1. Sympathetic impulses from the posterior hypothalamus might descend through the brain-stem and spinal cord to the stellate and cervical ganglia and back to the cerebral vessels causing a constrictive effect which was not balanced by corresponding parasympathetic impulses.

2. Injury to the hypothalamus might cause liberation of various vasoactive chemical agents into the cerebrospinal fluid.

3. Hypothalamic injury might stimulate a widespread sympathetic discharge, especially affecting the adrenal medulla, increasing the levels of circulating catecholamines. This was the most likely mechanism. Subarachnoid haemorrhage, by damaging adrenergic nerve endings but not alpha adrenergic receptor sites, might cause denervation hypersensitivity of the intracranial arteries to catecholamine circulating in the blood or cerebrospinal fluid.

Any one of these mechanisms might serve as the initial stimulus to intracranial arterial spasm. The resulting biochemical and ultrastructural changes might outlast the duration of the stimulus by a number of days. Furthermore, such a stimulus probably acted in addition to, or synergistically with, the effects of blood in the intracranial subarachnoid space. Finally, since intracranial vasospasm was most marked in the anterior part of the circle of Willis, such spasm might itself cause further ischaemic injury to the hypothalamus, thus perpetuating the period of spasm.

TWENTY-FIVE YEARS' EXPERIENCE WITH MIDDLE CEREBRAL ANEURYSMS

JOHN GILLINGHAM (Edinburgh) considered that the real challenge today in endeavouring to prevent recurrent haemorrhage from middle cerebral aneurysms was the avoidance of serious morbidity. Important advances in management had been the recognition of the importance of small warning haemorrhages, careful timing of angiography, production of low intracranial pressure and moderately low arterial pressure during anaesthesia and dissection of the aneurysm sac, the use of magnification methods, and better techniques of clipping or investing. The neck of the sac was particularly vulnerable after recurrent haemorrhage and was sensitive to manipulations which would cause or aggravate vasospasm. The author preferred complete investment of the sac with fine mesh gauze cotton to clipping. Magnification might lead to more gentle dissection but the outcome of operation was likely to be poor if there were too much manipulation. The series under review consisted of 81 consecutive cases. Operation had been carried out on or about the seventh day if the patient's condition were sufficiently satisfactory. The overall operation

mortality was five out of the 81 cases and all of these were in patients placed in Botterell grades 3, 4, or 5. One patient died from recurrent haemorrhage 24 hours after operation, one had a thrombosed middle cerebral artery after fracture of an atheromatous plaque, one died from an unrelated cause, one had fatal cerebral ischaemia and swelling due to myocardial insufficiency five days after surgery, and one died of recurrent haemorrhage two months after operation. There was an early return to normal activities in 35 of the 41 grade 1 cases, in seven of the nine grade 2 cases, in seven of the 27 grade 3 cases, and in none of those placed in grades 4 and 5. The best results were in those who underwent surgery after only one haemorrhage. Patients without hypertension or vascular disease fared better than did those with these conditions. The four late deaths in the grade 1 cases were due to contralateral middle cerebral artery thrombosis in one patient and unrelated causes in three. The two late deaths in grade 2 patients were the results of unrelated illnesses. The one late death in a grade 3 patient who was severely hypertensive was due to recurrent haemorrhage from an aneurysm sac. The incidence of epilepsy was 13.7%. It was concluded that technical factors leading to morbidity and mortality were excessive manipulation during dissection leading to haemorrhage with or without spasm, kinking of a major arterial trunk or its branches by a clip, inadequate investment, fracture of an atheromatous plaque, recurrent arterial spasm and ischaemia, or extradural haematoma. Favourable prognostic features are the early referral of grade 1 patients, operation within 10 days of the first haemorrhage, and minimal interference during dissection of the aneurysm sac.

THE 'A' PRINCIPLE—A NEW APPROACH TO THE MANAGEMENT OF INTRACRANIAL ANEURYSMS

W. J. ATKINSON (Haywards Heath) drew attention to patients whose long-term follow-up after successful aneurysm surgery demonstrated subtle personality changes interfering with the life of the patient and his family. In the atherosclerotic patient the pulse wave was transmitted directly to the aneurysm and was not 'taken up' by the expansion of the arterial wall. It was suggested that efforts to prevent high systolic pressure reaching aneurysms would prevent subsequent haemorrhage, and that this could be done by causing the arterial wall proximal to the aneurysm to expand and so take up the systolic pressure. Clamps were placed on the internal carotid artery in the neck for periods varying from 15 to 60 minutes to contuse and break up the muscle and intimal layers of the arterial wall. Six patients had been treated in this way with one death. Damage to the wall of the internal carotid artery may produce a false aneurysm