The Smoking Disease

The causative link between cigarette smoking and lung cancer has been verified in a dozen different types of study, confirmed in more than a score of countries, and amplified in hundreds of reports since E. L. Wynder and E. H. Graham in the U.S.A. and Richard Doll and (now Sir) A. Bradford Hill first established a thorough statistical basis for it. Links between smoking and many other diseases have since been detected with greater or less certainty. And reports digesting the research data have followed in profusion. They have undoubtedly influenced many people to give up smoking—especially doctors, who see its effects. But the enormous extent to which this habit continues, and especially its now fast growing prevalence among women, shows that information alone is not enough to deter many people from a course that may lead them to ill health in middle life and a premature death. Some 50,000 deaths a year in Great Britain may conservatively be attributed to the effects of smoking.

If the ill health and deaths that tobacco smoking causes or largely contributes to were the result of an epidemic infection, the Health Department would spare no efforts to eradicate the infecting agent. But it is fair to say that no government agency in any country has tackled the smoking epidemic with the vigour it devotes to, for example, an outbreak of smallpox. One reason for this may be that the diseases caused by smoking are regarded as self-inflicted. By taking thought, it is argued, we can avoid lighting a cigarette. Or can we?

To judge from the number of people who claim they tried to give up smoking but failed, the act of smoking seems commonly to be as much the symptom of a disease as does a smallpox papule. An appeal to reason is therefore unlikely to have much effect on people in the grip of an irrational, perhaps partly biochemical, mode of behaviour. But antismoking measures, whether they are special clinics, school lectures, or public advertisements, rarely get far away from the rational approach that comes naturally to the professional advisers who have hitherto inspired them. It may be significant that no large, national advertising campaign against smoking has been directed to the aim of showing that it is sexually unattractive and that to abstain from smoking is sexually rewarding. Yet this theme is familiar enough to professional persuaders.

The new report from the Royal College of Physicians of London, published this week, deserves to be widely read. It sets out the facts clearly and gives voluminous references to published papers. For the medical profession in particular it provides a useful summary of information on the diseases now known to be associated with smoking—especially of cigarettes, but pipes and cigars cannot be wholly excluded. Apart from cancer of the lung (squamous-cell and oat-cell cancers) the diseases for which the evidence shows a causative link with cigarette smoking include chronic bronchitis and emphysema, coronary-artery occlusion, angina pectoris, cancers of the mouth, pharynx, larynx, and oesophagus, cancer of the bladder, and pulmonary tuberculosis. Among patients with peptic ulcer those who smoke have a higher death rate than those who do not, and a mother’s smoking during pregnancy may retard the growth of the fetus.

Following an earlier report from the royal college, the present one brings the information up-to-date and makes some fresh suggestions to counter the smoking epidemic. Here it becomes less convincing. In particular the present report’s recommendation to establish smoking-control clinics, to be advised by a joint committee of the Health Department and the Medical Research Council, hardly seems realistic in view of the indifferent results achieved in the past by similar clinics with all the advantages of being staffed in the main by enthusiasts. The report makes sensible observations on the advice that individual doctors may give their patients and the example they set them, but many doctors have in fact been doing these things for years and the result has been little better than holding a deck-chair against a tidal wave. It is surely right, however, in criticizing a succession of Governments that have consistently refused the money to combat this epidemic by prolonged, contrivial action rather than brief Press campaigns, occasional posters, and snippets on television.

Though Governments depend on the votes of smokers as well as non-smokers, any administration that had the courage to attack this epidemic on the scale required might be surprised at the support it would receive from the smokers who are the unwilling victims of their craving and ashamed of the example they set their children. The twin fears at Westminster of antagonizing voters and losing revenue must give way to confidence in planning a national campaign against a form of
drug dependence on which young people are being continually hooked. At the same time more encouragement could well be given to research into the possibility of harmless smoking. Can it exist? A cigarette that gave the same or similar satisfaction as the tobacco-filled article but lacked its noxious effects would still be sociable and taxable. But it may prove to be a pipe dream.


Atypical Angina

In 1959 M. Prinzmetal called attention to an unusual form of angina pectoris in which the pain occurred at rest but was not related to physical exercise or emotional stress. Though not nearly as common as angina of effort, it is not a rare syndrome, and it has the same age and sex distribution. The pain generally lasts longer than in effort angina, and it is usually more severe, while the attacks are frequently recurrent in a cyclical and regular pattern. Prinzmetal referred to this syndrome as a variant form of angina pectoris. He noted that if myocardial infarction occurred in a patient with variant angina the pain usually disappeared—unlike that of effort angina. The electrocardiographic changes also differ; instead of the generalized ST depression of classical angina the variant angina ST elevation is accompanied by reciprocal depression in the standard leads. The occurrence of ST depression without reciprocal elevation in classical angina is explained by the diffuse distribution of areas of myocardial ischaemia. In variant angina the extent of the ischaemia is restricted to a discrete area supplied by a large, narrowed coronary artery, and Prinzmetal postulated that the syndrome is due to an increase in vascular tone. Quite a modest increase in tone in such a vessel could lead to complete though temporary obstruction. Angiographic studies in six patients with variant angina have confirmed this explanation by showing a single stenotic lesion of one major vessel without significant disease elsewhere.

The assumption that symptoms of variant angina are due to coronary spasm seems to contradict generally accepted concepts. Angina of effort develops only when there is severe myocardial hypoxia as a result of inadequate perfusion. In these circumstances the affected coronary vessels are already under the maximum physiological stimulus to dilate. This is probably true for classical angina, in which there is generalized disease of coronary vessels limiting their response to variations in tone and in which the ischaemic areas are scattered throughout the myocardium. Nevertheless, angina of effort as a result of myocardial hypoxia has been recorded in the absence of obstructive disease of visible coronary arteries. More recently a remarkable case of variant angina has been reported in which myocardial hypoxia was confirmed despite the absence of any coronary artery disease demonstrable by angiography. In this patient the pain occurred at rest or during sleep, but not with exercise. Marked ST elevation was associated with the pain, and episodes of venicular tachycardia and even venicular fibrillation were recorded. After an episode of syncope due to complete heart block associated with pain a pace-maker was inserted. No abnormalities of haemoglobin-oxygen dissociation were found, and the basis for this patient's variant angina remained unexplained.

It is possible that in these patients vascular disease is present but not seen, for the state of the microcirculation cannot be assessed by angiography. This is, however, unlikely in view of reports of such patients who have died after infarcts with no apparent disease of the large or small coronary arteries at necropsy. It is possible that myocardial ischaemia might arise from impaired oxygen diffusion or inappropriate oxygen utilization by the myocardial cells. Abnormalities in dissociation of oxygen from haemoglobin have been described in young women with angina and normal coronary angiograms. Furthermore, the energy needs of the myocardium may be abnormal. These are largely determined by the tension of the muscle wall of the ventricle, which is governed by the pressure within the cavity of the ventricle and the mean radius of the heart chamber. It has recently been suggested that the beneficial effect of nitroglycerine in angina is related to the reduction in ventricular volume and hence myocardial oxygen consumption associated with its use.

The pathogenesis of angina is more complex than a mere obstruction to perfusion of the myocardium by atheromatous narrowing of the coronary arteries. The lack of correlation between the degree of atheroma and the presence of clinical disease suggested this many years ago. Vascular spasm, changes in the microcirculation, and physicochemical disturbance within the myocardium are certainly relevant, but their frequency and importance await further elucidation.

Relapsing Polynephropathy and Corticosteroids

Though many cases of acute idiopathic polynephritis, or the Guillain-Barré syndrome, have been reported, clearcut criteria for its diagnosis have proved difficult to define. In some patients the interval between the onset of symptoms and maximum neurological loss may be only a day, while in others apparently identical in every other way the interval may be several months.

G. Guillain, J. A. Barré, and A. Strohl divided their cases into those with a relatively acute onset and the atypical forms. L. D. Osler and A. D. Sidell suggested that the diagnosis should not be made when the interval exceeded two weeks. However, H. Ravn found that the interval had no effect on the later course of patients with this syndrome. The longest interval in his series of 127 patients was 45 days. Similarly, D. E. Pleasure and colleagues, accepting cases with an interval of up to two months, found no difference in prognosis between those with a shorter and those with a longer interval. When the symptoms progress for more than