follow-up, however, suggests that history may be repeated; Ross and Gunning have now reported calcification extending from the aortic wall on to the valve cusps, producing stenosis of the graft.[2 8] The present incidence of thrombosis and embolism associated with prosthetic valves may, it seems, be being avoided only at the cost of eventual stenosis of a graft.

A further development in this field is the use by Ross of the patient's own pulmonary valve to replace his destroyed mitral valve.[2] Such an autograft should persist as living tissue and so avoid the hazard of eventual calcification. The problem then becomes how best to replace the excised pulmonary valve.

It is not easy for a cardiac surgeon to decide which of the artificial mitral valves he should use today, and this explains his reluctance to advise mitral valve replacement unless the patient's disability is severe. Time will probably narrow his choice of valve to an improved prosthesis or a pulmonary autograft.

Amines and Migraine

Like its predecessor,[1] the second migraine symposium, which was held in London on 24 November, was a useful review both of the clinical and of the biochemical and pharmacological aspects of the condition. Once again the role of amines in migraine was one of the principal topics discussed. Tyramine and dopamine, which are present in certain foods, were reported to be able to induce severe headaches in patients taking monoamine oxidase inhibitors.[2-4] Moreover, it was pointed out that many patients with migraine exclude from their diet foods such as chocolate and cheese, which contain varying amounts of tyramine and other amines. Patients who were apt to develop migraine if they ate these foods had been shown[5] to develop a migrainous headache within 24 hours if given tyramine by mouth. Normally tyramine is prevented from gaining access to the general circulation by the action of monoamine oxidase in the gut and the liver. Hence a possible explanation for the production of migraine by dietary factors might be a relative lack of monoamine oxidase in these sites. There is no evidence of this, however, and histochemical staining of temporal artery biopsy specimens in an attack of migraine show a normal content of monoamine oxidase in the vascular wall.[6] On the other hand, in-vitro studies on the adventitial layer of temporal artery taken from patients during an attack of migraine have shown a greater uptake of catecholamines than with temporal artery removed from other patients undergoing craniotomy for cerebral tumours.

When catecholamines and ergotamines are added to human cerebral arterioles removed at craniotomy there is no response, though when either of these agents is added to somatic arterioles definite vasoconstriction occurs. This finding does not support the accepted idea that the aura of migraine is due to ischaemia as a result of vasoconstriction[7]—unless vasoconstriction is a property of migrainous cerebral arterioles and not of normal ones. The therapeutic effect of ergotamine, however, is still thought to be due to its vasoconstrictor activity on the extracranial arteries,[8] and the development of new compounds for the treatment of migraine is based on their in-vitro effects on blood vessels. Other important properties of any new drug are that its effects on non-vascular smooth muscle, such as the uterus, should be minimal and that it should not induce nausea.[9]

The relationship of migraine to menstruation and to pregnancy suggests that hormones may also play a part in the condition, directly or indirectly. The results of treatment with oestrogens have been disappointing,[10-12] and androgenic steroids have also been unsuccessful.[12-16] Parenteral injections of progestogens are sometimes more effective, though the effect is of short duration. Giving progestogens by mouth, on the other hand, has had disappointing results,[17] despite some benefit having been reported previously.[18] Migraine remains an enigma, but further work on these biochemical and pharmacological aspects may help in its elucidation.

Life after Death

Man's response to the approach of death has a perennial fascination. In part this may rest in a false hope that by understanding its immediate antecedents we may come to know something of the nature of death. Sometimes the threat comes from outside—for instance, earthquake, battle, falls from great heights—and can be apprehended clearly. Men vary in their response to these experiences[1]: some remain calm and can continue to perceive their predicament in detail, while others develop fear of such intensity that awareness becomes clouded and behaviour incoherent, and yet others develop amnesia and other hysterical symptoms.

Illness is a threat to life which comes from within. The patient who has a serious heart disease is in a psychological as well as a physical predicament. He is aware of his heart to a greater extent than any of his other vital organs; he is aware of it as something which moves and thus epitomizes life. This internal threat to life cannot be countered as can many threats to life from outside, but at the same time the patient has not the reassurance that matters are entirely out of his hands, since he recognizes that intemperate activity endangers him.

R. G. Druss and D. S. Kornfeld[2] have recently described the psychiatric sequelae of cardiac arrest and compared these with the response of patients to other forms of heart disease. The only difference found was that patients whose hearts had stopped subsequently experienced nightmares of violence—as do those who have been wounded in battle. The significance of these nightmares is not yet known. They may be an

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