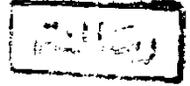


PULMONARY FUNCTION CHANGES
IN SMOKERS



THESIS

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BY

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TO MY PARENTS



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Introduction
&
AIM OF WORK

INTRODUCTION AND AIM OF THE WORK

Clinical findings and the chest roentgenogram establish the diagnosis of many lung diseases, yet the evaluation of patient with lung diseases is often incomplete without measurement of the physiologic consequences. It is difficult to judge the severity of the lung diseases without physiologic testing because there is often discrepancy between clinical signs, symptom roentgenographic findings, and physiologic parameters. Physiologic testing has many uses. As a sensitive index of the presence of lung diseases, it may be used to detect subclinical disease. This is especially true in chronic obstructive lung diseases, where abnormalities in small airways can be identified early in the course of the disease. Once a disease is clinically manifest, physiologic testing can characterize the type of dysfunction, quantify the impairment, relate an incremental change over time and evaluate response to therapy, Poe and Israel (1982).

Young asymptomatic smokers develop alteration in pulmonary functions that appear to precede the clinical development of chronic bronchitis and

emphysema. Particular smoking habit such as lighting and leaving cigarette in mouth, also increase the risk of disease, Lam and Coworkers (1982).

Several factors have been associated with development of chronic obstructive pulmonary diseases, but heavy cigarette smoking is the single most common cause of bronchial irritation. Even smoking in households or public areas may have a detrimental effect on non-smokers. The continued inhalation of tobacco smoke results in persistent bronchial inflammation and airway obstruction. The relative risk of mortality from chronic bronchitis in smokers is over 20 times the risk on non smokers.

The aim of this study is to detect changes which occur in pulmonary functions in current smokers.

Review of Literature

REVIEW OF LITERATURE

General effect of cigarette smoking on respiratory system:

Smoking is an extremely wide spread habit all over the world. Many studies had been done to demonstrate the effect of smoking on the respiratory system.

A report to the congress on smoking and health had documented the adverse relationship between smoking and pulmonary diseases (U.S. public services 1964).

It is a matter of every day clinical observation that the majority of patients with obstructive lung diseases give a significant history of tobacco smoking. Read and Selby (1961).

The association of cigarette smoking and chronic obstructive lung disease is now well established. McCarthy and Coworkers (1976).

Cigarette smoking had been found to be closely related to increased incidence of pulmonary cancer. Hammond and Horn (1954).

Salem and Co-workers (1973) found 14% of cigarette smokers in his work had bronchial carcinoma.

Chronic cigarette smoking generally leads to a decrease in the mucociliary transport that was correlated with structural changes in trachea and bronchi. Lourance and Co-workers (1971) Camner and Philipson (1972) Camner and Co-workers (1973), Wanner and Co-workers (1973). These changes include loss of ciliated epithelium, increased number of goblet cells, squameous metaplasia and increased quantity of mucous in tracheo-bronchial tree. Auerbach and Stout (1957), (1961).

Certain agents in the smoke serve as irritants that over a long period could easily lead to chronic infection and permanent changes in tracheo-bronchial tree as well as the alveolar epithelium. The effect of cigarette smoking vary greatly in degree of pathologic changes in trachea or bronchial structure especially if they had smoked over a period of eighteen years as much as one package or more per

day and conclusion was that smoking damages the pulmonary tissue and depresses physiologic function. Russel and Co-workers (1978).

Falk and Co-workers (1959) found that one of properties of cigarette smoking is its ability to interfere with normal function of ciliated mucous secreting epithelium. This effect is manifested primarily by slowing rate of flow, quantitative and qualitative changes in mucous secretion.

The effect of smoking on the tracheo-bronchial epithelium, not only causes carcinoma in situ but also presents a clear evidence of basal cell hyperplasia, increased stratification of surface mucosa, squamous cell metaplasia and other signs of inflammatory process of various degree. Aurbach and Stout (1957).

A number of clinical studies showed that daily chronic cough was rare in non-smokers, ex-smokers but the prevalence became more progressively greater with increase number of cigarettes smoked. Sputum

production followed the same pattern as cough. Wheeze seldom occurred in non-smokers, ex-smokers and light smokers but was found with increase frequency in moderate and heavy smokers. Oswald and Medrei (1955), Higgin (1957), Ferris and Anderson (1962), Hammond (1962), Ccline and Coworkers (1971).

In a number of epidemiological studies cough and sputum had been a frequent complaint in smoking females than non-smoking ones. Payne and Kjelsberg (1964), United State public health services (1967). Not only cough but also breathlessness on exertion was more frequently present in smoking female than non-smoking ones. Higgins (1957).

Higgins (1959) had suggested that increased incidence of bronchitis among smokers merely reflects a simple quantitative effect of tobacco smoking on bronchial mucosa. If this was so, it would be suggested that increased tobacco consumption and increased duration of smoking should be associated with an increased incidence of symptoms but it was demonestrated that neither of these two factors were true. Read and Selby (1961).

Women who smoked cigarette showed significant greater death rate as compared with those who had never smoked. The excess death rate among current cigarette smokers account for one every fourteen deaths (U.S. Pub. Services) (1967).

Many young smokers had a significantly tracheal decreased mucus velocity compared with that of age matched non-smokers Goodman and Coworkers (1978).

El Aaser and Coworkers (1974) reported a significant lowered occurrence of bronchial carcinoma among Goza smokers. Compared with cigarette smokers.

Salem and Coworkers (1973) stated that productive cough and mucoid sputum were the predominant symptoms, next in frequency was shortness of breath, which was more commonly accompanied with wheeze in Goza smokers. They proved that 24% of Goza smokers included in their study had pulmonary tuberculosis.

The pathological studies in cigarette smokers indicated that earliest pulmonary lesion lies in the small airways of less than 2 mm in diameter.

Disease in this region of bronchial tree was not detectable by spirographic parameters which are related to early part of flow rate. Read and Selby (1961).

Benjamin and Coworkers (1977) had reported that there was a wide variation in individual reaction to cigarette smoking. Higgin and Coworkers (1968) had suggested that a genetic factor cause variable response to tobacco smoke. Hankin and Coworkers (1982) had proved that a genetic factor directs the effect of cigarette smoke on bronchial tree and lung parenchyma.