Pseudo- and Segmental Occlusion of the Internal Carotid Artery: A New Classification, Surgical Treatment and Results

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Objectives: Occluded internal carotid arteries imply a high risk of ischaemic complications, but an “occluded” carotid artery is not always totally occluded. Pseudo- and segmental occlusions can be detected angiographically, and increasingly non-invasively, and include a variety of morphologic findings.

Methods and Materials: 128 patients with pseudo- or segmental occlusion were treated in a 13 year period. Three different types of pseudo- or segmental occlusion were identified. In most cases a subtotal stenosis (near-occlusion) at the carotid bifurcation is the underlying lesion (type I). In approximately 35% the internal carotid artery is totally occluded at the bifurcation, but collaterals prevent downstream occlusion (type II), or retrograde flow from the circle of Willis and ophthalmic artery preserves a patent petrous part and siphon (type III).

Results: In 79% patency of the arteries could be restored. Three patients (2.3%) died perioperatively, nine (7%) developed ischaemic stroke (7 ipsilateral, 2 contralateral), one intracerebral haemorrhage. The combined stroke-mortality rate was 8.6%. During follow-up (41 ± 29.9 months) four patients (4.5%) experienced a stroke (3 ipsilateral, 1 contralateral), one an intracranial (1.1%) haemorrhage and six transient ischaemic attacks (6.7%). The annual ipsilateral stroke rate was 0.9%, the cumulative patency rate of the entire series 78% after 73 months.

Conclusions: Although the surgical management carries an increased risk of complications (stroke, transient ischaemic attacks) compared to conventional carotid endarterectomy it is likely that the stroke risk can be reduced at least for symptomatic patients. Symptomatic internal carotid artery occlusion diagnosed non-invasively should be confirmed angiographically to exclude pseudo- or segmental occlusion.

Key Words: Carotid artery pseudo-occlusion; Carotid artery segmental occlusion; Carotid artery occlusion; Carotid endarterectomy.

Introduction

Internal carotid artery (ICA) occlusion is usually regarded as an end-stage process without further options for extracranial reconstruction. Efforts to restore blood flow in acutely occluded internal carotid arteries have led to high morbidity and mortality, especially in patients with acute or progressive stroke.1–3 Although some authors have reported satisfactory results in subgroups of patients without neurological deficits,4 surgery has on the whole been abandoned as a treatment for established internal carotid artery occlusion as well as for acute occlusion. The nature of these occluded arteries was thought to be stable and without further risk of embolisation. However, several studies have shown that, contrary to widespread belief, a totally occluded ICA implies a high risk of ischaemic complications. The annual ipsilateral stroke rate varies between 2 and 10%.5–11 Patients with high-grade (>90%) ICA stenoses are therefore now generally regarded as candidates for carotid thrombendarterectomy to prevent occlusion. The annual ipsilateral stroke risk of patients after successfully performed carotid thrombendarterectomy (TEA) varies from 1% to 3%.12–17

Occasionally pseudo-occlusion of the ICA is detected incidentally on angiography despite non-invasive evidence of total occlusion. Many of these patients present with ischaemic symptoms. Using an appropriate angiographic technique a patent petrous part, delayed visualisation of the distal ICA, or a subtotal stenosis of the carotid bifurcation can be identified. Success in the early 1980s in restoring blood flow in a few of our patients with initially diagnosed
total occlusion of the ICA, after incidentally finding that the ICA was patent, led us to perform careful angiographic examination of patients with non-invasively diagnosed occlusion of the ICA to exclude "pseudo"-occlusion. This report summarises our experiences in patients with "segmental"- and "pseudo"-occlusion of the ICA.

**Methods and Materials**

The angiograms of patients treated surgically for segmental and pseudo-occlusion of the ICA were studied retrospectively. Since 1984 arteriography in our institution was performed using a modified technique recommended by Countee and Vijayanathan. This technique consists of selective catheterisation or direct puncture of the common carotid artery, prolonged injection of contrast medium and prolonged lateral filming with visualisation of the carotid bifurcation, the petrous part and the siphon. Nowadays, using digital substraction angiography (DSA), the identification of pseudo- and segmental occlusions is simplified.

**Classification**

The angiographic results were compared with intraoperative findings and lead to a new classification with three categories of pseudo- and segmental occlusion:

- **Type I**: subtotal stenosis with delayed, but orthograde filling of the entire ICA.
- **Type II**: total occlusion of the ICA at the carotid bifurcation, but delayed orthograde filling of the cervical portion and the siphon via atypical collaterals of the proximal ICA (not always detected angiographically!).
- **Type III**: no visualisation of the cervical ICA, but patent petrous part and siphon, due to retrograde filling of the ICA via the Circle of Willis and ophthalmic artery.

Type I is defined as pseudo-occlusion, as it represents a subtotal stenosis, while type II and type III are termed as segmental occlusions. Occasionally it is difficult to differentiate angiographically between a type I and type II lesion. In most cases of type I a thread of contrast material is visualised in late sequences at the carotid bifurcation when the contrast filling of the external carotid branches is already over. In type II segmental occlusion the central carotid stump, the external branches and the filling of the ICA mainly via the Circle of Willis visible simultaneously.

**Surgical strategy**

Depending on the type of pseudo- or segmental occlusion simple TEA of the carotid bifurcation with saphenous vein patch plasty, vein graft interposition or occasionally ligation of the ICA was performed. In some cases with type III segmental occlusion the carotid bifurcation was only explored. If the ICA had to be ligated, TEA and vein patch plasty of the external carotid artery was performed.

In order to reduce the perioperative stroke risk the following strategy was employed:

1. ICA with a normal-size lumen and backflow with or without clot: expression of the clot from the distal part of the ICA followed by carotid TEA.
2. Hypoplastic ICA with a lumen and weak backflow: insertion, gentle inflation and careful withdrawal of a 2-French Fogarty catheter. If this was followed by vigorous backflow and occasional fresh clots then carotid endarterectomy, but if there was weak backflow then ligation or clipping of the ICA.
3. Hypoplastic ICA with organized fibrous clot, weak or no backflow: insertion, inflation and gentle withdrawal of a 2-French Fogarty catheter. If increased backflow and angiography showed a normal sized distal lumen then vein graft interposition or thrombendarterectomy. If no increase in backflow then ligation or clipping of the ICA.

**Material**

From 1982 to 1994, 128 patients (101 male, 27 female, mean age 60.6 years, range 43–77) underwent surgical treatment for pseudo-or segmental occlusion of the ICA (62 right, 66 left). Contralateral involvement of the ICA was present in 60 (46.9%) patients (remote or recent stroke 8, transient ischaemic attack (TIA) 11 cases), of whom 28 (21.9%) had significant ICA stenosis (>80%) and nine (7%) ICA occlusion. The contralateral ICA was treated surgically in 23.4% (30) of the patients, as a preceeding procedure in 10 cases.
(7.8%), shortly after treatment of the segmental or pseudoocclusion in 20 patients (15.6%). Ipsilateral ischaemic symptoms related to the side of pseudo- or segmental occlusion in 84.4% (stroke 46.1%, TIA 38.3%) led to intensified diagnostic investigations and detection of pseudo- or segmental occlusion. (7 patients presented with progressive stroke). All patients had one or more risk factors (hypertension 71%, diabetes 25%, hyperlipidaemia 44.5%, coronary heart disease 40.6%, previous myocardial infarction 19.5%). All patients were examined by a neurologist.

Cerebral angiography was performed routinely after non-invasive Doppler and colour-coded imaging to visualise the lesion and the collateral pathways. In no patient did an angiography-related complication occur. Eighty-one (63.3%) of the lesions were classified as type I, 13 (10.2%) as type II and 34 (26.6%) as type III. In at least two of the symptomatic patients a floating thrombus was identified at the petrous part or siphon (Fig. 4). In one patient a type I pseudo-occlusion had been diagnosed at an outside hospital 3 months previously. The lesion had been regarded as untreatable and anticoagulation was started. When the patient was admitted, he presented with type III segmental occlusion. Unfortunately there was no backflow from the petrous part of the ICA and the artery was ligated. This case underlines that type I and type III occlusions simply represent different stages in the progression of the atherosclerotic disease towards total occlusion.

Thrombendarterectomy (TEA) of the carotid bifurcation was performed in 82% (n = 105), saphenous vein graft interposition in 5.5% (n = 7). Ligation of the ICA could not be avoided in 12.5% (n = 16) mostly in type III segmental occlusion (Table 1).

Results

Three (2.3%) patients died perioperatively (30 days).

Fig. 1. Pseudo-occlusion type I, merely a subtotal occlusion of the ICA with orthograde filling in late sequences.
Fourteen (10.9%) patients experienced neurologic symptoms, nine suffered an ischaemic stroke (6 ipsilateral, 2 contralateral, 1 brainstem), one an intracerebral haemorrhage, four a TIA. One ICA in a female patient with an iatrogenic av-fistula was occluded using a detachable balloon. The overall perioperative stroke rate (+ 1 haemorrhage) was 7.8%, the mortality rate 2.3% (pulmonary artery embolism, intracerebral haemorrhage, extensive hemispheric stroke), the combined stroke-haemorrhage-mortality rate was 8.6% (Table 2). Eight carotid arteries (6.2%) occluded in the early postoperative period and 78.9% (n = 101) of the arteries operated upon were patent at discharge (Table 3).

Follow-up information could be obtained for 89 patients (mean follow-up time: 41.4 ± 29.9 months).

Fig. 2

(a) Segmental occlusion type II, retrograde flow via atypically originating ascending pharyngeal artery (two arrows), in late sequences orthograde filling of the ICA close to the bifurcation (arrow). (b) Intraoperative finding in segmental occlusion type II, longitudinal incision of the ICA, external carotid artery clamped, backflow of the ICA blocked with a dilator, atypically originating ascending pharyngeal artery (arrow), same case as (a).
22 had died (19 after discharge), 19 patients (14.8%) were lost to follow up, and eight were only treated within the last 6 months. Late follow-up examinations (clinical and ultrasound) were performed in 79 patients (61.7%). Two arteries had occluded (1 month, 23 months) and 65 (82.3%) were patent. Twelve (13.5%) out of 89 patients (including 10 patients who had died from known causes) had experienced cerebral events (Table 4). Three out of four strokes and six out of seven TIA occurred ipsilateral to the treated ICA. The ipsilateral annual stroke rate was 0.9% and the TIA rate 1.8%. In all patients with ipsilateral stroke the ICA was found to be patent without restenosis.

*Cumulative patency rates*

The cumulative patency rate for all patients with pseudo- and segmental occlusions was 78% after 73 months. The patency rate for type I occlusions (89%) was better than for type II (64%) and type III (57%), which is understandable, as in type II and III occlusions exploration of the carotid bifurcation and ligation of the ICA had been performed more often (Fig. 5).

**Discussion**

Chronic occlusion of the ICA is a condition not suitable for extracranial vascular surgery. However, an ICA diagnosed as “occluded” is not always totally occluded and can cause further ipsilateral hemispheric symptoms. Using improved and modified radiological techniques and modern technical facilities it became obvious that a non-invasively diagnosed or even a
long-term occluded ICA might occasionally be found to be patent not only in the petrous part of the artery. These cases have been classified as “pseudo-occlusion”, “segmental occlusion” or “nearly occluded ICA”. Specific angiographic signs were identified, including the “string sign” first published for ICA dissection, picked up in 1976 by Ehrenfeld and Wylie and today most often used and the “slim sign” reported by Lippman et al. Due to subtotal stenosis or collateral blood flow the petrous part and siphon of the ICA can remain patent. Delayed stroke on the side of pseudo- or segmental occlusion may be due to emboli arising from the extracranial ICA stump via external collateral vessels or the “tail” of a thrombus distal to the ICA occlusion.

With conventional plane film angiography these

![Fig. 4. Type I pseudo-occlusion with floating thrombus in the petrous part (arrow), removed thrombus material, postoperative angiogram.](image)

<table>
<thead>
<tr>
<th>Pseudo-occlusion</th>
<th>Type I (n=81)</th>
<th>Type II (n=13)</th>
<th>Type III (n=34)</th>
<th>Total (n=128)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombendarterectomy*</td>
<td>74 (91.3%)</td>
<td>10 (76.9%)</td>
<td>21 (61.8%)</td>
<td>105 (82.0%)</td>
</tr>
<tr>
<td>Vein graft interposition</td>
<td>2 (2.5%)</td>
<td>2 (15.4%)</td>
<td>3 (8.8%)</td>
<td>7 (5.5%)</td>
</tr>
<tr>
<td>Ligation of the ICA†</td>
<td>5 (6.2%)</td>
<td>1 (7.7%)</td>
<td>10 (29.4%)</td>
<td>16 (12.5%)</td>
</tr>
</tbody>
</table>

*Combined with vein patch plasty.
†Mostly combined with ECA reconstruction.
Table 2. Complication rate following surgical therapy of pseudo- and segmental occlusion of the ICA, combined stroke-haemorrhage mortality rate 7.8%

<table>
<thead>
<tr>
<th>Pseudo-occlusion</th>
<th>Type I (n=81)</th>
<th>Type II (n=13)</th>
<th>Type III (n=34)</th>
<th>Total (n=128)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality*</td>
<td>1 (1.2%)</td>
<td>-</td>
<td>2 (5.8%)</td>
<td>3 (2.3%)</td>
</tr>
<tr>
<td>Stroke</td>
<td>7 (8.6%)</td>
<td>1 (7.7%)</td>
<td>1 (2.9%)</td>
<td>9 (7.0%)</td>
</tr>
<tr>
<td>- stroke ipsilateral</td>
<td>5 (6.1%)</td>
<td>-</td>
<td>1 (2.9%)</td>
<td>6 (4.7%)</td>
</tr>
<tr>
<td>- stroke brainstem</td>
<td>1 (1.2%)</td>
<td>-</td>
<td>-</td>
<td>1 (0.8%)</td>
</tr>
<tr>
<td>- stroke contralateral</td>
<td>1 (1.2%)</td>
<td>1 (7.7%)</td>
<td>-</td>
<td>2 (1.6%)</td>
</tr>
<tr>
<td>- haemorrhage</td>
<td>1 (1.2%)</td>
<td>-</td>
<td>-</td>
<td>1 (0.8%)</td>
</tr>
<tr>
<td>TIA</td>
<td>3 (3.7%)</td>
<td>1 (7.7%)</td>
<td>-</td>
<td>4 (3.1%)</td>
</tr>
<tr>
<td>- ipsilateral</td>
<td>2 (2.5%)</td>
<td>1 (7.7%)</td>
<td>-</td>
<td>3 (2.3%)</td>
</tr>
<tr>
<td>- contralateral</td>
<td>1 (1.2%)</td>
<td>-</td>
<td>-</td>
<td>1 (0.8%)</td>
</tr>
</tbody>
</table>

*Pulmonary artery embolism, cerebral haemorrhage, stroke.

Fig. 5. Cumulative patency in 128 patients with segmental-or pseudoocclusion of the ICA, according to the type of occlusion. (©) type I (n = 81); (●) type II (n = 13); (○) type III (n = 34).

Table 3. Early results after treatment of pseudo- and segmental occlusion of the ICA

<table>
<thead>
<tr>
<th>Patients</th>
<th>128</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perioperative mortality</td>
<td>3</td>
</tr>
<tr>
<td>Ligation of ICA</td>
<td>16</td>
</tr>
<tr>
<td>Postoperative occlusion</td>
<td>8</td>
</tr>
<tr>
<td>- asymptomatic*</td>
<td>4</td>
</tr>
<tr>
<td>- TIA†</td>
<td>1</td>
</tr>
<tr>
<td>- stroke‡</td>
<td>3</td>
</tr>
<tr>
<td>ICA patent at discharge</td>
<td>101</td>
</tr>
</tbody>
</table>

*In one patient with av-sinus cavernous-fistula occlusion of the ICA by a detachable balloon.
†Included in Table 2.
‡ICA patent in three patients.

Table 4. Late follow-up results, mean follow-up 41.1 ± 29.91 months, annual ipsilateral stroke rate 0.9%, annual ipsilateral TIA rate 1.8%

<table>
<thead>
<tr>
<th>Patients</th>
<th>128</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early and late mortality</td>
<td>22</td>
</tr>
<tr>
<td>Lost for follow-up</td>
<td>19</td>
</tr>
<tr>
<td>6 months after operation</td>
<td>8</td>
</tr>
<tr>
<td>Clinical and ultrasound examination</td>
<td>79</td>
</tr>
<tr>
<td>- ICA patent</td>
<td>65</td>
</tr>
<tr>
<td>- ICA closed during follow-up</td>
<td>2</td>
</tr>
<tr>
<td>- ICA closed, ligated intraoperatively</td>
<td>9</td>
</tr>
<tr>
<td>- ICA closed perioperatively</td>
<td>3</td>
</tr>
<tr>
<td>Follow-up information available</td>
<td>89</td>
</tr>
<tr>
<td>Cerebral events</td>
<td>12</td>
</tr>
<tr>
<td>- stroke ipsilateral*</td>
<td>3</td>
</tr>
<tr>
<td>- stroke contralateral†</td>
<td>1</td>
</tr>
<tr>
<td>- haemorrhage contralateral†</td>
<td>1</td>
</tr>
<tr>
<td>- TIA ipsilateral‡</td>
<td>5</td>
</tr>
<tr>
<td>- TIA bilateral‡</td>
<td>1</td>
</tr>
</tbody>
</table>

*ICA patent in all cases.
†IPSilateral ICA occluded.
‡ICA patent in three patients.
occlusion does not in all cases reflect the true dimension, especially not the extent of the patent cervical ICA. Retrograde filling downstream from the carotid bifurcation depends on pressure gradients, turbulences and dilution of the contrast medium. Modern DSA simplifies the identification of these pseudo- and segmental occlusions of the ICA as smaller and more highly diluted amounts of contrast media can be detected. The above described technique by Countee and Vijayanathan is our standard procedure in patients with non-invasively diagnosed ICA occlusion undergoing cerebral angiography and in patients with symptoms related to the side of a presumed ICA occlusion. It is well known, that the stroke rate of cerebral angiography ranges between 1 and 2% but fortunately no patient of this series suffered a serious ischaemic cerebral complication.

A classification of ICA pseudo- and segmental occlusion was attempted by Archie. On the basis of pathologic intraoperative or anatomic findings, the author defined six different categories in 17 patients with 18 operations, including diameter and fibrotic reaction of the ICA, plaque and extent of thrombus. The classification presented here, based on the anatomic location and the extent of the “pseudo”- or “segmental” occlusion including collateral pathways, is simple and easier to understand. Based on delayed-film DSA and intraoperative findings we propose three types of “segmental” or “pseudo”-occlusion, depending on the location of the atherosclerotic lesion and collateral blood flow. Besides filling of the siphon via the ophthalmic artery, often combined with more extensive occlusion of the ICA (type II), in approximately 10% the angiographic findings indicated filling of the cervical ICA via an ascending pharyngeal or other unspecified artery (collateral from the vertebral artery via the occipital artery) originating atypically from the ICA 2-4 cm distal to the carotid bifurcation (type II). This was confirmed intraoperatively (Fig. 2b). The atypical origin of the pharyngeal artery from the ICA has occasionally been reported. Whereas type I and type III can be regarded as different stages in the time course progressing towards total ICA occlusion, these type II will probably remain patent in most cases.

Type III segmental occlusion may include cases of recent ICA occlusions as well as chronic conditions. Several authors underline that a recently occluded ICA can be treated surgically in many cases and patency can be achieved in 50-60%. Hugenholtz and Egli performed a grading of ICA collateral supply (5 grades) and pointed out that, in cases with a patent petrous part, patency of the ICA can be restored in approximately 50%. Depending on the age of the thrombus the treatment can be very simple. Fresh clots are flushed out by the backflow after TEA of the carotid bifurcation or can be easily removed with a gently withdrawn small Fogarty catheter. Occasionally, however, a weak backflow or a hypoplastic ICA is found, sometimes filled with a fibrous organised clot. These cases have to be treated extremely carefully, and the better treatment may be ligation of the ICA to prevent stroke or TIA from thrombus formation at a distal intimal flap after TEA. The high risk was identified in 12.5% of the cases of the whole series and in 29% of the patients with type-III-occlusion. A safe reconstruction and restoration of patency could not be achieved and ligation of the ICA was performed, mostly combined with a vein patch plasty of the ECA. In cases of a weak backflow no vigorous attempts have to be performed to restore patency of the artery. Once the clots have propagated up to the siphon surgical treatment is no longer appropriate.

In several of our cases with type III occlusion with patent petrous part and siphon, a chronic occlusion was found intraoperatively. In these cases no, or weak, backflow could be established and the ICA was therefore ligated. Apart from collateral flow via the opthalmic artery, other atypical collaterals may be responsible for keeping the petrous part patent. The intracavernous branches of the ICA may serve as collateral vessels. They can be remnants of the embryonic arteries due to incomplete regression or persistence (dural arteries, persistent hypoglossal artery, persistent trigeminal artery). Up to now, there is no reliable diagnostic parameter in these cases to differentiate between chronic and acute occlusion of the non-visualised cervical ICA except the patient’s history. If there have been recent ipsilateral hemispheric symptoms, acute occlusion can be assumed and exploration of the carotid bifurcation is indicated from our point of view. This easily explains the 50-60% success rate following surgery of recently occluded ICA and the number of ligated arteries in the type III segmental occlusions presented here. When we started to investigate the problem of pseudo-occlusion, all of our patients presented with acute ischaemic symptoms related to the side of presumed ICA occlusion. This rate is decreasing in recent years. Patients with an apparently occluded ICA are studied exactly non-invasively by Doppler ultrasound and Duplex examination, the angiograms are examined for discrete signs of pseudo- or segmental occlusion (string sign, retrograde filling of the siphon). The number of identified and treated pseudo- and segmental occlusions is now steadily increasing (62 patients (48.4%) were treated within the last 5 years) mainly because colour Doppler assisted Duplex
imaging has increased the sensitivity to detect slow residual flow in the distal ICA. Thus many, occasionally asymptomatic, type I pseudo- and type II segmental occlusions are now found by non-invasive means, whereas type III is detected angiographically only. Angiography is, however, still necessary and is performed in all of our patients, because the collateral flow has to be known and several authors stress that non-invasive studies cannot distinguish reliably enough between pseudo-occlusion and total occlusion. The accuracy of Doppler-ultrasound in detecting and differentiating total occlusion from pseudo-occlusion ranges between 75 and 85%, but recent reports publish an increasing sensitivity in detecting slow residual flow by colour-coded Duplex imaging.

The angiographic diagnosis of pseudo- and segmental occlusions contains pitfalls and traps, which is well known for many years. Already in 1971, Clark et al. published different types of pseudo-occluded arteries and suggested that the following signs may indicate patency of segments of the ICA:

1. Visualisation of a short proximal stump of the ICA with a tapered end beyond the carotid bifurcation.
2. Visualisation of a small thread of contrast material corresponding to the course of the occluded vessel.
3. Intracranial filling of the ICA by sources other than the ophthalmic artery. These angiographic findings correspond exactly to those of our type I and type III lesions.

Surgical management is, from our point of view, the treatment of choice for a detected pseudo- or segmental occlusion of the ICA, as most patients present hemispheric symptoms and a conservative management cannot prevent further embolisation. Most of the presented cases here were detected by careful diagnostic work-up of patients with recent ischaemic hemispheric symptoms despite a noninvasively or angiographically suspected ICA occlusion. In these cases embolisation will probably originate from the “stump” downstream of the occlusion which could be demonstrated in at least two of our cases with floating thrombus material in the petrous part of the ICA. (Fig. 4) In recent years an increasing number of asymptomatic patients have been detected probably due to increasing experience and high quality Duplex scanning. From our point of view surgery is indicated also for asymptomatic patients. We are convinced that type I and type III occlusion are only different steps in the progress towards total occlusion and those presently asymptomatic patients carry the risk of a symptomatic occlusion and further ipsilateral hemispheric symptoms related to the totally occluded artery. Even if there is some doubt, attempts to establish the diagnosis of a pseudo- or segmental occlusion should be made, and in special cases exploration is justified to restore the patency of the artery if possible. However, surgery has to be performed extremely carefully and in some cases (hypoplastic ICA, fibrous organised thrombus, weak backflow) ligation may be the better treatment. The high rate of ischaemic complications in our type I group may be explained by over vigorous efforts in previous years to restore blood flow.

Up to now long term follow-up results after treatment of pseudo- or segmental occlusion of the ICA have not been available. Although this cohort carries an elevated risk of intraoperative stroke and TIA, the long term results with an annual ipsilateral stroke risk of <1% underline that, in contrast to chronic ICA occlusion, a better prognosis can be expected.

From our data we can conclude:

1. Pseudo- or segmental occlusion of the ICA is not as rare as suspected.
2. In patients with non-invasively suspected ICA occlusion and ipsilateral ischaemic symptoms further diagnostic attempts should be performed to exclude segmental or pseudo-occlusion.
3. High quality colour-flow Duplex scanning can detect type I and II occlusions, but angiographic confirmation is still necessary for surgical intervention.
4. Exploration of the ICA is indicated to prevent total occlusion.
5. In selected cases ligation of the ICA is a better alternative than reconstruction.
6. Long-term results after successful treatment of pseudo- or segmental occlusion of the ICA correspond to those after carotid thrombendarterectomy.

The likely outcome of segmental and pseudo-occlusion of the ICA is progression to full-length occlusion. A substantial number of these patients suffer from ischaemic hemispheric symptoms and a totally occluded artery implies a further high risk of annual cerebral complications. Although, compared to conventional carotid endarterectomy, the surgical management carries an increased risk of perioperative ischaemic complications, we think surgery is indicated. Vascular surgeons and neurologists should consider and exclude pseudo- and segmental occlusion of the ICA in patients with assumed total
occlusion who present with related ischemic symptoms.

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