THE EVALUATION OF DISABILITY AMONG WORKERS EXPOSED TO THE HAZARD OF INDUSTRIAL LUNG DISEASE IN AN ASBESTOS FACTORY

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for the degree of
M.D.
(Chest Diseases)

By

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A C K N O W L E D G E M E N T

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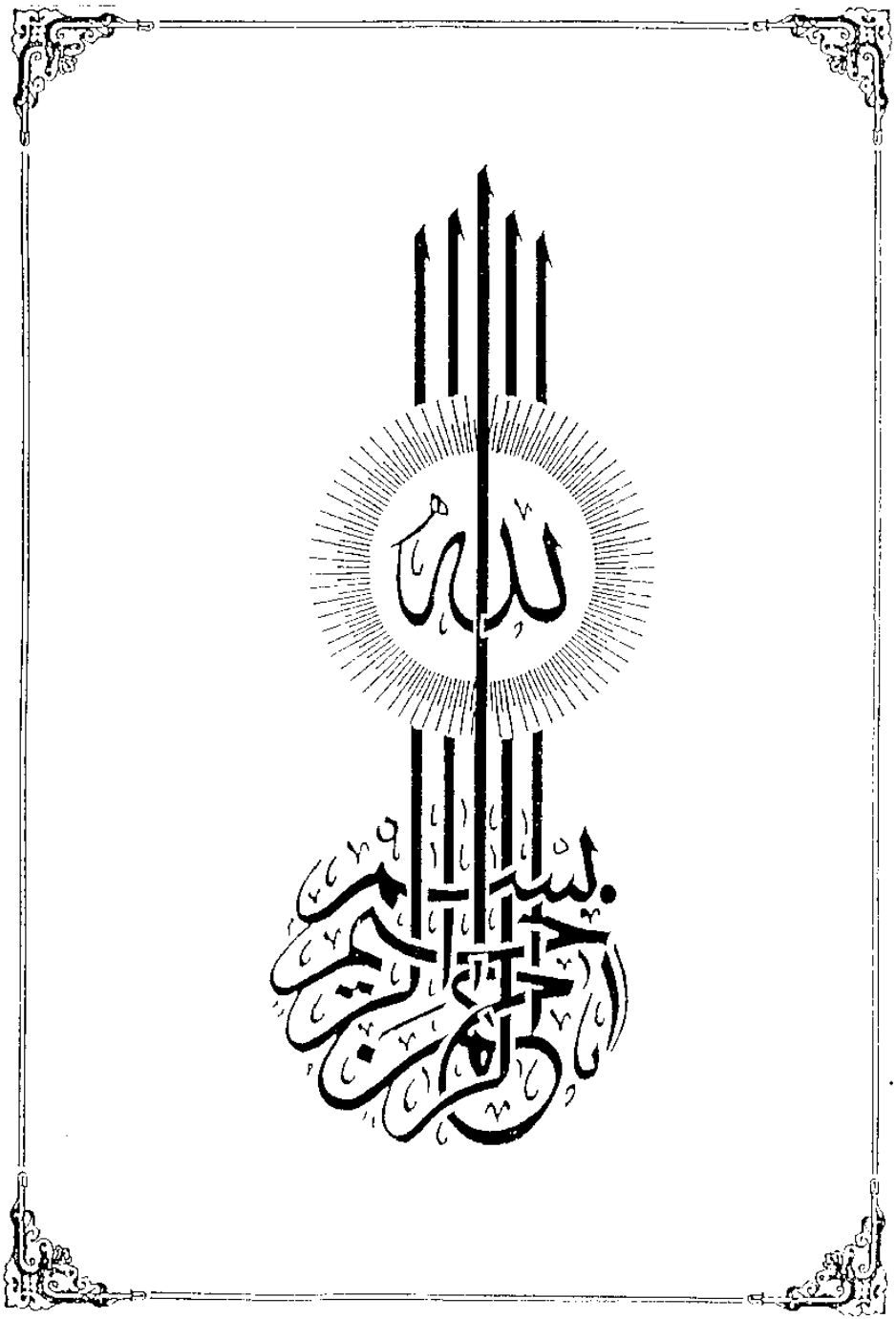

C O N T E N T S

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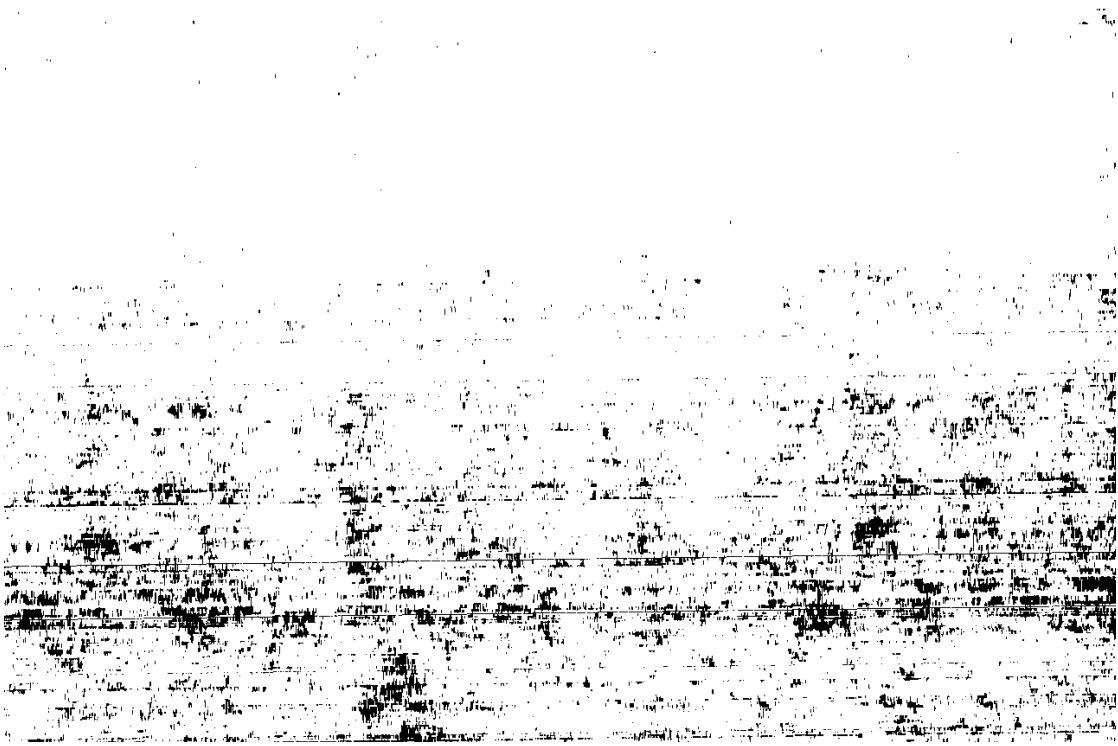
INTRODUCTION



I N T R O D U C T I O N

According to Comroe et al (1967)[24] the degree of functional impairment of the lungs will vary considerably among "disabled" patients because of the different work requirements of different occupations, heavy workers vs. accountants; the definition, therefore, of pulmonary disability as inability to perform the present job or the inability to perform any gainful occupation is too rigid for general application. Pulmonary insufficiency for the oxygenating function is rarely incapacitating if the insufficiency develops gradually over long periods of time, i.e. in chronic pulmonary diseases (Comroe et al, 1967)[24].

If "constant" cough and expectoration are excluded as well as haemorrhage (haemoptesis), then the most frequent cause of pulmonary disability is dyspnoea = difficulty breathing (Comroe et al, 1967)[24]. The clinical grading of dyspnoea, on an ordinary scale, based on quantitative

patients as the only measure for assessment of disability is open to error if not checked on by pulmonary function tests; this is particularly important when compensation is a consideration. In this respect the work of Gilson and Hugh-Jones (1955)[58] in coalworkers pneumoconiosis is a classical example where the pulmonary function tests correlated highly with clinical grades of dyspnea, that of the ventilatory capacity related most closely and even closer than the more sophesticated tests based on blood and mixed expired gas analysis.

Many patients would have insufficiency for the exchange of O_2 and CO_2 if their ventilation remained at "normal" levels. However, they avoid "insufficiency" for O_2 and CO_2 by hyperventilation. Strictly speaking, they do not have pulmonary insufficiency at this time, but only because of increased ventilation and additional work by their respiratory muscles (Comroe et al, 1967).[24] If evidence of increased ventilation does not show at rest it may become evident on exercise. Exercise tests performed on the classical lines, such as ergometry and

treadmill exercise require elaborate apparatus for measurement of blood gases and analysis of mixed expired gas at a steady state. Spirometers such as Fleish and Kipping, which analyse mixed expired gas for O_2 and CO_2 can measure the respiratory quotient which is a basic item of information in relation to the presence of a steady state. Such spirometers are used for measuring O_2 consumption during ergo-spirometry. In absence of such apparatus, the measurement of volume of ventilation instead of O_2 consumption at moderate and low work levels while performing a standardised exercise step test as advised by Hugh-Jones (1952)[70] and Gandevia (1962)[54] - provides an alternative (Cotes, 1965)[33]. Compensation of exposed workers should be settled by objective tests which do not require the claimants co-operation this may not be fulfilled by many pulmonary function tests with the exception of CO diffusion capacity tests or tests based on arterial blood studies. In the present work the adjusted standardized ventilation [illegible] which used as a parameter of gas transfer

as well as of ventilatory capacity (Campbell, 1968)[16].

The forced expiratory volume in the first second is a flow rate of established value, however, the calculation of the indirect maximal breathing capacity from this parameter has distinct practical advantages over the classical performance of the direct test in 15 seconds.

The portability and practicability of the peak flow meter in measuring the ventilatory capacity of a large number of cotton factory workers has been demonstrated by El-Gammal (1962)[37].

Assessment of gas exchange, during a steady state, at rest, measurement of dead space based on determination of Pa CO_2 by rebreathing and the use of standardised exercise test in addition to the use of simple spirometry has been described by El-Gammal (1968)[39] and has been followed by Aly (1969)[1] in examination of patients with pulmonary subistenosis. The same procedures are used in this work since then, with the exception of mixed expired gas analysis.

were advocated by the Ciba Guest Symposium* for the brief assessment of lung function.

The determination of lung volumes were intentionally neglected in the present work, since, in the words of Comroe et al (1967)[22] "Actually, they do not evaluate function since they are essentially anatomic measurements".

The description of radiological changes in industrial lung disease has been the subject of international conferences of experts. In the present work, the ILO (1958) Geneva conference classification has been followed up as a practical and inclusive classification which has been followed up in the Ein-Shams Chest Unit since 1965. The availability of the ILO standard radiographs in our chest unit showing the x-ray changes described in the classification are used as a yardstick for describing the radiological changes in exposed workers.

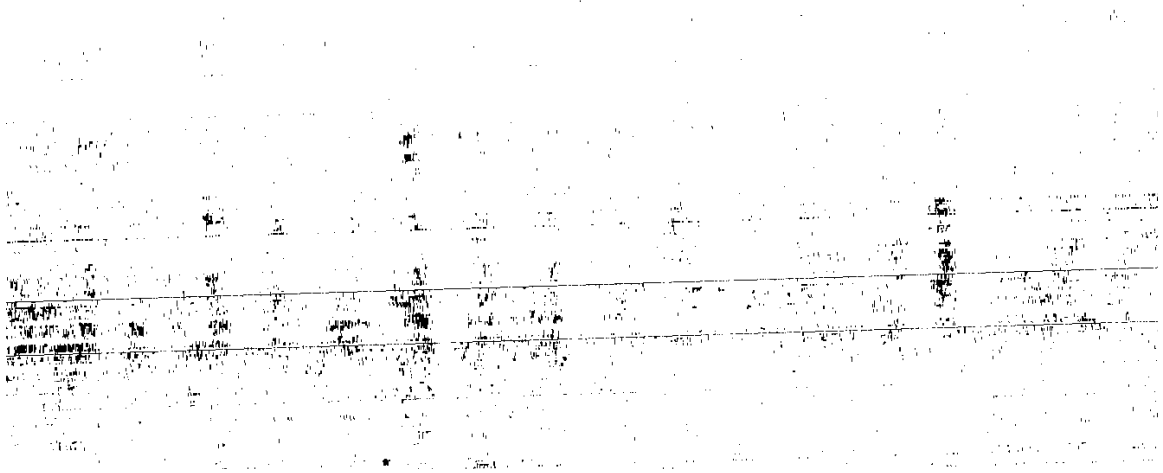
The selection of workers and the planning for the work are based on the methods advised by Gilson (1960)[29] for the

(*) Thorax (1959), 14 : 286.

investigation of disturbance of pulmonary function in industrial pulmonary diseases.

Environmental studies in Egypt are usually available from the work of the air pollution unit in the National Research Centre (Dokki). In the present work, study of dust concentration was quoted from a report by the air pollution unit.

It is hoped that the present work will fulfil the requirements demanded not only in the protocol of the thesis, but also for future investigation of exposed industrial workers in field work as well as in assessment of an individual case.



REVIEW OF LITERATURE

to these workers a minimum length of 20 - 50 microns for the asbestos fibres is required to produce fibrosis in the guinea-pig.

Lynch (1955)[94] is of the opinion that most of asbestos-plant dust when inhaled is expelled, but a remarkable quantity of fragments of length up to 100 microns or more reaches the terminal respiratory units and that this material exerts physical rather than chemical influence. He stated that " while it now appears that free silica is in a crude sense chemically poisonous to living tissues when deposited in them, asbestos crystal fragments may traumatize living tissues by their physical properties. Hence, the present belief that it is the larger particles that do the damage here as contrasted with the harmful effects of the smaller particles in silicosis ".

Gough and Hepplestone (1960)[64] presented experimental evidences which favour again the hypothesis of mechanical irritation : Erionite ($Na\ CaK_2$) fibres, containing very little silica, caused a similar fibrotic reaction to asbestos on

intratracheal injection. The potency of free silica increases with diminishing particle sizes, but asbestos fibres less than 10-20 microns long are practically innocuous. Aluminium hydroxide neutralizes the effect of quartz but not asbestos. Non-fibrous serpentine is inert in the tissues but has the same chemical composition as the active fibrous chrysotile (a hydrated magnesium silicate). Thus, though minerals causing asbestosis vary widely in their chemical composition they are all fibrous in form, favouring a mechanical hypothesis. Furthermore, long fibres must be flexible, inflexible fibres of glasswool are non-fibrogenic. Long, flexible fibres only appear to act in the lung and peritoneum; that is sites normally subjected to regular movements.

ASBESTOSIS - PATHOLOGY

Site of Lesion :

Whereas Gloyne (1933),[60] Gough and Heppleston (1960)[63] described the most marked lesions in asbestosis as occurring in the lower lobes, Wagner (1960)[134] found in his