Observations on the Effect of Systemic Blood Pressure on Intracranial Circulation in Patients with Cerebrovascular Insufficiency*

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HEMIPARESIS may develop after carotid angiography or after dissection around the intracranial portion of the internal carotid artery either for isolation of an aneurysm or removal of a tumor. Angiospasm or transient occlusion of the vessel and the resultant ischemia of the brain may be the causative factor. Hemiparesis may be persistent, even though supportive care is given and the blood pressure is maintained at a normal level. In the cases reported here, an attempt was made to elevate the blood pressure to a hypertensive level. The immediate response to this type of treatment in alleviation of hemiparesis has been gratifying.

Case Reports

Case 1. In 1956, the patient, a 54-year-old left-handed man, developed right retroorbital headache. A diagnosis of pituitary chromophobe adenoma was made. He was treated by irradiation therapy and his symptoms disappeared. In 1964, he had recurrence of headaches and partial right third and fourth nerve palsies. At the time of operation a cystic tumor was found encircling the right optic nerve and the right internal carotid artery, compressing the oculomotor and trochlear nerves. The tumor was dissected and partially removed from the carotid artery, optic nerve, and adjacent structures. Immediately after the operation the patient was lethargic and had a left hemiparesis. His blood pressure at this time was 140/80 mm Hg. An intravenous infusion of metaraminol bitartrate was started immediately and the blood pressure was raised to 170/100 mm Hg (Fig. 1). Within 5 to 10 minutes the patient awakened and began to move his left leg and his left arm. Whenever the blood pressure was permitted to drop below a systolic level of 130 mm Hg the hemiparesis would recur. After 3 days, however, the blood pressure could be lowered to a systolic level of 120 mm Hg without recurrence of a significant degree of weakness. The infusion of metaraminol bitartrate could not be discontinued because of ensuing rebound hypotension. An intravenous infusion of levarterenol bitartrate was started, and over a period of 24 hours was tapered off. The blood pressure remained elevated in the vicinity of 140/80 mm Hg.

Case 2. A 41-year-old, right-handed man had transient episodes of numbness of the right hand, face, and leg for about a year.

FIG. 1. Graphic representation of the patient's response to elevation of blood pressure (Case 1).
Skull x-rays and a right retrograde brachial arteriogram showed a pattern consistent with two meningiomas, one parasagittal and one a tuberculum sella meningioma. A left percutaneous carotid arteriogram was performed without any difficulty. A discrete stenosis of the intracranial portion of the left internal carotid artery was seen (Fig. 2). Forty-five minutes after angiography the patient was asphasic and had a right hemiplegia. Various supportive measures were of no avail. Two hours later 5 mg of metaraminol bitartrate was given intravenously. The blood pressure rose to 220/100 mm Hg (Fig. 3). Within 5 minutes the patient began to move his right side, develop good strength in the extremities, and started to talk. As his blood pressure decreased to a level of 100/70 mm Hg, the patient had again become asphasic and hemiplegic. When the blood pressure was elevated again the patient began to speak and was able to move his right side. Three days after the onset of hemiplegia and asphasia he was doing well with a blood pressure maintained at a level of 130/90 mm Hg. He was operated upon and the meningiomas of the falk and tuberculum sellae were removed. During induction of anesthesia and during the operative procedure an attempt was made to keep the blood pressure at about a systolic level of 140 mm Hg, but it was quite labile and on several occasions it dropped below a systolic level of 100 mm Hg. After the operation the patient was asphasic and hemiplegic; he no longer responded to artificially elevated blood pressure and died 2 days later. Autopsy showed a massive infarction of the left cerebral hemisphere. The internal carotid and the proximal portion of the anterior cerebral artery were thrombosed (Fig. 4).

This patient's initial symptoms were caused by cerebrovascular insufficiency. The arteriogram added insult to an already present marginal blood supply. With elevation of the blood pressure, after the onset of hemiplegia, the cerebral blood flow was increased and an impending infarction of brain was prevented. The drop in blood pressure during the operative procedure, however, caused irreversible changes in the brain and led to cerebral infarction.

**Case 3.** A 44-year-old, right-handed woman had an aneurysm or the right internal carotid at the junction of the posterior communicating artery (Fig. 5). The aneurysm was trapped intracranially. Immediately the patient was given an intravenous metaraminol bitartrate infusion, and the systolic