Eur J Vasc Endovasc Surg **34**, 540–545 (2007)
doi:10.1016/j.ejvs.2007.05.020, available online at http://www.sciencedirect.com on ScienceDirect

Evaluation of Changes of Systemic Blood Pressure and Shunt Incidence in CEA

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Background. *Induced hypertension is widely recommended as a protective measure in carotid endarterectomy (CEA) to prevent shunt insertion. In this study changes of systemic blood pressure were evaluated in relation to the shunt rate when CEA was performed under local anaesthesia.*

Materials and methods. *In 930 CEAs performed for a high-grade (>70%) ICA stenosis under local anaesthesia the mean systemic blood pressure was measured preoperatively (RR1) and directly before carotid cross-clamping (RR2). A ratio was calculated from these values (RRR = RR2/RR1). A shunt was only inserted for clinical signs of cerebral ischemia. If that became necessary later after cross-clamping had been tolerated primarily, the blood pressure during this period was also recorded (RR3). Also the presence of a contralateral ICA occlusion and baseline blood pressure levels were considered as factors with potential impact on shunt necessity.*

Results. *Among the 638 male (69%) and 292 female (31%) patients with a median age of 70 years (ranging from 52 to 91 years) 82 (9%) had a contralateral ICA occlusion.*

A shunt was used in 177 operations (19%) and significantly more frequent in patients with a contralateral ICA occlusion (39/82 = 48% vs. 138/848 = 16%, p < 0,001).

RRR was significantly reduced in patients who needed a shunt (0.95 (0.41–1.53) vs. 1.0 (0.54–1.9), p = 0.002) which was only true for patients with a patent contralateral ICA.

The shunt rate did not differ when contrasting RRR thresholds (<0.7 vs. >1.3) or preoperative blood pressure levels (<100 mmHg vs. >120 mmHg) were compared.

RRR did not differ between directly or delayed shunted patients. RR3 did not differ significantly from RR2.

A regression analysis identified the presence of a contralateral ICA occlusion as the only independent parameter influencing shunt insertion.

Conclusions. *Changes in systemic blood pressure during CEA under local anaesthesia seem to influence shunting rather marginally. The value of induced hypertension to prevent cerebral ischemia should be newly discussed.*

Keywords: Induced hypertension; Blood pressure changes; Carotid endarterectomy; Local anaesthesia.

Introduction

The benefit in terms of stroke prophylaxis gained by carotid endarterectomy (CEA) in patients presenting with either symptomatic[1] or asymptomatic[2] high-grade internal carotid artery (ICA) stensosis depends on the perioperative complication rate. New neurological deficits after CEA are caused by several mechanisms. Cerebral emboli are either lodged during preparation of the extracranial vessels[3] or postoperatively after a symptom free interval.[4] Postoperative intracerebral haemorrhages occur rarely, but have the worst prognosis.[5] Unrecognized episodes of cerebral ischemia during carotid cross-clamping might also be responsible for perioperative strokes.[6]

Since cerebral autoregulation is impaired in high-grade ICA stenosis,[7] cerebral perfusion depends mainly on systemic blood pressure which, therefore, should be maintained in the high-normal range throughout the procedure and particularly during carotid cross-clamping. For that purpose anaesthesiologic textbooks recommend to raise the blood pressure up to even 40% above baseline.[8] This regime is said to be even more important in patients with pre-existing pronounced hypertension and a contralateral ICA occlusion or severe stenosis where collateral blood flow is further compromised.[9]

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1078–5884/000540 + 06 $32.00/0

However, sufficient brain perfusion during CEA should be ensured timely by routine or selective insertion of an intraluminal shunt which is based on various techniques of neuromonitoring like EEG or SSEP.[10] When the operation is performed under local anaesthesia the patient's clinical condition is simply controlled to discover cerebral malperfusion.[11] Consequently, the independent effect of blood pressure increase as a protective measure can hardly be assessed if the operation is undertaken without neuromonitoring or when routine shunting is used.

In the following study changes of systemic blood pressure during CEA under local anaesthesia were evaluated with respect to the shunt incidence which also might clarify the potential value of induced hypertension for cerebral protection.

Materials and Methods

In a six year period between January 2000 and December 2006 a total number of 930 consecutive patients underwent CEA under local anaesthesia for a high-grade (>70%) ICA stenosis who constitute the study population. The data was collected prospectively and analyzed retrospectively.

There were 38 operations (4%) for recurrent ICA stenosis. Mostly, a standard endarterectomy followed by a Dacron patchplasty was performed (869/930). In the remaining operations either the eversion technique or direct suture of the arterotomy were used.

The degree of ipsilaterat ICA stenosis and patency of contralateral ICA were determined primarily by colour coded ultrasound using the DEGUM-criteria. Whenever the findings differed between examiners or an ICA occlusion was suspected, an additional MRI or intraarterial angiography was performed.

Patients received some sedative medication (midozolam 7.5 mg p.o.) before surgery started. Local anaesthesia comprised a blockage of the superficial and deep cervical plexus.

Cerebral ischemic tolerance was tested by temporary ICA cross-clamping. The decision for shunting was made by the responsible surgeon and independent from any pre-existing clinical parameter, but solely based on the neurological condition of the awake patient at the time of test-clamping or later during actual carotid cross-clamping. The occurrence of any new neurological symptom or aggrevation of present symptoms were regarded as an indicator for shunting. Hemispheric deficits were differentiated from unconsciousness. The delay at which symptoms occurred after cross-clamping was recorded.

Preoperatively, the mean systemic blood pressure was measured under standard conditions on the ward repeatedly (RR1). If there was a pressure difference between both arms, the higher value was used.

Generally, blood pressure was measured every 5 minutes during the operation invasively via a pressure transducer after having punctured the radial artery. Anaesthesiologists were not asked to follow a specific regime, but to maintain stable circulatory conditions primarily to prevent cardiac complications.

Circulatory instability was defined when differences regarding the peak systolic blood pressure of more than 50 mmHg were found throughout the time of surgery.

The mean systemic blood pressure was recorded again directly before carotid cross-clamping (RR2). A blood pressure ratio (RRR) was calculated from these two variables (RRR = RR2/RR1). If a shunt insertion became necessary only during cross-clamping, the mean systemic blood pressure 5 minutes before or directly before this incident took place (when it was less than 5 minutes) was documented (RR3).

Different contrasting thresholds of RRR were used to further evaluate the impact of systemic blood pressure on shunting. The levels of $RRR > 1.3$ and $RRR < 0.7$ were chosen as a clinically relevant increase or decrease of the intraoperative blood pressure of each 30% compared to the preoperative baseline value.

Furthermore, a receiver operating characterictic (ROC)-analysis regarding RRR was performed indicating its differentiating value for shunting where the area under the ROC curve is a non-parametric measure of discrimination generated from the respective test sets (AUC).

Anatomical characteristics like the presence of a contralateral ICA occlusion and pre-existing hypertension were also considered in the analysis.

For statistical analysis SPSS package, version 14.0 was used. Continous data is presented as median and range in this not-normally distributed population (according to Kolmogorov test) Group comparison was performed by Chi-square test, Mann–Whitney test for unpaired, non-parametric variables or Wilcoxon test for paired, non-parametric variables when appropriate. A binary linear regression model was applied to assess whether the included variables (RR1, RR2, RRR, presence of a contralateral ICA occlusion) influenced shunt incidence independently. Differences were considered significant at a level of $p < 0.05$.

Results

There were 638 males (69%) and 292 females (31%) with a median age of 70 years ranging from 52 to 91 years. More than 3 quarters (718/930) of the patients had severe comorbidities as indicated by the ASA classification (ASA III and IV).

The operation was performed due to a symptomatic stenosis (strictly ipsilateral hemispheric) in 37% (210 transitoric ischemic attacks, 134 strokes). Sixty-three percent of the patients were neurologically asymptomatic at the time of surgery where all patients, which exhibited contralateral symptoms, symptoms which had occurred more than 6 months earlier and unspecific symptoms like dizziness, vertigo or cognitive impairment, were included.

In 82 patients (9%) a contralateral ICA occlusion was present.

Seven temporary deficits (0.8%) and 13 new strokes (1.4%) with a permanent neurological deterioration occurred in these 930 operations.

Only a single patient suffered from a myocardial infarction perioperatively who had a lethal course.

A shunt was used in 177 operations (19%). More patients showed general neurological deficits like unconsciousness ($n = 97$, 55%) compared to hemispheric deficits ($n = 80$, 45%). In the majority of cases ($n = 123$, 70%) these symptoms occurred directly after carotid cross-clamping. In the other patients ($n = 54$, 30%), there was a median delay of 10 minutes (3–25 minutes).

The shunt rate was significantly higher in presence of a contralateral ICA occlusion ($39/82 = 48\%$ vs. $138/848 = 16\%$, $p < 0.001$).

Overall, blood pressure was instable in 158 patients during the course of the operation (17%).

Median baseline RR (RR1) was 100 mmHg (60–145 mmHg). Median RR before cross-clamping (RR2) was also 100 mmHg (55–155 mmHg). The blood pressure ratio (RRR) ranged from 0.41 to 1.9 with a median value of 1.0. For those cases with a delayed shunt insertion median systemic RR during cross-clamping (RR3) was 103 mmHg (53–140 mmHg).

Baseline RR (RR1) was significantly higher for shunted patients whereas RR before cross-clamping (RR2) was significantly increased in those not needing a shunt.

RRR was significantly lower for patients who needed a shunt (Table 1) whereas the range of values was similar in shunted and not-shunted patients (Fig. 1).

The ROC-analysis revealed RRR as a weak indicator for shunting with an AUC of 0.421 (95% CI: 0.371–0.471) (Fig. 2).

Table 1. Mean systemic blood pressures and blood pressure ratio (median and range) comparing patient who needed or not needed a shunt

Parameter	Shunt	No shunt	p
RR1 (mmHg)	103 (73–140)	100 (60–145)	0.021
RR2 (mmHg)	96 (55–150)	100 (58–155)	0.007
RRR	0.95 (0.41–1.53)	1.0 (0.54–1.9)	0.002

When the condition of the contralateral ICA is considered additionally, RRR was significantly different between shunted and not-shunted patients only for those who had a patent contralateral ICA: median RRR 0.95 (0.41–1.46) vs. 1.0 (0.54–1.9) respectively, $p = 0.001$. In patients with a contralateral ICA occlusion RRR did not differ between those with or without shunt insertion: median RRR 0.98 (0.66–1.53) vs. 0.98 (0.56–1.67) respectively, $p = 0.5$.

RRR did not differ in shunted patients depending on whether the shunt was inserted directly after

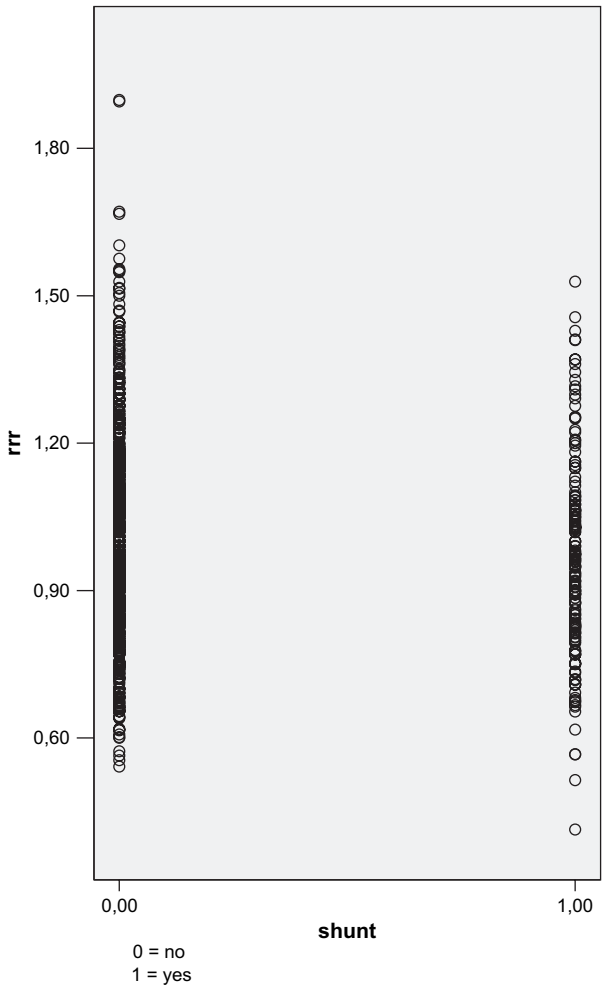

Fig. 1. Distribution of RRR values in shunted and not shunted patients.

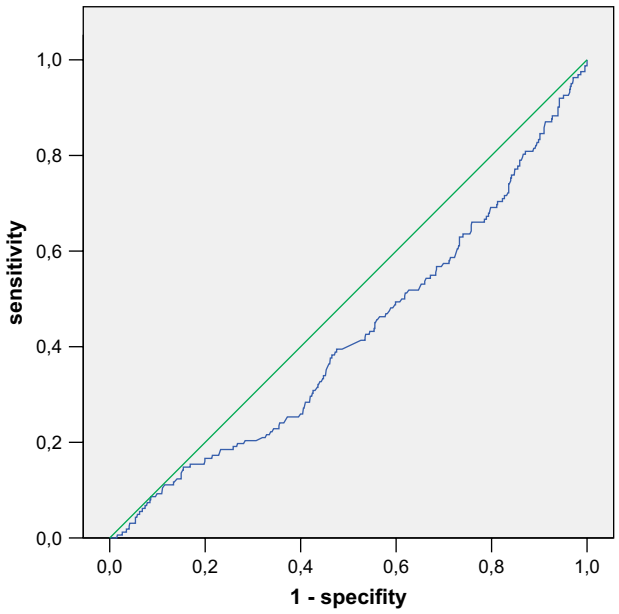

Fig. 2. ROC-curve regarding RRR.

cross-clamping or with a delay: median RRR 0.94 (0.41−1.53) vs. 0.97 (0.65−1.46), $p = 0{,}484$. In those patients who received a shunt after delay RR3 did not differ significantly from RR2 ($p = 0.991$).

The shunt rate did not differ significantly when various RRR thresholds were compared: 25% for RRR < 0.7 (13/52) vs. 17% for RRR > 1.3 (12/70), $p = 0.288$.

When comparing hypotensive ($n = 372$) and hypertensive ($n = 64$) patients (RR1 < 100 mmHg or > 120 mmHg respectively) it was noticed that the shunt rate did not differ significantly (17% vs. 26%, $p = 0.074$), although RRR was significantly reduced in patients with a pre-existing hypertensive condition: median RRR 0.8 (0.41−1.09) vs. 1.09 (0.62−1.9), $p < 0.001$.

Among the parameters which were entered in the regression model, only the presence of a contralateral ICA occlusion revealed an independent impact on shunt necessity (Table 2).

Table 2. Binary linear regression model for shunt necessity

Parameter	Regression coefficient	Standard error	p
Contralateral ICA occlusion	−1.554	0.256	<0.001
RR1 (mmHg)	0.043	0.028	0.124
RR2 (mmHg)	−0.041	0.028	0.142
RRR	2.583	2.745	0.347
Constant factor	−2.889	2.876	0.315

Discussion

Pathophysiologically, induced hypertension seems to be a reasonable measure to ensure sufficient cerebral perfusion in presence of a high-grade ICA stenosis. However, its significance in carotid surgery in order to prevent shunt insertion has not been examined profoundly, yet.[12,13] The present study showed that a decreased intraoperative blood pressure compared to baseline was associated with a more frequent shunt insertion suggesting an underlying pressure related reduction of the cerebral perfusion. But astonishingly, this was only true for those patients with a patent contralateral ICA, although one would have expected sufficient cerebral blood supply through collateral pathways in this particular subgroup of patients. It might be hypothesized that patients with a contralateral ICA occlusion have established those collaterals over a longer period of time by which cerebral perfusion is maintained even during carotid cross-clamping in contrast to those patients with a patent contralateral ICA where ipsilateral ICA clamping becomes more serious as the brain is not as ''preconditioned'' by chronic malperfusion.

The study results also showed that the shunt rate did not differ between patients who had either a 30% increase or decrease of blood pressure intraoperatively meaning that hypertension is not necessarily protective. This is documented by the ROC-analysis, too. Moreover, in patients who developed neurological deficits after first having tolerated cross-clamping no later blood pressure impairment was detected to explain those symptoms.

Patients with pre-existing hypertension who are said to be more dependent on a high normal pressure level did not have a significantly higher shunt rate despite an even poorer intraoperative blood pressure control.

Except from case reports, meaningful clinical studies dealing with continuous blood pressure surveillance during CEA to support a regime of hypertension during carotid cross-clamping are missing.[14] Moreover, that recommendation is mainly based on investigations performed in CEA under general anaesthesia where the induction of anaesthesia itself was associated with a decrease of cerebral blood flow of more than 30% as assessed by an intravenous tracer (133Xenon) technique.[15] On the other hand, an ischemic EEG pattern observed during carotid cross-clamping could be reversed by elevation of the blood pressure to 20% above the preoperative level.[16] If EEG changes persist despite such means, some authors suggest applying additional narcotics to attenuate cerebral metabolism and therefore to protect the brain for this period of time.[17,18]

Whether ischemic EEG changes always represent a state of serious cerebral malperfusion requiring an instant shunt insertion to prevent neurological complications, was examined by Deriu *et al.*[19] They found that patients who developed such EEG signs tolerated a delay of 7.3 ± 3.5 minutes before shunting without a statistical significant increase of the perioperative stroke rate. However, 4 out of 95 patients exhibited an ischemic neurological deficit at awakening which was eventually reversible, but could have been also permanent at worst.

Regarding all these studies one has to consider that the significance of EEG recordings used for the purpose of neuromonitoring during CEA might be restricted by various factors (previous strokes, impact of drugs etc.) leading to false positive or negative results.[20,21] This uncertainty does not exist if the operation is performed under local anaesthesia with the patient's neurological condition serving as a direct indicator for cerebral ischemia.[22,23] To our knowledge this is the first published systematic study which investigates the impact of blood pressure on the shunt rate.

Induced hypertension by vasopressors or haemodilution is also used as part of the treatment modalities for acute stroke to increase blood flow to the ischemic penumbra[24] provided that upper limits are respected to prevent other adverse effects like arrhythmias or intracerebral haemorrhages.[25] But just as in carotid surgery, only a few pilot studies[26,27] or case reports[28] have been able to demonstrate a significant positive effect of blood pressure elevation on neurological outcome measured by cognitive function or by the reduction of hypoperfused brain tissue on PWI scans. Another observational report showed a worse outcome, though, when mean blood pressure was increased by 30% after ischemic stroke.[29] A systemic review of the literature revealed inconclusive results regarding the theoretical benefit of induced hypertension.[30] In fact, both low blood pressure and high blood pressure are independent prognostic factors for poor outcome.[31,32]

There are some limitations adherent to this study.

First, intraoperative hypertension was not a prerequisite of the study because an increased risk of myocardial ischemia has been observed with a routine application of vasopressor agents, at least under general anaesthesia.[33,34] The perioperative blood pressure course was only followed and analyzed retrospectively so that it is not known whether shunt insertion could have been prevented if blood pressure had been further increased in patients being neurologically symptomatic during cross-camping. It would have been unethical, though, to withhold a shunt in those patients, because of the risk of an ischemic stroke, only to investigate the potentially protective effect of induced hypertension.

Secondly, there was only a slight difference between mean pre- and intraoperative systemic blood pressure because anaesthesiologists apparently tried to keep it stable. Consequently, the ratio of those measurements was thought to represent the individual variation of blood pressure better. However, its overall clinical relevance might be questionable, as the absolute difference between patients needing and not needing a shunt was marginal, though statistically significant.

Thirdly, although this study deals with a comparatively large series of CEA performed under local anaesthesia, the overall number of patients in subgroups like having a contralateral ICA occlusion or pre-existing hypertension was still low to draw firm conclusions.

In summary, this study revealed that patients needing a shunt during CEA under local anaesthesia had a minor blood pressure impairment compared to the preoperative level. However, several details were found that allow to discuss the value of intraoperative induced hypertension. It seems that there is more to consider than simply rising the blood pressure in order to protect patients against cerebral malperfusion.

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Accepted 27 May 2007
Available online 9 July 2007