Preoperative risk assessment for carotid occlusion by transcranial Doppler ultrasound

S Schneweis, H Urbach, L Solymosi, F Ries

Abstract

***Objectives*—An endovascular carotid balloon occlusion test with continuous intracranial monitoring by transcranial Doppler sonography was performed in 55 patients for prediction of tolerance of a required permanent occlusion of the carotid artery.**

***Methods*—Blood flow velocities of the ipsilateral middle cerebral artery during occlusion were recorded and compared with clinical tolerance during an occlusion test as well as with postoperative outcome after an eventual permanent occlusion. To stress the capacity of the cerebral circulation to tolerate the occlusion acetazolamide was injected before occlusion in all patients.**

***Results*—The onset of neurological symptoms during temporary occlusion was dependent on the percentage fall of mean blood flow velocity relative to baseline rather than on absolute flow velocities during the time of occlusion. Patients with a fall of mean flow velocity of less than 30% tolerated temporary and permanent occlusion, with the exception of two patients who developed an infarction due to thromboembolism after iatrogenic sacrifice of the carotid artery. Patients with a major decrease developed neurological symptoms during occlusion in 55% and, in cases of carotid ligation, a haemodynamic infarction occurred.**

***Conclusion*—The results show that transcranial Doppler monitoring as a part of an endovascular balloon occlusion test may be a reliable technique for preoperative risk assessment for permanent occlusion of the carotid artery.**

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Keywords: carotid occlusion; preoperative risk assessment; balloon occlusion test

Department of Neurology
S Schneweis
F Ries

Department of Neuroradiology, University of Bonn, Germany
H Urbach
L Solymosi

Correspondence to:
Dr Susanne Schneweis, Neurologische Universitätsklinik, Sigmund-Freud-Straße 25, D-53105 Bonn, Germany.

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Permanent occlusion of the carotid artery is sometimes required in the surgery of intracranial aneurysms and tumours as well as in neck surgery. Haemodynamic or embolic infarction after sacrifice of the carotid artery is the predominant risk of this procedure. Temporary occlusion of the carotid artery therefore has been recommended to predict the patient's tolerance to permanent occlusion. Techniques such as manual compression, ligation clamp, and balloon catheters have been used for a temporary carotid occlusion.[1 5] The occlusion by a double lumen balloon catheter is one of the safest and most reliable methods. By contrast with manual compression, adequate occlusion of the internal carotid artery can be proved angiographically. However, 5%-20% of the patients who tolerate temporary occlusion will develop an infarction after permanent occlusion of the carotid artery.[2] Therefore, additional techniques such as EEG, HMPAO-SPECT, xenon-CT, PET, stump pressure monitoring, and transcranial Doppler sonography were used for preoperative risk assessment.[1 4 6–14] Transcranial Doppler is a non-invasive method and allows continuous monitoring of haemodynamic changes that take place during the occlusion procedure. Vasomotor reactivity as well as collateralisation by the circle of Willis are two mechanisms of clinical tolerance of acute carotid occlusion.

A decline in cerebral blood flow as a result of reduced perfusion pressure leads to a dilatation of the cerebral resistance vessels. This mechanism of autoregulation can be simulated by stimulation with the carbonic anhydrase inhibitor acetazolamide (ACZ) as well as by hypercapnia.[15–18] Once the cerebral vasomotor reactivity capacity is exhausted, however, the brain is in danger of ischaemia in cases of additionally reduced perfusion pressure.

We report the results of 55 endovascular balloon occlusion tests monitored with transcranial Doppler. The aim of the study was to evaluate the validity of transcranial Doppler in the prediction of clinical tolerance in cases of permanent carotid occlusion. A standardised stimulation with ACZ was used as an additional stress test for the cerebrovascular reserve capacity.

Patients and methods

We examined 55 patients (31 men, 24 women, mean age 53·5, range 22–73 years) with a balloon occlusion test of the carotid artery under continuous monitoring with transcranial Doppler. Patients had cervical tumours (38), intracranial aneurysms (seven), intracranial tumours (nine), and an iatrogenic trauma of the intracranial internal carotid artery (one). Previous to the test occlusion all patients underwent a diagnostic angiography of both carotid arteries. During the test procedure continuous monitoring of the ipsilateral middle cerebral artery by a 2 MHz pulsed wave Doppler probe was performed. The M1 segment of the middle cerebral artery was insonated in a depth of 45 to 55 mm. Time

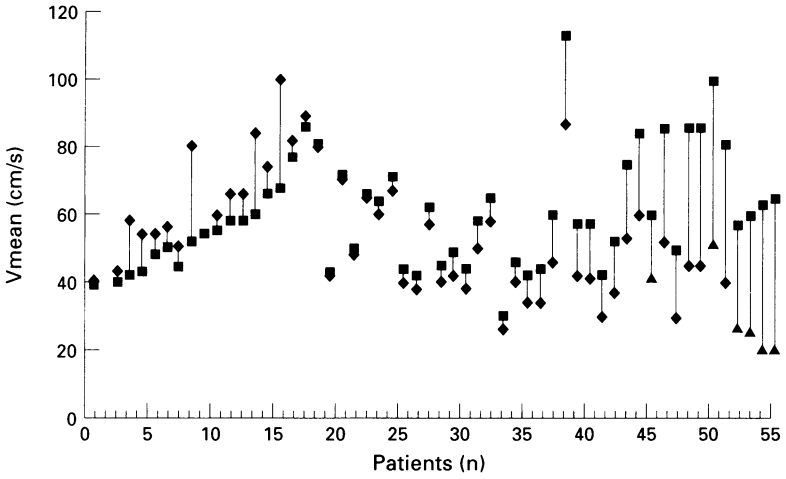
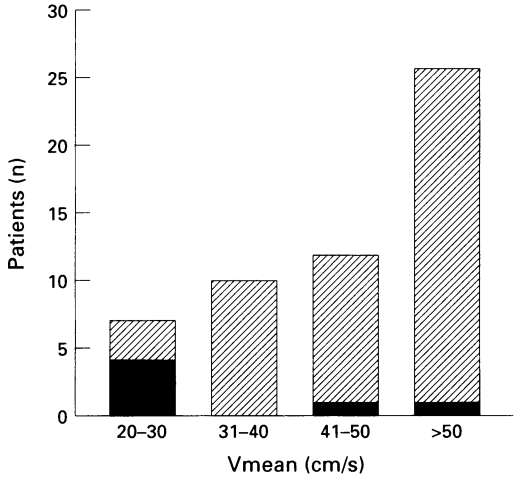


Figure 1 Mean flow velocities (cm/s) at rest and during test occlusion in 55 patients. Vmean at rest (■); Vmean during occlusion in patients who tolerated test occlusion (◆); Vmean during occlusion in patients with neurological symptoms during test occlusion (▲).

Figure 2 Incidence of neurological deficits during test occlusion related to Vmean (cm/s). All patients = hatched columns, patients with neurological deficit during test occlusion = black columns.



averaged maximal flow velocity (Vmean) as well as the systolic peak velocity and the end diastolic velocity were recorded. A stable Vmean value over a period of 10 minutes at rest was taken as the baseline value. Ten minutes before inflation of the balloon ACZ was injected intravenously in all patients. (ACZ is a carbonic anhydrase inhibitor that induces a dose dependent increase in cerebral blood flow by vasodilatation of the precapillary arterioles.) The internal carotid artery was then occluded with a double lumen balloon catheter for 10 minutes. Repeated clinical neurological examinations before and during the occlusion test were performed. Flow velocities during test occlusion were recorded continuously. In cases of increasing or decreasing velocities during the occlusion an average value was calculated. After release of the balloon transcranial Doppler monitoring was continued for another three minutes. Systolic and diastolic blood pressure as well as heart rate were recorded before, during, and after occlusion. The test occlusion was cut off immediately when a neurological deficit occurred during occlusion.

Mean flow velocity of the middle cerebral artery at rest and during occlusion was recorded and documented graphically. The percentage drop of Vmean during occlusion relative to the baseline value was determined. The absolute value of Vmean during occlusion was related to the percentage drop in each patient. The results of transcranial monitoring were compared with the occurrence of neurological symptoms during the occlusion test. According to the extent of the drop of Vmean during occlusion, patients were separated into two different groups. The minimal decrease in mean flow velocity in a patient who developed neurological symptoms during test occlusion (positive occlusion test) was taken as the borderline value concerning tolerance (group A) or intolerance (group B) of occlusion.

The data were analysed with the Mann-Whitney (Wilcoxon) *W* test of differences in medians.

Clinical outcome of patients after an eventual permanent occlusion of the carotid artery was compared with the results of test occlusion. In cases of an ischaemic event after permanent occlusion, cerebral CT was done to differentiate embolic and haemodynamic infarctions according to morphological criteria.[19]

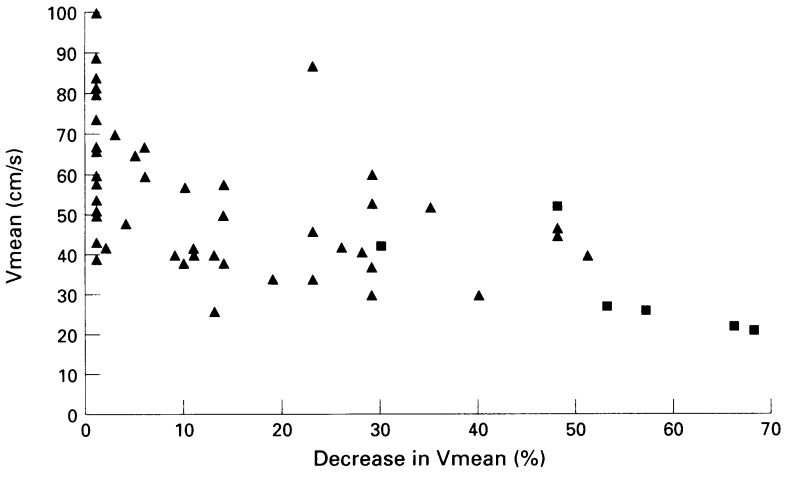


Figure 3 Relation between absolute flow velocities during occlusion and percentage drop of flow velocity. Patients with neurological symptoms during test occlusion (■); patients who tolerated test occlusion (▲).

Results

MEAN FLOW VELOCITIES DURING TEST OCCLUSION

Figure 1 summarises mean flow velocities at rest and during test occlusion. Of 55 patients studied six (11%) developed neurological symptoms during test occlusion. Baseline values of Vmean in these patients were not significantly different from baseline values in patients who tolerated test occlusion (P = 0·9); Vmean during occlusion, however, was significantly lower in patients with clinical intolerance of test occlusion than in those who tolerated the occlusion (P = 0·002). Values for Vmean during occlusion in patients with a positive occlusion test were between 21 and 52 cm/s: four patients had a Vmean between 20–30 cm/s, one between 41–50 cm/s, and one more than 50 cm/s. In patients who tolerated test occlusion without neurological

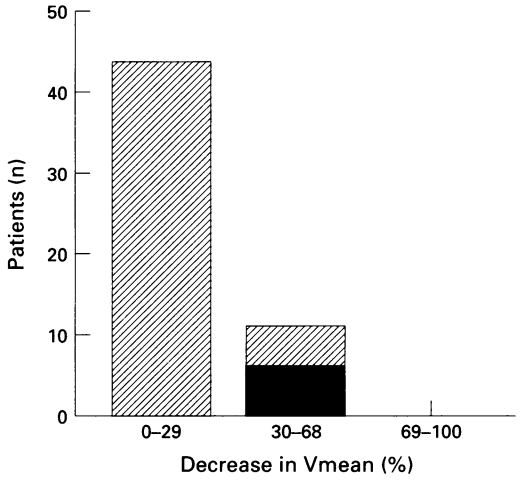


Figure 4 Incidence of neurological deficits during test occlusion (positive occlusion test) related to the extent of decrease of Vmean (%) in the ipsilateral middle cerebral artery. All patients = hatched columns, patients with neurological deficits during test occlusion = black column.

deficits mean flow velocity during the time of occlusion was between 20–30 cm/s in three, between 31–40 cm/s in 10, between 41 and 50 cm/s in 11, and over 50 cm/s in 25 patients (fig 2). Absolute values of Vmean during occlusion were related to the percentage drop of Vmean relative to baseline indicating which patients developed neurological deficits during test occlusion (fig 3). The minimal decrease of Vmean in patients with a positive occlusion test was a drop of 30% relative to baseline. Of 55 patients 44 (80%) showed a drop of mean flow velocity of less than 30% during balloon occlusion (group A), in 11 patients (20%) the decrease of Vmean in the ipsilateral middle cerebral artery was between 30% and 68% compared with baseline (group B), and six (55%) of these patients had clinical signs of ischaemia during test occlusion (fig 4). The percentage drop of Vmean during occlusion was significantly higher in those patients with a positive occlusion test than in those who tolerated test occlusion ($P < 0{\cdot}0001$).

POSITIVE OCCLUSION TEST

During test occlusion six of 55 patients developed neurological symptoms of ischaemia. In five of the six patients symptoms were contralateral to the occluded side and ipsilateral in the other patient. Neurological deficits occurred either in the first two minutes after inflation of the balloon (in three of six patients) or after an occlusion time of six to seven minutes (in three of six patients). All symptoms were transient and disappeared within minutes after deflation of the balloon (table).

CLINICAL OUTCOME AFTER PERMANENT OCCLUSION

Ten patients of group A subsequently underwent a therapeutic permanent occlusion of the carotid artery, seven of them (70%) without ischaemic complications. In two patients (20%), however, an embolic infarction occurred. One patient of group A, who was operated on abroad, died of unknown causes. In group B, two patients had to be permanently occluded. In one patient an external carotid-internal carotid bypass was made before permanent occlusion; in the other patient, who had to be occluded to stop bleeding from the carotid artery, a haemodynamic infarction occurred.

Discussion

Stroke is a severe complication in permanent occlusion of the carotid artery, which may be an unavoidable procedure in brain and neck surgery. Inadequate haemodynamic perfusion and thromboembolism are the two mechanisms of stroke. The risk of stroke after iatrogenic carotid occlusion in unselected patients varies between 30% and 54%.[20 21] A previous temporary endovascular balloon occlusion test leads to a reduction in postoperative infarctions and stroke related mortality.[2] By contrast with manual compression, adequate occlusion of the internal carotid artery can be assured by this procedure. Clinical tolerance and the functional aspect of the circle of Willis can be tested. A delay in the appearance of the cerebral veins on the occluded side is evidence of an insufficient collateral cross filling.[22] Intraoperative and postoperative changing of perfusion requirements, however, is not considered. Between 5% and 20% of the patients with a negative balloon occlusion test will develop cerebral infarction after permanent occlusion of the carotid artery. In 20% of these patients the onset of infarction is delayed by more than 48 hours.[8] This may be due to haemodynamic alterations such as hypotension, reduced cardiac output in cases of heart failure, and arrhythmia as well as to postoperative blood loss and sedation. Therefore, various attempts were made to validate additional techniques for improving the preoperative risk assessment. EEG is an easily available method, which can be used for monitoring before and during the time of occlusion. The sensitivity, however, seems to be poor: To induce EEG abnormalities a considerable decrease in cere-

Data for patients with positive occlusion tests

Patient (age/sex)	*Onset of symptoms (s)*	*Neurological deficit*	*Transient (yes/no)*	*Vmean (cm/s)**	*Fall in Vmean (%)*
44/M	120	Ipsilateral hypaesthesia Ipsilateral hemianopsia	Yes	26	57
44/M	360	Contralateral hemiparesis	Yes	21	68
65/M	420	Contralateral hemiparesis Dysphasia	Yes	42	30
67/M	30	Contralateral hemiparesis Aphasia	Yes	52	48
65/W	420	Contralateral hemiparesis Dysphasia	Yes	27	53
55/M	30	Contralateral hemiparesis	Yes	21	67

*Flow velocities during test occlusion.

bral perfusion is necessary.[23] Cerebral perfusion is indirectly reflected by the regional cerebral blood flow. Recent studies with xenon enhanced cerebral CT claimed an increased risk of stroke after carotid occlusion with regional cerebral blood flow concentrations below 30 ml/100 g/min. In cases of low baseline values regional cerebral blood flow below this borderline may be tolerated.[9 24] Slowing of EEG, however, was only found when regional cerebral blood flow was in the range of 16–22 ml/100 g/min.[23] Stable xenon enhanced CT was also used to improve the prediction of a patient's tolerance of carotid occlusion, but the number of infarctions after permanent occlusion was only slightly reduced by this assessment.[9] ^{99m}Tc HMPAO-SPECT is supposed to be a very sensitive method in balloon occlusion tests, but the specifity of changes between baseline and occlusion SPECT was reported to be very poor.[10 22 25 26] Also, this technique, as well as PET, has the disadvantage of not always being available on site and the patient may have to be moved to a different location. Transcranial Doppler ultrasound is a non-invasive technique which is usually available and which allows continuous monitoring before, during, and after the occlusion test. The correlation between middle cerebral artery velocities and regional cerebral blood flow, however, is controversial.[27–30]

Our results suggest that the drop of Vmean in the ipsilateral middle cerebral artery during balloon occlusion has a predictive value of clinical tolerance. A decrease of 30% relative to baseline is supposed to be a critical borderline value for haemodynamic complications. None of the patients with a fall in Vmean below 30% relative to baseline (group A) developed ischaemic complications during test occlusion or had a haemodynamic infarction when permanently occluded. Although absolute flow velocities during occlusion were significantly lower in patients with a positive occlusion test, the absolute values are less predictive. In cases with lower baseline values flow velocities of 20–30 cm/s are tolerated. By contrast a decrease of more than 30% from baseline can be followed by cerebral ischaemia even with flow velocities greater than 50 cm/s.

In our study the tolerated drop of Vmean was lower than in previous data, in which an increase in haemodynamic complications was noted when the velocity in the middle cerebral artery fell by more than 60% of baseline.[7] In our study four of six patients with haemodynamic ischaemic symptoms during test occlusion had a fall in Vmean of less than 60% from baseline. This is probably due to different factors in the study design: an occlusion time of less than 10 minutes, as well as a manual compression, which cannot assure complete occlusion of the carotid artery, can result in false negative occlusion tests. The use of ACZ in our study affects the vasomotor reactivity capacity. Under the influence of ACZ, the pial arteries and the other resistance vessels of the brain dilate, while the proximal conducting vessels contract slightly.[30] Through this mechanism cerebral autoregulation may be partially exhausted, thus increasing the liability to ischaemic injury.[30 31]

Three of six patients developed neurological symptoms during test occlusion after an occlusion time of six to seven minutes. The interval until the onset of neurological symptoms was not dependent on the extent of fall in Vmean. A similar decrease in flow velocity during occlusion was followed by an individually different time of clinical tolerance. This supports the hypothesis of two regulatory mechanisms in acute carotid occlusion: a decline in cerebral perfusion pressure may firstly be prevented by cerebral autoregulation mechanisms. In cases of continued occlusion, clinical tolerance also depends on the patency of collateral channels.

By continuous monitoring with transcranial Doppler, the time course of changes in cerebral haemodynamics during occlusion, which are related to collateral function and autoregulation, can be documented. The indirect monitoring of these variables gives additional information about regulatory mechanisms, which take place after an acute carotid occlusion. Our results disclose that there may be significant alterations in flow velocity during the time of occlusion. Transcranial Doppler monitoring during an endovascular balloon occlusion test is a reliable technique, which is useful as a part of a preoperative risk assessment. From our results we would consider a decrease of 30% and more relative to baseline as a risk for haemodynamic infarction after permanent occlusion. Stimulation of vasomotor reactivity by ACZ seems to be a procedure which can be used as an additional stress test.

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