain of adverse medical, legal and societal consequences, reminds the legal and social responsibility of the employer or the producer not to endanger the workmen or the community of the society. He or it is not absolved of the inherent responsibility to the exposed workmen or the society at large. They have the responsibility legal, moral and social to provide protective measures to the workmen and to the public or all those who are exposed to the harmful consequences of their products. Mere adoption of regulations for the enforcement has no real meaning and efficacy without die professional, industrial and governmental resources and legal and moral determination to implement such regulations.

20. The preamble and Article 38 of the Constitution of India the supreme law, envisions social justice as its arch to ensure life to be meaningful and liveable with human dignity. Jurisprudence is the eye of law giving an insight into the environment of which it is the expression. It relates the law to the spirit of the time and makes it richer. Law is the ultimate aim of every civilised society as a key system in a given era, to meet the needs and demands of its time. Justice, according to law, comprehends social urge and commitment. The Constitution commands justice, liberty, equality and fraternity as supreme values to usher in the egalitarian social, economic and political democracy. Social justice, equality and dignity of person are corner stones of social democracy. The concept 'social justice' which the Constitution of India engrafted, consists of diverse principles essential for the orderly growth and development of personality of every citizen. "Social justice" is thus an integral part of "justice" in generic sense. Justice is the genus, of which social justice is one of its species. Social justice is a dynamic device to mitigate the sufferings of the poor, weak, Dalits, Tribals and deprived sections of the society and to elevate them to the level of equality to live a life with dignity of person. Social justice is not a simple or single idea of a society but is an essential part of complex of social change to relieve the poor etc. from handicaps, penury to ward off distress, and to make their life liveable, for greater good of the society at large. In other words, the aim of social justice is to attain substantial degree of social, economic and political equality, which is the legitimate expectations. Social security, just and humane conditions of work and leisure to workman are part of his meaningful right to life and to achieve self-expression of his

personality and to enjoy the life with dignity, the State should provide facilities and opportunities to them to reach at least minimum standard of health, economic security and civilised living while sharing according to the capacity, social and cultural heritage.

21. In a developing society like ours steeped with unbridgeable and ever widening gaps of inequality in status and of opportunity, law is calalist. rubican to the poor etc. to reach the ladder of social justice, Justice K. Subba Rao, the former Chief Justice of this Court, in his "Social Justice and Law" at page 2, had stated that "Social Justice is one of the disciplines of justice and the discipline of justice relates to the society." What is due

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cannot be ascertained by absolute standard which keeps changing depending upon the time, place and circumstance. The constitutional concern of social justice as an elastic continuous process is to accord justice to all sections of the society by providing facilities and opportunities to remove handicaps and disabilities with which the poor etc. are languishing to secure dignity of their person. The Constitution, therefore, Mandates the State to accord justice to all members of the society in all facets of human activity. The concept of social justice embeds equality to flavour and enliven practical content of 'life'. Social justice and equality are complementary to each other so that both should maintain their vitality. Rule of law, therefore, is a potent instrument of social justice to bring about equality in results.

22. Article 1 of the Universal Declaration of Human Rights asserts human sensitivity and moral responsibility of every State that "all human beings are born free and equal in dignity and rights. They are endowed with reason and conscience and should act towards one another in a spirit of brotherhood." The Charter of the United Nations thus reinforces the faith in fundamental human rights and in the dignity and worth of the human person envisaged in the directive principles of State policy as part of the constitution. The jurisprudence of personhood or philosophy of the right to life envisaged under Article 21, enlarges its sweep to encompass human personality in its full blossom with invigorated health which is a wealth to the workman to can his livelihood to sustain the dignity of person and to live a life with dignity and equality.

23. Article 38(1) lays down the foundation for human rights and enjoins the State to promote the welfare of the people by securing and protecting, as effectively as it may, a social order in which justice, social, economic and political, shall inform all the institutions of the national life. Art.46 directs the State to protect the poor from social injustice and all forms of exploitation. Article 39(e) charges that the policy of the State shall be to secure "the health and strength of the workers". Article 42 mandates that the States shall make provision, statutory or executive "to secure just and humane conditions of work". Article 43 directs that the State shall "endeavour to secure to all workers, by suitable legislation or economic organisation or any other way to ensure decent standard of life and full enjoyment of leisure and social and cultural opportunities to the workers". Article 48-A enjoins the State to protect and improve the environment. As human resources are valuable national assets for peace, industrial or material production, national wealth, progress, social stability, descent standard of life of worker is an input. Art. 25(2) of the universal declaration of human rights

ensures right to standard of adequate living for health and well-being of the individual including medical care, sickness and disability, Article 2(b) of the International Convention on Political, Social and Cultural Rights protects the right of worker to enjoy just and favourable conditions of work ensuring safe and healthy working conditions.

24. The expression 'life' assured in Art.21 of the Constitution does not connote mere animal existence or continued drudgery through life. It has a much wider meaning which includes right to livelihood, better standard of life, hygienic conditions

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in work place and leisure. In *Olga Tellis v. Bombay Municipal Corporation*, 1985(3) SCC 545, this Court held that no person can live without the means of living i.e. means of livelihood. If the right to livelihood is not treated as a part of the constitutional right to life, the easiest way of depriving a person of his right to life would be to deprive him of his means of livelihood to the point of abrogation. Such deprivation would not only denude the life of its effective content of meaningfulness but it would make life impossible to live, leave aside what makes life liveable. The right to life with human dignity encompasses within its fold, some of the finer facets of human civilisation which makes life worth living. The expanded connotation of life would mean the tradition and cultural heritage of the persons concerned. In *State of H.P. v. Umed Ram Sharma*, (1986)2 SCC 68, this Court held that the right to life includes the quality of life as understood in its richness and fullness by the ambit of the constitution. Access to road was held to be an access to life itself in that state.

25. In *Sunil Batra v. Delhi Administration*, (1978) 4 SCC 494, considering the effect of solitary confinement of a prisoner sentenced to death and the meaning of the word 'life' enshrined under Article 21, the Constitution Bench held that the quality of-life covered by Article 21 is something more than the dynamic meaning attached to life and liberty. The same view was reiterated in *Board of Trustees of the port of Bombay v. D.R. Nadkarni*, (1983) 1 SCC 124, *Vikrant Deo Singh Tomar v. State of Bihar*, (1988) Suppl.SCC 734, *R. Autyanuprasi v. Union of India*, (1989)1 Suppl. SCC 251. In *Charles Sobraj v. Supdt. Central Jail, Tihar*, AIR 1978 SC 1514, this Court held that the right to life includes right to human dignity. The right against torture, cruel or unusual punishment or degraded treatment was held to violate the right to life. In *Bandhua Mukti Morcha v. Union of India*, (1984) 3 SCC 161 at 183-84, this Court held that the right to live with human dignity, enshrined in Article 21, derives its life-breath from the directive principles of the State policy and particularly Clauses (e) and (f) of Article 39 and Articles 41 and 42. In *C.E.S.C. Ltd. & Ors. v. Subhash Chandra Bose*, 1992(1) SCC 441, considered the gamut of operational efficacy of Human Rights and the constitutional rights, the right to medical aid and health and held that the right to social justice are fundamental rights. Right to free legal aid to the poor and indigent worker was held to be a fundamental right in *Khatri (11) v. State of Bihar*, (1981)1 SCC 627. Right to education was held to be a fundamental right vide *Maharashtra State B.O.S. & H.S. Education v. K.S. Gandhi*, 1991(2) SCC 716. and *Unni Krishnan v. State of A.P.*, (1993)1 SCC 645.

26. The right to health to a worker is an integral facet of meaningful right to life to have not only a meaningful existence but also robust health and vigour without which worker would lead life of misery. Lack of health denudes

his livelihood. Compelling economic necessity to work in an industry exposed to health hazards due to indigence to bread-winning to himself and his dependents, should not be at the cost of the health and vigour of the workman. Facilities and opportunities, as enjoined in Article 38, should be provided to protect the health of the workman. Provision for medical test and treatment invigorates the health of the worker for

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higher production or efficient service. Continued treatment, while in service or after retirement is a moral, legal and constitutional concomitant duty of the employer and the State. Therefore, it must be held that the right to health and medical care is a fundamental right under Article 21 read with Articles 39(c), 41 and 43 of the Constitution and make the life of the workman meaningful and purposeful with dignity of person. Right to life includes protection of the health and strength of the worker is a minimum requirement to enable a person to live with human dignity. The State, be it Union or State government or an industry, public or private, is enjoined to take all such action which will promote health, strength and vigour of the workman during the period of employment and leisure and health even after retirement as basic essentials to live the life with health and happiness. The health and strength of the worker is an integral facet of right to life. Denial thereof denudes the workman the finer facets of life violating Art.21. The right to human dignity, development of personality, social protection, right to rest and leisure are fundamental human rights to a workman assured by the Charter of Human Rights, in the Preamble and Arts.38 and 39 of the Constitution. Facilities for medical care and health against sickness ensures stable manpower for economic development and would generate devotion to duty and dedication to give the workers' best physically as well as mentally in production of goods or services. Health of the worker enables him to enjoy the fruit of his labour, keeping him physically fit and mentally alert for leading a successful life, economically, socially and culturally. Medical facilities to protect the health of the workers are, therefore, the fundamental and human rights to the workmen.

27. Therefore, we hold that right to health, medical aid to protect the health and vigour to a worker while in service or post retirement is a fundamental right under Article 21, read with Articles 39(e), 41, 43, 48A and all related Articles and fundamental human rights to make the life of the workman meaningful and purposeful with dignity of person.

28. In *M. C. Mehta v. Union of India*, (1987) 4 SCC 463, when tanneries were discharging effluents into the river Ganges, this Court, in a public interest litigation, while directing to implement Water (Prevention and Control of Pollution) Act or Environment (Protection) Act, prevented the tanneries etc. by appropriate directions from discharging effluents into the river Ganga, directed establishment of primary treatment plants etc. and such of these industries that did not comply with the directions were ordered to be closed. when ecological balance was getting upset by destroying forest due to working the mines, this Court directed closer of the mines. In *Pt Parmanand Katara v. Union of India*, (1989)4 SCC 286, Ohs court directed even private doctors or hospitals to extend services to protect the life of the patient, be an innocent or a criminal liable for punishment in accordance with law. In *National Textile Workers' Union v. P.R. Ramakrishnan*,

1983(1) SCR 922, the Constitution Bench, per majority, held that the role of a company in modern economy and their increasing impact of individuals and groups through the ramifications of their activities, began to be increasingly recognised. The socio-economic objectives set out in Part IV of the constitution guide and shape the new corporate phi-

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losophy. "Today social scientists and thinkers regard a company as a living vital and dynamic social organism with firm and deep rooted affiliations with the rest of the community in which it functions. It would be wrong to look upon it as something belonging to the shareholders." It was further held that "it is not only the shareholders who have supplied capital who are interested in the enterprise which is being run by a company but the workers who supply labour are also equally, if not, more interested because what is produced by the enterprise is the result of labour as well as capital. In fact, the owners of capital bear only limited financial risk and otherwise contribute nothing to production while labour contributes a major share of the product. While the former invest only a part of their moneys, the latter invest their sweat and toil, in fact their life itself. The workers, therefore, have a special place in a socialist pattern of society. They are not mere vendors of toil, they are not a marketable commodity to be purchased by the owners of capital. They are producers of wealth as much as capital may very much more. They supply labour without which capital would be impotent and they, at the least, equal partners with capital in the enterprise. Our constitution has shown profound concern for the workers and given them a pride of place in the new socioeconomic order envisaged in the Preamble and the Directive Principles of State Policy. The Preamble contains the profound declaration pregnant with meaning and hope for millions of peasants and workers that India shall be a socialist democratic republic where social and economic justice will inform all the institutions of national life and there will be equality of status and opportunity for all and every endeavour shall be made to promote fraternity ensuring the dignity of the individual. " In that case, the question was whether the labour is entitled to be heard before a company is closed and liquidator is appointed. In considering that question vis-a-vis Article 43-A of the constitution, this Court, per majority, held that they are entitled to be heard before appointing a liquidator in a winding up proceedings of the company.

29. In Workmen of Meenakshi Mills Lid v. Meenakshi Mills Ltd. (1992) 3 SC(: 3 36, a Bench of three Judges considered the vires of Section 25-N of the Industrial Disputes Act on the anvil of Article 19(1)(f) of the Constitution. It was held that the right of the Management under Article 19(1)(f) is subject to the mandates contained in Articles 38, 39-A, 41 and 43. Accordingly, the fundamental right, under Article 19(1)(g) was held to be subject to the directive principles and s.25-N does not suffer from the vice of unconstitutionality.

30. It would thus be clear that in an appropriate case, the Court would give appropriate directions to the employer, be it the State or its undertaking or private employer to make the right to life meaningful; to prevent pollution of work place; protection of the environment; protection of the health of the workman or to preserve free and unpolluted water for the safety and health of the people. The authorities or even private persons or industry are bound by

the directions issued by this Court under Article 32 and Article 142 of the Constitution.

31. Yet another contentions of the petitioners is that the workman affected by asbestosis are suffering from lung cancer

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and related ailments and they were not properly diagnosed. They be sent to national institute and such of those found suffering from diseases developed due to asbestos, proper compensation paid. It is needless to reiterate that they need to be re-examined and cause for the disease and the nature of the disease diagnosed. Thereon each one of them whether entitled to damages? The employer is vicariously liable to pay damages is unquestionable. The award of compensation in proceedings under Article 32 or 226 is a remedy available in public law. In Rudul Sah v. State of Bihar, 1983(3) SCR 508, it was held that this Court under Article 32 can grant compensation for the deprivation of personal liberty, though ordinary process of court, may be available to enforce the right and money claim could be granted by this Court. Accordingly compensation was awarded. This view was reiterated in Nilabati Behera v. State of Orissa, (1993) 2 SCC 746 and awarded monetary compensation for custodial death lifting the State immunity from the purview of public law. It is, therefore, settled law that in public law claim for compensation is a remedy available under Article 32 or 226 for the enforcement and protection of fundamental and human rights. The defence of sovereign immunity is inapplicable and alien to the concept of guarantee of fundamental rights. There is no question of defence being available for constitutional remedy. It is a practical and inexpensive mode of redress available for the contravention made by the State, its servants, its instrumentalities, a company or a person in the purported exercise of their powers and enforcement of the rights claimed either under the statutes or licence issued under the statute or for the enforcement of any right or duty under the constitution or the law.

32. The Government of India issued model Rule 123-A under the Factories Act for adoption. Under the directions issued by this Court from time to time, all the State governments have by now amended their respective rules and adopted the same as part of it but still there are yearning gaps in their effective implementation in that behalf. It is, therefore, necessary to issue appropriate directions. In the light of the rules "All Safety in the Use of Asbestos" issued by the I.L.O., the same shall be binding on all the industries. As a fact, the 13th respondent-Ferodo Ltd admitted in its written submissions that all the major industries in India have formed an association called the "Asbestos Information Centre" (AIC) affiliated to the Asbestos International Association(AIA), London. The AIA has been publishing a code of conduct for its members in accordance with the international practice and all the members of AIC have been following the same. In view of that admission, they are bound by the directions issued by the ILO referred to in the body of the judgment. In that view, it is not necessary to issue any direction to Union or State Governments to constitute a committee to convert the dry process of manufacturing into wet process but they are bound by the rules not only specifically referred to in the judgment but all the rules in that behalf in the above I.L.O. rules. The Employees State Insurance Act and the Workmen's Compensation Act provide for payment of mandatory compensation for the injury or death caused to the workman

while in employment. Since the Act does not provide for payment of compensation after cessation of employment, it becomes necessary to protect such persons from the respective dates of cessation of their employment till date. Liquidated damages by way of compensation are accepted principles of

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compensation. In the light of the law above laid down and also on the doctrine of tortious liability, the respective factories or companies shall be bound to compensate the workmen for the health hazards which is the cause for the disease with which the workmen are suffering from or had suffered pending the writ petitions. Therefore, the factory or establishment shall be responsible to pay liquidated damages to the concerned workmen.

33. The writ petition is, therefore, allowed. All the industries are directed (1) To maintain and keep maintaining the health record of every worker up to a minimum period of 40 years from the beginning of the employment or 15 years after retirement or cessation of the employment whichever is later; (2) The Membrane Filter test, to detect asbestos fibre should be adopted by all the factories or establishments at par with the Metalliferous Mines Regulations, 1961; and Vienna Convention and Rules issued thereunder; (3) All the factories whether covered by the Employees State Insurance Act or Workmen's Compensation Act or otherwise are directed to compulsorily insure health coverage to every worker; (4) The Union and the State Governments are directed to review the standards of permissible exposure limit value of fibre/cc in tune with the international standards reducing the permissible content as prayed in the writ petition referred to at the beginning. The review shall be continued after every 10 years and also as and when the I.L.O. gives directions in this behalf consistent with its recommendations or any Conventions; (5) The Union and all the State Governments are directed to consider inclusion of such of those small scale factory or factories or industries to protect health hazards of the worker engaged in the manufacture of asbestos or its ancillary produce; (6) The appropriate Inspector of Factories in particular of the State of Gujarat, is directed to send all the workers, examined by the concerned ESI hospital, for re-examination by the National Institute of Occupational Health to detect whether all or any of them are suffering from asbestosis. In case of the positive finding that all or any of them are suffering from the occupational health hazards, each such worker shall be entitled to compensation in a sum of rupees one lakh payable by the concerned factory or industry or establishment within a period of three months from the date of certification by the National Institute of Occupational Health.

34. The writ petitions are accordingly allowed. No costs.

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