Cowardice about Smoking

Sir Keith Joseph's statement on the Government's proposed action on tobacco smoking (p. 735) will come as a disappointment to those who had hoped for a firmer lead. The warning to be printed on all cigarette packets, "Smoking can damage your health", is some advance, but its equivocation is typical of the weak approach to a serious problem shown by successive Governments in the last ten years. An outside observer might conclude that the recent statement was a panic response to a new situation: in fact it comes 21 years after the classical indictment of the cigarette,1 14 years after a report by the Medical Research Council,2 and 9 years after the first report of the Royal College of Physicians,3 and at the end of a decade of warning by the Department's own Chief Medical Officer and of pressure by M.P.'s on successive Ministers of Health.

In tackling the dangers of smoking any Government has to steer a difficult course between avoiding restrictions on individual freedom and doing its utmost to discourage the habit. Yet the fact is still ignored that smoking is a socially contagious disease: young people start the habit because their parents, teachers, or teenage idols do it, or because they are persuaded by advertisements emphasizing adulthood or agreeable sexual attributes. Little, moreover, is done to deter them; it is only too easy for teenagers to buy cigarettes from shops or slot-machines, and the law on smoking under age is poorly enforced. Why is no legislation proposed for stopping placing slot-machines in public places (p. 735)? Why are there no proposals to prohibit cigarette advertising further? Why are there still comparatively few restrictions on smoking in public places—a feature where Britain seriously lags behind much of the Continent?

Statements by successive Ministers of Health in the last ten years have typified this cowardice about any effective action on smoking.4 In 1962 Mr. Enoch Powell said that the suggestions of the first report by the Royal College of Physicians were under consideration by the Government; in 1966 Mr. Kenneth Robinson—who had pressed the Government particularly hard over smoking when he was in opposition—said the same about proposals on limiting it in public places; while in 1968 Mr. Richard Crossman said that the preparation of adequate measures was "by no means easy." Three years later all Sir Keith Joseph can offer the country besides the agreed warning—which may never be printed if Sir Gerald Nabarro does not withdraw his Private Member's Bill—is a standing scientific liaison committee and some more money for the Health Education Council. In the meantime the Government is considering how to tackle the "very difficult question" of how to dissuade the young from starting to smoke. With a justified sense of déjà vu, doctors might be forgiven for asking whether the Government will still be shilly-shallying in 1981.

Nervous Disorders and Thorn Cells

In 1950 F. A. Bassen and A. L. Kornzweig4 described in two siblings of consanguineous parents a morphological malformation of the red blood cells. These cells had "protoplasmic projections varying in size and shape." One of the siblings also presented clinical features thought to conform to Friedreich's ataxia with retinitis pigmentosa. K. Singer and colleagues,5 who two years later described similarly misshapen erythrocytes in a child of parents who were second cousins, considered that the "protoplasmic projections" bore a resemblance to thorns and called this abnormal type of red cell "acanthocyte" (acanthos—thorn in Greek).6 This name was later corrected to "acanthocyte."

The large, spiculate projections of the acanthocytes are different from the regularly spaced, small protuberances of the normal erythrocyte becoming crenated by exposure to hypertonic salt solution. When the patient of Singer and colleagues was reviewed six years later, "a phenomenally low serum cholesterol value" was discovered, and this suggested that "an inborn error of fat metabolism produces a harmful effect on erythrocytes."4 Bassen and Kornzweig reviewed their cases in 1957 and found that both siblings now presented similar neurological abnormalities.3 They observed that all

4 see British Medical Journal, 1962, 1, 808; 1966, 2, 1540; 1968, 2, 652.
three cases of acanthocytosis known at the time were associated with coeliac disease in early life, progressive ataxic neuropathy, and retinitis pigmentosa. Shortly afterwards another case also presenting all these features was reported.3 In 1960 a 17-month-old girl with steatorrhoea was found to have acanthocytosis.4 An exceptionally low total blood cholesterol led to detailed lipid studies, which disclosed an absence of serum betalipoproteins. Since then a number of families have been described showing acanthocytosis, plasma betalipoprotein deficiency, a neuromuscular disorder characterized most often by ataxia of gait, trunk, and extremities, proprioceptive sensory loss, tendon areflexia, Babinski signs, kyphosis, and retinitis pigmentosa. The additional features are steatorrhoea and low levels of blood cholesterol.5,6

In 1967 a new hereditary syndrome of acanthocytosis was recognized in which neither the betalipoproteins nor blood cholesterol were reduced and neurological symptoms were characterized by choreiform movements of the Huntington's type and muscular atrophy of the Charcot-Marie-Tooth type (wasting of peripheral limb muscles).14-17 E. M. R. Critchley, who encountered one such family in Kentucky, has now with colleagues described a case in the United Kingdom presenting a number of these features.18 The dominant symptom was a severe persistent semifunctional activity of the facial, mandibular, and lingual muscles. He suggests that "all patients with oro-facial tics and involuntary movements" should have the peripheral blood examined for acanthocytes. Since these neurological phenomena are common in the elderly, such screening should, perhaps, be restricted to younger people.19 Thus acanthocytosis may be associated with two groups of neurological disorders in only one of which deficiency of serum betalipoprotein is to be found.

One year before the publication of the first paper by Bassen and Kornzweig erythrocytes were described "having one or more large spiny projections"—that is, having identical morphological features as acanthocytes.20 S. O. Schwartz and S. A. Moto found these cells in a high proportion of cases of uraemia, carcinoma of the stomach, and bleeding peptic ulcer. They designated these red cells as "burr" cells, and they are now known to occur in a number of conditions, some of which are associated with haemolysis. Recently they were also found in hypothyroidism.21 Since acanthocytes and "burr" cells are morphologically indistinguishable, one of these terms is redundant. A "Medical Registrar General of Terminological Births and Deaths"—an office suggested by Richard Asher—might have resolved this vocabulary dilemma.22 At present it is by no means certain whether this type of poikilocytosis is due to some abnormality of the plasma components23 or to an abnormality of the surface membrane of the erythrocytes.24 When exposed in vitro to a non-ionic detergent, these abnormal cells rapidly assume the biconcave circular shape of normal erythrocytes.25

Bleeding Duodenal Ulcers

Most experienced surgeons in Great Britain have the impression that duodenal ulcers are not like they used to be. The enormous indurated penetrating ulcer, burrowing into pancreas, liver, gall bladder, or even the anterior abdominal wall is now rarely encountered in the operating theatre, where the usual finding is a relatively shallow ulcer on the posterior duodenal wall. As well as this the complications of duodenal ulcer, stenosis and perforation, have also become less common. All this may be because patients are being referred to surgeons at an earlier stage of the natural history of the disease rather than that the pathology of the ulcer itself is undergoing change. However, the bleeding from duodenal ulceration remains the single most common cause of haemorrhage from the upper alimentary tract in this country, so that its correct management and prognosis continue to be important practical problems.

K. F. R. Schiller, S. C. Truelove, and D. G. Williams1 have reported an important study of 2,149 emergency admissions for haematemesis or melaena in a 15-year period in the Oxford region. About one-third of the patients had duodenal ulcer, and, interestingly enough, the fatality rate remained virtually constant throughout this period. Deaths due to haemorrhage from a duodenal ulcer amounted to 5-5%, of cases as compared with 8-9% due to haemorrhage from all causes in this series. The highest mortality occurred in patients with oesophageal varices (43%), and bleeding from carcinoma of the stomach and from stomal ulcer also carried high fatality rates. Poor prognosis was associated with advanced age of the patient, a low blood pressure, gross anaemia on arrival in hospital, and continued bleeding after admission. These facts are generally accepted, so that it is usual to advise operation for patients who continue to bleed or in whom bleeding recurs while in hospital, especially if they are elderly, since these are the patients who are especially prone to die unless the source of haemorrhage is treated operatively.2,3

References

3 Druze, G., Revue d'Hematologie, 1959, 14, 3.