SEROUS MENINGITIS*

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Neurologists and neurosurgeons have long been troubled by a heterogeneous group of patients who showed evidence of increased intracranial pressure but who proved not to have space-occupying lesions. Some of these patients have a history of an infection contiguous to the intracranial cavity in ears, mastoids, or sinuses. They are suspected of having a brain abscess. A few have had an injury to the head and are thought to have subdural hematomas. Others have no history of infection or injury and are considered to have brain tumors.

These cases have been variously referred to as "pseudo-tumors," serous arachnoiditis, serous meningitis, chronic arachnoiditis, toxic hydrocephalus, otitic hydrocephalus, meningitis sympathica, aseptic meningitis and so on. The wide variety of terminology clearly indicates the general confusion regarding them.

Quincke\textsuperscript{10} reported ten cases of serous meningitis and emphasized the spinal fluid findings and the increase of intracranial pressure. Passot,\textsuperscript{9} in a thesis, distinguished between the purulent and non-purulent complications of otitis media. He differentiated them by spinal fluid examination. The non-purulent group was considered due to an excess of cerebrospinal fluid in the subarachnoid spaces. Warrington\textsuperscript{13} in 1914 described five groups of intracranial serous effusions of inflammatory origin: those due to otitis media, to tuberculosis, to specific fever, to head injury, and finally an unexplained group. As his title indicates, he considered a near or distant focus of infection as the primary origin. Claude\textsuperscript{11} discussed the subject thoroughly. He gave trauma, infection, and encephalitis as the etiologic factors and emphasized the value of air studies in differential diagnosis.

More recently Symonds,\textsuperscript{12} Davidoff and Dyke,\textsuperscript{3} McAlpine,\textsuperscript{8} Howell,\textsuperscript{6} and Sahs and Hyndman\textsuperscript{11} have contributed to this subject. The majority of authors have advocated conservative treatment, once the condition was recognized, with repeated spinal drainages and dehydration of the patient. Davidoff and Dyke,\textsuperscript{2} in contrast, advocated subtemporal decompression as a routine measure. The need for air studies to differentiate space-expanding lesions is admitted by most authors, although Symonds\textsuperscript{12} did not use them in his three cases and McAlpine\textsuperscript{8} in only two of his five cases.

It is obvious from a review of the literature that these patients all recovered spontaneously or at least with conservative treatment. However, in many instances it is not clear as to whether impairment of vision occurred in the patients who were treated expectantly or conservatively. McAlpine\textsuperscript{8}
reports optic atrophy in one of his cases and it is to be suspected that this complication may have occurred more often than had been reported.

CASE REPORTS

Case 1. An early experience with a case of "pseudo-tumor" was that of Mrs. J. H. G., a 49-year-old woman, first seen in February 1935. At that time she complained of failing vision, nausea and vomiting, and dizziness during the preceding year with tinnitus for one month. The past history was not relevant except for periodic frontal headaches occurring on an average of once monthly since the age of 18. There had been no change in the intensity and frequency of these headaches during the preceding year. General physical and neurological examinations revealed no significant findings except bilateral papilledema of 3 to 4 diopters with reduction of vision to 0.2 in each eye.

Under the assumption that we were dealing with an unlocalized brain tumor, ventriculography was performed. Both ventricles were tapped and each contained about 15 cc. of fluid. X-rays taken after replacement of the fluid by air failed to disclose any abnormalities of the ventricular system. A few days later formal right subtemporal decompression was carried out. The brain appeared normal but the leptomeninges were thought to be somewhat thickened and opaque, and there was an unusual amount of subarachnoid fluid.

Convalescence was uneventful and the patient left the hospital on the 17th postoperative day. She lives in another city and has not been seen since three months after the operation. At that time the margins of the optic discs were blurred but there was no elevation. Vision was 1.0 in the right eye and 0.8 in the left. The decompression bulged moderately. We have heard from her at intervals since; the last report was in August 1944. Her vision had not changed. There was still slight bulging of the decompression, indicating the persistence of a mild degree of increased intracranial pressure.

Comment. It does not seem likely that this woman's vision would have been spared without decompression. No etiologic factors for her intracranial condition were ever discovered.

Case 2. A similar case, but with chronic infection present elsewhere in the body as a possible etiologic factor, was that of F. H., an 11-year-old boy seen on the children's orthopedic service at the Cook County Hospital in March 1935. He had been under treatment for chronic osteomyelitis of the left femur and humerus of 2 years' duration. Headaches had been present for 3 weeks, and 2 weeks before he had developed diplopia and begun to vomit frequently. Examination revealed slight weakness of the right side of the face, bilateral abducens palsy, and bilateral papilledema of 2 to 3 diopters. X-rays of the chest were negative.

He was thought to have a metastatic brain abscess, and ventriculography was performed. The ventricles appeared to communicate at the time of the air injection but the roentgenograms showed poor filling of the left ventricle with no air in the third. A left subtemporal exploration and decompression were carried out. There was more than the usual amount of subarachnoid fluid but the brain itself appeared normal without any evidence of an intracranial lesion. When a cannula was introduced into the temporal lobe, the temporal horn of the ventricle was readily entered and about 80 cc. of clear fluid were aspirated.

The child made an uneventful convalescence and the papilledema and abducens palsy rapidly receded. Bulging of the decompression was marked for the first month; the bulging could be collapsed temporarily by spinal drainage. Gradually the decompression ceased to bulge and by dismissal 2 months after operation it was flat.

Comment. This patient might well have been treated by conservative means. However, the ventriculograms misled us as they appeared to substantiate the clinical impression of a left temporal lobe abscess. Accordingly surgery was performed without any further delay.