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Advances in skull base surgery have resulted from the ability to perform large scale en bloc resections of neoplasms affecting the clivus, sphenoid wings, and other deep structures. Such tumors frequently involve the internal carotid artery (ICA), and complete resection may require dissection or sacrifice of the ICA. Occlusion of the ICA without bypass graft placement is performed for the treatment of certain aneurysms. These and other less common indications for ICA sacrifice necessitate our ability to predict the result of ICA occlusion.

Without any type of temporary test occlusion, the incidence of stroke after permanent carotid artery occlusion ranges from 17% to 30% (1-5). In 1911, Matas (6) described temporary arterial occlusion by manual compression of the common carotid artery to determine tolerance for permanent arterial occlusion. Serbinenko (7) introduced the concept of endovascular arterial occlusion using small endovascular balloons in the early 1970s. His novel method of endovascular arterial occlusion has since been widely adopted and remains the current foundation for temporary arterial test occlusion.

When carotid artery Balloon Test Occlusion (BTO) is clinically tolerated, the morbidity and mortality associated with permanent arterial occlusion are reduced but, unfortunately, not eliminated. Since the report of endovascular temporary arterial occlusion presented by Serbinenko (7) was published, a variety of adjunctive methods have been tested to improve the sensitivity and specificity of clinical neurologic evaluation alone for the detection of insufficient cerebral blood flow to allow safe permanent arterial occlusion. Evaluation of regional cerebral blood flow during BTO using radioactive xenon with external probes (8, 9), stable xenon-enhanced CT (10-14), technetium-99m hexamethylpropyleneamine oxime single photon emission CT (15-17), and [15O] labeled H2O positron emission tomography (18) has been reported. Other indirect indicators of regional cerebral blood flow, including perfusion CT and MR imaging, angiography of collateral vessels (9, 19-21), measurement of arterial stump pressure (10, 22-26), EEG (27, 28), and transcranial Doppler ultrasonography (8), have been tested. Pharmacologic induction of hypotension during BTO has also been used to attempt to elicit clinical signs of inadequate perfusion (29, 30).

Although it seems logical that cerebral blood flow evaluation and/or other adjunctive tests described above performed during BTO should help to reveal marginal areas of perfusion that might lead to neurologic deficits after permanent arterial occlusion, this has not been convincingly shown. After BTO with clinical evaluation only, 198 patients underwent permanent ICA occlusion without bypass graft placement, with a 3.0% incidence of permanent neurologic deficits (20, 21, 31). After BTO combined with cerebral blood flow analysis or induction of hypotension, 120 patients underwent arterial occlusion without graft placement and had a 6.7% incidence of permanent complications (9, 15, 18, 29, 30, 32). Because the numbers of patients is relatively small and reported incidences of neurologic complications are low, statistically significant differences between the different techniques are very difficult to show. Considering the wide variations in occlusion techniques, post-occlusion care, patient selection, and limited numbers of patients in reported series, it is impossible to prove superiority for any particular method of BTO.

Indications

The indications for BTO include an aneurysm or pseudoaneurysm arising from the ICA (treatment by permanent ICA occlusion [Hunterian ligation] planned; at risk for inadvertent ICA occlusion during a difficult open or endovascular surgical approach); cranial and cervical neoplasms with ICA involvement; hemorrhage related to trauma, infection, or neoplasm; arterial dissection when anticoagulant therapy is contraindicated; and carotid-cavernous fistula, which may not be treatable with arterial preservation.

Threshold: 100%. A review should be conducted whenever BTO is performed for other indications.

Efficacy

Technical success or efficacy is defined as the ability to complete the BTO procedure. Atherosclerotic changes identified during the diagnostic angiographic portion of the procedure may prevent safe performance of BTO and should not be considered as a technical failure.

Threshold for technical success: 95%.

Clinical success or efficacy is defined as the ability to accurately predict the outcome of permanent arterial occlusion in terms of whether a permanent post-occlusion neurologic deficit develops.

Threshold for clinical success: 95%.

BTO of the vertebral arteries may also be performed for similar indications. Depending on the sizes of the vertebral arteries, tolerance for permanent vertebral artery occlusion is generally greater than that of the ICA. Thresholds for indications, success, and complications associated with
BTO of the ICA can be applied to BTO of the vertebral artery as well.

Safety

Reported complications associated with BTO include arterial dissection (both asymptomatic and symptomatic), transient and permanent neurologic deficits, death, and puncture site hematoma. The largest reported series of carotid artery BTO examined 500 cases treated by multiple operators at the University of Pittsburgh (13). They reported rates of 1.2% transient and 0.4% permanent neurologic complications, which are within the ranges reported for diagnostic angiography alone (33–35). Using other techniques, complication rates from carotid artery test occlusion as high as 15% have been reported (36). With careful attention to technique, complications associated with BTO should only slightly exceed those associated with diagnostic neuroangiography (37). A review should be prompted when the complication rate surpasses the following threshold value.

| Indicator                              | Threshold (%)
|----------------------------------------|----------------
| Asymptomatic arterial dissection       | 3              |
| Transient neurologic deficit           | 5              |
| (persisting after balloon deflation)   |                |
| Permanent neurologic deficit           | 5              |
| Death                                  | 0              |

References