HEALTH HAZARDS AMONG A GROUP OF WOODWORKERS

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قالسوا

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INTRODUCTION

INTRODUCTION

Accustomed as he has been from time immemorial to daily contact with wood, man has always regarded this material as entirely innocuous. Nowadays, however, it is known not necessarily the case, and that in particular the dust produced in woodworking may prove hazardous in high concentrations. Wood industry has got its general adverse health effects, as all mechanized industries, which can be from abrasions up to amputations and foreign body impaction especially to the eye. The wood dust is hazardous to the respiratory tract, and also may cause contact dermatitis. Not less hazardous are the organic solvents used in this industry. These solvents include toluene, xylene, thinner, glue which is from ammonia and formaldehyde; and adhesives.

The clinical symptoms, in which wood dust is assumed to be a causative agent are different according to different authorities. South African boxwood (S.A.B) was introduced into the British Isles. in 1905 for use in Shuttle making for the Lancashire cotton industry where it caused widespread disability. It was found that the machinery used in making the wooden shuttles produced a dust, the inhalation of which led to drowsiness in the workmen so that they nearly fell off their benches. Pieces of this wood were studied by Marvey Gibson, at the depart of botany in Liverpool University, and an alkaloid was discovered in the wood which induced a gradual slwoing of the heart beat.

It is undoubtedly accounted for the symptoms complained of by the workmen (May 1908 cited by Hunter, 1976). The complaints of the workmen were different; some complained of headache, somnolence, running of the nose or an irritation in the nasal passages, running of the eyes, sneezing, attacks of cough, slight dysponea expiratory in nature, a sense of tightness across the chest and faintness. In order to deal with the problem Legge 1934 (Cited by Hunter, 1976) applied local exhaust ventilation to the benches of the workmen. This did not entirely suppress the dust, and other types of wood had to be substituted. In 1941 Davidson (cited by Hunter 1976) reported an outbreak of contact dermatitis in men handling a consignment of badly-seasoned iroko logs. All complained of itching of exposed skin surfaces, while some suffered also from intense irritation of covered parts, especially the neck, axillae, antecubital fossae, genitalia and backs of the knees. In some cases there was marked oedema of the face with irritation of the conjunctiva and blepharospasm. In 1936 Bridge described the effects of bronchial spasm in men handling mansonia wood. In 1941 Davidson reported nine cases of bronchial asthma out of fifty. In 1949 Doing recorded small outbreaks of asthma in factories where Western red cedar was handled. In British Columbia, Canada, Cedar posioning occurs as asthma, dermatitis or both (Strong, 1954). One should always investigate whether woodcutters' disease may not be due

contact sensitivity to lichens growing on trees (Champion, 1965)

The studies about the allergic effects to the skin and respiratory system of wood dust are continued by different authorities in order to specify the exposure limit values, pathogenesis of diseases caused by wood dust, and plans of preventing these diseases. The American Conference of Governmental Industrial Hygienists has proposed the adoption of TLV for non-allergenic wood dust of 5 mg./m³. The USSR health authorities have adopted MAC of 2 mg./m³ for wood dust containing 10% or more of free silica, and of 4 mg./m³ when the free silica content in wood dust is below 10% (Kadlec and Hanslian, 1983). The biological effects of wood dust give rise to many different morbid symptoms, the nature of which depends on the quantity and composition of the constitutent substances which range from hydrocarbons to polycyclic compounds (Kadlec and Hanslian, 1983).

The literature contains reports of reversible bronchial obstruction caused by exposure to the wood dust of boxwood, cottonwood, and evergreens used as Christmas trees. Sosman et al. (1969) reviewed these reports and considered that a hypersensitivity reaction plus a non-specific irritant effect are responsible. In 1967 Cohen et al. described an interstitial granulomatous pneumonitis following prolonged inhalation of sawdust of redwood trees. Schlueter et al. (1972) described a hypersensitivity pneumonitis

in men working with wood plup, and name it woodpulp working disease. In 1973 Chan-Yeung et al. described in detail asthmatic symptoms and rhinitis suffered by men working in the dust of a species of western red cedar. Michaels (1967) studied autopsy material of wood workers and suggested that there may be a pneumnoconiosis associated with furniture manufacture and the timber trade. Acheson et al. (1968) have reported a significant increase in adenocarcinoma of the nasal cavity and accessory sinuses, especially the ethmoid, among wood workers in U.K. At present the dust of oak and beech is suspected to be the aetiologic wood types (Hunter 1976). In 1972 Acheson et al. confirmed this observation (Hamilton and Hardy 1974).

In 1975 Carl Zenz described wood workers pneumnoconiosis (Maple bark disease, sequosis, wood pulp workers, disease, wood workers lung). Workers engaged in wood-working operations with maple trees, particularly sawing, debarking and chipping the logs, were exposed to clouds of dust particles, 85% of which have been shown to be composed of the fungus Cryptostroma corticale. The inhalation of large concentrations of this organism produces the classic signs and symptoms described under farmer's lung, and eventually may result in irreversible pulmonary fibrosis and death. The spores of the fungus are 4-5 microns in diameter and can be found growing beneath the bark of the maple logs.

Not a less serious hazard of wood industry is due to exposure to noise. The extraudiotary effects of noise include annoyance, headache, hypertension, cardiovascular diseases. The audiotary effect is occupational impairment of hearing. In 1976 Hunter described occupational impairment of hearing: It occurs in workers exposed to noise and vibration in many industries. True occupational deafness develops slowly in response to a frequently repeated noise and affects a considerable number of workers engaged in the same work. It must be distinguished from deafness occurring as a result of an accident, such as an explosion, which causes a very intense sudden noise which causes sudden deafness.

According to the WHO report (1983 series 684), vegatable dust is defined as an aerosol derived from plant material regardless of the nature of the particles or the circumstances of their emission into the air. The harmful effects on the airways and lungs caused by inhaled as aerosol vegetable dust were classified into: An allergic (atopic) response that occurs either in the upper airways (hay fever) or in the bronchi (asthma), or in both. The second type of response is byssinosis. The third type involves immunological changes in the lung parenchyma causing diseases collectively known as extrinsic allergic pneumonitis or hypersensitivity pneumonitis. The fourth type of response is simple non-specific irritation in the respiratory tract, which may, with prolonged exposure lead to non-specific chronic obstructive

pulmonary disease (COPD). Wood dust is one of the dusts (grain cereals and their products, wood dust, tea and tobacco) which cause occupational asthma. Wood dust may also cause COPD and extrinsic allergic pneumonitis. The number of harmful vegetable dusts is so vast that it is virtually impossible to study the adverse health effect for all of them. Wood dust is one of those studied because of its distribution, abundance, frequency of exposure to it, its potential to cause serious functional disability.

From this historical background, it is clear how variable the hazards to which wood workers are exposed, and hence how essential to study their health condition and to suggest preventive measures.

LITERATURE REVIEW

DEFINITION OF TERMS AND ABBREVIATIONS

FEV_{1.0}

Fored expiratory volume in the first second (in litres). It is the volume of gas expired over the first second of a forced expiration following a full inspiration. The FEV_{1-0} in healthy adult males, depending on their age and size, is in the range of 1.2 L. to 5.7 L. In females in the range of 0.8 L. to 4.0 L.

FVC

Forced Vital Capacity (in litres) is the maximal volume of gas which can be expelled from the lung during a forced expiration starting from total lung capacity. Normally one should be able to exhale at least 75% of FVC in the first second, and 95% in the first three seconds. The first 25% of an FVC maneuver is totally effort-dependent, and the last 75% of the FVC maneuver is relatively effort-independent.

FEV%

Percentage expired i.e. FEV/FVCX100.

PEFR

Peak expiratory flow rate in (L/min.) (Cotes, 1975. Burton et al. 1977).

Allergic

rhinitis

A condition characterized by sudden onset on exposure to an exciting cause (dust, cold etc.)