CONSIDERATIONS CONCERNING INCREASING CEREBRAL BLOOD FLOW IN CEREBRAL INSUFFICIENCY*

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Experiences in the past and more recent clinical and experimental information indicate that it seems possible to increase the blood flow to a cerebral hemisphere by means of repeated temporary carotid compression. Considerations concerning this observation are briefly summarized and illustrated in this paper.

I. Past Experience. Rudolph Matas employed carotid compression by several mechanical means for inducing collateral flow to the ipsilateral hemisphere, prior to carotid ligation. He pointed out that Halsted had first used such a compression method, the aim being "to obtain by gradual occlusions a secondary development of the collateral circulation in parts liable to suffer by insufficient vascular supply."

Halsted applied an aluminum band to a common carotid artery, almost occluding it, and observed: "Even in this case slight head symptoms persisted for several months making it seem likely that complete occlusion would have been followed by severe symptoms if not by death." Halsted concluded that "The partial occlusion of arteries discloses a suggestive and, I believe, a promising field for investigation in physiology and experimental pathology."

Dandy also found that patients could tolerate a gradual occlusion of a carotid artery but not an acute, complete interruption of flow. He stated, "regardless of age, the adequacy of the collateral circulation must be known before operation (carotid ligation). If inadequate, it can be made adequate by partially occluding the internal carotid, thus forcing the collateral branches to take up the increased burden and later permit its total ligation without hazard."

II. Clinical-Pathologic Evidence. A common clinical observation is the ability of patients to recover from cerebral vascular occlusions because of an improvement in the cerebral circulation. Neurologic defects often reverse in patients with occlusions in various parts of the circulation. A silver clip, for example, placed upon the anterior choroidal artery may result in a definite but temporary neurologic effect.

The neuropathologists have recognized that the infarcted area in a brain

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is of lesser area than that normally supplied by the involved artery. Also, the vessels within the infarcted area contain fluid blood indicating that collateral circulation has been present.

Our experience has also shown that patients with carotid occlusions (partial or complete) caused by atheromatous disease at the carotid bifurcation develop an increasing tolerance to contralateral digital carotid compression. Syncope with or without chronic convulsive movements usually occurs abruptly after 12 or 15 seconds of total occlusion of the contralateral vessel upon the first compression test. In some of these patients cardiac slowing with a fall in blood pressure occurs, unaffected by atropine. In others, the cardiac effect is minimal. These responses are quite different from the cardiac arrest resulting within several seconds after compression of the “hypersensitive carotid sinus.” The response to digital compression in patients with occlusions gradually becomes less marked and requires a longer period of compression. Subsequently, in some cases, the syncopal effect cannot be induced. Finally, the symptoms of dizziness, blurred vision and faintness fail to appear. These responses seem to occur in the younger age groups, possibly related to the absence of advanced generalized arteriosclerosis.

The observation has also been made that following carotid ligation, early compression of the contralateral carotid produces syncope. A tolerance then develops because of increasing collateral blood flow. 8

Kussmaul and Tenner 15 in 1859 reviewed in considerable detail the experiences of the past concerning temporary carotid occlusions. These authors described the use of carotid compression by Caleb Parry 18 in the treatment of hysteria and convulsions, the experiments of Jacobi 14 and many others. None had made mention of convulsions occurring, although such effects as “dimness of sight, dizziness, stupor, weakness in the legs, staggering, swooning, loss of consciousness and sudden apoplectic falling down” were observed. Kussmaul and Tenner emphasized that compression of both carotids also may result in “all the symptoms of a slight epileptic attack” as well as unconsciousness. Not all the experiments produced this effect since “collateral circulation is kept up by the vertebral arteries.” It is of interest that after 100 years, these same observations are remade on the basis of one compressed carotid and one that is obstructed by disease.

Until the observations of Weiss and Baker, 21 who stressed the importance of the carotid sinus reflex mediated through the 9th nerve, the carotid artery claimed little attention in this century. Although these authors recognized a “cerebral” type of “reflex,” the etiology was ascribed to a “cerebral vasoconstriction” produced by an ill-defined neural mechanism. Now after another 20 years, we must return to the older 19th century concepts which recognized the effects of cerebral ischemia produced mechanically.

III. Angiographic Evidence. Angiographic studies have confirmed earlier pathologic studies which showed that a collateral blood supply may be demonstrated following the occlusion of major vessels. A “cross-over” effect may occur in an occlusion of the carotid artery (Fig. 1). The middle cerebral