MANIPULATION HEMIPLEGIA
AN UNTOWARD COMPLICATION IN THE SURGERY OF FOCAL EPILEPSY

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No professional man gets greater pleasure from the record of his successes than a surgeon. How delightful when the uninformed refer to them as miracles! And how inaccurate! But no one regrets his failures more. If there is a further clause that the modern surgeon might add to the Oath of Hippocrates, it is this:

“I will faithfully record and analyze my failures in the care of the sick, seeking the cause so that those who follow may be warned of danger.”

This is such an analysis. The idea that manipulation, in the neighborhood of certain arteries, is an adequate cause of sudden hemiplegia is a tentative hypothesis. It is one that has forced itself upon us gradually during years of work in a new field. Others will explain it and verify it, or find another cause for this untoward complication of surgery in deep Sylvian areas of the brain.

From time to time during the decade that followed 1930, focal cerebral seizures were treated by excision of portions of the temporal lobe by one of us (W.P.). But realization of how many epileptic patients were suffering from temporal lobe seizures came only gradually. The development of modern surgical therapy for this group of patients dates from the mid-nineteen forties. This advance was made possible when the cause of lesions of the temporal lobe became apparent and assistance from electroencephalographers and neuroradiologists was at hand.

The results were in general encouraging, sometimes brilliant. Occasionally, associated abnormality of behavior disappeared. But no attempt will be made to assess the results. This has been done statistically elsewhere.† All of these patients had tried medical therapy faithfully before surgery was recommended. About half of them now consider themselves cured, others greatly benefitted.

The distressing complication of hemiplegia or hemiparesis described here made its appearance with increasing frequency as the removals became more extensive. Consequently the first author (W.P.) feels bound to report his untoward experiences in the hope that analysis may bring an understanding that will keep us all from such unhappy pitfalls. It may be pointed out at once that there is only a single instance of this complication when the operation was restricted to simple partial temporal lobectomy. In all the other cases the complication occurred when, under the guidance of repeated electrograms, the excision of epileptogenic gray matter was extended across the fissure of Sylvius.

MATERIAL

The second author (R.L.) has reviewed all operations carried out in the temporal regions by W.P. from 1948 when the first hemiparesis occurred through 1955. During

— See Penfield,9 also Penfield and Jasper.14 A general follow-up analysis of results in all cases of focal epilepsy treated by Penfield and his associates during a 6-year period, 1945–1950, was reported by Penfield and Paine.15 Follow-up analyses of results in temporal lobe epilepsy were reported by Penfield and Flanigin,13 and by Rasmussen and Jasper.17
this period 161 such operations were performed; hemiparesis developed in 8 of these, an incidence of 5 per cent. In some cases the paralysis has since cleared completely, so that the disability was serious in 23 per cent. The hemiparesis has commonly been associated with complete homonymous hemianopsia, and with some degree of aphasia in the 2 cases of operation on the dominant hemisphere.

The third author (T.R.) has added a final section on the surgical technique including the precautions he believes important to avoid the complication. He has now carried out just over 110 operations for temporal lobe epilepsy (not included in this series) without this complication. In about half of his cases the excision was simple lobectomy. In the others the removal was extended across the Sylvian fissure to include epileptogenic tissue beyond the temporal lobe.

TEMPORAL LOBE EPILEPSY

A new field of therapy has been opened by recognizing, as temporal lobe seizures, the epileptic attacks that are caused by local discharge arising in one temporal lobe or in the adjacent gray matter deep within the Sylvian fissure, including the insula and bordering areas of frontoparietal cortex. Although focal attacks of this type have been referred to as temporal lobe seizures, it would be more descriptive, anatomically, to call them temporo-Sylvian seizures.10

The operation of radical excision has met with gratifying success and in the majority of cases the cause proved to be partial atrophy and sclerosis of the cortex of the temporal lobe. This abnormality is found most often on the mesial surface and in the superior surface of the temporal lobe that lies within the Sylvian fissure and circular sulcus. We have given this lesion the name incisural sclerosis, because it is evidently produced by compression of the arteries that cross the incisura of the tentorium. The lesion most often dates from birth. This conclusion is borne out by the frequency of radiographic evidence of smallness of the middle cranial fossa that contains the affected lobe. In spite of that fact, however, the attacks may not make their appearance until the second or third decade of life.

The mechanism of production of the lesion is presumed to be as follows: The cerebral anoxemia produced by passage of the head through the birth canal is rendered more severe in the incisural zone by herniation of the inferior mesial margin of the temporal lobe through the incisura of the tentorium. Herniation, of course, injures the involved cortex directly, but it also embarrasses the flow of blood through the arteries that cross the edge of the tentorium (anterior choroidal, also lateral branches of posterior cerebral arteries). Arterial compression is probably the cause also when abnormality is present in the insula and the adjacent parietal and frontal opercula and the orbital surface of the frontal lobe.

SURGICAL EXCISION OF EPILEPTOGENIC CORTEX

Removal of the anterior 5 or 6 cm. of the temporal lobe, including all of the inferior mesial surface (uncus, amygdala and hippocampal gyrus) may result in an upper quadrantic homonymous hemianopsia which often is incomplete. This is a visual defect that most patients do not discover, unless it is drawn to their attention. If the removal extends back 7 or 8 cm. from the anterior end of the middle fossa, the hemianopsia usually involves the lower quadrant as well and is then usually noticeable by the patient. Simple removal of the temporal lobe does not interfere with motor function in any way.

In the 8 cases to be reported, paresis or paralysis occurred in the opposite arm and leg during dissection of a very deep scar. In 6 cases it appeared to occur during the dissection near the middle cerebral artery. In no case was there hemorrhage from, or ligation of, a major artery. The probable mechanism will be discussed after presentation of the details of the cases.

CASES

Case 1. A.B., a woman aged 37, had had a brain abscess at the age of 6. It was drained successfully and later she began to have focal cerebral