LIGATION OF THE INTERNAL CAROTID ARTERY IN THE NECK

PREVENTION OF CERTAIN COMPLICATIONS

JAMES L. POPPEN, M.D.

Department of Neurosurgery, The Lahey Clinic, Boston, Massachusetts

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The sudden interruption of normal blood flow through the internal carotid arteries extracranially is recognized as a hazardous procedure in that it may result in temporary or permanent disability. The fact that many ingenious methods of ligation of the internal carotid artery have been described indicates that occlusion with a single ligature is not safe. In my experience there is no safe method of ligation. It should be executed only when an absolute emergency arises in an attempt to save life after every means known today have been utilized to determine the adequacy of the collateral circulation of the circle of Willis to carry sufficient blood supply to the hemisphere in which the direct flow will be greatly diminished by the ligation of an internal carotid artery.

Unfortunately, the circle of Willis in many instances is malformed, especially in the region of the posterior communicating arteries and, to a lesser degree, of the anterior communicating arteries. In these patients, ligation of an internal carotid artery is almost certain to be attended with a high morbidity as well as mortality, regardless of the method of ligation or occlusion.

The competency of the collateral circulation can usually be predicted with reasonable accuracy by temporary interruption of the blood flow through the internal carotid artery with digital compression. False impressions may be gathered from digital compression of an internal carotid artery. A patient with normal collateral circulation will tolerate complete occlusion by digital compression indefinitely; usually 10 minutes suffices to determine whether or not a patient can tolerate occlusion permanently. In many patients, untoward symptoms, such as dizziness, slowing of the pulse, numbness or weakness of the contralateral side or syncope, may be initiated within a few seconds or minutes after digital compression has been instituted. It is important to determine whether these symptoms are the result of ischemia from a faulty collateral circulation or of vasospasm produced by an irritable carotid sinus. The differential diagnosis can usually be determined by occluding the internal carotid artery well above or well below the carotid sinus. If similar subjective symptoms are produced by pressure well away from the carotid sinus, one must assume that the symptoms are the result of inadequacy of the collateral circulation. Another relatively satisfactory method is to interrupt the carotid sinus temporarily with local infiltration of procaine. It is difficult at times to be certain that the carotid sinus has been adequately anesthetized. If no untoward subjective symp-
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Symptoms occur from the digital compression following infiltration of the carotid sinus with a local anesthetic agent, one is justified in assuming that an irritable carotid sinus was responsible for producing the symptoms, and not a faulty collateral circulation.

The indications for ligation of the internal carotid artery in the neck are becoming more frequent since intracranial saccular aneurysms are being visualized before death by the use of arteriograms.\(^3\),\(^4\),\(^5\),\(^6\),\(^7\) Ligation of the internal carotid artery may be necessary in an acute emergency arising from gunshot wounds, lacerations, or erosion of the wall of an internal carotid artery by a malignant lesion or a knife wound. In these patients immediate ligation is necessary to save life, repair of the rupture being rarely possible.

In the elective ligation such as deemed necessary for intracranial lesions, the method of occlusion is highly important. I fully realize that any method utilized in a large number of patients may seem perfectly safe. However, in my previous experiences\(^1\),\(^2\) with partial occlusion or occlusion in stages, satisfactory results were obtained in the first 40 patients, then 3 tragic delayed hemiplegias followed. Every effort should be made to prevent temporary or partial complete disability from ligation of the internal carotid artery if a patient has adequate collateral circulation which has been demonstrated to be true for a period of 36 to 48 hours, and then the patient be subjected to a sudden catastrophe due to thrombosis or embolus. It is important, therefore, when the collateral circulation has been demonstrated to be satisfactory, that every precaution is utilized to prevent these delayed tragedies. The cause of thrombosis in many instances is injury of the intima of the artery at the site of ligation. I believe that ligation of the internal carotid artery is less dangerous than ligation of the common carotid artery. The reason for this impression is that the caliber of the common carotid artery is usually \(1 \frac{1}{2}\) to 2 times that of the internal carotid artery. I realize that theoretically the danger of ligation of the common carotid artery should be materially less since interruption of the internal carotid artery diminishes the blood flow into the ipsilateral hemisphere much more completely than does ligation of the common carotid artery (Fig. 1). With the latter the blood flow is still possible by retrograde circulation through the external carotid artery into the internal carotid artery (Fig. 2), whereas with ligation of the internal carotid artery all blood that supplies the hemisphere is interrupted from the neck, with the possible exception of the slight collateral circulation of the external carotid artery through the ophthalmic artery.

Ligation of the internal carotid artery is less dangerous than ligation of the common carotid artery because first, the caliber of the common carotid artery is greater than that of the internal carotid artery, and second, atheromatous plaques in the common carotid artery in the immediate vicinity of the carotid bulb are much more common than in the internal carotid artery. A ligature placed over an atheromatous plaque may well cause a fracture of the plaque, resulting in injury to the intima, thus developing an excellent precursor to ascending thrombosis or embolus. That
this actually happens was vividly demonstrated in a patient subjected to thyroidectomy on the general surgical service, during which procedure the common carotid artery was retracted laterally in the usual manner. The patient did not regain consciousness; when he recovered from anesthesia it was obvious that a complete contralateral hemiplegia had developed. Death occurred within 24 hours. Postmortem examination demonstrated a recent thrombosis in the entire right internal carotid artery, extending from

![Diagram of arteries](image)

**Fig. 1 (left).** Complete ligation of internal carotid artery.
**Fig. 2 (right).** Ligation of common carotid artery with blood flow into internal carotid from external carotid artery.

a fractured atheromatous plaque in the common carotid artery just below its bifurcation at the site of retraction. It is important, therefore, during manipulation of the carotid system that extreme gentleness be used in palpating the artery; nevertheless, it should be done to prevent placing a ligature in the immediate vicinity of an atheromatous plaque.

Another instance of injury to all layers of a large vessel from ligation was demonstrated by the formation of a pulsating mass in the necks in 3 patients, 3 hours, 6 months and 12 months, respectively, following ligation. Obviously, the ligature had cut through the wall of the artery in the first instance at the site of a markedly sclerotic plaque. In the other 2 patients, aneurysms had developed at the ligature. In one patient sudden rupture of the aneurysm
took place and death occurred. The other patient was saved by excision of the aneurysmal sac in the neck.

In well over 100 ligations of the carotid system, permanent partial hemiplegias took place on the contralateral side in 4 patients. Three of these patients, however, are able to carry out their usual activities, but they do have definite residual symptoms as a result of the thrombosis.

One patient with hemiplegia on the ipsilateral side following ligation died as the result of rupture of an aneurysm on the opposite side. It is this episode that has made us insist that bilateral arteriograms be taken of all patients subjected to ligation of an internal carotid artery for intracranial aneurysms so that we can be certain that another saccular aneurysm is not present on the opposite side. Fortunately, in our surgical experience at the clinic with 152 cases of intracranial aneurysms, 3 per cent of the aneurysms were bilateral and 7 per cent multiple but unilateral.

The following technic was utilized for 50 patients who have survived for more than 3 years.

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**Fig. 3.** (a) Line of incision. This is usually carried from the tip of the mastoid just anterior to the sternocleidomastoid muscle downward to a level 2 or 3 cm. below the bifurcation of the common carotid artery. (b) Polyethylene tubing inserted into sheath of superior cervical ganglion. (c) No. 00 catgut suture used to prevent displacement of tubing. At end of 3 days, the polyethylene tubing can be readily pulled out through the incision. Tubing is used as an accurate means of perfusing the ganglion continuously with a slow drip of procaine in an attempt to prevent possible vasospasm of intracranial vessels.
The anesthesia is of especial importance. It is necessary that the patient is cooperative, and therefore, regional or local anesthesia is preferable. After adequate premedication, most emotional patients can be managed if they are made aware of the importance of their cooperation. Many patients become very restless if the drapes are applied so that proper oxygenation is not possible. Adequate air or oxygen is imperative to prevent building up of carbon dioxide under the sterile drapes.

An oblique incision is made anterior to the sternocleidomastoid muscle, starting immediately below the tip of the mastoid process and carrying it down below the level of the bifurcation of the carotid artery (Fig. 3 a). The carotid sheath is allowed to remain in contact with the carotid arteries, dissecting the internal jugular vein and the vagus nerve from the sheath. It is usually necessary to ligate the common facial vein as well as the lingual and the superior thyroid veins to allow adequate exposure of the internal carotid artery and the carotid bulb. The carotid sinus is anesthetized by infiltrating a small amount of procaine directly into it. The external carotid artery is separated from the internal carotid artery by incising the adventitial layer and separating it from the lateral wall of the external carotid. This allows the full thickness of the adventitia to be utilized in forming a natural band for constricting the internal carotid artery. The lumen of the internal carotid artery is then obliterated by interrupted black silk sutures, as shown in Fig. 4 a, b and c. Three layers usually suffice to occlude the lumen completely. It is, of course, obvious that the adventitial layer will stretch as time goes on and allow the lumen to reopen. I have no factual evidence as to the length of time this would take. To prevent this, however, two large black silk sutures are placed, one on the extreme cephalad end and the other on the extreme caudad end of the occlusion, usually 3 to 3.5 cm. apart (Fig. 4 e). These ligatures are tied snugly but not so tight as to injure the intima. At the conclusion of the ligation the sheath over the superior cervical ganglion is opened to allow a small polyethylene tubing to be inserted so that a constant drip of procaine may perfuse the ganglion for 2 or 3 days following the operation (Fig. 3 b and c). The tubing is kept in place with plain, No. 2 or 3 catgut sutures so that the tubing can be readily removed at the end of 3 days. This is done with the hope that it will prevent vasospasm and allow maximum collateral circulation during the crucial time following ligation.

The patient is in the horizontal position throughout the operation so as to maintain a normal blood pressure during and following the operation. Careful hemostasis is instituted so that heparin may be given if necessary following operation. It had been my opinion that heparin should be given 4 hours postoperatively and continued for 3 or 4 days. It was necessary to modify this opinion following an experience in which heparin was given in a case in which the aneurysm had recently ruptured. Several hours after administration of the heparin a fatal subarachnoid hemorrhage took place. Heparin is given to all patients 4 to 6 hours following ligation who have not
had a subarachnoid hemorrhage within 3 weeks previous to the ligation and also to all patients who have large, inoperable intracranial aneurysms and who have shown no signs of subarachnoid hemorrhage but who have local neurologic changes.

The blood pressure must be carefully watched throughout the operation and following the operation. There is very little question that ligation performed at the time of a lowered blood pressure is conducive to serious sequelae. The patient is returned to the ward with the bed in shock position and the patient is kept in an oxygen tent 12 to 24 hours. Proper oxygenation is worthwhile in a patient whose rate of circulation is greatly lowered as a result of the ligation.

The operating room is kept prepared for the immediate removal of the ligatures if untoward signs are noted in the first 4 hours. If hemiplegia occurs after 4 hours, removal of the ligatures has not been beneficial. Symptoms from ischemia as a result of occlusion usually manifest themselves well within that time.

Following operation the site of ligation in the neck must be carefully handled. No attempt should be made to palpate the neck since, if thrombosis has occurred at the site of ligation, a thrombus may be dislodged.

Thrombosis and embolus formation probably occur less frequently in cases of ligation of the internal carotid artery in which immediate division of the artery is carried out rather than ligation in continuity. There is no recourse, however, if hemiplegia occurs in 2 to 3 hours following ligation, as happens in a few cases.

![Diagram](image-url)  
**Fig. 4.** Imbrication of the large vessel by utilizing the adventitial layer.
Ligation of the internal carotid artery in a patient in shock will increase both the mortality and the morbidity. The blood pressure should be elevated by transfusions if ligation becomes necessary as a life-saving mechanism in an actively leaking aneurysm. An actual "blow-out" of an intracranial aneurysmal wall will result in death so quickly that operation is out of the question unless by pure chance the rupture occurred as the internal carotid artery was exposed immediately prior to ligation. It is doubtful that ligation at such a time would save the patient's life.

REFERENCES