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The occurrence of massive, life-endangering epistaxis following closed craniocerebral trauma is extremely uncommon. It was first reported in 1928 by Birley, with a note by Trotter. In 1958, Hamilton noted only 3 previous reports in the literature. We have found a total of only 9 cases, including the present one.

The onset of bleeding may be insidious and remote in time from the trauma. It may be extremely difficult to identify the location of the bleeding and the pathologic anatomy that is involved. The more common type of epistaxis following craniocerebral trauma is that arising from the anterior ethmoidal or sphenopalatine arteries. The rate and rapidity of this hemorrhage usually does not become life-endangering and it is likely to be controlled by nasal packing. The massive type of hemorrhage, from which the patient rapidly goes into shock, has its origin directly from the internal carotid artery. In all the cases reviewed, the blood reached the nasopharynx via the sphenoidal air sinus. The literature is abundant with reports of cases of fistulous communications between the internal carotid artery and the cavernous sinus. However, the vast majority of these lack the prerequisite for massive epistaxis, namely, a fracture across the floor of the anterior cranial fossa and into the sphenoidal air sinus. The purpose of this paper is to bring this interesting and challenging entity to the current literature and to discuss the clinical picture in the following case, which is remarkably similar to other cases reported.

CASE REPORT

R.A.H., an 18-year-old man enlisted in the Navy, was admitted on Jan. 5, 1960 complaining of blindness of the left eye of 3 week's duration. He had been injured in an automobile accident on Dec. 14, 1959, sustaining severe blunt trauma to the left orbit. He was taken to a hospital where the findings were a semicomatose state and bleeding from a laceration above the left eye. Within 4 days, his level of consciousness returned to normal and he was discharged. The following day, swelling about the left orbit decreased sufficiently to allow the eye to open and the patient noted that this eye was blind. Because of this, he reported to the U. S. Naval Hospital in St. Albans for evaluation.

Physical examination revealed a healed laceration below the left eyebrow and resolving ecchymosis of the left bulbar conjunctiva. The left pupil was dilated and did not react to light. There was no perception of light in the left eye. The fundi were normal. Bilateral 6th cranial nerve palsy was present, complete on the left and partial on the right. Roentgenograms of the skull revealed several fracture lines in the medial aspect of the left orbit extending to the floor of the anterior fossa. The ethmoid air cells were cloudy on the left; the sphenoid sinus was clear (Fig. 1).

Course. The patient was being followed to evaluate the right 6th nerve palsy when, on Jan. 16, 1960, he suddenly had severe epistaxis from the left nostril which ceased without packing and did not require transfusion. Examination of the nose and nasopharynx revealed no bleeding point. In the following 20 days, two more minor episodes of epistaxis occurred. Then on Feb. 5, 1960, a sudden massive epistaxis began at 2 A.M. Bilateral anterior and posterior packs seemed to control the hemorrhage but four units of blood were required for the treatment of shock. No bruist was audible over the skull the following day. The packing was removed on the 2nd day and the patient did well until Feb. 13, 1960, when another massive hemorrhage from the nose and mouth occurred, requiring three units of blood. The patient now noted that he could tell when he was going to bleed by a "tickling" sensation in his nasopharynx. A bruist was heard over both eyes, louder on the right. Then, on Feb. 14, 1960, the third massive hemorrhage occurred, requiring three units of blood were given. Several hours later, as preparation for carotid angiography was being made, he bled again, losing blood so rapidly into the pharynx that rapid, bedside tracheotomy was required to relieve obstruction of airway. The patient was transfused with six more units of blood and the left common carotid artery was ligated under local anesthesia. The bleeding ceased immediately.

The patient demonstrated no untoward effects from the carotid ligation and was ambulatory within 48 hours postoperatively. Within 2 weeks the palsy of the left 6th nerve improved to 60 per cent of normal, the right to 90 per cent. He remained blind in the left eye. A faint bruit persisted over both eyes, louder on the right. Compression of the right carotid artery made the bruist much louder over the left eye and fainter over the right.

Right carotid angiography revealed excellent bilateral

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opacification of the anterior and middle cerebral arteries but no flow into the left internal carotid artery or the cavernous sinus. Left carotid angiograms were then performed under direct vision with cannulation of the internal carotid. The left internal carotid artery was demonstrated to be thrombosed at the level of the base of the skull, probably the result of the common carotid ligation (Fig. 2). A strong pulsatile flow to this point was shown by direct pressure recordings to come entirely from the external carotid by retrograde, collateral circulation. The external carotid artery was ligated just beyond the origin of the superior thyroid artery, allowing only a small, pulsatile flow to continue into the internal carotid.

The patient recovered uneventfully from these procedures. Re-examination disclosed no change in the intracranial bruit, the source of which is not obvious. Except for the lesions of the optic and abducens nerves he has remained asymptomatic to date, and no further therapy is planned.

DISCUSSION

As the case report illustrates, this clinical syndrome can present a challenge both in diagnosis and treatment. Unilateral blindness, a fracture of the roof of the orbit and massive epistaxis are the characteristic triad.

Table 1 illustrates several points of interest concerning the 8 cases reported previously, together with the present case. Epistaxis was delayed in 3 instances, the longest interval being 3 months. The number of hemorrhages varied from 2 to 14 and there was a tendency toward increasing severity. The optic nerve was involved in every case, usually with homolateral blindness. Injury to the oculomotor nerve was apparent in 6 instances, although pupillary dilatation was the only evidence in the present case. Impairment of the 6th nerve was present in 4 cases, being bilateral in 2 of these. Damage to the olfactory nerve also was noted in 4 patients. The other cranial nerves were involved as follows: 4th, 3; 5th, 2; and 7th, 1. A cranial bruit was noted in 5 cases, not mentioned in 3 and said to have been absent in 1. The earliest period at which the bruit was detected was 7 weeks, the latest, 6 months. Cranial fracture was identified in the anterior fossa in 6 instances, the middle fossa in 2. The usual treatment was ligation of the cervical carotid artery. There were 3 fatal results, all from hemorrhage: 1 apparently because of incomplete occlusion of the common carotid, in spite of additional intracranial clipping of the carotid; 1 in a case in which no ligation was performed, and 1 for undetermined reasons. In 1 instance, intracranial clipping of the carotid artery apparently was responsible for a successful result after cervical ligation of the common and internal carotid arteries had been followed by another epistaxis.

Intracranial hemorrhage of sufficient extent to be detected clinically has not been reported, nor is intracranial hemorrhage mentioned as a nec-