Interventional Stroke Therapy

Olav Jansen
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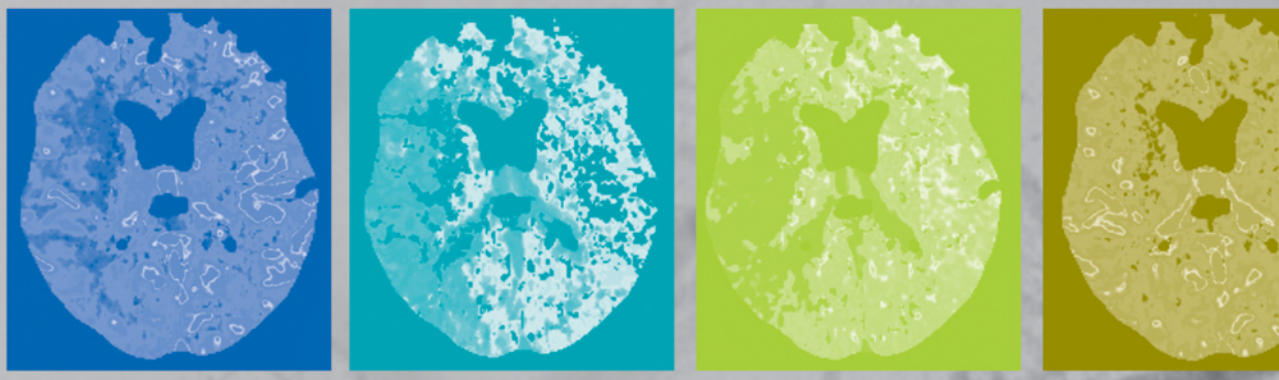

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Preface

The first endovascular recanalization was performed 30years ago by Hermann Zeumer. His technique for the local intra-arterial fibrinolysis of vertebrobasilar and middle cerebral artery occlusions was a major milestone in the development of interventional treatments for stroke.

Advances in both the diagnosis and treatment of stroke have followed in quick succession during the past three decades.

Initially, cranial CT could distinguish only between hemorrhage and ischemia and could supply information on the etiology of strokes. MRI, developed approximately 10 years after cranial CT, was initially important only for the evaluation of brainstem infarctions. But some 20 years ago MRI gained the ability to provide high-contrast diffusion-weighted images of a fresh cerebral infarction. The capabilities and individual components of "stroke MRI" significantly advanced our understanding of the pathophysiology of stroke and especially ischemic stroke. Today, the options of CT angiography and perfusion CT (*stroke CT*) have made computed tomography the first-line imaging study in stroke patients–especially for the relatively common ischemic strokes in the anterior circulation.

There has been equally rapid growth in the range of available interventional devices and techniques, fibrinolytic agents, platelet-aggregation inhibitors, and platelet-receptor inhibitors.

The wide range of techniques available for the diagnosis and interventional treatment of acute stroke prompted us to publish an "inventory" of these options in the form of a textbook. While we know that the techniques are still in flux, this is an opportune time to review current methods in detail, rate their efficacies, and make progress toward uniformity.

Fortunately, we had access to a strong "network" of neuroradiologists in which highly experienced colleagues were able to collaborate in the creation of this book.

The book is intended for all colleagues who seek concise, clinically relevant information in their day-to-day practice of diagnosing and treating stroke patients.

Kiel and Munich
Winter, 2013

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Abbreviations

A1	A1 segment of the anterior cerebral artery
ACA	Anterior cerebral artery
ACST	Asymptomatic Carotid Surgery Trial
ACT	Activated clotting time
ADC	Apparent diffusion coefficient
AICA	Anterior inferior cerebellar artery
AP	Anteroposterior
APSAC	Anisoylated plasminogen-streptokinase activator complex
aPTT	Activated partial thromboplastin time
ASK	Australian Streptokinase Study
ASPECT	Alberta Stroke Program Early CT Score
atm	Atmospheres
AV fistula	Arteriovenous fistula
BASICS	Basilar Artery International Cooperation Study
b.w.	Body weight
CASES	Canadian Activase for Stroke Effectiveness Study
CCA	Common carotid artery
CE	Communauté Européenne (certification mark)
CI	Confidence interval
CNS	Central nervous system
CREST	Carotid Revascularization Endarterectomy vs. Stenting Trial
CSF	Cerebrospinal fluid
CT	Computed tomography
CTA	Computed tomographic angiography
CTP	CT perfusion imaging
CVST	Cerebral venous and sinus thrombosis
DAC	Distal access catheter
DSA	Digital subtraction angiography
DSC-PWI	Dynamic susceptibility-weighted perfusion MRI
DWI	Diffusion-weighted imaging
DW MRI	Diffusion-weighted magnetic resonance imaging
ECA	External carotid artery
ECASS	European Cooperative Acute Stroke Study
ECG	Electrocardiography
ECASS	European Cooperative Acute Stroke Study
ECST	European Carotid Surgery Trial
EMEA	European Medicines Evaluation Agency
EVA	Endarterectomy versus Angioplasty in Patients with Symptomatic Severe Carotid Stenosis
FII, FVII	Coagulation factor II, factor VII (etc.)
FDA	Food and Drug Administration
FLAIR	Fluid-attenuated inversion recovery
FOV	Field of view
GESICA	Grupo de Estudio de la Sobrevida en la Insuficiencia Cardiac en Argentina
h	Hour
HMCAS	Hyperdense middle cerebral artery sign
i.a.	Intra-arterial
IAT	Intra-arterial thrombolysis
ICA	Internal carotid artery
ICSS	International Carotid Stenting Study
ICU	Intensive care unit
INR	International normalized ratio
IU	International units
i.v.	Intravenous
IVT	Intravenous thrombolysis
lys-plas	Human-derived Lys-plasminogen
M1	M1 segment of the middle cerebral artery
MAST-E	Multicenter Acute Stroke Trial–Europe Study Group 1996
MAST-I	Multicenter Acute Stroke Trial–Italy 1995
mbar	Millibars
MCA	Middle cerebral artery
MIP	Maximum intensity projection
MPR	Multiplanar reconstruction
MPRAGE	Magnetization-prepared rapid acquisition gradient echo
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
mRS	Modified Rankin Scale, Modified Rankin Score
MT	Mechanical thrombectomy
MTT	Mean transit time
NASCET	North American Symptomatic Carotid Endarterectomy Trial
NIHSS	National Institutes of Health Stroke Scale
NINDS	National Institute of Neurological Disorders and Stroke
NMDA	N-methyl-d-aspartate
PA	Posteroanterior
PAI-1	Plasminogen activator inhibitor-1
PCA	Posterior cerebral artery
PI	Platelet inhibitor
PICA	Posterior inferior cerebellar artery
PSV	Peak systolic velocity
PTA	Peripheral transluminal angioplasty
PTT	Partial thromboplastin time
PWI	Perfusion-weighted imaging

RSAR	Removable stent-assistant revascularization
rtPA	Recombinant tissue plasminogen activator
s	Seconds
SITS-MOST	Safe Implementation of Thrombolysis in Stroke–Monitoring Study
SK	Streptokinase
SWAN	Susceptibility-weighted angiography
SWI	Susceptibility-weighted imaging
SPACE	Stent-Protected Angioplasty versus Carotid Endarterectomy
TIA	Transient ischemic attack
TOF MRA	Time-of-flight magnetic resonance angiography
tPA	Tissue-type plasminogen activator
TTD	Time to drain
TTP	Time to peak
U	Units
UK	Urokinase
WASID	Warfarin-Aspirin for Symptomatic Intracranial Disease

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Neuroimaging of Stroke

1 Internal Carotid Artery Occlusion

O. Jansen

Introduction

Segmental Anatomy of the Internal Carotid Artery

The intradural portion of the internal carotid artery (ICA) is divided into four segments that are named and numbered as (from distal to proximal):

- *C1 segment:* the segment below the division of the carotid artery into the middle and anterior cerebral arteries (the "carotid T")
- *C2 segment:* the distal horizontal segment of the cavernous part of the ICA
- *C3 segment:* the vertical segment of the cavernous part
- *C4 segment:* the proximal horizontal segment of the cavernous part

The more proximal portions of the ICA are referred to in clinical parlance (from distal to proximal) as the petrous part; the subpetrous, cervical, or suprabulbar ICA; and finally the carotid bulb and origin of the ICA.

Definition of Carotid Artery Occlusion

Note

Every occlusion site correlates with an underlying cause that is typical for that location.

For example, occlusions of the ICA at its origin or at the carotid bulb are almost always caused by atherosclerotic stenosis. Most occlusions of the cervical ICA are caused by an acute carotid dissection, which always terminates at the entrance to the petrous part of the vessel. An acute occlusion of the petrous part is rare but may be caused by atherosclerosis at that level. The same applies to acute occlusions of the cavernous part (C2–C4), which are generally caused by the occlusion of a local, preexisting atherosclerotic stenosis.

On the other hand, acute occlusions of the C1 segment or the carotid T are almost always due to embolism from a cardiac source or, less commonly, a carotid source. The carotid T is the first site of relative narrowing in the carotid circulation where an embolus of substantial size may become lodged at the arterial junction, causing an acute vascular occlusion, and may subsequently grow due to proximal and/or distal thrombosis. As a result, these carotid T or C1 occlusions typically present with a large thrombus burden.

Traumatic or spontaneous intracranial carotid dissections are a very rare cause of acute carotid occlusions. Spontaneous intracranial carotid dissections, which are even rarer than traumatic dissections, may occur in patients with fibromuscular dysplasia or may be secondary to vasculitis.

Frequency of Carotid Artery Occlusions

Acute occlusions of the ICA, whether intra- or extracranial, have been recognized as the cause of ~5% of all strokes. Like acute basilar artery thrombosis, they are quite rare but may have severe manifestations due to the potential magnitude of the acute cerebral perfusion deficit, especially with a carotid-T occlusion, as well as the severity of acute neurologic deficits and the potential size of the resulting brain infarction.

In patients with a well-formed circle of Willis that would allow for the development of a collateral circulation, acute carotid occlusions proximal to the C1 segment may be asymptomatic or may produce fluctuating clinical symptoms. The latter presentation reflects a deficiency of collateral flow that is blood pressure–dependent and may lead to a hemodynamic infarction pattern.

Imaging Studies

Computed Tomography

As in all patients with acute stroke, noncontrast computed tomography (CT) is the initial imaging study of choice for patients with an acute carotid artery occlusion

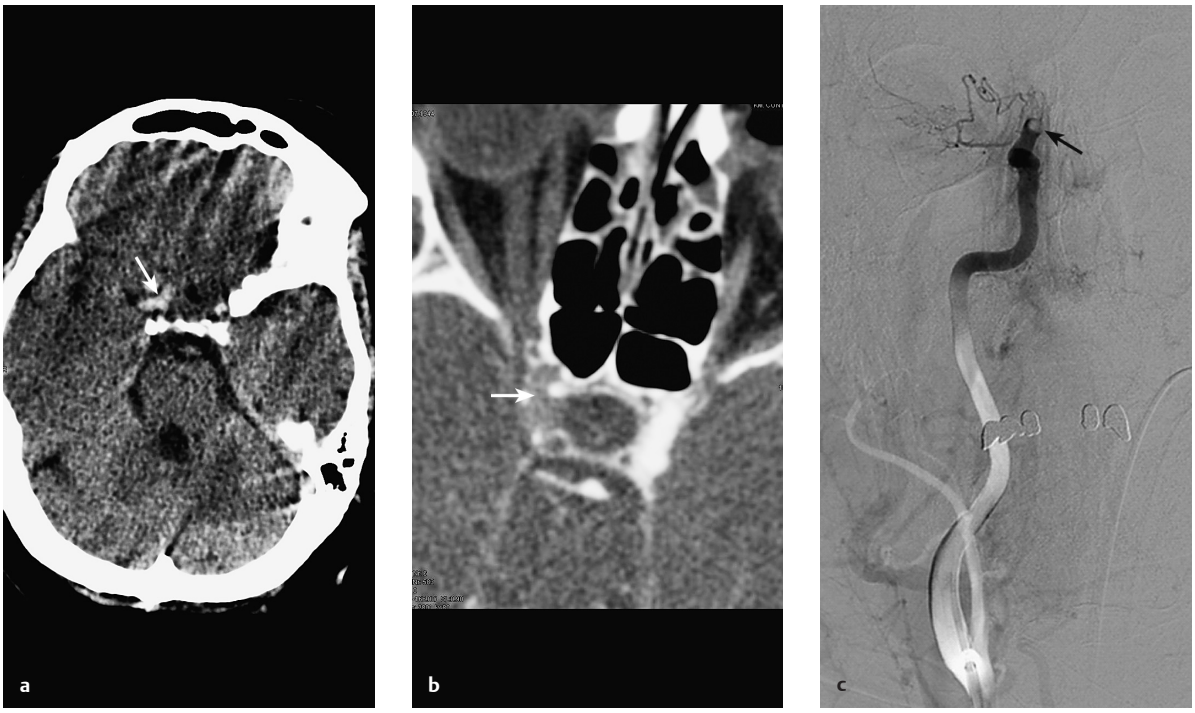

Fig. 1.1a–c Acute embolic occlusion of the carotid T.
a The hyperdense embolus is clearly visualized on cranial CT (*arrow*).
b CTA (source image) documents lack of opacification of the carotid siphon (*arrow*).
c DSA demonstrates the occlusion site in the C1 segment of the ICA and defines the proximal end of the thrombus (*arrow*).

(see Chapter 2). It can exclude hemorrhagic stroke and other causes of an acute neurologic deficit.

If the acute carotid occlusion is located proximal to the C1 segment, CT scans usually show no abnormalities other than possible early infarct signs (see Chapter 2 for more details). If collateral flow is deficient and clinical symptoms have been present for some time, CT images of these proximal carotid occlusions may demonstrate a chain of small infarctions in the terminal carotid branches above the level of the ventricles (cella media).

By contrast, the intravascular thrombi causing an acute occlusion of the carotid T and/or C1 segment almost always produce a hyperdense artery sign on noncontrast CT (see Chapter 2). This is particularly true when we note that the large thrombus burden of these occlusions allows for direct visualization of the thrombus even in relatively thick CT slices (**Fig. 1.1a**).

CT Angiography

CT angiography (CTA) is the CT technique of choice for the detection of carotid occlusion in acute stroke patients. Lack of opacification of the ICA and the imaging correlate of the carotid occlusion can be demonstrated during interpretation of the CT source images as well as with 2D and 3D reconstructions (**Fig. 1.1b**). On the other hand, accurate localization of the carotid occlusion may be difficult with CTA. Ordinarily, CTA is performed in such a way that data acquisition for the CT slices takes place during the first pass of the contrast material through the arterial circulation. In patients with a distal carotid artery occlusion, the more proximal carotid segments are occupied by a blood column that admits contrast material at a very slow rate, if at all. This happens too slowly to be detected during data acquisition for CTA.

Practical Tip

Even if some small distal branch vessels from the ICA are still patent, such as the ophthalmic artery, meningohypophyseal trunk, posterior communicating artery, or anterior choroidal artery, overall blood flow in the ICA is still so diminished that the artery will show a general lack of opacification and appear occluded on CTA. It may be helpful in these cases to carefully reevaluate the noncontrast CT for hyperdense vessel signs or even view the CTA source images with a different window setting to permit a more accurate localization of the occlusion site.

Based on the phenomenon of slow blood flow in the ICA described above, CTA may also yield false-positive results. This can easily cause a pseudo-occlusion to be missed. If necessary, a second CTA dataset can be acquired immediately after the first to help localize the occlusion or aid differentiation from a pseudo-occlusion.

As a rule, magnetic resonance imaging (MRI) with MR angiography (MRA) is rarely used in patients with acute stroke. The problems of imprecise localization and differentiation from pseudo-occlusion described above for CT and CTA apply with equal validity to MRI and MRA.

Angiography

The definitive diagnosis and localization of an ICA occlusion can be established by catheter angiography using digital subtraction (DSA) technique (**Fig. 1.1c**). However, the differentiation between occlusion and pseudo-occlusion relies on special techniques that require a cooperative or sedated patient or the use of general anesthesia.

Practical Tip

The diagnostic catheter should be placed just proximal to the presumed ICA occlusion site. This presents no difficulty with extracranial carotid occlusions. The contrast material is then injected at sufficiently high concentration (undiluted if necessary), and the acquisition time of the DSA series should be long enough to detect the slow trickle of contrast material into a blood column, if present, or into the collapsed poststenotic lumen of a pseudo-occlusion. An acquisition time of up to 10–15 seconds at a film rate of 1–0.5 frames/s may be necessary to avoid missing a pseudo-occlusion. Meanwhile, the development of a collateral circulation via extra- or intracranial vessels should be documented to detect, say, retrograde filling of the carotid siphon via the ophthalmic artery (**Fig. 1.2**).

Caution

DSA findings may be misinterpreted in patients with acute distal occlusions of the ICA unless the correct protocol is followed.

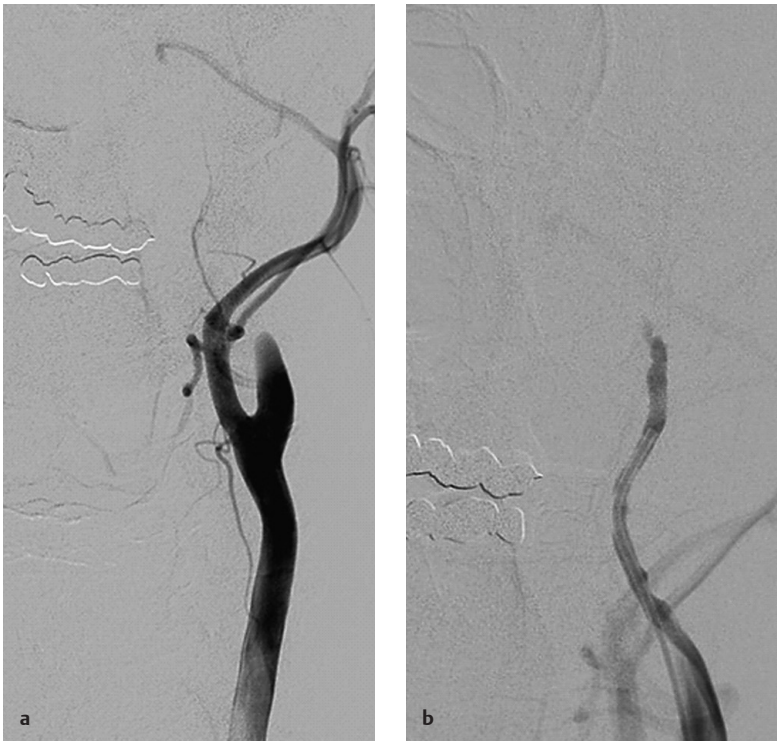

Fig. 1.2a,b Old occlusion of the cervical carotid artery on DSA.

a Survey image with contrast injection documents an apparent occlusion with pseudotapering at the top of the carotid bulb.

b Selective catheterization of the carotid stump confirms a definitive occlusion in the upper cervical portion of the ICA.

An occlusion of the ICA that also occludes the distal branches (ophthalmic artery, etc.) leads to the stasis of blood throughout the ICA lumen. When the diagnostic catheter is placed into the common carotid artery (CCA) or even the proximal ICA, there will be no distal runoff of injected contrast material and even a very long angiographic series may create the impression of a proximal occlusion. Not infrequently, the injected contrast material may layer along the standing blood column in the extracranial ICA, mimicking the tapered sign or string sign of an extracranial ICA dissection (see the section on Digital Subtraction Angiography, p. 33) (**Fig. 1.2a**). Doubts can be resolved only by a more distal catheterization of the ICA lumen, perhaps advancing the microcatheter past the petrous part of the artery and repeating the angiographic series so that the occlusion site can be accurately located (**Fig. 1.2b**). Sometimes the location and length of an occlusion are clearly appreciated only when endovascular recanalization is performed. By carefully injecting contrast through the microcatheter and advancing the microcatheter and microwire across the occlusion site into the intracranial circulation, the operator can define the entire lumen of the ICA, the occlusion, and the length of the thrombus. Meanwhile the location of the occlusion and whether it can be passed with a catheter can provide clues to the etiopathogenesis of the occlusion.

Pseudo-occlusion of the Internal Carotid Artery

Note

By definition, a pseudo-occlusion of the ICA is present when noninvasive vascular imaging demonstrates a vascular occlusion but invasive catheter angiography documents a high-grade stenosis or partial occlusion.

Three grades of ICA pseudo-occlusion have been described (Kniemeyer et al. 1996):

- *Grade I:* a high-grade stenosis of the ICA in which the original lumen of the carotid artery is preserved (**Fig. 1.3**).
- *Grade II:* a segmental occlusion of the actual carotid lumen, but the distal lumen still carries antegrade flow through vasa vasorum in the carotid wall. Usually the carotid lumen is markedly reduced (**Fig. 1.4**).
- *Grade III:* an actual carotid occlusion in which the ICA stump is perfused by retrograde flow from the external carotid artery (ECA), usually as far as the occlusion site (**Fig. 1.5**).

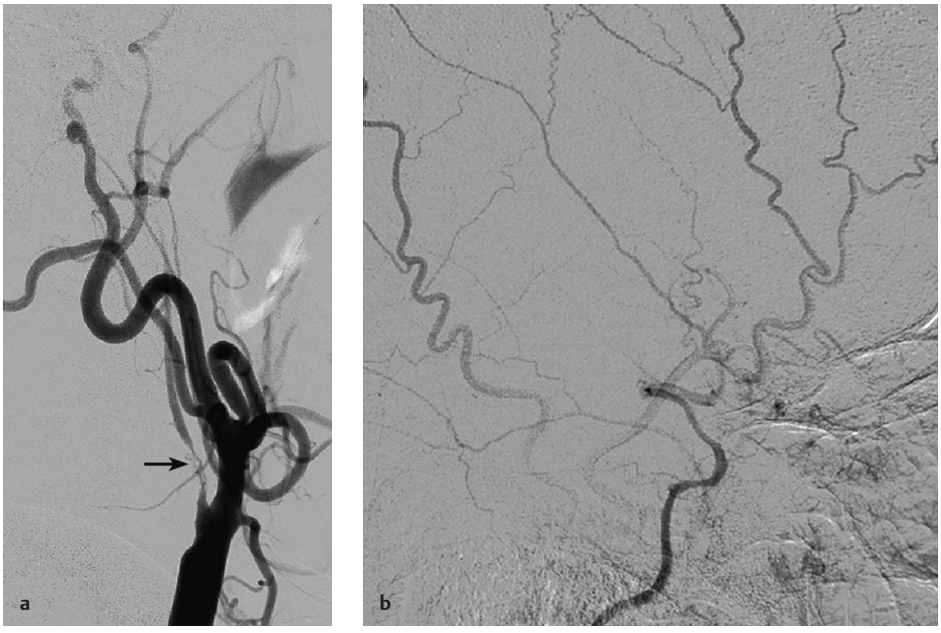

Fig. 1.3a,b Grade I pseudo-occlusion.

a Catheter angiography demonstrates a high-grade proximal stenosis of the ICA (*arrow*). The carotid lumen is preserved but markedly reduced in caliber.

b A long DSA series can still trace the narrowed carotid lumen into the cranial cavity.

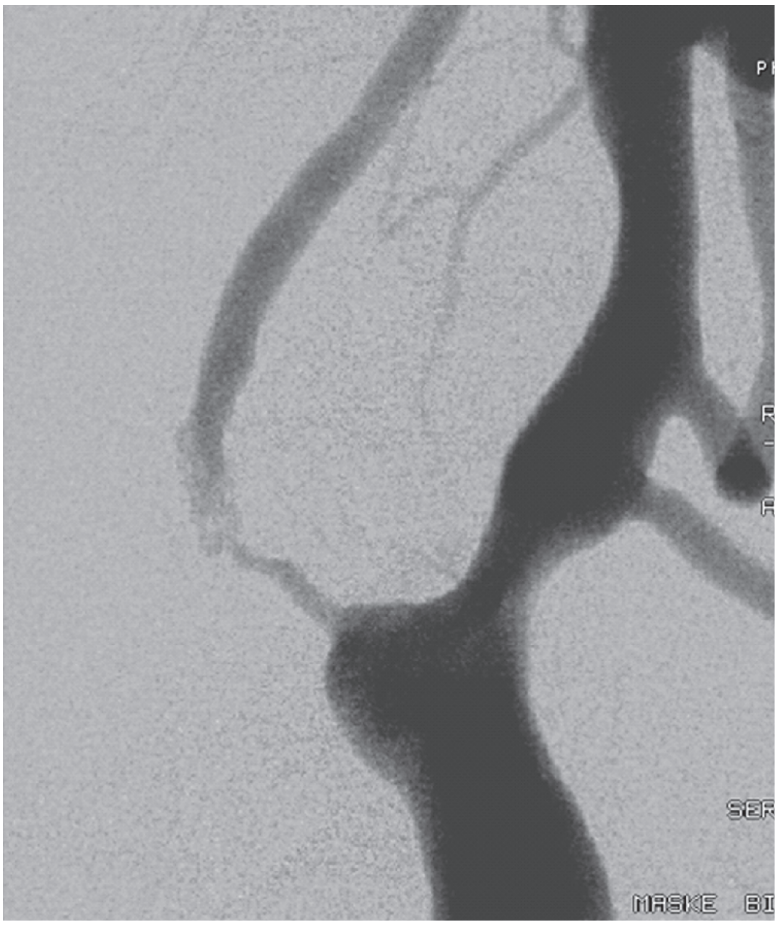

Fig. 1.4 Grade II pseudo-occlusion. Magnified DSA image of the common carotid artery demonstrates the recruitment of vasa vasorum. The original carotid lumen is occluded.

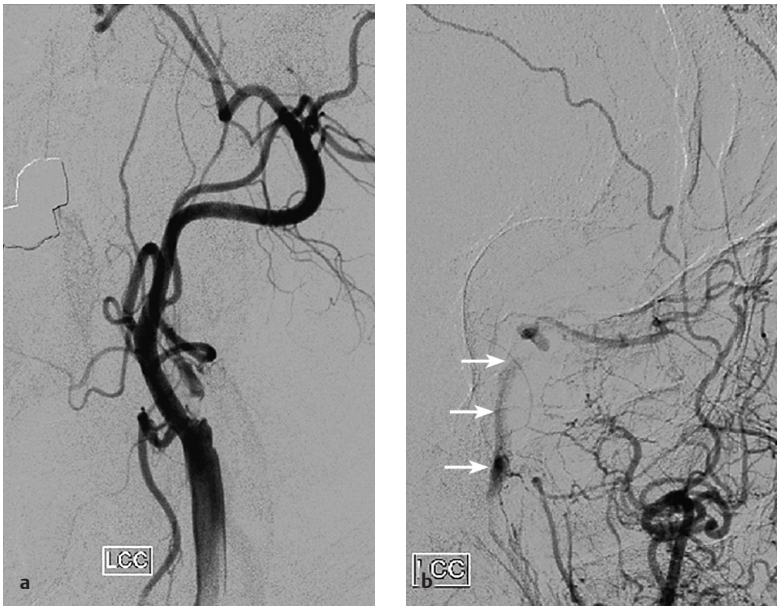

Fig. 1.5a,b Grade III pseudo-occlusion.
a Survey angiogram shows residual filling of the proximal ICA stump through very small lumina.
b The actual lumen of the cervical and petrous ICA is filled by retrograde flow from ECA collaterals (*arrows*).