Prediction of tolerance to carotid artery occlusion using transcranial Doppler ultrasound

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Surgical sacrifice of the carotid artery is a frequently anticipated event during the treatment of certain aneurysms and tumors. The ability to predict tolerance to carotid artery occlusion is therefore of benefit when planning procedures in which the carotid artery is at risk. A trial of carotid artery occlusion using an angiographic balloon during concurrent neurological examination or blood flow studies is an accepted method for testing tolerance, but it carries the risks of an angiogram and cannot be performed at the bedside. Transcranial Doppler ultrasound (TCD) is a noninvasive modality that permits measurement of blood velocity in cerebral vessels. The immediate effects of carotid artery occlusion on middle cerebral artery (MCA) perfusion can be obtained by insonating this artery during manual carotid artery compression.

To compare the TCD response to carotid artery compression with the data obtained with more formal testing, the MCA of 22 patients was insonated during manual carotid artery compression and the results compared with the clinical tolerance to balloon occlusion in all patients and to blood flow studied by single photon emission computerized tomography before or during balloon occlusion in 14 of the 22 patients. Surgery was planned to treat giant unruptured aneurysms in 17 cases, intracranial tumors in three, a carotid-cavernous fistula in one, and a carotid artery injury in one. Fifteen patients showed a reduction in TCD flow velocities by no more than 65%; of these, 14 (93%) clinically tolerated the balloon occlusion test. Of the seven patients showing a TCD flow velocity decrease of more than 65%, six (86%) developed a transient focal deficit during the occlusion. It is concluded that the change in MCA velocity measured with TCD studies during manual carotid artery occlusion is a useful predictor of the clinical and blood flow responses to a trial of carotid artery occlusion with an angiographic balloon.

KEY WORDS - carotid artery occlusion - transcranial Doppler ultrasound - balloon occlusion test - middle cerebral artery

Carotid artery occlusion is frequently a planned event during the treatment of certain complex aneurysms and tumors.\(^1\)\(^2\)\(^3\) and prediction of clinical tolerance to this procedure has accordingly received much attention. The Matas test, in which the carotid artery is manually occluded for 10 to 15 minutes during patient observation, was perhaps the earliest and most straightforward test but suffered from lack of sensitivity.\(^1\)\(^2\) More recently, trial occlusion of the carotid artery with an angiographic balloon has been performed with concurrent neurological examination or measurement of cerebral blood flow.\(^1\)\(^2\) Both methods require interventional angiography and transport of the patient to an examination suite, and neither can be performed frequently. However, an indication of the adequacy of perfusion during carotid artery occlusion can be obtained at the bedside noninvasively by insonating the middle cerebral artery (MCA) by means of transcranial Doppler ultrasound (TCD) studies during brief manual compression of the ipsilateral carotid artery.\(^6\)

The purpose of this report is to compare this quick bedside test with the results of formal balloon occlusion and of single-photon emission computerized tomography (SPECT) measurements. Our aim was to further the interpretation of TCD studies in which MCA flow velocity is noted to fall significantly during provocative maneuvers.

Clinical Material and Methods

Patient Population

Twenty-two adult patients were studied during evaluation for elective carotid artery occlusion. The indications for surgery were giant unruptured aneurysms
in 17 cases, intracranial tumors in three, a carotid-cavernous fistula in one, and a carotid artery injury in one.

**Balloon Occlusion Test**

All patients underwent trial occlusion of the cervical internal carotid artery with an angiographic balloon while awake and under constant neurological observation. The occlusion was continued for 30 to 45 minutes or until a focal deficit was observed.

**Transcranial Doppler Ultrasound Protocol**

All patients received trial manual compression of the carotid artery prior to balloon occlusion with simultaneous monitoring of the ipsilateral MCA according to standard criteria 15,16 (Fig. 1). The carotid artery was slowly and gently compressed for a period of 3 to 5 seconds. The portion of the MCA chosen for insonation was well distal to the carotid artery bifurcation (50 to 55 mm deep to the ultrasound probe) so that no potentially confounding collateral flow through the anterior and posterior communicating arteries would be detected in the sample volume. The mean percentage drop in velocity during compression compared to baseline was calculated.

**SPECT Studies**

In 14 patients, SPECT studies were obtained at baseline and during trial balloon occlusion. Acquisition and processing of the images were performed as described previously. 17 A relative quantification of perfusion asymmetries was calculated for each study. 17 Four transaxial slices, approximately 1.5 cm apart, were selected, beginning at the level of the superior border of the corpus callosum and extending inferiorly. Each slice was divided into eight pie-shaped regions of similar size. The two middle regions of each slice were considered representative of the MCA distribution. The average count per pixel in each of these middle regions was divided by the average count per pixel in the whole slice, with the average of these values obtained for the four representative slices. The asymmetry index was defined as the normalized count per pixel of the following calculation: (occluded side - nonoccluded side)/nonoccluded side; this formula was used for both the temporary occlusion study and the baseline study. The SPECT index was then calculated for each MCA region by subtracting the asymmetry index of the occlusion study from that of the baseline study; the maximum decrease between baseline and occluded studies was used for this analysis.

**Data Analysis**

Visual inspection of the data suggested that a threshold of velocity decrease of 65% would best identify the group of patients who would not tolerate balloon occlusion. Sensitivity, specificity, and positive predictive value were calculated using this parameter.

**Results**

Of the 22 patients studied, 15 showed a fall in MCA flow velocity of no more than 65%; 14 (93%) of these patients clinically tolerated the balloon occlusion test. The remaining seven patients showed a fall in MCA flow velocity of more than 65%; six (86%) of these failed the balloon occlusion test and developed a focal deficit before completion of the 45-minute observation period (Fig. 2). The 95% confidence interval for the percentage drop in MCA flow velocity was 37% ± 19% (mean ± standard error of the mean) in the group tolerating balloon occlusion and 78% ± 58% in the group not tolerating balloon occlusion. The sensitivity of the TCD occlusion test compared to the balloon occlusion test was 86%, the specificity 93%, and the positive predictive value 86%.

There was a linear correlation between the SPECT
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![Graph comparing decreases in middle cerebral artery (MCA) flow velocity during manual carotid artery compression to clinical tolerance of a trial of carotid artery occlusion. Some data points have been spread slightly for clarity.](image)

Fig. 2. Graph comparing decreases in middle cerebral artery (MCA) flow velocity during manual carotid artery compression to clinical tolerance of a trial of carotid artery occlusion. Some data points have been spread slightly for clarity.

![Scatterplot comparing decreases in middle cerebral artery (MCA) flow velocity during transient manual compression of the ipsilateral carotid artery compared to the single-photon emission computerized tomography index described in the text. There is a linear correlation with a correlation coefficient of 0.80.](image)

Fig. 3. Scatterplot comparing decreases in middle cerebral artery (MCA) flow velocity during transient manual compression of the ipsilateral carotid artery compared to the single-photon emission computerized tomography index described in the text. There is a linear correlation with a correlation coefficient of 0.80.

index and the percentage decrease in MCA flow velocity, with a correlation coefficient of 0.82 (p < 0.001) (Fig. 3).

**Discussion**

Blood flow velocity in the MCA can be measured with TCD during manual carotid artery compression to directly observe the hemodynamic effects of carotid artery occlusion. A normal response to this compression is an attenuation of velocity to 40% to 60% of baseline; attenuation of velocity to 0% can be compelling evidence of dependence upon carotid artery perfusion. This study has confirmed that the degree of fall in velocity during carotid artery compression correlates well with more formal testing in the angiographic suite with balloon occlusion and SPECT measurement of cerebral blood flow.

Although TCD compression can be performed quickly and serially at the bedside, there are several technical limitations. In some patients ultrasonic study windows are poor, and the resulting attenuated signals can be deceptive and may vanish artifactually even when flow is not significantly affected. This seemed to be the case in the one patient in our study whose flow velocity fell to 0% but who tolerated the balloon occlusion test. This was an elderly woman with poor ultrasonic study windows who underwent TCD early in our experience. Another difficulty arises when carotid artery compression cannot easily be performed; accordingly, we do not test patients with extensive cervical tumors. The TCD compression test monitors only one branch in the MCA territory alone. We have seen a patient in whom the MCA flow velocity changed minimally during carotid artery compression, while the SPECT studies showed a decrease in the posterior cerebral distribution; this was believed to be due to a fetal origin of the posterior cerebral artery.

The threshold of 65% in our data was chosen from examination of the small number of patients in the groups tolerating and not tolerating balloon occlusion, and the relatively large confidence intervals confirm the resulting uncertainty in the 65% threshold. Nevertheless, this threshold is similar to those in other studies demonstrating electroencephalographic (EEG) changes, stumps pressures, collateral flow, and clinical outcome during carotid artery cross-clamping at carotid endarterectomy. In a recent review it was noted that a decrease in MCA flow velocity of at least 65% during carotid endarterectomy was necessary to cause changes in the somatosensory evoked cortical responses, and several authors have shown severe EEG changes occurring significantly more frequently when MCA velocity falls by more than 60%, 18, 22, 64%, 18 and 70%. 18 Stump pressure has also been noted to correlate with the degree of MCA flow velocity decrease, 18, 22 with a reduction of 60% corresponding to a fall in stump pressure to 25 mm Hg. A decrease in velocity by more than 64% is also associated with impaired collateral flow when evaluated by TCD examination. Finally, Ringelstein reported a group of 35 patients in whom flow velocity fell by less than 33% and in whom no deficit was noted despite prolonged occlusion times. Halsey reported an increase in the occurrence of infarct in patients treated without shunting during carotid endarterectomy when the velocity fell by more than 60%. Although approximate and based on data obtained while the patients were under general anesthesia, these thresholds suggest that a fall in MCA flow velocity by more than 60% to 70% during carotid artery occlusion is associated with a higher incidence of hemodynamic effects and are similar to the thresholds we found in our data.

The response of the cerebral vasculature to momentary carotid artery occlusion is not static and changes quickly due to autoregulation.
and Maurer, et al., noted a gradual increase over 5 to 20 seconds in MCA flow velocity during the occlusion period due to autoregulation; they based their analyses on these late changes, although we used the velocity decrease seen immediately upon carotid occlusion. Both the immediate and late changes in velocity seem to correlate with hemodynamic impairment of the cerebral circulation. The determination of which index best predicts long-term tolerance to carotid artery occlusion awaits further study.

We believe that the TCD compression test is relatively safe when performed gently and slowly, and have seen no morbidity in a large number of test compressions performed for a variety of reasons. A survey of the literature concerning carotid artery compression for provocative testing has revealed little morbidity during compression. Nevertheless, the clinician must ensure that the diagnostic benefit derived from a carotid artery compression test outweighs the small risk of its application. In selected cases it may be prudent to first perform a duplex ultrasound examination to rule out the occurrence of significant atherosclerotic disease; in one series this practice permitted performance of a carotid artery compressive maneuver in 40% of the patients.

Whether the internal or common carotid artery is occluded during manual compression is difficult to fully determine, and adds a level of uncertainty and a chance of a false-negative result. Nevertheless, we believe that there are advantages to a quick bedside evaluation of dependence upon carotid artery compression that can be incorporated into clinical decision-making. For example, despite uncertainties in the degree of occlusion, it is our opinion that a fall in velocity by more than 80% is compelling evidence for intolerance to carotid artery occlusion.

Although a fall in MCA velocity by more than 65% seems to predict intolerance to balloon occlusion, this study is relatively small and false-negative results can occur. Replacing the balloon occlusion test with the TCD test in all situations is therefore not warranted, although changes in TCD blood flow velocity confirm the results of balloon occlusion and might possibly be used to shorten occlusion time or to provide frequent serial evaluations. In addition, the TCD test can suggest the degree of hemodynamic dependence upon a carotid artery when the risk of undertaking an angiographic trial occlusion is not otherwise indicated.

Perhaps the greatest use of our results, however, is in the interpretation of TCD studies. We now believe that a fall in MCA flow velocity arising from maneuvers during a TCD examination is hemodynamically significant if greater than 65% of baseline.

The assessment of MCA flow velocity during manual carotid artery compression seems to correlate well with the clinical tolerance of carotid artery occlusion during formal testing and also correlates with a SPECT index of changes in asymmetry. This makes available a relatively noninvasive bedside assessment of tolerance to carotid artery occlusion that we consider reliably predicts intolerance when the velocity falls to 80% or less of baseline levels. The statistical threshold of 65% agrees with data of previous studies, and provides an interpretation of the importance of observing a fall in TCD flow velocity.

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References

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