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. Hemi-paresis 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 retro-orbital 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.

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Skull x-rays and a right retrograde brachial arteriogram showed a pattern consistent with two meningiomas, one parasagittal and one a tuberculum sellae 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 aphasic 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 aphasic 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 aphasia 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 falx 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 aphasic 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

Fig. 2. Segmental stenosis of intracranial portion of the internal carotid artery (Case 2).

Fig. 3. Graphic representation of the patient's response to elevation of blood pressure (Case 2).
pressure was maintained at 150 mm Hg. She did quite well and had no neurological deficit. A day later the systolic pressure had diminished to 110 mm Hg. She then became quite lethargic and a short while later developed a left hemiplegia. Metaraminol was restarted, and as the blood pressure went up to 170/110 mm Hg she began to move her left side. Within 2 hours she had shown remarkable improvement. Three days later metaraminol administration was gradually tapered, and the patient made an excellent recovery.

Case 4. M. D., a 52-year-old, right-handed woman, had a sudden onset of a severe frontal headache. She lost consciousness for a short time. When she regained her senses she complained of stiffness of the neck, suboccipital headache, nausea, and vomiting. The neurological examination was normal.

Lumbar puncture showed a bloody spinal fluid under 240 mm of water pressure. Bilateral carotid arteriograms showed no apparent aneurysm. The right internal carotid artery and its branches showed spasm. Upon injection of the left carotid artery the patient became confused and within a few minutes lapsed into a comatose state. She had a right hemiplegia and assumed a decerebrate posture. An intravenous injection of papaverine and the inhalation of carbogen (95% O₂, 5% CO₂), did not improve her condition. She was then given an intramuscular injection of 10 mg metaraminol bitartrate. Within 15 to 30 minutes she became more responsive, but had a receptive aphasia. Her blood pressure at this time was 250/120 mm Hg. Her condition progressively improved, and her recovery was complete within 2 or 3 days.

Discussion

In 1951, Thompson and Smith\textsuperscript{11} showed that ligation of the middle cerebral artery in normotensive monkeys caused no neurological deficit. However, if these animals were rendered hypotensive they showed profound neurological abnormalities. Corday, et al.,\textsuperscript{3,4} demonstrated electroencephalographic changes after the reduction of blood pressure in monkeys whose carotid and vertebral arteries were mechanically narrowed on one side. These electroencephalographic abnormalities disappeared shortly after the elevation of blood pressure by transfusion or by the use of vasopressor drugs. Injury to the brain\textsuperscript{7,12} has long been known to occur in patients who develop a state of shock of hemorrhagic, cardiogenic, or hypoglycemic origin. In fact, in some patients the initial presenting symptom of coronary thrombosis is that of disturbance of the cerebral function brought on by ischemia to the brain.\textsuperscript{1,2,6} The
judicious use of vasodepressor drugs in patients with hypertension and cerebrovascular disease may lead to a sudden drop in blood pressure and cause ischemia of the brain and marked neurological abnormalities.

Shanbrom and Levy clearly demonstrated the value of elevation of blood pressure in patients with cerebrovascular insufficiency. They used norepinephrine, stating that the cerebral vasoconstricting effect of this drug in patients with arteriosclerosis was minimal.

Cerebral blood flow is dependent on two major factors: the amount of blood pressure and the resistance to blood flow. The resistance to blood flow is independent on intracranial pressure, the viscosity of blood, chemical factors, and the neurogenic control of the caliber of the blood vessels. Although with increased blood pressure there is a concomitant increase in resistance to blood flow, clinically it appears that the elevation of blood pressure outgains the factor of resistance. It is possible that in an ischemic area, because of the increase in CO₂ tension, the vessels are maximally dilated and the increased blood pressure allows additional blood flow to this area. The rationale behind the use of hypertensive drugs is twofold. First, by the elevation of the blood pressure the blood flow to the brain is increased. Second, by prevention of cerebral ischemia, increased intracranial pressure and the resultant increase in resistance to the blood flow is eliminated. It is a well-known fact that ischemia of the brain leads to cerebral edema and intracranial hypertension.

Patients who develop hemiparesis after angiography or after intracranial manipulation of the carotid artery at the time of operation may have cerebrovascular insufficiency with cerebral ischemia. If a supported state of hypertension can be achieved, severe neurological deficit can be prevented. Usually in a few days enough collateral circulation develops so that normal blood pressure can sustain sufficient blood supply to the ischemic area.

In the surgical approach to aneurysms, as soon as the lesion is clipped or trapped, intravenous metaraminol is used and the systolic blood pressure is raised to 50 or 60 mm Hg above the normal pressure for that patient. This may be continued for about 48 to 72 hours, and then the drug is gradually tapered. In patients who develop hemiparesis after angiography the artificial elevation of blood pressure for 12 to 24 hours is usually sufficient. In other cases in which hemiparesis develops after an operation in the region of the sella turcica, a hypertensive regimen is started in the recovery room as soon as the weakness is discovered. The dosage of drug required to keep blood pressure up to a satisfactory level is variable. The strength of the solution is usually 100 mg of metaraminol bitartrate per 250 cc of saline solution.

Metaraminol bitartrate is used initially because its vasoconstricting effect on cerebral circulation is less than norepinephrine and it can sustain a relatively constant blood pressure level. However, this drug acts by increasing the use of the patient's own supply of catecholamines. After a day or so, withdrawal of metaraminol leaves the body depleted of catecholamines, and a precipitous drop in blood pressure results. To avoid this rebound hypotension, towards the latter part of therapy norepinephrine should be used for at least 24 hours to replete the body's catecholamines and then gradually tapered off.

Summary

The intracranial circulation in patients with cerebrovascular insufficiency varies with the systemic blood pressure. Certain cases of cerebrovascular insufficiency of acute nature can be treated by maintenance of an elevated blood pressure. In several patients dramatic results have been obtained with this method of therapy. This technique has been used in patients with postangiographic hemiparesis or hemiplegia, in others with neurological deficit after ligation of intracranial aneurysms, and in some cases at the time of operation for intracranial aneurysms. We have reported four cases and described the method of treatment.

References

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Effect of Systemic BP on Intracranial Circulation