# Cerebral Blood Flow Change Before and After Carotid Angioplasty and Stenting (CAS) in Cases with Contralateral Carotid Artery Occlusion

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**Key words:** CAS, contralateral carotid artery occlusion, CBF.

***Summary***

*Contralateral carotid artery occlusion is thought to represent a significant risk factor in carotid endarterectomy (CEA). There is also evidence that intraoperative and postoperative hypotention may cause contralateral hemodynamic ischemia. As such, contralateral carotid artery occlusion is regarded as a risk factor for carotid angioplasty and stenting (CAS). In this paper, we report on five cases of severe ICA stenosis with contralateral carotid artery occlusions. Cerebral blood flow(CBF) and cerebral vasoreactivity(CVR) of the contralateral carotid artery occlusions were measured before and after CAS. Additionally, the influence that ipsilateral CAS exerted on the occluded side was examined.*

*$^{123}$I-IMP SPECT was performed before and after CAS, both at rest and at the time of acetazoramide administration. The CBF was evaluated quantitatively using the ARG method. The mean CBF of the treated side rose from 30.0 ± 7.1 ml/100g/min to 34.4 ± 8.3 ml/100g/min ($p<0.05$), and the mean CBF of the occluded side similarly rose from 28.3 ± 6.1 ml/100g/min to 31.7 ± 6.4 ml/100g/min ($p<0.05$). Correspondingly, the regional CVR (rCVR) increased from 5.9% ± 16.3% to 35.0% ± 16.4%($p<0.05$) on the treated side, and from 3.7% ± 14.7% to 10.7% ± 16.9% ($p<0.05$) on the occluded side.*

*This demonstrates that ipsilateral CAS seems to improve both CBF and CVR on the contralateral occluded side. The fact that some cases developed cross flow from the anterior communicating artery was both remarkable and significant. Where there was poor cross flow from the anterior communicating artery, improvement in cerebral vaso reactivity was limited.*

## Introduction

As noted by NASCET[2], a carotid artery occlusion of the contralateral side is considered to be a significant risk factors for CEA, and an important factor in choosing CAS. This is evidenced in the SAPPHIRE trial[7], where contralateral carotid artery occlusion was one of the inclusion criteria. At our institute, we perform CAS in those cases where risk factors make CEA a non-preferable option. Carotid artery stenosis with contralateral carotid artery occlusion is such a risk so is consequently treated by CAS as a first choice. The authors recognize that patients with a contralateral carotid artery occlusion may be considered at risk of cerebral hyperperfusion syndrome[4,1], whether treated by CEA or CAS, and that temporary hypotension and bradycardia occur in 19-22% of post CAS patients[3,5]. Under these circumstances, the occluded side may be subject to haemodynamic ischemia, and when there is the possibility of a hemodynamic complication, it is very important to understand and monitor CBF and CVR changes both before and after CAS.

The bilateral haemodynamic benefit of carotid endarterectomy in patients with contralateral internal carotid artery occlusions has been reported in various publications [6], but there are no definitive reports about CAS and CVR. We experienced five cases, all of whom underwent CBF and CVR measurement and evaluation both before and after CAS. Using our findings and data, we report on the bilateral hemodynamic benefit of CAS in patients with contralateral carotid artery occlusions.

## Material and Methods

From May 1999 to August 2004, we treated five cases who had contralateral internal carotid artery occlusions, each undergoing a quantitative CBF-SPECT before and after CAS, at rest and at the time of acetazolamide administration. All cases were male. The age of the patients varied from 55 to 76 years, the mean age was 67, and the mean stenosis rate was 77.4% of the diameter. All cases were symptomatic; the stenosed side was symptomatic in three cases, the occluded side was symptomatic in one case, and progressing dementia was seen in the remaining one case. All cases were treated with a SMART stent (Cordis/Johnson and Johnson).

CBF at rest was assessed by means of $^{123}$I-IMP SPECT, both before and after CAS. We also similarly measured CVR after Acetazolamide administration (15 mg/Kg of acetazolamide was given intravenously) to evaluate the haemodynamic reserve. rCBF measurement was performed using the $^{123}$I-IMP ARG method in four cases and the $^{123}$I-IMP Dual-table ARG method in one case. The regional CVR was calculated as follows. rCVR(%) = (rCBF at Acetazolamide adminstration – resting rCBF at rest) x 100 / rCBF at rest. Both the rCBF and rCVR of the middle cerebral artery territory were

Table 1 **CBF Change in CAS Side: at rest / at acetazolamide administration.**

| Case No | Pre CAS, Rest* (ml/100g/min) | Post CAS, Rest (ml/100g/min) | Pre CAS, AZM** (ml/100g/min) | Post CAS, AZM (ml/100g/min) |
|---|---|---|---|---|
| 1 | 34.0 | 34.9 | 33.8 | 47.8 |
| 2 | 28.5 | 32.6 | 32.4 | 45.0 |
| 3 | 20.6 | 24.0 | 26.7 | 36.6 |
| 4 | 39.5 | 47.1 | 39.7 | 50.8 |
| 5 | 27.5 | 33.5 | 23.8 | 46.7 |
| Mean | 30 | 34.4 | 31.3 | 45.4 |

*Rest*: at rest, AZM**: at acetazolamide administration*

Table 2 **CBF Change in Occluded Side: at rest / at acetazolamide administration.**

| Case No | Pre CAS, Rest* (ml/100g/min) | Post CAS, Rest (ml/100g/min) | Pre CAS, AZM** (ml/100g/min) | Post CAS, AZM (ml/100g/min) |
|---|---|---|---|---|
| 1 | 35.6 | 40.0 | 36.0 | 48.9 |
| 2 | 24.6 | 27.4 | 22.0 | 31.7 |
| 3 | 21.9 | 23.9 | 25.5 | 30.3 |
| 4 | 33.8 | 35.6 | 33.0 | 36.9 |
| 5 | 25.4 | 31.5 | 19.5 | 26.7 |
| Mean | 38.3 | 31.7 | 27.2 | 34.9 |

*Rest*: at rest, AZM**: at acetazolamide administration*

measured. The median times between the preoperative CBF SPECT studies and CAS therapy were 33.4 days (CBF at rest), and 32.6 days (CBF after Acetazolamide administration). Postoperative CBF SPECT studies were similarly performed at 8.4 days and 10.2 days respectively. Four vessel angiography was performed pre treatment in all cases, and collateral circulation was evaluated from the anterior and posterior communicating arteries, and leptomeningeal anastomosis. Wilcoxon's rank order sum test was used for statistical analysis.

## Results

### *Quantitative rCBF Change before and after CAS*

Table 1 shows the rCBF change before and after CAS of the stenosed side (treated side), at rest and after acetazolamide administration. The mean rCBF increased from 30.0 ± 7.1 ml/100g/min to 34.4 ± 8.3 ml/100g/min ($p<0.05$) at rest. The mean rCBF increased from 31.3 ± 6.2 ml/100g/min to 45.4 ± 5.3 ml/100g/min ($p<0.05$) after acetazolamide administration.

Table 2 shows the rCBF change before and after CAS of the occluded side (contralateral side), at rest and after acetazolamide administration. The mean rCBF rose from 28.3 ± 6.1 ml/100g/min to 31.7 ± 6.4 ml/100g/min ($p<0.05$) at rest. The mean rCBF rose from 27.2 ± 7.1 ml/100g/min to 34.9 ± 8.6 ml/100g/min ($p<0.05$) after acerazolamide administration.

### *Cerebral Vasoreactivity Change before and after CAS*

Table 3 shows rCVR change before and after CAS on both the stenosed and occluded sides. The mean rCVR of the stenosed side elevated from 5.9 ± 16.3 % to 35.0 ± 16.4% ($p<0.05$). The mean rCVR of the occluded side grew from –3.7 ± 14.7 % to 10.7 ± 16.9%. ($p<0.05$).

Table 3 **CVR Change in CAS Side and Occluded Side.**

| Case No | CAS*, Pre CAS* % | CAS, Post CAS % | Contralateral** Pre CAS % | Contralateral Post CAS % |
|---|---|---|---|---|
| 1 | -0,6 | 37 | 1.1 | 22.3 |
| 2 | 13.7 | 37 | -10.6 | 15.7 |
| 3 | 29.6 | 52.5 | 16.4 | 26.8 |
| 4 | 0.5 | 7.9 | -2.4 | 3.7 |
| 5 | -13.5 | 39.4 | -23.4 | -15.2 |
| Mean | 6 | 35 | -3.7 | 10.7 |

*CAS*: CAS side (treated side), Contralateral**: Contralateral side (occluded side)*

Table 4 **Collateral Circulation in Occluded Side.**

| Case No | via Ant. Com Artery* | EC-IC Anastomosis | via Post. Com Artery** | Leptomeningeal Anastomosis |
|---|---|---|---|---|
| 1 | + | + | – | + |
| 2 | + | + | – | + |
| 3 | + | – | – | + |
| 4 | + | – | + | – |
| 5 | – | + | – | – |

*via Ant. Com. Artery*: via anterior communicating artery*
*via Post. Com. Artery**: via posterior communicating artery*

The degree of improvement in rCVR tended to vary according to the individual. Arterial steal was found in three of the five cases on the occluded side, prior to treatment, but in only one case after treatment.

*Angiographical Collateral Circulation.*

Table 4 shows collateral circulation of the occluded side. Collateral circulation was evaluated from the anterior and posterior communicating arteries, and leptomeningeal anastomosis. In the fifth case, collateral cross flow via the anterior communicating artery was absent, as was absent of collateral flow from the posterior communicating artery and leptommeningeal anastomosis.

## Discussion

In severely stenosed carotid arteries with contralateral carotid artery occlusions, both contralateral CBF (on the occluded side) and rCVR have been shown to demonstrate improvement as a result of performing ipsilateral CAS. Improvement of contralateral CBF after CEA has often been reported [6], but the authors found that similar improvement occurs after CAS. Additionally, not only CBF but also rCVR was seen to show improvement. From the results in cases of bilateral carotid artery occlusive disease noted in this study, it seems reasonable to expect an improvement in CBF and rCVR on both sides by performing CAS on one side. The improvement in rCVR is a distinct benefit of CAS treatment.

The improvement of rCVR may prevent postoperative haemodynamic ischemia resulting from postoperative hypotension. However, the degree of improvement in rCVR was different in each individual case. On the occluded side, the arterial steal phenomenon was found in only one case postoperatively (case 5). In this case, cross flow via the anterior communicating artery was not found angiographically either before or after CAS. If the collateral flow provided by the circle of Willis is unhampered, we can expect improvement in the contralateral rCVR after CAS. In cases where angiographic cross flow via the anterior communicating artery was seen prior to CAS, CVR will improve after CAS on the occluded side. In such cases, the risk of post CAS haemodynamic ischemia is low, making CAS a relatively safe choice even in cases with bilateral occlusions. But where there is poor cross flow from the anterior communicating artery, any improvement in CBF or rCVR will be limited. Hypotension due to carotid sinus stimulation is 19% to 22% after CAS [3,5]. If collateral circulation via the anterior communicating artery does not develop, post procedure hypotension may have a potential risk of developing into brain ischemia on the contralateral side. Clearly, monitoring and checking for postoperative hypotension is important in such cases.

## Conclusions

In severely stenosed carotid arteries with contralateral carotid artery occlusion, contralateral (on the occluded side) CBF and rCVR was improved by performing ipsilateral CAS. The resultant developing cross flow, in some cases, from the anterior communicating artery is quite remarkable and responsible for this phenomenon. Where there is significantly poor cross flow from the anterior communicating artery, any improvement in CVR is limited. Such cases needed to be carefully watched for postoperative hypotension.

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