either group. In 8 of the 32 individuals (4 with COPD and 4 without) the changes in V/Q were of sufficient magnitude to cause a significant decrease in PaO₂, averaging 10.4 mm Hg decrease immediately after smoking; there were no changes in PaCO₂ or pH. In six of these eight, the response was primarily in the airways as indicated by increased A-a, QS/QT and alveolar shunting. In two of the eight, the response was primarily vascular as judged by an increase in physiologic dead space, wasted ventilation, alveolar dead space and a decrease in the effective gas exchange compartment of the lungs.

An additional six patients with COPD were studied to determine the effect of cigarette smoking on levels of carboxyhemoglobin and cardiac output. Smoking one cigarette increased the carboxyhemoglobin from an average of 4.26 percent to 5.36 percent. Three of the subjects had a mean decrease of 1.1 L/minute in cardiac output and three a mean increase of 2.21 L/minute as a result of smoking. If we assume an individual with a pre-smoking PaO₂ of 70 mm and 14 grams of hemoglobin he will sustain a decrease of 7 ml/L in O₂ if his PaO₂ drops by 10 mm Hg as seen in our eight hypoxia responding patients, an additional loss of 16.13 ml/L due to carboxyhemoglobin and 4.2 ml/liter due to leftward displacement of the oxyhemoglobin dissociation curve. This is a net loss of 27.33 ml of O₂/L of blood or 15 percent. A mean decrease of 1.1 L/minute in cardiac output will further decrease oxygen delivery by 161 ml O₂/minute. In some instances, significant decrease in O₂ delivery for cellular metabolism can result from smoking.

Discussion

In response to questions, Anderson noted marked differences in the pattern of smoking in the subjects studied. This was not measured. They simply made sure that each subject smoked 6 cm of each cigarette and that the puffs were taken at intervals of no greater than 30 seconds. Subjects who already had a low PaO₂ responded in the same way with respect to their A-a gradient as those with normal PaO₂. Only two subjects had PaO₂'s less than 80 mm Hg and they responded in much the same way as those with normal PaO₂. He felt that the decrease in ventilation was the most likely explanation for the observed changes in PaO₂, venous admixture and the A-a gradient.

Smoking and Chronic Respiratory Disease: Finding in Surveys Carried Out in 1957 and 1966 in Staveley in Derbyshire, England


Many studies have shown a higher prevalence of respiratory symptoms and lower average ventilatory lung function in smokers than in nonsmokers. Fewer studies have assessed changes in lung function over the years or the rate of development or remission of symptoms in relation to smoking habits.

Surveys of chronic respiratory disease were conducted in 1957 and 1966 in representative samples of the male population of Staveley, Derbyshire, England.

In 1957 two age groups, 25-34 and 55-64, were sampled. In 1966 each ten-year age group from 25-74 was included. On each occasion there were four occupational groups. Three of these were men who had been engaged in dusty jobs: (1) miners and ex-miners, (2) foundry and ex-foundry workers, and (3) men who had worked in both mining and foundry work or in other dusty jobs or with chemicals. The fourth occupational group was men who had worked only in nondusty jobs. In 1966, in addition to the 1966 representative cross-sectional sample, a nine-year follow-up was made of all those who had been examined in 1957.

There were 756 men who were seen in the 1957 sample. In 1966, 594 (78.6 percent) were seen again; 102 (13.5 percent) had died, 42 (5.6 percent) had left the area, and 18 (2.4 percent) refused to cooperate. About 1 percent of the results were excluded because of incompleteness. There were 992 men in the new 1966 sample. Nine hundred and four (91.3 percent) were seen. After excluding records with missing data, 878 were finally analyzed.

The methods used were similar on the two occasions. They included assessment of respiratory symptoms, chest illnesses and smoking habits in a standardized manner using a prototype of the MRC respiratory symptoms questionnaire; occupational and residential histories; measurement of the forced expiratory volume and forced vital capacity, standing and sitting height, and weight; and chest radiography. In 1966 the questionnaires and lung function tests were carried out by four physicians. Two of them had seen all the members of the sample in 1957. On each occasion subjects were allocated to each observer at random so that observer variation could be adequately studied.

The age-adjusted prevalence of cough, sputum, chest illness during the past three years, wheezing in the chest and breathlessness was higher in smokers than in nonsmokers. The trend of increasing prevalence with increasing cigarette consumption was striking only for cough and sputum. The age-adjusted FEV₁ values were lower in smokers than in nonsmokers or ex-smokers. There were only small differences in FVC between the different smoking categories. Consequently, the FEV₁ /
The regression of FEV on age showed that some age groups of smokers had appreciably lower mean or no difference between them. It is likely that the inconsistency is partly due to the confounding effect of occupation. The regression of FEV on age showed that the decline was least for nonsmokers. There was little difference in the slope of the regression between light and heavy smokers.

The rate of development of symptoms in those who in 1957 had reported freedom from each symptom and the rate of disappearance of symptoms in those who had reported symptoms at that time were studied in relation to smoking habits. A higher proportion of smokers than of nonsmokers or ex-smokers developed symptoms during the nine years. A higher proportion of nonsmokers and of ex-smokers than of continuing smokers were relieved of symptoms during the follow-up period.

The effect of giving up smoking was also studied. Among the younger men who reported giving up smoking between 1957 and 1966, a higher proportion reported no symptoms in 1966 than had done so in 1957. But among the older men there was no change in the proportion who reported no symptoms in 1966. The findings suggest that the benefits of giving up smoking on respiratory symptoms are less in those who have smoked for many years than in those who have smoked for shorter periods.

Mortality during the nine years was related to smoking habits as recorded in 1957. In the men aged 55-64 the proportion of smokers who died from all causes during the nine years was approximately double that of the nonsmokers or ex-smokers.

The validity of the symptoms and lung function tests used was assessed in relation to mortality. There was a progressive increase in the proportion who died with (1) increase in prevalence of symptoms reported in 1957, (2) increase in breathlessness grade, and (3) decrease in FEV.

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Discussion

Speizer stated that the rate of deterioration of lung function was not influenced by the number or severity of chest infections over a long period. However, many factors should be considered in the causation of deterioration of pulmonary function, and it is extremely difficult to isolate any one factor, such as infection, and assess its significance.

SESSION VIII. PATHOLOGY

A Quantitative Study of the Relationship between Chronic Bronchitis, Emphysema and Smoking

M. S. Dunnill, M.D., and R. Ryder, M.D.

The lungs of 353 patients at autopsy were examined using the point counting technique1 to estimate the percentage bronchial mucous gland volume and the percentage volume of emphysema in the lung parenchyma. The age and sex of each patient was known and in 179 cases a smoking history was available.

The mean percentage volume of bronchial mucous glands for the whole series was 17.0 (SD 7.1). In the 106 known smokers, the value was 17.6 (SD 6.6) and in 73 non-smokers 14.5 (SD 6.6), a difference that was small but statistically significant (P < 0.005). There was no significant difference in the mean bronchial mucous gland volume with age of the type observed by Thurlbeck.2 The mean value obtained for all women was slightly less than for all men due to the high proportion of women non-smokers.

Emphysema, mainly of the centrilobular type, was present in 219 cases. The volume of the lung parenchyma occupied by the emphysematous spaces ranged from 0.5 to 95.0 percent. The difference in incidence between smokers and non-smokers was very striking, emphysema being present in 80 out of 106 smokers and in only 21 out of 73 non-smokers (P < 0.001). The mean value for the percentage volume of parenchyma involved with emphysema in smokers was 10.8 and in non-smokers 1.7. In smokers the disease began between 30 and 39 years and was almost universal by 50 years whereas in non-smokers it was rare even in the ninth decade. These differences were highly significant over the whole series and when men and women were considered separately.

There was no obvious direct relationship between bronchial mucous gland volume and the volume of lung parenchyma involved in emphysema. However, the mean percentage bronchial mucous gland volume in the 219 cases which showed emphysema was 18.3 compared with 14.8 in those cases without emphysema, a difference which was very significant (P < 0.001).