SPASM OF THE INTERNAL CAROTID ARTERY

MAJOR ARTHUR D. ECKER, M.C., A.U.S.

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It has long been known that sudden occlusion of the internal carotid artery may produce enough interference with the blood supply of the cerebral hemisphere on the same side as to cause severe symptoms. However, the role of minor trauma to the internal carotid artery in the production of cerebral dysfunctions appears to have been given insufficient attention, although Makins² did raise the question whether spasm might be involved in cases of contusion of this vessel. The purpose of this report is to record a series of observations in which cerebral symptoms were produced from penetrating war wounds of the neck in which there was neither laceration nor thrombosis of the internal carotid artery.

CASES OF WOUNDS OF THE NECK

Case 1. This soldier suffered a bullet wound on 7 June 1944 at Normandy. The missile was a 0.30 calibre machine gun bullet, which entered the left cheek and came to rest just under the base of the skull, and just medial to the left mastoid process (Fig. 1). Little is known about the patient’s condition for the first three days. He arrived in a fixed hospital in England on 10 June 1944. At this time he was very drowsy and apparently was suffering from
motor aphasia, although he responded with muscular movements to pain or noise. The blood pressure was 156/100, and the pulse rate 76. There were right hemiparesis, which was more marked in the upper limb, and transitory right ankle clonus. The corneal, pupillary, plantar and swallowing reflexes were normal. The right pupil was dilated, but responded to light. There was blood in the patient’s mouth; the eardrums were normal. There was ecchymosis of the left side of the neck. The respiratory rate was 12.

X-ray films revealed the retained bullet just beneath the base of the skull and just medial to the left mastoid process, but on the films there was evidence of neither fracture of the skull nor intracranial foreign body (Fig. 1). Lumbar puncture revealed clear and colorless fluid under pressure of 300 mm. of water. On unilateral jugular compression and release on each side, there was prompt rise of spinal fluid pressure to 400 mm. and a prompt fall. The patient seemed brighter following lumbar puncture. For a few hours, the paresis of the right arm—especially the fingers—seemed to increase, and a tentative diagnosis of “intracranial hemorrhage (possibly epidural)” was made. However, within another few hours the respiratory rate increased to 20, the paresis of the right upper limb diminished, and the patient seemed less drowsy.

On 11 June 1944 the bullet was removed, but the internal carotid artery was not carefully examined. In the course of the next two weeks, the patient’s general condition gradually improved so that he began to talk and say a few words. By 18 July 1944 the patient had regained moderate speech, and was able to take care of himself on the ward. On this date an air encephalogram was made. The spinal fluid contained 45 mg. total protein per 100 cc. The film (Fig. 2) revealed slight dilatation of the left lateral ventricle and of the third ventricle.

In summary, a “near miss” of the left internal carotid artery caused aphasia, right hemiplegia, increased intracranial pressure and residual left ventricular dilatation.

Case 2. This soldier was wounded in action in France on 23 June 1944. He suffered a perforating wound due to a small missile of unknown type. The missile entered the left cheek, caused a comminuted fracture of the left side of the mandible near the angle of the jaw, and left the patient’s body from the right side of the neck. There was no fracture of the skull. The patient suffered immediate right hemiplegia, aphasia and respiratory difficulty. A tracheotomy was performed at a field hospital.

On 7 July 1944, the patient came under the care of the author in a fixed hospital in England. At that time examination revealed restlessness, aphasia, flaccid right hemiplegia, slight hyperactivity of the right tendon reflexes and the presence of Babinski’s sign on the right. The small wound in the posteroinferior portion of the left cheek and on the right side of the neck were healed. The tracheotomy wound was healing. For a few days the patient could not phonate, but the ability to make sounds soon returned although speech did not return for a few weeks.

On 16 July 1944, encephalography was carried out and revealed considerable dilatation of the left lateral ventricle and of the third ventricle and slight dilatation of the right lateral ventricle (Fig. 3). The subarachnoid markings were normal. Cerebral arteriography was carried out on both right and left sides on 7 August 1944. The middle and anterior cerebral arteries and their branches on the left side were smaller than those on the right. Around 15 August 1944 the patient began to say a few words and there was slight return of strength of the right limbs, more in the lower than the upper.

In summary, a “near miss” of the left internal carotid artery caused aphasia, right hemiplegia, dilatation of the lateral ventricles more on the left than on the right, and diminution in calibre of the left middle and anterior cerebral arteries.
Case 3. A soldier suffered a penetrating wound on the right side of the neck 17 July 1944 and had immediate left hemiplegia. When he was examined at a fixed hospital 20 July 1944, a metallic fragment less than 1 cm. in diameter was found in the soft tissues of the neck anterolateral to and at the level of the 5th vertebra. At this time there was no clinically demonstrable residuum of the hemiplegia.

**CASE OF A BLUNT HEAD INJURY**

Case 4. This soldier fell off a truck at 3:00 o'clock on the morning of 22 June 1944. He was unconscious for a short time but soon regained responsiveness, although he remained disoriented. There was bleeding from the left ear but no laceration of the scalp. In the next three hours he became progressively less responsive. At 6:00 o'clock he was in a deep coma, had stertorous breathing and episodes of decerebrate rigidity. His left pupil was greatly dilated and did not respond to light. Both Babinski and Hoffmann signs were present on each side. An X-ray film revealed a fracture of the skull in the left temporoparietal area extending across the posterior division of the middle meningeal artery.

At 7:30 o'clock, that is, 4½ hours after the injury, an emergency left temporal craniotomy was performed without anesthesia. An extradural blood clot, 2.5 cm. deep and about 7 cm. in diameter, was evacuated and the bleeding from the meningeal vessels was controlled. There was immediate improvement in the size and reactivity of the left pupil so that these were normal within 15 minutes after the removal of the clot. However, the patient failed to regain consciousness. His rectal temperature was 103.6°F, but later responded to cooling of the body. The blood pressure remained normal and there were no postoperative episodes of decerebrate rigidity. However, the pulse rate and respiratory rate remained high and the patient died 36 hours after the injury.
Pathologic Findings. Gross. A complete autopsy was performed and revealed no visceral lesion except bilateral pulmonary edema (weight of right lung 1110 gm., weight of left lung 855 gm.). At the site of the operation there was a small amount of epidural blood, which was nowhere more than 1 cm. deep. The brain convolutions were markedly flattened and sections of the brain revealed dilatation of its blood vessels without hemorrhage. The fracture line extended into the base of the skull along the petrous portion of the temporal bone. The portion of the left internal carotid artery lying within the carotid canal and that portion immediately beyond its emergence from the canal were marked by hemorrhage into the wall of the vessel. The right internal carotid artery was normal.

Microscopic. Study of the sections of the left internal carotid artery revealed recent hemorrhage in two different areas of the outer portion of the adventitial connective tissue. There was no evidence of inflammatory reaction or organization. The outlines of the cells of the muscularis were obscure. The cells of the middle and outer thirds of the media were void of nuclei. The subintimal third of the media contained scattered nuclei, many of which were pyknotic. The intima was marked by a few scattered endothelial cells which were decreased in number and some of which were pyknotic. There was neither focal necrosis nor inflammatory reaction. The hemorrhage in the adventitia was probably terminal. Sections of the right internal carotid artery were used as controls (Fig. 4). These microscopic studies were made by Major Oscar Wollenman, M.C., A.U.S., and the findings are consistent with ischemic necrosis.

DISCUSSION

In Cases 1, 2 and 3 there were wounds of the neck with “near misses” of the internal carotid artery by missiles. There is no reason to believe that the internal carotid behaves differently from arteries of similar size supplying
the limbs in which the spasm is known to occur following such trauma. Cohen has written of such lesions:

The spasm is rarely maximal, the lumen being merely narrowed and the distal limb is rarely seriously endangered. It would appear reasonable to regard this form of spasm as consequent upon the sudden stretching of the vessel by lateral displacement due to the exploding force of the missile.

Because of the extreme susceptibility of the brain tissues to anoxia, a relatively minor impairment of its blood supply will yield profound dysfunction, thus differing from the situation in an extremity. In Cases 1, 2 and 3 there was evidence of neither laceration nor thrombosis of the carotid artery but very likely spasm took place.

**Increased Intracranial Pressure.** Moniz has been impressed with intracranial hypertension following carotid thrombosis. He cites a case of severe traumatism of the head with so much traction on the carotid artery as to produce tears of the intima, which initiated a thrombotic process. A lesser amount of traction may cause spasm of the artery. The swelling of the brain in these cases is probably due to a combination of cerebral edema and congestion. The morphology and pathogenesis of these vascular disturbances have been characterized as vasoparalysis of the central nervous system. In Case 1 intracranial pressure was found to be increased three days after the wounding. In Case 4 the autopsy revealed evidence of markedly increased intracranial pressure 36 hours after the injury.

**Ventricular Dilatation.** A recent study of traumatic dilatation of the cerebral ventricle indicates that it may be well due to a generalized effect on the brain, although its microscopic pathology has not yet been identified. In Case 1 there was unilateral and in Case 2 bilateral dilatation of the lateral ventricle although there was no evidence of skull fracture in either case. It is, therefore, suggested that impairment of cerebral arterial circulation may be a significant factor in traumatic dilatation of the cerebral ventricle.

Neurosurgeons have recognized for some time the high frequency of death in cases of hemorrhage from the middle meningeal artery. It has been reasonable to suppose that death was due not to the hemorrhage itself but to the associated brain injury. In Case 4 an unsuspected lesion, ischemic necrosis of the internal carotid artery, was discovered at necropsy. In Fig. 3 of Holbourn's recent paper on the mechanics of head injury, shear-strain due to a rotation in the coronal plane caused by a blow above the ear is seen to be maximal near the midline at the base of the skull. In other words, the nature of the blow that is most likely to cause a fracture of the skull across the meningeal vessels and extradural hemorrhage is the same as that in which the brain is most likely to pull on the circle of Willis and the internal carotid artery. As Cohen has shown, traction on an artery is one of the commonest causes of arterial spasm. In all fatal cases of head injury special attention should be devoted to both gross and microscopic study of the arteries at the base of the brain. As a matter of fact, abnormalities may not
be demonstrable in cases where death ensues promptly. The further demonstration of changes in cerebral arteries in head injury may await the more frequent use of cerebral arteriography. Wakeley and Orley\(^7\) state:

In mild cases of cerebral concussion or contusion arteriography reveals a contraction of the blood vessels. In grave cases and in the presence of cerebral tumefaction the blood vessels are flattened and fill incompletely owing to vascular paralysis and mechanical compression.

**SUMMARY**

There have been presented 4 cases of dysfunction of the brain which are considered to be due to relatively mild trauma of the internal carotid artery. The first three were cases of penetrating wounds of the neck without laceration or thrombosis of the artery or any evidence of direct head injury. The fourth case was a blunt head injury with a fracture of the skull and post-mortem evidence of brain swelling and ischemic necrosis of the wall of an internal carotid artery. It is considered that the common denominator in all 4 cases was the application of force to the carotid artery resulting in spasm of this vessel. Within 10 minutes there resulted dysfunction of the cerebral hemisphere on the same side which persisted for a period of time varying with the degree of anoxemia produced. There was swelling of the brain which probably reached its maximum in 24 hours and lasted at least three days. This brain swelling may be due, in part, to a vasoparalysis of the central nervous system which, in turn, may be closely associated with narrowing of the cerebral arteries. Furthermore, dilatation of the cerebral ventricles, especially that on the same side, may be a permanent residuum.

**CONCLUSION**

A syndrome of spasm of the internal carotid artery is described and consists of:

1. Dysfunction of a cerebral hemisphere.
2. Brain swelling, in the acute stage.
3. Internal hydrocephalus, in a late stage.
4. Narrowing of the cerebral arteries.

Cases are presented that illustrate the association of this syndrome with not only penetrating wounds of the neck but also blunt injuries of the head.

**REFERENCES**