INTRASELLAR ANEURYSMS SIMULATING HYPOPHYSEAL TUMOURS

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Although many striking examples of aneurysms that compress the optic nerves, chiasm, or optic tracts have been reported, few have been described in which differentiation from an expanding pituitary tumour is difficult or impossible without the aid of arteriography or even direct surgical exposure. Cushing recorded a case in 1912 in which there was an aneurysm lying in contact with an hypophyseal adenoma, and added the significant comment that an aneurysm can play the same role as an adenoma and "by its compression effects cause outspoken hypopituitarism." In order for an aneurysm to be mistaken for an adenoma or craniopharyngioma it must expand the sella turcica, interrupt the decussating optic fibres, and compress the pituitary gland. Furthermore, it must do this without telltale bleeding, calcification of its wall, pain in the forehead or ocular palsies from injury to the nerves in the cavernous sinus. This syndrome has been described so rarely that it justifies reporting 3 personal cases, reviewing the 19 that have been published, and adding 13 unpublished cases that have been submitted to us by other neurosurgeons in answer to letters of inquiry (Table 1). Twenty-nine of the 35 examples that we are discussing occurred in individuals in whom a clinical diagnosis of hypophyseal tumour had been made and the aneurysm was revealed by exploration or post mortem. We have included 3 that have been discovered in recent years in which a clinical diagnosis of pituitary adenoma had been made, but was corrected prior to operation thanks to the policy of carrying out routine arteriography. In 2 others no diagnosis was recorded, but in 1, at least, clinical evidence pointed clearly to an intrasellar tumour. In only a single case was the possibility of an aneurysm considered on the basis of ocular palsies, but exploratory craniotomy had to be performed to rule out a pituitary tumour. In addition, Dr. James G. Love has written that there have been at least 7 other cases seen at the Mayo Clinic which he plans to report. Five others were cited at the recent meeting of the Scandinavian Neurosurgical Congress in Stockholm after this paper was read.

In our 3 cases aneurysm was revealed in the first by its spontaneous rupture the day before a scheduled transfrontal exploration of the pituitary; in the second, as a result of its rupture in the course of a transsphenoidal approach to an expanding intrasellar lesion that had been diagnosed as a typical chromophobe adenoma by both the roentgenologists and the endocrinologists; and in the third, at re-exploration for a supposed recurrent craniopharyngioma. The first patient, who entered the hospital in 1935, would to-day be submitted to angiography because he gave the history of a transitory episode of blindness while chopping in the woods the winter before and a subsequent attack of numbness of the left side of his body followed by severe occipital headache. In the other two it is difficult to see how a clinical diagnosis of aneurysm could have been made, unless we had adopted the policy of ordering arteriograms in every instance of suspected hypophyseal tumour. Our second case has taught us that this is the only way of avoiding an occasional surgical disaster if the exploration is to be carried out by the trans-
ANEURYSMS SIMULATING HYPOPHYSEAL TUMOURS

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On the 5th hospital day he had a severe right-sided seizure, followed by a second the next morning. This resulted in right hemiparesis. Late that afternoon there was a terminal seizure followed by gasping respirations and coma. Twist-drill puncture revealed a ventricle full of blood under high pressure just before he died. The clinical diagnosis was haemorrhage into a pituitary adenoma. The postmortem appearance of the brain is illustrated in Fig. 1 and shows the large aneurysm that filled the sella turcica, arising from the infraclinoid portion of the left internal carotid artery.

**Fig. 1.** William P. Postmortem view of base of brain to illustrate aneurysm.

Sphenoidal approach. Even arteriography may fail to disclose the aneurysm, as will be pointed out below, in the not too rare event that the sac is thrombosed.

**PERSONAL CASES**

**#344547.** William P., aged 44. A robust lumberjack was admitted in April, 1935, complaining of progressive loss of vision. Fourteen months previously he had suffered a sudden 15-minute episode of nearly complete blindness with equally rapid recovery, so that he had gone right on with his work. Two months later he suffered another sudden episode of dizziness and numbness of his right side with severe occipital headache. This cleared in a few days, but headache would return on strenuous exertion. Six months prior to admission his left eye had become totally blind, with dimming of lateral vision in the right eye. He had continued to work in the woods until a few days before he entered the hospital.

On admission there was no perception of light in the left eye and the temporal field was cut in the right eye through the centre of the macula. Neurological findings were not otherwise abnormal. He had sired seven children and there was no obvious endocrine dysfunction. Lumbar puncture pressure was normal with a clear fluid, no cells, and protein of 56 mg. per cent. Radiograms of the skull were suggestive of acromegaly with large sinuses, thickened cranial bones, and prominent jaw and brow ridge. The sella turcica was ballooned out with spreading of the anterior and atrophy of the posterior clinoids.

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**#763909.** Ellen O'B., aged 61. In 1953, when first seen on the Endocrine Service, this patient had no visual complaints but entered the clinic because of abnormal fatigability suggestive of Addisonian weakness. She was noted to have very scant body hair and all her laboratory tests indicated a severe degree of panhypopituitarism (Table 2). Successive roentgenograms showed progressive ballooning and destruction of the sella turcica. The films taken in 1953 and 21 months later, shown in Fig. 2, were reproduced in Holmes and Robbins' textbook of roentgenology as a typical example of progressive sellar destruction by a pituitary adenoma. In 1955 a pneumoencephalogram showed a mass protruding slightly above the diaphragma sellae which also seemed typical of an enlarging adenoma. Dr. Anne Forbes made the diagnosis of chromophobe adenoma with severe panhypopituitarism. On treatment with cortisone and thyroid extract she had improved and was able to work again. The area of the pituitary was treated by two courses of radiation.

Examination of the eyes when she was first seen in 1953 revealed normal fields with visual acuity 20/20 in O.D., and 20/30 in O.S. Four years later she had a dense bitemporal hemianopsia with visual acuity still 20/30 in O.D., but down to counting fingers at 4 feet in O.S. A left abducens weakness developed between 1955 and 1957.

She was finally admitted on the Neurosurgical Service in 1957. At this time neurological findings were not remarkable except for the impairment of vision described above. Because of Dr. Oscar Hirsch's remarkably low operative mortality, the Neurological and Endocrine Services have preferred to have their patients operated upon by the transsphenoidal route. Transnasal operation was performed on Oct. 19, 1957 by Dr. Hirsch and Dr. Hannibal Hamlin. On entering the eroded floor of the sella a gush of blood was encountered with loss of several hundred cc. before it could be controlled. The bleeding had stopped when the packs were removed several days later.