Long-term Follow-up Study of Patients with Cavernous Sinus Aneurysm Treated by Proximal Occlusion

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Abstract

Proximal occlusion of the internal carotid artery (ICA) is still the treatment of choice for a large cavernous sinus aneurysm. Endovascular occlusion or trapping of the ICA with or without an extracranial-intracranial bypass is sometimes performed. We analyzed the results of the long-term follow up of 11 patients with a giant or large cavernous sinus aneurysm treated by only proximal occlusion between 1975 and 1989. Proximal occlusion of the carotid artery was performed by Salverstone clamping. The follow-up period ranged from 6 to 21 years (mean 13.9 years). Eight of the 11 patients showed improvement of cranial nerves paresis or headache, and four became asymptomatic. None of the original aneurysms ruptured. The final outcomes were nine good recovery, one moderately disabled, and one severely disabled by the Glasgow Outcome Scale. The causes of morbidity were early ischemia and subarachnoid hemorrhage from a newly formed aneurysm. Late complications included ischemia in two patients, and new formation and enlargement of aneurysms at a site other than the original aneurysm in two patients, 13 and 17 years later. Therapeutic carotid artery occlusion requires strict test ICA occlusion. In addition, long-term follow up by periodical cerebral angiography using magnetic resonance, computed tomography, or digital subtraction angiography is necessary, and postoperative medical treatment is important to reduce the risk of late complications.

Key words: cavernous sinus aneurysm, proximal occlusion, new aneurysm formation, late complication, giant aneurysm, Salverstone clamp

Introduction

Direct clipping of a giant aneurysm of the internal carotid artery (ICA) is sometimes difficult because of the broad neck and the complex anatomical relations with the surrounding cranial nerves and the cavernous sinus. Improved microsurgical techniques for cavernous sinus lesions now allow direct clipping of these aneurysms, but the selection of treatment remains controversial. Endovascular treatment has recently been used for such giant aneurysms, but endovascular obliteration with preservation of the parent artery is also difficult. Therefore, occlusion or trapping of the ICA is performed after the test occlusion of ICA, However, the necessity for extracranial-intracranial (EC-IC) bypass is possible after proximal occlusion testing, because testing of patient tolerance of occlusion is still imperfect using only the temporary occlusion test.

Proximal occlusion of the carotid artery with Salverstone clamp is one of the classic methods of treatment, used before the intravascular technique became common. Follow-up studies of patients treated by carotid artery occlusion for intracranial aneurysms have clarified the efficacy and the serious problems of this method. Nowadays, this treatment is selected only for aneurysms that appear to be difficult to clip, such as giant or extradural ICA aneurysms. If the patient tolerates the ICA test occlusion, intravascular balloon occlusion without bypass surgery is usually performed. The present study analyzed the results of long-term follow up (mean follow-up period of more than 13 years) of patients with a giant or large cavernous sinus aneurysm treated by only proximal occlusion.
to assess the problems of proximal occlusion and the potential treatment of complications.

Clinical Materials and Methods

Selverstone clamping of the carotid artery was used for the treatment of aneurysms and other vascular lesions in 16 patients at the Kagoshima University Hospital and its affiliated hospitals between 1975 and 1989. This study included 11 patients, 10 females and one male aged 52 to 75 years (mean 60 years), with an aneurysm of cavernous portion of ICA treated by proximal occlusion with Selverstone clamping. All aneurysms were giant or large. One patient received additional carotid ligation because of incomplete carotid artery occlusion by the Selverstone clamp.

Manual compression of the carotid artery (Matas test) was performed for 5 minutes initially, and the occlusion time was gradually prolonged up to 30 minutes under electroencephalography monitoring before the operation. The Selverstone clamp was placed on the common carotid artery (CCA) in eight patients and the external carotid artery (ECA) was ligated, except in one aged patient. The clamp was placed on the ICA distal to the bifurcation of CCA in the other three patients (Table 1). Gradual occlusion taking about a week was performed. Before complete occlusion, low molecular weight dextran was administered. The blood pressure after complete occlusion was maintained at almost the same value compared to the preoperative blood pressure by the hypervolemic transfusion with low molecular weight dextran for several days.

All patients underwent pre- and postoperative angiography and nine patients underwent preoperative computed tomography (CT). The clinical records and imaging studies were reviewed. The follow-up period ranged from 6 to 21 years (mean 13.9 years). Follow-up imaging studies were performed mainly by CT. Five of the 11 patients underwent angiography because of neurological aggravation or newly occurred stroke during the follow-up period. Follow-up investigation was performed using clinical records and mail or telephone interviews.

Results

The clinical outcomes are shown in Table 1. The final outcomes were nine good recovery, one moderately disabled, and one severely disabled by the

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/Sex</th>
<th>Initial symptoms</th>
<th>Method of carotid artery occlusion</th>
<th>Final symptoms</th>
<th>Aneurysm on CT</th>
<th>Follow up (yrs)</th>
<th>Outcome*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>54/F</td>
<td>headache, transient ptosis</td>
<td>Selverstone clamp on CCA with ligation of ECA</td>
<td>asymptomatic</td>
<td>completely thromosed</td>
<td>21</td>
<td>GR</td>
</tr>
<tr>
<td>2</td>
<td>52/M</td>
<td>headache, nasal bleeding</td>
<td>Selverstone clamp on ICA</td>
<td>asymptomatic</td>
<td>completely thromosed</td>
<td>19</td>
<td>GR</td>
</tr>
<tr>
<td>3</td>
<td>50/F</td>
<td>orbital pain, ptosis</td>
<td>Selverstone clamp on CCA with ligation of ECA</td>
<td>ptosis</td>
<td>partially thromosed</td>
<td>16</td>
<td>GR</td>
</tr>
<tr>
<td>4</td>
<td>61/F</td>
<td>diplopia</td>
<td>Selverstone clamp on CCA with ligation of ECA</td>
<td>partially improved</td>
<td>partially thromosed</td>
<td>16</td>
<td>GR</td>
</tr>
<tr>
<td>5</td>
<td>53/F</td>
<td>ptosis, diplopia</td>
<td>Selverstone clamp on CCA with ligation of ECA</td>
<td>asymptomatic</td>
<td>completely thromosed</td>
<td>16</td>
<td>GR</td>
</tr>
<tr>
<td>6</td>
<td>52/F</td>
<td>orbital pain</td>
<td>Selverstone clamp on CCA with ligation of ECA</td>
<td>asymptomatic</td>
<td>completely thromosed</td>
<td>15</td>
<td>GR</td>
</tr>
<tr>
<td>7</td>
<td>61/F</td>
<td>diplopia</td>
<td>Selverstone clamp on CCA with ligation of ECA</td>
<td>no change</td>
<td>completely thromosed</td>
<td>15</td>
<td>SD</td>
</tr>
<tr>
<td>8</td>
<td>75/F</td>
<td>ptosis</td>
<td>Selverstone clamp on CCA with ligation of ECA</td>
<td>partially improved</td>
<td>partially thromosed</td>
<td>10</td>
<td>GR</td>
</tr>
<tr>
<td>9</td>
<td>67/F</td>
<td>diplopia</td>
<td>Selverstone clamp on ICA</td>
<td>no change</td>
<td>completely thromosed</td>
<td>10</td>
<td>MD</td>
</tr>
<tr>
<td>10</td>
<td>69/F</td>
<td>ptosis</td>
<td>Selverstone clamp on ICA</td>
<td>partially improved</td>
<td>completely thromosed</td>
<td>9</td>
<td>GR</td>
</tr>
<tr>
<td>11</td>
<td>57/F</td>
<td>ptosis</td>
<td>Selverstone clamp on CCA with ligation of ECA</td>
<td>no change</td>
<td>completely thromosed</td>
<td>6</td>
<td>GR</td>
</tr>
</tbody>
</table>

*By Glasgow Outcome Scale. **Additional common carotid artery (CCA) ligation was performed after Selverstone clamping. CT: computed tomography, ECA: external carotid artery, GR: good recovery, ICA: internal carotid artery, MD: moderately disabled, SD: severely disabled.

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Table 2 Complications after treatment of cavernous sinus aneurysm by proximal occlusion

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Early complications</th>
<th>Late complications</th>
<th>Interval</th>
<th>Second surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>TIAs</td>
<td>none</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>prolonged RIND</td>
<td>contralateral cavernous ICA giant aneurysm (symptomatic)</td>
<td>17 yrs</td>
<td>ICA ligation with high-flow bypass</td>
</tr>
<tr>
<td>3</td>
<td>none</td>
<td>re-enlargement of original aneurysm</td>
<td>5 mos</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>TIAs</td>
<td>TIAs</td>
<td>3 yrs</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>none</td>
<td>re-enlargement of original aneurysm</td>
<td>11 yrs</td>
<td>CCA ligation and removal of clamp</td>
</tr>
<tr>
<td>6</td>
<td>none</td>
<td>none</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>TIAs</td>
<td>SAH from ruptured AcomA aneurysm</td>
<td>3 mos</td>
<td>clipping</td>
</tr>
<tr>
<td>8</td>
<td>none</td>
<td>none</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>completed stroke</td>
<td>none</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>none</td>
<td>none</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>none</td>
<td>none</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Glasgow Outcome Scale. During the follow-up period, no bleeding occurred from the original giant or large ICA aneurysms.

Initially, all patients showed some cranial nerve signs, except for one patient who had headache and nasal bleeding. Seven patients showed improvement of the cranial nerve signs. Four patients became symptom-free. The improvement of symptoms was observed immediately after the complete occlusion of the carotid artery, usually within 1 week or 2. Orbital pain and headache commonly improved.

Early ischemic complications occurred within a few days after carotid artery occlusion in four patients (Table 2). Two patients had transient ischemic attacks (TIAs), one had prolonged reversible ischemic neurological deficit, and one had minor completed stroke. Early ischemic complications were ipsilateral to the carotid artery occlusion.

Patients with successful carotid artery occlusion or recovery from early ischemic attack may suffer delayed strokes or neurological aggravation. In our study, such late complications occurred in five of the 11 patients at 3 months to 17 years after carotid artery occlusion (Table 2). Two patients had TIAs. Case 7 suffered transient right hemiparesis for 30 minutes, 3 months after the carotid artery occlusion. Case 4 had right transient hemiparesis with dysphasia for 1 hour, 3 years after the carotid artery occlusion. In both cases, the ischemic side was ipsilateral to the occluded side and CT showed the aneurysms as high density. Two were new formations or enlargement of aneurysms at sites other than the original cavernous sinus aneurysm. In Case 2, the aneurysm of the contralateral cavernous portion of the ICA was enlarged and became symptomatic, 17 years after the proximal occlusion. Case 7 suffered subarachnoid hemorrhage (SAH) from a ruptured anterior communicating artery (AcomA) aneurysm, 13 years after the proximal occlusion. Other late complications were aggravation of cranial nerve signs due to re-enlargement of the original giant aneurysms. Case 3 had an associated vascular anomaly of the persistent primitive trigeminal artery (PTA). Complication in Case 5 was caused by incomplete occlusion with the Silverstone clamp, so the CCA was ligated and the clamp was removed after balloon test. After the second operation, she became symptom-free.

Representative Case Reports

Case 2: A 52-year-old male presented with headache and repeated nasal bleeding in May 1978. Left carotid angiography revealed a large cavernous sinus aneurysm. Proximal ICA occlusion with a Silverstone clamp was performed. Postoperatively, he had right hemiparesis and speech disturbance which recovered after about 1 month. The patient's headache disappeared and the nasal bleeding stopped. In July 1995, he presented with right visual disturbance, ptosis, and diplopia. CT showed an isodense mass with homogeneous contrast enhancement at the level of the right cavernous sinus. Four-vessel angiography revealed a giant aneurysm in the right cavernous portion (Fig. 1 left). Occlusion of the left ICA and absence of the left giant aneurysm were confirmed (Fig. 1 center). Right EC-IC high-flow bypass with a saphenous vein graft was performed, and the proximal right ICA was ligated. Postoperative angiography showed good bypass
Fig. 1 Case 2. Carotid angiograms taken 17 years after proximal internal carotid artery (ICA) occlusion by Silverstone clamp. Left: Right internal carotid angiogram showing a giant aneurysm at the cavernous portion of the right ICA. Center: The distal ICA of the left side is filled through the collateral circulation from the external carotid artery, and the aneurysmal shadow of the left ICA is not visible. Right: Postoperative right external carotid angiogram showing good patency of the extracranial-intracranial high-flow bypass and no aneurysmal shadow.

flow (Fig. 1 right), and the aneurysm was completely thrombosed without ischemic complications.

Case 7: A 61-year-old female presented with diplopia on left lateral gaze in May 1984. Neurological examination revealed a deficit of the left 6th cranial nerve. CT showed an isodense mass with homogeneous contrast enhancement at the level of the left cavernous sinus. Left carotid angiography revealed a giant cavernous sinus aneurysm. Right retrograde brachial panangiography showed no aneurysms. Carotid occlusion with a Silverstone clamp was performed uneventfully. Three months after the operation, the patient suffered from transient right hemiparesis persisting for 30 minutes. She was in good health until May 24, 1997, when she experienced severe headache followed by loss of consciousness. CT showed diffuse SAH but no enhanced mass at the level of the left cavernous sinus. Right carotid angiography revealed an AcomA aneurysm (Fig. 2). The aneurysm was clipped but the patient was severely disabled because of right hemiparesis caused by vasospasm.

Case 3: A 59-year-old female presented with right orbital pain and ptosis in July 1981. CT showed an isodense mass with homogeneous contrast enhancement at the level of the right cavernous sinus (Fig. 3A, B). Right carotid angiography revealed a giant cavernous sinus aneurysm and PTA (Fig. 4 left).

Fig. 2 Case 7. Right internal carotid angiogram taken 13 years after proximal occlusion by Silverstone clamp, revealing an anterior communicating artery aneurysm directed to the left side.
Carotid artery occlusion with a Selverstone clamp was performed uneventfully. The orbital pain was disappeared and ptosis was improved. The CT density of the aneurysm became high with homogeneous enhancement, 2 weeks after carotid artery occlusion (Fig. 3C, D). Five months after the operation, the patient again suffered from orbital pain and aggravated ptosis. The aneurysm showed slightly high density with ring-like enhancement and partial solid enhancement (Fig. 3E, F). Right carotid angiography showed no filling of the aneurysm, but left vertebral angiography showed a small aneurysmal lumen at the junction of the right ICA and PTA (Fig. 4 right). Direct clipping to the PTA was intended, but the orbital pain improved and the patient did not agree to surgery. After that, the orbital pain disappeared and ptosis was gradually improved. Her neurological findings are stable and follow-up CT, 11 years later, showed partially thrombosed aneurysm (Fig. 3G, H).

Discussion
This long-term follow-up study of carotid artery occlusion with the Selverstone clamp identified some serious problems. The most serious problem is the potential for ischemic complications. In our series, mortality was 0%, but the rate of early ischemic complications was 9.1% permanent morbidity and 36.4% temporary morbidity. Before the introduction

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Fig. 4 Case 3. Cerebral angiograms. left: Right carotid angiogram revealing a giant cavernous sinus aneurysm and primitive trigeminal artery (PTA) (arrow), and the upper part of the basilar artery visualized through the PTA. right: Left vertebral angiogram, performed when the neurological findings aggravated, showing the small aneurysmal lumen at the junction of the right internal carotid artery and PTA (arrows).

The procedure was to gradually prolong the period of the compression of the carotid artery from 5 to 30 minutes preoperatively, followed by gradual carotid artery occlusion. However, abrupt occlusion is better than gradual occlusion to reduce thromboembolism.9 In our study, all four early ischemias occurred ipsilateral to the carotid artery occlusion. Some incidents, especially the TIAs, were thought to be thromboembolic. One reason for the high rate of transient early ischemias may be the gradual occlusion. Before the use of the balloon test, opening or stopping of clamping at once whenever ischemic symptoms were noted was an advantage.2,30 Nowadays, carotid artery occlusion is performed more safely after balloon test. If the patient can tolerate the test occlusion, abrupt occlusion of the proximal vessel is possible, or EC-IC bypass is first performed. Mortality and morbidity caused by ischemic complication were remarkably reduced after the introduction of the balloon test (Table 3).3,4,10,14,16,31

Ischemic complications may occur both early and late after carotid artery occlusion.16,25,31 We experienced two cases of late ischemia, both of ipsilateral stroke to the carotid artery ligation. In Case 7, the aneurysm was high density on CT, indicating fresh and fragile thrombus formation, at the time of TIA. The ischemia was thought to be embolic based on the density of the aneurysmal lumen on CT. In Case 4, TIA occurred twice with hemiparesis of the right side and dysphasia, 4 days and 3

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Number of patients</th>
<th>Ligated artery</th>
<th>Aneurysm</th>
<th>Morbidity (%)</th>
<th>Temporary morbidity (%)</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mount (1959)</td>
<td>65</td>
<td>ICA, CCA</td>
<td>various</td>
<td>18.5</td>
<td>—</td>
<td>4.6</td>
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<td>Poppen and Fager (1960)</td>
<td>101</td>
<td>ICA</td>
<td>ICA</td>
<td>6.9</td>
<td>—</td>
<td>3.9</td>
</tr>
<tr>
<td>German and Black (1965)</td>
<td>35</td>
<td>ICA, CCA</td>
<td>ICA</td>
<td>2.9</td>
<td>2.9</td>
<td>6</td>
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<td>Gurdjian et al. (1965)</td>
<td>27</td>
<td>CCA</td>
<td>ICA</td>
<td>4.0</td>
<td>—</td>
<td>11.1</td>
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<tr>
<td>Nishioka (1966)</td>
<td>785</td>
<td>ICA, CCA</td>
<td>various</td>
<td>30.0</td>
<td>—</td>
<td>9.2</td>
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<tr>
<td>Miller et al. (1977)</td>
<td>72</td>
<td>ICA, CCA</td>
<td>various</td>
<td>5.0</td>
<td>21.0</td>
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<td>Giannotta et al. (1979)</td>
<td>21</td>
<td>ICA</td>
<td>ICA (inaccessible)</td>
<td>9.5</td>
<td>—</td>
<td>0</td>
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<td>Roski et al. (1981)</td>
<td>57</td>
<td>ICA, CCA</td>
<td>various</td>
<td>16.0</td>
<td>26.1</td>
<td>5</td>
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<td>Weil et al. (1987)</td>
<td>21</td>
<td>ICA</td>
<td>ICA (inaccessible)</td>
<td>0</td>
<td>14.3</td>
<td>0</td>
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<td>Little et al. (1989)</td>
<td>15</td>
<td>ICA</td>
<td>cavernous ICA</td>
<td>3.5</td>
<td>—</td>
<td>0</td>
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<tr>
<td>Drake et al. (1994)</td>
<td>112</td>
<td>ICA</td>
<td>ICA (giant)</td>
<td>2.7</td>
<td>5.5</td>
<td>0</td>
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<td>Larson et al. (1995)</td>
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<td>ICA</td>
<td>ICA</td>
<td>3.4</td>
<td>10</td>
<td>3.4</td>
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<tr>
<td>Hacene-Bey et al. (1997)</td>
<td>9</td>
<td>ICA</td>
<td>ICA (inaccessible)</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Dare et al. (1998)</td>
<td>20</td>
<td>ICA</td>
<td>ICA and others</td>
<td>15</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Present series</td>
<td>11</td>
<td>ICA, CCA</td>
<td>cavernous ICA</td>
<td>9.1</td>
<td>36.4</td>
<td>0</td>
</tr>
</tbody>
</table>

*Studies after introduction of the balloon test occlusion. CCA: common carotid artery, ICA: internal carotid artery.

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years after carotid artery occlusion. The aneurysm was high density on CT and partially enhanced at the second TlAs. Both ischemia were thought to be embolic. Both patients received anti-hypertension and anti-platelet treatment and no more ischemic attacks occurred. Embolic stroke is difficult to prevent. Proximal occlusion close to the origin of the aneurysm and aneurysmal packing are possible methods to reduce embolic stroke, but there is no definitive medical treatment. Administration of low-dose warfarin for several weeks is recommended. However, the potential complication of long-term anticoagulation may outweigh the potential benefits. Although the preventive effect is not confirmed, anti-platelet therapy is a possible treatment after therapeutic carotid artery occlusion.

In our study, the classical preoperative test occlusion was performed. Manual compression of the carotid artery in the neck is helpful for identifying patients who cannot tolerate occlusion, but has very little predictive value for selecting patients who can tolerate occlusion. The provocative balloon test occlusion combined with induced hypotension and cerebral blood flow mapping is a promising predictor of complications of ICA occlusion. Judging from the results of such strict test occlusion, ICA occlusion is possible without bypass surgery. However, completely reliable prediction of whether a patient can tolerate permanent occlusion of the ICA is not possible yet.

Partial contrast enhancement in the aneurysmal lumen on CT indicated incomplete thrombosis. Four giant aneurysms could not be thrombosed completely by proximal occlusion with the Silverstone clamp. Giant aneurysm at the junction of the ICA and PTA is extremely rare, but complete thrombosis cannot be obtained only by proximal ICA occlusion. Before the technique of intravascular surgery, direct intracranial clipping of PTA was performed. Nowadays, PTA is occluded by intravascular balloon or direct aneurysm balloon packing. Apart from insufficient occlusion by the Silverstone clamp, as in Case 5, incomplete thrombosis may result from insufficient decrease in the distal intraluminal pressure, as in our other two cases. One case of incomplete thrombosis was due to occluded CCA by ECA ligation. Incomplete thrombosis results in limited improvement of cranial nerve paresis, and embolus from the intralumen of an aneurysm may cause late ischemia. Additionally, the residual aneurysmal lumen is in danger of re-enlargement.

The potential for aneurysmal formation or enlargement after carotid artery occlusion is a considerable problem. In the present study, the mean follow-up period was more than 13 years, and two of the 11 patients eventually showed symptoms originating from aneurysms other than the original cavernous sinus aneurysm. There are many previous cases of newly formed or enlarged aneurysms after carotid artery ligation. The common locations are the contralateral ICA and AcomA. Some aneurysms were termed ‘de novo aneurysm.’ The incidence of the aneurysmal formation or enlargement after carotid artery ligation is not clear, but de novo aneurysm formation after ligation ranges from 1.4% to 4%. Two newly formed or enlarged aneurysms were observed in four of 27 cases of therapeutic carotid artery occlusion during a mean follow-up period of 10 years, and two de novo aneurysms occurred in 58 patients. The incidence of true de novo aneurysm unrelated to the therapeutic occlusion of proximal major vessels is 0.9–1.1%. Therefore, carotid artery occlusion contributes to the formation or enlargement of an aneurysm. The vast majority of reported cases included only symptomatic aneurysms, except for one asymptomatic case. If follow-up angiography is performed periodically, the incidence of new formation or enlargement of aneurysms after carotid artery occlusion may increase.

Hemodynamic stress caused by increased blood flow of the ICA contralateral to the AcomA is thought to be the cause of aneurysmal formation and enlargement. However, the observer error and technical factors are also problems. In both our cases, the repeat angiography showed good circulation of the ligated side without contralateral cross circulation. The effect of hemodynamic stress is apparently not so responsible for newly formed or enlarged aneurysm, but the hemodynamic stress before completing the collateral circulation may contribute to form or enlarge the aneurysm. In Case 7, right retrograde panangiography might not have excluded the small AcomA aneurysm completely.

Hypertension is also a risk factor for aneurysmal formation or enlargement, and is sometimes induced by carotid artery occlusion. In our study, seven of 11 patients received anti-hypertensive therapy after carotid artery ligation and both patients who developed aneurysms showed hypertension. Unfortunately, Case 7 refused further medical treatment 3 years after carotid artery occlusion. Ruptured aneurysm results in mortality or severe morbidity, so aneurysmal formation or enlargement is a serious late complication of carotid artery occlusion. Therefore, when a therapeutic carotid artery occlusion is selected, long-term follow up by cerebral angiography; using magnetic resonance imaging; and considering the clinical situation may be helpful in determining the adequacy of follow-up angiography.

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(MR) angiography, CT angiography, or digital subtraction angiography is necessary. The Selverstone clamp is ferromagnetic, so MR angiography cannot be used for follow-up angiography. Medical treatment for hypertension is also important to prevent de novo formation or enlargement of an aneurysm.

This long-term follow-up study showed relatively good outcomes for the treatment of cavernous sinus aneurysms even by the classical method of proximal occlusion using the Selverstone clamp. However, the use of this method entails the risk of dangerous complications including acute and late ischemic complications. De novo formation or enlargement of an aneurysm at a site other than the original aneurysm is another risk that must be considered. Even if the initial treatment is successful, long-term follow up including cerebral angiography and postoperative medication for hypertension and thromboembolism are required.

References

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have suggested that a bypass operation should be performed in all cases of carotid occlusion. In my opinion, this is simply not necessary for a large number of patients.

I agree with the authors that endovascular balloon test occlusion has been very useful in identifying patients who are able to tolerate carotid occlusion and those patients who clearly require augmentation of collateral blood flow through a bypass. To date, however, there is no test occlusion procedure that is flawless in its ability to identify all patients who will safely tolerate carotid occlusion.

At our institution, we perform balloon test occlusions on all patients for whom we consider therapeutic carotid occlusion, and combine the test occlusion with cerebral blood flow studies to stratify patients into low, moderate and high risk for ischemic complications. For the low risk patients in whom we perform endovascular carotid occlusion without bypass, we believe aggressive medical therapy with volume expansion and induced hypertension to augment collateral blood flow and antiplatelet or anticoagulant therapy for a limited time assists in the reduction of ischemic complications. For the moderate or high-risk patients, we perform one of a variety of types of bypass procedures to augment collateral flow. Although this strategy has clearly reduced the risk of complications, those complications have not been eliminated despite these adjuncts.

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This is a report on long-term follow up after gradual occlusion of the carotid artery with Seldinger clamp, following a manual occlusion test for giant intracavernous aneurysms. Both Seldinger clamping and manual occlusion testing are classical methods and not routinely used nowadays. But as the treatment for this type of aneurysm is still controversial in many ways, and there is no definitive test available for the occlusion, the long-term results in this paper provide an important piece of information. The results show that a variety of complications can occur in the long run, as late as 17 years after surgery. The authors say that 'relatively good' results were obtained, but it is my feeling that when a complication occurs in one case, this has to be taken very seriously. So, even for a case in which preoperative tests seem to guarantee permanent carotid occlusion, preventive bypass surgery is necessary.

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The authors present a meticulous and thoughtful analysis of their long-term (mean 13.9 years) experience with 11 patients with cavernous sinus aneurysm treated by gradual proximal occlusion using the Silverstone clamp. Serious problems caused by gradual carotid artery occlusion were ischemic complications and aneurysmal formation or enlargement. They concluded that this classical method showed relatively good outcome, strict test internal carotid artery (ICA) occlusion before therapeutic carotid artery occlusion is required to prevent early ischemic complication, and long-term follow up by periodical cerebral angiography is necessary to detect aneurysm formation or enlargement. The main issue of sacrificing the ICA is the identification of an individual who has poor collateral circulation requiring extracranial-intracranial bypass. The most reliable and available method for decision of the bypass is test balloon occlusion of the ICA during compressed spectral assay (digitized electroencephalography) monitoring.

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The authors have analyzed the results of long-term follow up of 11 patients with either giant or large cavernous sinus aneurysm treated exclusively with proximal occlusion. Proximal occlusion of the carotid artery was performed using the Silverstone clamping technique. The follow-up period covered 13.9 years. Good recovery was detected in 9 patients (81.8%) while 2 patients developed disabilities in the final outcome. The paper is a retrospective study of surgical results that should be interpreted carefully. The method used to collect the information about the patient outcome (either by mail or telephone interviews) may not be the best one because it is subject to errors and misinterpretation. Three patients (27%) presented with ischemic complications despite the pre-surgical evaluation using the Matas test. This must be highlighted because it indicates that this test is not accurate to predict low flow complications in the long run. The use of modern methods, such as the transcranial Doppler, combined with the Matas test, may improve the results by providing better selection for patients treated by artery occlusion. We suggest a prospective study using more modern methods of evaluation to clarify the results of this treatment.

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Neur Med Chir (Tokyo) 40, February, 2000