from Israel through the work of Professor K. J. Mann at Haddassah University Hospital has shown that 60% of the work presenting to the family doctor can be adequately performed by a well trained community nurse. The Health Commission of New South Wales is building medical and nursing centres, which will be used by family doctors, who will consult in them assisted by community nurses. In situations, however, where a family doctor cannot be attracted to a town, the community nurse will practise unaided, except by a visiting doctor. The residents of that town will come to identify the medical and nursing centre as the place from which their health care emanates. (This concept has been pioneered by A. E. Mooney, Esq., B.E.M., at Ardlethan in the Riverina.)

Nurses are also assuming more responsibility in hospitals, and it is not uncommon to see the nurse in North America accepting her own case load, particularly in paediatrics and intensive care units.

Beard, in his paper in this issue, also discusses the wastage of nurses who have finished their training. Revin’s classic work in England showed—and experience supports his view—that the greatest cause of wastage was unsympathetic supervision by charge nurses and executive nurses within the hospital. It is most encouraging nowadays to see how well matrons and directors of nursing are adjusting to the rapidly changing attitudes of the young nurses, who no longer accept the hierarchical management attitudes which applied previously in nursing.

Lastly, comment is necessary on the changes proposed for nurse education in Australia. Colleges of Advanced Education are to train some nurses, at least, in the future. Such training programmes will enable nurses to study together with other health professionals. In this way Australia is following the example of North America. One hopes that in doing this the training authority will have regard to the immense value the nurse obtains during her training through contact with the patient, and will recognize that as many hours as possible in the training course must be spent with patients, rather than in the classroom. Despite growing technological demands, nursing remains an art requiring skills that can be learned only through close association with people. Any diminution in these skills would be a sad loss for the community, particularly the sick and their relatives.

Finally, giving true recognition to the invaluable role nurses play and affirming their stature in the health services of this country may well be the factor that stimulates their rational use, and hence improves recruitment and prevents wastage, thereby overcoming the apparent shortage.

COMMENTS

THE GRAVE FACTS ABOUT SMOKING

It is now just over ten years since the Advisory Committee of the United States Surgeon General issued its famous report of January, 1964, which definitely linked cigarette smoking with lung cancer, and 20 years since scientific evidence indicating that cigarette smoking is a major health problem came to widespread public attention. As a result of the continuing growth of scientific evidence on the hazards of cigarette smoking and the educational programmes to disseminate this knowledge, millions of people in the United States have stopped smoking, and millions of others who would otherwise have taken up smoking have not done so. The evidence is now clear that people who have stopped smoking cigarettes have lower death rates from smoking-related diseases than those who continue to smoke. Several further reviews and reports were compiled in the years since 1964, and now, ten years later, a new document, The Health Consequences of Smoking, 1974,1 has been released. This confirms the evidence in previous reports that cigarette smoking is a serious health hazard and contributes to the development of various forms of cancer, cardiovascular disease and respiratory disease.

In reviewing this latest major report from the United States on the hazards of smoking, it is appropriate to draw attention to the special Supplement to this issue of the Journal. This Supplement, which contains material prepared by the Australian Council on Smoking and Health, is commended to our readers. It may well be read in conjunction with the following summary of the report from the United States.

Atherosclerotic cardiovascular disease is the leading cause of death in the United States, accounting for greater than 50% of annual deaths. Cigarette smoking is a major risk factor for the development of coronary heart disease (CHD), with male cigarette smokers running a twofold risk of dying from CHD compared to non-smokers. There is a dose-response relationship, and cigarette smoking acts both independently of and synergistically with the other two major risk factors, hypercholesterolaemia and hypertension, to produce these effects on CHD morbidity and mortality. The relative importance of cigarette smoking in the development of CHD in young men (less than 50 years old) is greater than that for any other risk factor. Cigarette smoke, carbon monoxide and nicotine induce some of the biochemical, anatomical and pathophysiological changes seen in CHD, but prospective epidemiological studies document that cessation of cigarette smoking results in reduced mortality from CHD. Most studies suggest that pipe and cigar smokers exhibit a slightly higher risk of development of CHD than non-smokers, but a significantly lower risk than cigarette smokers. The most recent research shows that cigarette smoking acts independently of and in conjunction with certain cardiac arrhythmias to increase the risk of mortality from CHD in men. Smokers also have a greater probability of dying from

CHD at an earlier age than non-smokers. Women who smoke cigarettes have a greater risk of sudden death from CHD than do non-smoking women. Experimental studies demonstrate that (i) the elevated levels of carboxyhaemoglobin seen in smokers may result in significantly decreased cardiac work performance and precipitation of ischaemic ECG changes and arrhythmias where clinical and subclinical CHD exists; (ii) nicotine acts indirectly to cause elevation of plasma free fatty acids; (iii) there are strong associations between cigarette smoking and development of peripheral vascular disease and atherosclerotic brain infarction.

Cigarette smoking has been identified as the major cause of lung cancer, which is the most common site of cancer in males in the 35 to 74 years age group, and cancer is the second leading cause of death in this age group in the United States. There is a dose-response relationship between cigarette consumption and the risk of development of lung cancer, the risk for smokers in general ranging from 7 to 14 times that for non-smokers, while male heavy smokers may have up to 20 times the risk. While the incidence of lung cancer in women is lower, there is similarly an association between cigarette smoking and lung cancer mortality in females. The most common types of cancer seen are oat-cell and epidermoid carcinomas, but there is some evidence that adenocarcinomas may also be associated with cigarette smoking. Certainly, autopsy studies have shown that changes in the bronchial mucosa which precede bronchocarcinoma are more common in smokers than in non-smokers. Cell and tissue culture studies have demonstrated that constituents of tobacco and cigarette smoke condensate may produce malignant transformation, and numerous complete carcinogens and cocarcinogens (tumour promoters) have been isolated from and identified in cigarette smoke condensate. There is also an increased risk of development of lung cancer in pipe and cigar smokers compared to non-smokers, with a dose-response relationship, but the risk is less than that of cigarette smokers. This lesser risk is consistent with differences in inhalation patterns of these two groups of smokers.

Recent epidemiological data suggest that the incidence of lung cancer in women continues to rise in correlation with the trend towards an increase in smoking among women. Data from experimental studies in animals suggest that chronic respiratory infections may enhance the carcinogenicity of components of cigarette smoke, as may alterations in the immune system. Cigarette smoke components induce aryl hydrocarbon hydroxylase activity in pulmonary alveolar macrophages, but the role of this in tumourgenesis or as a host defence mechanism is presently clear.

There is a markedly increased mortality from chronic obstructive pulmonary disease (chronic bronchitis and emphysema) for male smokers compared to non-smokers, and cigarette smoking is confirmed as the primary cause of chronic bronchitis and emphysema. Heavy cigarette smokers run a relative risk of mortality from chronic bronchitis ranging from 3 to 8 times that of non-smokers, and a relative risk of mortality from emphysema ranging from 8 to 20 times that of non-smokers, being a dose-response relationship. Both male and female smokers suffer from symptoms of chronic obstructive pulmonary disease (cough, sputum production, dyspnoea, etc.), with a dose-response relationship, more frequently than do non-smokers, and they also have greater impairment of pulmonary function than do non-smokers. However, cessation of smoking results in lower death rates from these diseases as well as improved pulmonary function and a decrease in the prevalence of pulmonary symptoms. Although air pollution may contribute to symptoms, cigarette smoking is far more important in producing respiratory disease, and occupational exposures to cotton fibre, asbestos and particularly coal dust act in concert with cigarette smoking. The smoke may impair the function of the pulmonary surfactant system, pulmonary clearance, ciliary movement and alveolar macrophages. Postoperative complications and spontaneous pneumothorax are more common in cigarette smokers. Pipe and cigar smokers have higher mortality rates from chronic bronchitis and emphysema than do non-smokers, but lower rates than those of cigarette smokers, while their prevalence of respiratory symptoms is higher than that of non-smokers. Those smokers who retain their cigarettes in their mouths continuously while smoking ("droopers") have a higher prevalence of chronic bronchitis than those who remove the cigarette from their mouths between puffs.

There is also an association between mortality from oral cancer and all forms of tobacco usage, which may act in concert with alcohol consumption to increase the risk of oral cancer, and this relationship also holds for cancer of the oesophagus. An association also exists between smoking and mortality from pancreatic cancer and cancer of the larynx, while metabolites of tryptophan, which are affected by cigarette smoking, can be carcinogenic in the bladders of mice.

Finally, on a brighter note, no firm relationship between stomach cancer and cigarette smoking has been established. Yet.

**POPULATION AND AUSTRALIA**

A COMMITTEE under the chairmanship of Professor W. D. Borrie of the Australian National University has now published, at the invitation of the Australian Government, the First Report on an inquiry into all aspects of population growth in Australia and into such matters as would contribute usefully to the formulation and application of national policies. Chapters XV to XVII, pages 543 to 706, deal with Australia's countries overseas with which Australia is likely to have relations, and many of the points made about them are not strictly relevant to Australia's development. Chapters I to IV and XI to XIII seem to be those relevant to medical observers.

In Chapter I is given a profile of the Australian population. The age distribution is almost entirely dependent on past fertility and migration with a notable deficiency of persons corresponding to the low birth rates of the 1930s. The concentration of the population in the cities is noted.

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