COMBINED SUPRA- AND INFRACLINOID ANEURYSMS
OF INTERNAL CAROTID ARTERY

REPORT OF A CASE OF UNUSUAL CONGENITAL DILATATION OF INTRA-
CRANIAL PORTION OF CAROTID ARTERY AND INJURIES TO
VISUAL, OCULOMOTOR, SENSORY, AND TASTE FIBRES

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Following Sir Geoffrey Jefferson's classic description in 1938 \(^{12}\) of infraclinoid aneurysms of the carotid syphon it has been possible to make an exact localization of their position within the cavernous sinus by the neurological signs. Jefferson divided these lesions into three main groups. If the aneurysm is situated in the most rostral portion of the sinus, only the ophthalmic division of the trigeminus is compressed, with resulting pain and more or less hypaesthesia in the forehead and eye. When the aneurysm is posterior, the maxillary nerve is likely to be involved in addition, and when still further caudal or when it fills the entire sinus, there is sensory loss over the mandibular area and often paralysis of the motor root as well. The oculomotor nerve is nearly always involved, with resulting ptosis and pupillary dilatation. Regardless of the anteroposterior position of the aneurysm, there may be a paralysis of the trochlear and abducent nerves and a complete external and internal ophthalmoplegia. Vision and taste are not ordinarily affected by infraclinoid aneurysms, but are possible complications when the aneurysm bulges upward to press upon the optic nerve or sufficiently far back in the middle fossa to compress the greater superficial petrosal ramus of the facial nerve on the petrous ridge. This latter possibility has not been mentioned heretofore.

Upward expansion of an aneurysm in the cavernous sinus to the level of the anterior clinoid, where it may cause added erosion or tilting of this structure with compression of the optic nerve, has not often been described. The dense layer of dura mater that covers the sinus tends to confine aneurysmal expansions either to the infra- or the supraclinoid portion of the carotid trunk. Jefferson \(^{11,12,13}\) and Dandy \(^{6}\) have reviewed the world literature and recorded 25 examples of this combined syndrome, which are listed in Table 1. Four of these were in Jefferson’s personal series and 3 in Dandy’s (one other published with Heuer \(^{9}\)). The blindness has usually been of the monocular type. Occasionally there has been impingement on the crossing fibres from the inferior nasal portion of the retina of the opposite eye. These curved forward into the optic nerve for a short distance in front of the chiasm as they decussate. When compressed there is loss of the upper temporal
field of vision in the contralateral eye, as described by Traquair.\textsuperscript{20} One example of this defect is included in Table 1 (Jefferson, 1955, Case 2). In addition, there may be a central scotoma (Dandy, 1944, Case 1; Jefferson, 1955, Case 3) or damage to the optic tract with hemianopsia (McKinney \textit{et al.}, 1936; Dandy, 1944, Case 1), but the aneurysm is not likely to extend this far caudally. Papilloedema has also been reported (Blessig, 1877; McKinney \textit{et al.}, 1936).

Fatal hemorrhage has been rare, because of the dense dural covering over the cavernous sinus, but one such complication has been recorded by Beadles\textsuperscript{2} (Nettleship’s case, in which exsanguination took place through the nose). Aneurysms in the carotid syphon may rupture into the cavernous sinus, giving rise to an arteriovenous communication. As this type of lesion causes visual damage by another mechanism, we have not included any cases of this sort.

Most of the aneurysms giving rise to the combined visual, oculomotor, and sensory syndrome were studied before the days of angiography. Only 17 have been operated on and but 8 of these were exposed intracranially. Jefferson’s first patient, whose lesion was verified by intracranial exploration, developed a hemiparesis and died a month later. Postmortem examination revealed a huge aneurysm, almost completely thrombosed, which filled the middle fossa and compressed the temporal lobe. Another patient was submitted to carotid ligation in the neck with satisfactory improvement, but the 2 others were not operated upon. Dandy carried out combined cervical and intracranial occlusion of the internal carotid in 2 patients, with little improvement in the one whose clinical result is mentioned. He performed a subtemporal decompression in his third patient and in another reported with Heuer.\textsuperscript{9} Three other aneurysms were exposed at intracranial operation by Reinhardt,\textsuperscript{17} Zollinger and Cutler,\textsuperscript{23} and Gardner.\textsuperscript{7} The first 2 patients died, but Gardner’s survived despite gross bleeding on opening the aneurysm, which was controlled by packing and cervical ligation.

In view of the rarity of large infraclinoid aneurysms that bulge upward out of the cavernous sinus to involve the optic nerve, and of the lack of surgical experience in dealing with them, it seems worth while to report the following case, especially as it demonstrates the added neurological sign of loss of taste, which has not been observed heretofore.

\textbf{CASE REPORT}

\textit{MGH U-857750}. Chester P., aged 16 years, entered the hospital in June 1954. Otherwise in good health, he had noticed a gradual loss of vision in his left eye over the preceding year. One week prior to admission a severe pounding left temporal headache developed which prevented sleep. The next day, as the headache subsided somewhat, a sense of numbness spread over the entire left trigeminal area, the left eyelid drooped, and all movements of this eye were lost. There were also further reduction in vision of the left eye and loss of taste on the left side of his tongue. He did not become nauseated, vomit or have a stiff neck.

\textit{Examination}. Blood pressure was 120/62; temperature was normal. There were two small naevi on the left upper lip and temple. The left eye seemed slightly more