Temporary Balloon Test Occlusion of the Internal Carotid Artery: Experience in 500 Cases

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PURPOSE: To describe experience with 500 temporary balloon occlusions of the internal carotid artery, with particular emphasis on the techniques and complications. **METHODS:** Temporary occlusion of the internal carotid artery was accomplished endovascularly using various balloon-catheter combinations. These temporary balloon occlusions were combined, when possible, with cerebral blood flow analysis with stable xenon–enhanced CT. **RESULTS:** Complications related to this procedure occurred in 16 (3.2%) patients. Eight (1.6%) patients had asymptomatic complications. There were 8 who experienced neurologic changes. Six (1.2%) of these were transient; two (0.4%) were permanent. There were no deaths. **CONCLUSIONS:** Temporary balloon occlusion of the internal carotid artery, believed helpful in identifying patients at risk of stroke during abrupt carotid artery sacrifice, can be performed with an acceptably low complication rate.

Index terms: Arteries, carotid, internal; Arteries, therapeutic blockade; Interventional neuroradiology, provocative testing

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Temporary balloon occlusion (TBO) of the internal carotid artery has become a well-accepted part of the preoperative evaluation of patients with aneurysm or tumor involving the neck or skull base in whom arterial sacrifice or prolonged temporary occlusion is considered a possible part of the surgical or endovascular therapy. Temporary balloon occlusion in conjunction with cerebral blood flow analysis is believed to help identify the subset of patients who will not tolerate permanent carotid occlusion. In this paper we review our experience with 500 temporary carotid occlusions and evaluate the associated technique and complications.

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Materials and Methods

All patients receive a baseline neurologic examination. A baseline stable xenon–enhanced computed tomography (Xe CT) cerebral blood flow examination is obtained as well. A 6F Terumo sheath (Meditech, Watertown, Mass) is first placed in the femoral artery by standard Seldinger technique. Diagnostic angiography precedes TBO and includes evaluation of both carotid arteries and the vertebrobasilar system. The angiogram is assessed for collateral vascular anatomy, vascular encasement, tumor vascularity, atherosclerotic disease, and secondary vascular abnormalities. The angiographic catheter is withdrawn and replaced with a balloon catheter, which will be used for TBO. Selection of the balloon catheter is based on anatomic findings from the diagnostic study as well as the intended site of endovascular occlusion (1). Under most circumstances, the internal carotid artery is temporarily occluded in a high cervical position at approximately the C-1 to C-2 level.

When the balloon catheter has been appropriately positioned, 7000 units of heparin are administered intravenously. A digital "road map" of the carotid artery is obtained to establish its approximate size. The balloon is inflated carefully during fluoroscopic observation, with the intent of producing minimal balloon distension that will produce carotid occlusion. With double-lumen catheters we simultaneously monitor intraarterial pressures and watch the arterial waveform during balloon inflation. The end point for balloon inflation is reached when maximal

damping of the arterial waveform occurs. Occlusion can be substantiated easily by infusing a small amount of contrast through the central catheter lumen and fluoroscopically observing a static contrast column. With single-lumen balloon systems, arterial pressure measurements and waveform monitoring are not possible. In this situation, occlusion can be documented by injecting contrast through the balloon catheter guiding sheath (which must be placed in the carotid artery for coaxial introduction of the balloon).

After arterial occlusion has been established, the patient's neurologic status is assessed and monitored continuously throughout the 15-minute period of endovascular occlusion. If there is a change in neurologic status, the balloon is immediately deflated and the procedure terminated. An internal carotid artery–occluded, stable Xe CT cerebral blood flow exam is subsequently performed on the group of patients who tolerate the 15-minute TBO without clinical change. Patients are transported from the angiographic suite to the CT scanner with the balloon deflated but still positioned in the internal carotid artery. A constant heparinized saline drip is continued through the central lumen of the balloon catheter when a double lumen catheter is used. During transportation to the CT scanner suite, careful attention is directed to preventing movement of the patient's head.

After the patient has been positioned in the CT scanner, the balloon is again inflated with the identical volume used to produce balloon occlusion during the clinical TBO. Digital scout radiographs of the head and neck in both anteroposterior and lateral projections are used to confirm adequate balloon position and reinflation. A Xe CT cerebral blood flow study is performed with the balloon inflated (GE Medical Systems, Milwaukee, Wis). The Xe CT cerebral blood flow method has been previously described (1,2). Balloon occlusion during this portion of the examination is limited to approximately 5 to 7 minutes. When the internal carotid artery–occluded cerebral blood flow study is completed, the balloon catheter is deflated and removed. Protamine sulfate reverses the residual heparin. The sheath is withdrawn and hemostasis achieved.

Results

Between June 1985 and January 1993, 500 TBOs of the internal carotid artery were performed at the University of Pittsburgh. Complications related to this procedure occurred in 16 (3.2%) patients (Table 1). Eight patients (1.6%) experienced complications that were asymptomatic, including dissection (Fig 1), embolus, and pseudoaneurysm (Fig 2).

Symptomatic neurologic complications occurred in an additional eight (1.6%) patients. Six (1.2%) experienced transient neurologic deficits which cleared completely. Two (0.4%) had permanent deficits which only partially resolved.

TABLE 1: Complications associated with 500 temporary balloon occlusions of the internal carotid artery

	No. of Patients (%)
Asymptomatic	8 (1.6)
Dissection	6 (1.2)
Pseudoaneurysm	1 (0.2)
Embolus	1 (0.2)
Neurologic Deficit	8 (1.6)
Transient	6 (1.2)
Permanent	2 (0.4)
Death	0

No significant complications could be attributed to the Xe CT cerebral blood flow portion of the exam. Groin hematomas were rare; none required surgical intervention. No deaths occurred in this series.

Discussion

TBO Technique

In the 500 temporary endovascular occlusions performed at this institution, experience has been gained largely with three different nondetachable balloon occlusion catheters: 5F (93-110-5F) Swan-Ganz (Edwards Laboratories, Anasco, Puerto Rico); ITC-NDSB (1509) (Interventional Therapeutics, San Francisco, Calif); and 5F (OB/5/2/100) and 7F (OB/7/2/100)

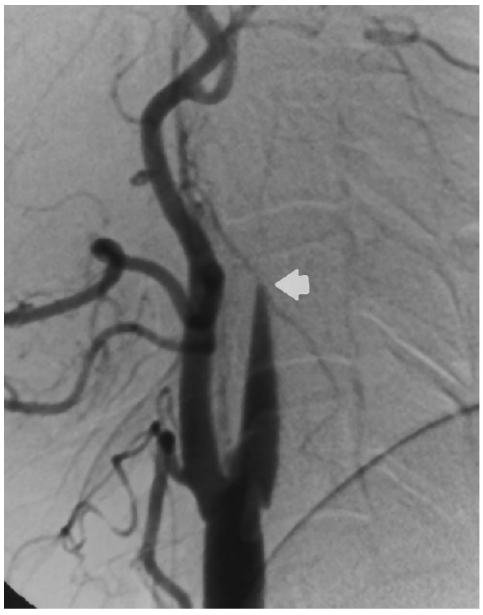

Fig 1. Common carotid arteriogram after temporary balloon occlusion reveals an asymptomatic internal carotid artery dissection and total occlusion (*arrow*).

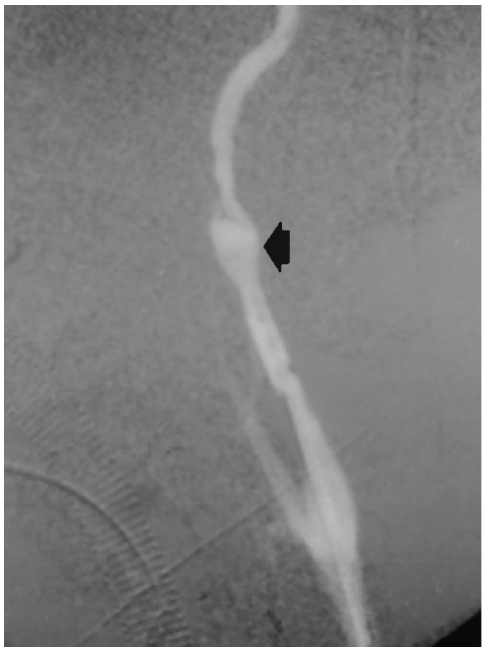

Fig 2. An intraoperative carotid arteriogram demonstrates a pseudoaneurysm (*arrow*) at the site of prior temporary balloon occlusion.

Meditech balloon catheters (Boston Scientific, Meditech, Watertown, Mass).

By far our most commonly used balloon is the Swan-Ganz because of its low cost, ease of introduction (without need of an exchange wire), double-lumen configuration (allowing pressure measurements and contrast injection beyond the point of balloon occlusion), and reasonably compliant balloon properties. The tip of the 5F Swan-Ganz catheter is easily formed over steam into a common cerebral configuration (eg, JB 2), which facilitates selection of the innominate and left carotid origins. Slight inflation/deflation usually allows flow-guided positioning of the balloon in the internal carotid artery at the C-1/C-2 level. If necessary, a 0.010-inch guide wire (Seeker Lite, Target Therapeutics, Fremont, Calif) can be passed through the Swan-Ganz lumen to help engage the internal carotid artery origin and/or direct the catheter. Alternatively, a 5F Meditech balloon catheter can be placed into the internal carotid artery over a 0.025-inch exchange wire. This balloon catheter system also has a double-lumen configuration. Both the Swan-Ganz and Meditech catheters have latex balloons that exhibit similar compliance properties (4).

In situations in which arterial narrowing or vessel tortuosity preclude the relatively large or stiff balloon-catheter systems, the ITC-NDSB (1509) is a good alternative. This balloon-catheter system is the most compliant of those used, requiring the least pressure during inflation and generating minimal pressure on the vessel wall when inflated beyond the volume needed for occlusion (4). Its small size (1.5 mm for the 1509) and supple shaft allow safe positioning beyond tortuous areas of the internal carotid artery in most cases. Drawbacks of this system include the need for a 7.3F (minimum size) introductory guiding sheath and the fact that it is of single-lumen configuration (precluding the possibility of contrast injection through the catheter or distal pressure measurements during balloon occlusion).

Several authors describe use of the 7F Meditech balloon system (8, 11). However, it should be noted that this balloon produces considerably more radial vessel wall pressure on the vessel if any overinflation occurs during balloon occlusion (4). This increased radial wall pressure raises the risk of vessel injury with the attendant possibility for embolic events or pseudoaneurysm formation.

Once the balloon system is in place, a digital roadmap of the internal carotid artery is made through the central catheter lumen (or guiding sheath in the case of a single-lumen catheter configuration). When using double-lumen balloon systems (Swan-Ganz and Meditech) the central lumen is connected to a transducer for continuous pressure monitoring.

Anticoagulation is achieved with an intravenous bolus of 7000 U of heparin. This quantity exceeds that used in our original publications (3, 6) and that commonly used by several other authors (11, 13, 14). This dose of heparin was an empiric choice attempting to ensure improved anticoagulation in all patients and was prompted by several transient ischemic attacks early in our experience when using a 5000-U bolus. We now have used this amount of heparin in more than 300 patients without hemorrhagic complication and with a reduced rate of transient ischemic attacks.

Proper balloon inflation is critical to safe execution of this procedure. Previous publications suggested that internal carotid artery occlusion could be judged by change in balloon appearance from spherical to slightly elongated (6, 7). We no longer recommend this method of validation of internal carotid artery occlusion and

believe this degree of balloon extension may contribute to intimal injury. Minimal inflation to create full damping of the arterial pressure wave (measured through the balloon catheter lumen) will produce occlusion without overextension of the arterial lumen. Complete occlusion should be confirmed by injecting contrast material through the central catheter lumen (or guiding sheath in the case of the ITC-NDSB), producing a static contrast column. Flushing the internal carotid artery with heparinized saline prevents prolonged exposure of the intima to contrast after confirming occlusion.

A baseline neurologic examination is obtained on all patients before TBO. Continuous neurologic testing is performed throughout the 15-minute test occlusion (by the same individual who obtains the baseline exam). Neurologic change demands immediate balloon deflation and termination of the procedure. Temporary occlusion times from the literature vary from 15 to 120 minutes (7, 9–11). We have elected to avoid prolonged occlusion because vascular injury has been documented in animals during balloon occlusion and is directly related to increasing occlusion time (Gyorke et al, "A Comparison of Temporary Arterial Occlusions Using Intraluminal Occlusive Balloon Catheters vs Temporary Aneurysm Clips in Pigs," presented at the 31st annual meeting of the American Society of Neuroradiology, Vancouver, Canada, May 16–20, 1993). Our occlusion time of 15 minutes was chosen to show acute collateral insufficiency and give a conservative margin of safety for internal carotid artery–occluded, stable Xe CT cerebral blood flow analysis that follows the clinical test occlusion. (Occlusion time for stable Xe CT is approximately 5 to 7 minutes). Without cerebral blood flow analysis, many authors have chosen a longer temporary occlusion time. If there is no neurologic change during the temporary occlusion, the TBO is followed, when possible, with an internal carotid artery–occluded, stable Xe CT cerebral blood flow exam. After the Xe CT cerebral blood flow analysis, the balloon is deflated and then removed. Sheath removal follows reversal of heparinization with intravenous protamine (10 mg of protamine per 1000 U of heparin is given in a dose calculated based on a 1.5-hour heparin half-life) (12).

Complications

A total of 16 (3.2%) complications occurred in this series of 500 TBOs. Complications are divided into eight (1.6%) patients who remained asymptomatic and 8 (1.6%) with neurologic deficits (Table 1). In the asymptomatic group, there were six dissections (Fig 1), one middle cerebral artery embolus, and one pseudoaneurysm (Fig 2) that were found on follow-up angiograms or at the time of surgery. In the symptomatic group, seven of eight (1.4%) neurologic deficits were immediate, occurring before catheter removal. Six (1.2%) of these were transient and two (0.4%) were permanent. There were no deaths in this series.

Comparing our complications with those of other series is difficult because no other similar large series exists. The incidence of neurologic complications from seven published reports (8–11, 13–15) range from 0% to 8.3% (Table 2). A cumulative total of 181 TBOs are reported, and there are four (2.2%) neurologic complications from this group. This compares favorably with the 1.6% incidence of neurologic complications in our series.

Our 1.6% (0.4% permanent) incidence of neurologic complications is similar to complication rates recently reported for diagnostic cerebral angiography. Dion (16), in a prospective evaluation of 1002 cerebral angiograms, found a 1.3% (0.1% permanent) incidence of neurologic complication. Ernest (17) prospectively evaluated 1517 cerebral angiograms and reported a 2.6% (0.33% permanent) incidence of neurologic complication.

Our asymptomatic complications are unique. The large number of follow-up angiograms after surgery may explain the identification of this classification of complications not reported by other authors. Without question, positioning and inflation of the balloon occlusion device in

TABLE 2: Complications in recent temporary balloon occlusion series

Series	No. of Cases	Complications
Moody (8) 1991	6	0
Monsein (9) 1991	11	0
Simonson (10) 1992	12	1 (8.3%) transient
Fox (13) 1987	67	2 (2.9%) transient
Anon (14) 1992	40	1 (2.5%) permanent
Gonzalez (15) 1990	27	0
Peterman (11) 1991	17	0
Total	**181**	**4 (2.2%)**

the internal carotid artery offers ample opportunity for vascular damage. The internal carotid artery–occluded Xe CT exam requires patient transport to the CT suite with the balloon catheter deflated in the internal carotid artery. Reinflation in the CT suite for 5 to 7 minutes follows CT positioning. Although these maneuvers potentially increase the risk of balloon-associated complications, our results do not appear to be adversely affected in comparison with TBOs without cerebral blood flow analysis (8–11, 13–15). Most neurologic (symptomatic) complications occurred early in the course of the 15-minute, clinically monitored TBO performed before the Xe CT cerebral blood flow study. These were distinguished from simple clinical TBO failures by their persistence (greater than 1 hour) or occurrence after the termination of balloon occlusion. Rarely did neurologic events have their onset after the termination of the TBO.

Implications of TBO and Cerebral Blood Flow Analysis

Before the advent of temporary test occlusion of the internal carotid artery, abrupt internal carotid artery occlusion was associated with a high morbidity and mortality rate. Linskey et al, using a literature control series of abrupt internal carotid artery occlusions without testing, found a cumulative stroke rate of 26% and mortality rate of 12% (25). Strokes were common and often large, resulting in a high associated mortality rate.

Patients who fail TBO have been shown to be at extremely high risk of stroke associated with internal carotid artery sacrifice or prolonged temporary occlusion. For obvious reasons, there are few patients in this category who have undergone unprotected internal carotid artery occlusion. At the University of Pittsburgh, 100% of these have experienced an associated neurologic deficit (3, 5, 24).

Alternatively, those patients clinically passing TBO have demonstrated a relatively low risk of stroke associated with internal carotid artery sacrifice. A literature review of internal carotid artery sacrifice after clinical passage for a TBO alone revealed nine (4.7%) permanent strokes in 192 patients (13, 14, 26–28). The mortality rate was zero.

The addition of cerebral blood flow analysis to TBO has been postulated to improve the detection of patients at risk of ischemic stroke after internal carotid artery sacrifice (23). Linskey et al looked at 30 patients who underwent permanent internal carotid artery sacrifice after passing combined TBO and internal carotid artery–occluded, stable Xe CT cerebral blood flow analysis (25). The permanent stroke rate in this group was 3% (zero mortality) and indicates that TBO, with or without cerebral blood flow analysis, has improved the safety of selected internal carotid artery sacrifice over internal carotid artery sacrifice without testing.

There remains the issue of whether it is necessary to detect the category of patients clinically passing TBO yet having low cerebral blood flow (ipsilateral cerebral blood flow below 30 mL/100 g per minute). Normal cerebral blood flow is approximately 54 mL/100 g per minute with a standard deviation of 12 mL/100 g per minute (18–22). Patients having an ipsilateral decrease in cerebral blood flow during TBO below 30 mL/100 g per minute have been considered, at our institution, to be at a risk of ischemic stroke that is intermediate between those failing TBO and those passing both TBO and cerebral blood flow analysis. This intermediate risk is suggested by Sen et al, in whose study patients undergoing internal carotid artery bypass grafting underwent prolonged, temporary internal carotid artery occlusion of 1 hour, 45 minutes to 5 hours (24). Seven percent of those passing both TBO and stable Xe CT cerebral blood flow awoke with a neurologic deficit. In the group of patients clinically passing TBO but having a cerebral blood flow of less than 30 mL/100 g per minute, there was a 56% immediate neurologic deficit rate. All patients (100%) clinically failing TBO awoke with a deficit.

The group of patients comprising this intermediate category make up approximately 11% of the population undergoing TBO (5). This intermediate category seems to be real and, from the data above, worthy of identification. However, results of the comparison of complications associated with internal carotid artery sacrifice in patients having TBO alone versus TBO with cerebral blood flow analysis are not statistically significant at present (23). Continued evaluation of these techniques, as well as alternative methods (such as evaluation of hemispheric venous outflow rates during TBO), is warranted.

Conclusion

Analysis of balloon test occlusion coupled with stable Xe CT cerebral blood flow in 500 patients reveals a low complication rate, not significantly different from TBO alone or diagnostic cerebral angiography. Several catheter systems are available that allow TBO of the internal carotid artery to be performed safely in almost all situations. Knowledge of the physical characteristics of these various systems, along with operator experience, helps to avoid pitfalls and complications. Finally, TBO has markedly improved our surgical safety by identifying those at high risk of hemodynamic stroke associated with internal carotid artery sacrifice.

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