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Young asymptomatic smokers with relatively short smoking histories, show recognisable and significant pulmonary changes. The earliest changes occur in the small airways and can be detected by relatively simple and inexpensive tests such as the forced expiratory spirogram. These changes are related to the total amount of tobacco smoked and increase progressively with continued smoking.

The rapid onset of airway changes in these young smokers, even within as short a period as 2 years of smoking may reflect a special susceptibility of the growing lung to inhaled smoke. The mechanisms by which these changes are brought about are not clear, but inflammation, qualitative and quantitative alterations in the secretion of mucus and modification of surface characteristics may play an important part in their pathogenesis.

To what extent these changes are reversible in these early stages, needs to be studied. Preliminary studies done in this laboratory, and other published reports 120,154 suggest that at least some of these effects such as the changes in PEF may be reversed within as short a period as six weeks. It is difficult to evaluate whether this reversal is complete or partial,
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exchange. If so, it should be possible to demonstrate the beneficial effect of breathing oxygen on the heart rates and oxygen uptakes of smokers during maximal work loads.

As observed in the present study and in published reports, the immediate response of the large airways to cigarette smoking is constriction, whereas the small airway response is generally dilatation but may be variable. Histamine release as indicated by a fall in blood basophils and eosinophils is probably one of the mechanisms by which airway constriction is brought about. The constriction that histamine would be expected to produce in the small airways may be masked by a sympathomimetic dilator action caused by catecholamine release, due to the nicotine absorbed.

There seem to be large differences in the magnitude of the immediate response among individual subjects, as determined by the extent of the fall in PEF and blood basophil and eosinophil counts immediately after smoking. Nevertheless, it appears unlikely that the magnitude of this immediate response would help to detect individual susceptibility to prolonged smoking.

The fall in static compliance and decrease in the static P-V hysteresis observed in the experimental animals immediately after smoking, indicate alteration of the surface properties of the lung, probably by
inactivation of surfactant.

Three lines of investigation follow naturally from these findings. One is to clarify the mechanism whereby small airway dilatation occurs. As this is probably mediated through the action of catecholamines on the beta receptors, more studies using beta blockers would be useful. Zuskin et al. (1974)\textsuperscript{243} have shown that propranolol could potentiate the effect of smoking. This underlines the danger to patients who are being treated with beta blockers, as marked small airway constriction with resultant severe impairment in gas exchange may occur in them, following smoking.\textsuperscript{243}

The other line of investigation is the elucidation of the action of cigarette smoke on pulmonary surfactant. This substance may have a significant role to play in maintaining the patency of small airways. In addition to the Type II alveolar epithelial cells which lie in the alveolar walls, surfactant is also thought to be produced by Clara cells which lie in the walls of the bronchioles.\textsuperscript{140,174} Reduction of Clara cell secretion either due to inactivation of the secretion itself, or due to damage to the secretory cells by the toxic constituents of tobacco smoke such as hydrogen cyanide, could increase the surface tension of the lining of the small airways and thus cause their walls to collapse.

Lastly, in view of the fall in blood basophil and
eosinophil counts soon after smoking, it would be of interest to undertake studies on the possibility of tobacco smoke acting as an allergen. Very few studies have been done on this aspect although contact dermatitis and various cardiovascular disturbances have been reported as allergic manifestations of tobacco smoking. A study of 834 patients, smokers and nonsmokers, suffering from bronchial asthma and chronic bronchitis revealed a highly significant increased frequency of positive skin tests against tobacco extract in smokers, regardless of whether asthma was present or not. Allergisation may therefore participate in the development of chronic bronchitis in smokers. It is also known that smoking or exposure to smoke from tobacco can aggravate the symptoms of the asthmatic patient.

However, in order to gain adequate information on the sensitivity of an individual to the immediate effects of cigarette smoke, one would have to test these effects on a large group of subjects, both nonsmokers and smokers. Such studies are handicapped for two reasons. Firstly, it is not justifiable to perform such studies on young nonsmokers for obvious ethical reasons. Hence, the studies would have to be restricted to smokers. The other drawback is that those subjects who are sensitive to the immediate effects of tobacco smoke are likely to have stopped
or reduced smoking due to symptoms, so that those who continue to smoke would be mostly those who are less sensitive.

The findings summarised above emphasise the need for initiating programmes to prevent people from starting smoking. Smoking has been described as a conditioned behavioural event with positive and negative reinforcers. The positive reinforcers are the pleasurable symptoms caused by the pharmacological effects of nicotine whereas the negative reinforcers are the impairment in one's own health, or knowledge pertaining to the health hazards of cigarette smoke. Smoking habits of parents, siblings and friends, and peer group pressure are the most important factors in starting smoking. Since the habit usually starts in teenagers, programmes to prevent individuals from starting smoking should concentrate on this age group. This may be more effective if objective evidence regarding the harmful effects is presented. However, health education of teenagers alone is not enough. The smoking habits of children and teenagers are influenced by those of adults, particularly by those of parents, teachers and doctors, whom they try to emulate. Therefore, adults should set an example not only by stopping smoking themselves, but also by actively discouraging young people from starting smoking.
An additional important reason for intensifying antismoking programmes is the fact that a much larger number of cigarette smokers than nonsmokers become smokers of marijuana and opium. Moreover, smoking is known to potentiate the harmful effects of other atmospheric pollutants such as industrial dusts and smog. 77,112,125

Attempts to protect those who continue to smoke despite all warnings, should also be an important part of these measures. Low risk cigarettes within the limits of consumer acceptability in which toxic components of the smoke such as carbon monoxide, nicotine, oxides of nitrogen, hydrogen cyanide, carcinogens, and other undesirable particulate matter are reduced, may be advocated. 77,81 Smoking without inhaling may also help to reduce the ill effects. 129

While many antismoking measures have been adopted in other countries, such attempts have not been undertaken seriously in India, although recently the Health Report issued by the Ministry of Health in 1975 requires a statutory warning regarding the health hazards of cigarettes to be displayed on cigarette packets. 144

While the role of tobacco smoking in the development of lung cancer is widely known, the part it plays in the etiology of noncancerous respiratory diseases, particularly chronic bronchitis is generally not fully
recognised. Although unlike lung cancer, the latter disease has a slow and smouldering course, it nevertheless leads to prolonged and severe morbidity with distressing symptoms. The contribution of the early small airway changes that occur in chronic bronchitis to the development of arterial hypoxemia and the consequent decrease in maximal oxygen uptakes even in asymptomatic smokers, has not been adequately recognised. A reduction in maximal oxygen uptake per se is a significant handicap to young smokers in their teens and twenties, as it limits their performance in competitive athletics particularly of the endurance type, as compared to their nonsmoking counterparts. Moreover, the decrease in work capacity in men of working age and the crippling nature of the established disease inevitably lead to severe and permanent disability in the individual affected. These also result in a recurring drain on the resources of the nation, in terms of medical care and loss of working time. Personal air pollution by tobacco smoking is thus a health hazard of major importance.