CEREBROSPINAL FLUID ENZYME STUDIES
AN AID IN THE SURGICAL MANAGEMENT OF INTERNAL CAROTID ARTERY OCCLUSION

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Recently, attempts have been made to alleviate occlusive disease of the internal carotid artery by reconstructive surgery or a shunting procedure. The results of such therapy are difficult to evaluate, since a certain number of patients with thrombosis of the internal carotid artery recover spontaneously. Moreover, the maximal time interval between the onset of symptoms and operation that is compatible with a favorable result is not clearly defined. Lyons and Galbraith suggested that a subclavian-internal carotid shunt was of value in relieving neurologic symptoms if performed before a cerebral infarction was developed.

It is difficult, in many cases, to differentiate clinically between intermittent cerebral ischemia without infarction and infarction. An objective laboratory determination that aided in this differentiation would be of value in selecting those patients with internal carotid artery disease who might best benefit from surgical treatment. The measurement of certain enzyme activities in the cerebrospinal fluid may constitute such a prognostic aid.

We, and others, have reported elevated glutamic oxalacetic transaminase and lactic dehydrogenase activities in the cerebrospinal fluid of patients with cerebrovascular disease. The levels of these enzymes in the cerebrospinal fluid appeared, in several cases, to parallel the amount of brain-tissue destruction observed at autopsy. It was thought that serial determinations of glutamic oxalacetic transaminase or lactic dehydrogenase activity in the cerebrospinal fluid might indicate the occurrence of a cerebral infarction, and foretell the end result of a surgical attempt to re-establish the circulation in a case of internal carotid artery occlusion. Accordingly, the cerebrospinal fluid enzyme activities of 3 patients operated on for thrombosis of the internal carotid artery were determined, and the relationship between the changes in these activities and the clinical courses will be the subject of this report.

MATERIALS AND METHODS

Cerebrospinal fluid was removed by lumbar puncture from each patient as soon after admission to the hospital as practicable, and before arteriography. The lumbar puncture was repeated every few days, and occa-

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CEREBROSPINAL FLUID ENZYME STUDIES

sionally daily if warranted by a change in the patient’s hospital course. Each sample of cerebrospinal fluid studied had a normal cell content and a negative serologic test for syphilis.

The glutamic oxalacetic transaminase and lactic dehydrogenase activities in the cerebrospinal fluid were each determined by spectrophotometric methods at 37°C. These methods have been described in detail elsewhere. Normal glutamic oxalacetic transaminase activity, as determined in this laboratory, averaged \(43 \pm 12\) \(\mu\text{M.}\) of oxalacetate formed per hour, per 100 ml., ranging from 25 to 62. Normal lactic dehydrogenase activity ranged from 31 to 87 \(\mu\text{M.}\) of lactate formed per hour, per 100 ml., with a mean of 68 \(\pm 15\).

CASE REPORTS AND RESULTS

Case 1. The first patient studied was a 59-year-old right-handed man who was admitted because of the abrupt onset of complete aphasia and right hemiparesis. One year before, he had suffered an almost identical attack, from which he made an excellent recovery.

Examination revealed an obese, aphasic man who was unable to move his right extremities. There was a marked facial weakness. Lumbar puncture yielded clear cerebrospinal fluid under a pressure of 150 mm. Percutaneous left carotid arteriography disclosed a block at the bifurcation of the common carotid artery.

Surgical exploration of the left carotid system, 12 hours after the onset of symptoms, revealed a firm thrombus in the common carotid artery at its bifurcation. The artery was opened, and the clot was found to extend into the internal carotid throughout its entire length in the neck. Since the thrombus reached at least to the foramen lacerum, no corrective procedure was thought possible.

The postoperative course was uneventful. No improvement was noted in either the hemiparesis or the aphasia. Periodic re-examinations conducted during the following 9 months revealed no change in his neurologic deficit.

The cerebrospinal fluid enzyme changes are shown in Fig. 1. It will be noted that the initial level of lactic dehydrogenase activity was high, and reached a maximum on the 5th day, then gradually declined. The glutamic oxalacetic transaminase activity, after rising slowly in the first week, had not yet reached a peak value on the 15th day. The cerebrospinal fluid protein varied between 35 and 45 mg. per 100 ml., in a random fashion.

![Fig. 1. Case 1. Enzyme activities of cerebrospinal fluid. S indicates time of surgery. The upper limits of normal for glutamic oxalacetic transaminase and lactic dehydrogenase are 62 and 87 \(\mu\text{M.}\) respectively.](image-url)