OBSERVATIONS ON UNILATERAL COMPRESSION AND PALPATION OF THE CAROTID BIFURCATION*

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When a ligation of the common or the internal carotid artery is desirable, evaluation of the competency of the cerebral circulation before the ligation is extremely important. Compression of the carotid arteries for testing the adequacy of the circulation gives valuable information. Conditioning of the cerebral circulation by repeated compressions of the carotid, as suggested by Matas,\textsuperscript{7} Dandy\textsuperscript{1} and others, is possible and it is a valuable means of obtaining better results.

During the past 50 years there have been varying opinions concerning the results of unilateral palpation and/or compression of the carotid bifurcation. Before the days of “the irritable carotid sinus,” syncopal effects from compression of the carotid bifurcation were thought to be caused by ischemia of the brain. Since the outstanding work of Hering,\textsuperscript{6} Heymans\textsuperscript{6} and others on the carotid sinus as one of the governing mechanisms of the blood flow to the brain, there has been a tendency to consider all abnormalities resulting from palpation and/or compression of the carotid bifurcation, the consequences of “irritable carotid sinus.” Manipulations of the bifurcation have been described as a means for relief of anginal pain by Freedberg and Rice-\textsuperscript{2}man.\textsuperscript{2} Others ascribed the production of anginal pain to such palpation or compression.\textsuperscript{3,8} Hypersensitivity of the carotid sinus may be present with hypertensive disease and arteriosclerosis, according to Weiss and his co-workers.\textsuperscript{11}

With a view to clarify the confused state of our present-day knowledge concerning this subject it was thought advisable to record our experiences with palpation and compression of the carotid bifurcation in patients with unilateral occlusion of the internal carotid artery and in cases in which a carotid ligation was carried out for certain intracranial aneurysms, arteriovenous malformations and arteriovenous fistulae.

Unilateral palpation and/or compression of the carotid bifurcation may result in several different effects. In normal patients there may be no abnormality noted. In others, a syncopal effect may be produced. Syncope as a result of such manipulations may be caused by (1) an irritable carotid sinus, (2) an ischemia of the brain produced by the compressive occlusion of the internal carotid artery and (3) a combination of irritable carotid sinus and ischemic effects, or a combination of (1) and (2).

(1) The importance of the carotid sinus as a governing mechanism of

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blood flow to the brain was first appreciated by Hering and later analyzed more critically by Heymans and his co-workers. Experimentally, a lowering of the intrasinus pressure by compression of the common carotid artery, proximal to the sinus, results in cardiac acceleration, vasoconstriction and a rise in arterial pressure with liberation of adrenalin. Pressure against the carotid bifurcation causes a slowing of the heart rate, vasodilatation and a fall in blood pressure. In men, increased irritability of the carotid sinus region may result in pathological states which are: (1) a bradycardia and asystole with eventual syncope, (2) a precipitous drop in blood pressure associated with syncope but without bradycardia, (3) a stimulation of brain centers causing unconsciousness and syncope through glossopharyngeal influences unassociated with bradycardia or fall in blood pressure (the cerebral type), and (4) a combination of the above types. It is difficult to say whether the so-called "cerebral type" is a valid form of the effect of irritable carotid sinus. The presence of bradycardia and asystole and a drop in blood pressure can be easily recorded, but an unconscious state resulting from stimulation of the brain-stem centers from an irritable carotid sinus is more difficult to prove, particularly if the compression were severe enough to produce an ischemic effect, which may also cause an unconscious state.

(2) When the carotid arteries are compressed sufficiently to cause an occlusion of the internal carotid artery an ischemic effect may result, characterized by syncope in about 15-25 seconds of compression. If the opposite carotid artery is adequate, if the basilar circulation is adequate, compression of the internal carotid artery on a given side may not cause any abnormality in the absence of an irritable carotid sinus. If, on the other hand, there is a partial occlusion of the internal carotid artery on the opposite side, if there is a thrombosis of the internal carotid artery on the opposite side, if there is a generalized atheromatosis of the brain so that compression of the carotid on either side causes an inadequate amount of the blood flow to the circle of Willis, a cerebrovascular insufficiency with syncope may occur. In the presence of inadequate posterior and anterior communicating arteries in the circle of Willis, cerebrovascular insufficiency may occur when the carotid arteries in the neck are compressed on the side of the small or absent communicating vessels. We have been particularly impressed by the ischemic type of syncope in the past 5 years because of the amazing frequency of internal carotid artery thrombosis in the neck. In most of these patients compression-occlusion of the patent carotid artery causes syncope. The same is also true in most patients who have had one carotid artery, either internal or common, ligated for a cerebrovascular abnormality. Compression of the normal carotid artery for 15-25 seconds in such a case frequently results in syncope soon after the ligation. In some patients tested 6 months to 2 years after the ligation there has developed sufficient conditioning of the cerebral circulation so that compression of the patent carotid artery does not cause a completely unconscious state but does result in many instances in dizziness, extreme nervousness and a feeling of faintness.