bookbinders. A further series of photographs was then shown of dissections of the upper brain-stem showing the 5th nerves and the structures in the vicinity of the apex of the petrous bone. Serial photographs and radiographs had been taken while the crushing was in progress and again at the end of each procedure when the crushing force had been released. An attempt was then made to correlate the findings in these experiments with the clinical features of six patients with clinical and radiological evidence of petrous dislocation. It was concluded that, because of certain types of deformation of the base of the skull, the petrous bone might be dislodged in a posteromedial direction from its normal position due to opening of the petrosphenoid and petrosquamous sutures. As a result of this bony displacement, the 5th and 6th nerves might be damaged by stretching or by involvement in the fracture line. Extension of the fractures laterally into the middle ear produced deafness and facial weakness and the function of the temporomandibular joint might be disturbed by disruption of the petrosquamous fissure in the glenoid fossa. Medially the displaced petrous apex might damage the carotid artery and cause a carotid cavernous fistula. It was suggested that this syndrome and its variants might occur more frequently than had been hitherto suspected.

SURVEY AND FOLLOW-UP OF 225 CONSECUTIVE PATIENTS WITH A DEPRESSED SKULL FRACTURE

R. BRAAKMAN (Rotterdam) had undertaken a retrospective study of cases of depressed fracture of the skull who had been admitted to his department during the past 10 years. He included only those patients admitted within 48 hours of the accident and assessed their present condition in relation to the injury. The 225 cases under review were also compared with the 400 Glasgow cases reported by Miller and Jennett in 1968.

As in the Glasgow series 85% of the cases were male and of the 196 survivors information was available about 177; 44% were under 16 years of age. Only 14% of fractures were classified as closed, whereas Lewin had found closed and compound fractures in equal numbers. In this, and also regarding dural penetration, the Rotterdam and Glasgow figures were similar.

The incidence of epilepsy, both early and late, was less than in the Glasgow series, although the mortality rate was significantly higher. In relation to practical management, as a result of this study, they felt that bone fragments should usually be replaced. There were five infections in 109 cases with replacement and the same number in 56 cases where the fragments had been completely removed. Antibiotics were administered to some cases but not to others without any apparent plan or, as far as could be seen, any influence on the incidence of infection in the two groups.

REFERENCES

Kriss, F. C., Taren, J. A., and Kahn, E. A. (1969). Primary repair of compound skull fractures by replacement of bone fragments. J. Neurosurg., 30, 698.

Miller, J. D., and Jennett, W. B. (1968). Complications of

depressed skull fracture. Lancet, 2, 991.

HEMICRANIECTOMY IN THE TREATMENT OF ACUTE SUBDURAL HAEMATOMA

J. RANSOHOFF and V. BENJAMIN (New York) gave an account of their experiences of a regime of treatment for patients with acute subdural haematoma demanding surgery for the preservation of life within the first 24 hours after injury. They referred to the very high mortality reported in this type of head injury, which, in their own department, previously amounted to 75% with very few patients restored to normal life. Death had resulted from brain-stem compression, torsion, and secondary haemorrhage, but, at necropsy, few had shown what had been regarded as primary brain-stem lesions. General factors over which the surgeon had no control were:

- 1. The severity of the initial brain injury.
- 2. The rapidity of haematoma expansion.
- 3. The age and poor general condition of the patient. Factors which the surgeon should be able to influence were:
 - 1. Delay in diagnosis and treatment.
 - 2. Inadequate removal of clot and pulped brain.
 - 3. Failure to control haemorrhage.
 - 4. No provision for the accommodation of secondary cerebral oedema.

Diagnostic evaluation, including emergency cerebral angiography, proceeded simultaneously with general resuscitation, intubation, and respiratory assistance when necessary and the use of intravenous dehydrating agents (mannitol, 2 g/kg, intravenously). Operation was undertaken immediately on patients shown by angiography to have large, unilateral subdural haematomas. Essentially, this consisted of the removal, using the air drill, of a very large fronto-temporo-occipital bone flap through a skin incision from the glabella to the inion and thence laterally. The squamous temporal bone was removed to the base of the skull. An equally large dural flap was hinged medially. Clot and pulped brain (only) were removed and haemorrhage from bridging veins and lacerated brain controlled. Subsequently electrolytes and blood gases were controlled and tracheostomy performed in 48 hours if necessary. Corticosteroids and anticonvulsants were administered.

Twenty patients had been so treated during a two-year period. On admission 10 showed bilateral and five unilateral decerebration, and five made no response to stimulation. There were eight deaths, one coma vigil, and one hemiplegia. Eight patients (40%) returned to their pre-accident employment and two made a partial recovery. There were no survivors over the age of 65, with a haematoma larger than 500 ml., when treatment had been delayed or when in the immediate preoperative period both apnoea and bilaterally dilated and fixed pupils were present.

STEREOTAXIC TREATMENT OF PAIN

M. P. A. M. DE GROOD (Tilburg) outlined current views on the three main afferent pathways for pain.

1. The classical spinothalamic tract terminating in the posteroventral thalamic nuclei. Lesions in this group of