Chapter 9
Carotid Artery Disease

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1 Anatomy

Familiarity with the extra- and intracranial vessel anatomy is decisive for successful treatment of carotid artery disease. The right common carotid artery originates from the bifurcation of the brachiocephalic trunk, while the left one arises directly from the aortic arch. The common carotid artery does not have any side branches. Usually at the level of the upper edge of the thyroid cartilage, it divides into the internal and external carotid arteries. The internal carotid artery supplies the anterior part of the brain, the eye and its appendages, and sends branches to the forehead and nose. Its size in the adult is equal to that of the external carotid artery, but it can be identified due to the absence of side branches in its extracranial course up to the intracranial branching point. Due to its course, the internal carotid can be divided into four parts:

– *Cervical part*: this portion ascends laterally behind the hypopharynx—where it can be palpated—and in front of the transverse processes of the cervical vertebrae to the carotid canal in the petrous part of the temporal bone.

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- *Petrous part*: in its passage through the carotid canal and along the side of the body of the sphenoid bone, it describes a double curvature.
- Cavernous part: This part ascends toward the posterior clinoid process, then passes forward by the side of the body of the sphenoid bone, and again curves upward on the medial side of the anterior clinoid. The ophthalmic artery arises from the internal carotid, just as that vessel is emerging from the cavernous sinus.
- *Cerebral part*: It begins distal to the origin of the ophthalmic artery and continues until the bifurcation into the anterior and middle cerebral artery.

2 Disease Definition

Carotid artery disease describes any alteration of the carotid artery that is clinically indicated to treat. A carotid artery stenosis is a narrowing constriction of the inner surface of the carotid artery, usually caused by atherosclerosis. The carotid bifurcation is a common site for a build up of plaque that can narrow the common or internal carotid artery. Carotid artery dissection is a spontaneous or traumatic separation of the layers of the artery wall. An aneurysm of the carotid artery is a localized, blood-filled, balloon-like bulge of a blood vessel. Aneurysms can be hereditary or caused by disease, both of which lead to the weakening of the blood vessels wall.

3 Disease Distribution

Cerebral ischemic events are the most frequent cause of stroke (80–85%) and currently the third leading cause of death in industrialized countries. It is well accepted that the presence of carotid stenosis is responsible for 20–30% of all strokes. Atherosclerosis causes about 90% of all carotid artery stenoses. Only about 10% are the result from a non-atherosclerotic disease.

Carotid artery dissection is the most common cause of stroke in young adults between 20 and 40 years of age. A total of 70% of all carotid dissections appear in this group.

The most common reason for extracranial aneurysms of the internal carotid artery is atherosclerosis. These aneurysms are mostly seen over the age of 50 and carry a high risk for thromboembolic stroke. The rupture risk is high in mycotic carotid aneurysms, whereas traumatic ones tend to stabilize and show regression.

4 Classification: Degree of Stenosis

Duplex ultrasound has 90–95% sensitivity and specificity in validation of stenoses of the internal carotid artery >50% compared to angiography. Its specificity and sensitivity for differentiating high-grade stenosis from occlusion is more than 90%.

In angiography, the degree of carotid stenosis can be measured using two different methods:

- European Carotid Surgery Trial (ECST) method: Measurement of the minimal lumen diameter in relation to the assumed diameter of the internal carotid artery at the site of maximal stenosis.
- North American Symptomatic Carotid Endarterectomy Trial (NASCET) method: Relation between the minimal lumen diameter and the diameter of the internal carotid artery distal to the stenosis.

Due to the fact that the anatomic relation between the carotid bulb and the distal internal carotid artery is constant, a conversion of these methods is possible:

$$\text{ECST-stenosis (\%)} = 0.6 \times \text{NASCET-stenosis (\%)} + 40\%$$

5 Clinical Findings

The clinical symptoms of stenosis of the internal carotid artery are characterized by ischemia in the corresponding region of cerebral flow. Symptomatic stenosis of the internal carotid artery is defined by

neurological syndromes of the ipsilateral hemisphere 6 months prior to the intervention:

- Transitory ischemic attack (TIA) = focal neurological deficit for less than 24 h.
- Amaurosis = monocular visual loss
- Stroke = neurological deficit for more than 24 h

With more widespread use of modern imaging techniques for the brain, up to one-third of patients with symptoms lasting less than 24 h have been found to have an infarction. This has led to a new tissue-based definition of TIA: a transient episode of neurological dysfunction caused by a focal ischemia without acute infarction.

6 Diagnosis

Carotid artery disease is usually diagnosed by color duplex ultrasound. In addition to the assessment of the degree of stenosis, this method also makes it possible to identify the plaque morphology (e.g., soft plaque, calcified plaque, or an ulcerated surface). Patients who should undergo duplex evaluation include:

- Symptomatic patients who have suffered a focal neurological deficit or amaurosis fugax.
- In asymptomatic patients, there are no standard diagnostic recommendations to date, except when a bypass operation is planned. In this situation, a duplex ultrasound examination is recommended in patients with history of cerebral ischemia or carotid bruit on auscultation. Besides duplex ultrasound should be performed in patients over 65 years, patients with stenosis of the left main coronary artery or peripheral arterial occlusive disease.

When duplex ultrasound results are unclear, diagnostic accuracy can be increased using supplementary computed-tomography angiography (CTA) or magnetic resonance angiography (MRA).

Cerebral computed tomography (CT) or magnetic resonance imaging (MRI) can also help exclude other causes of neurological symptoms, such as hemorrhage or tumor.

7 Management

7.1 Medical Treatment

Medical treatment is indicated in every patient in order to limit atherosclerotic progression and reduce the risk of a neurological event. This treatment recommendation is independent of the decision on whether to offer interventional or surgical revascularization therapy. Treatments currently available include inhibition of platelets using acetylsalicylic acid, dipyridamole plus aspirin, or clopidogrel. In addition, treatment with statins is advised due to their anti-inflammatory and thus plaque-stabilizing effect on the vascular bed.

Medical treatment alone is recommended in patients with stenosis of the internal carotid artery who either have a low risk of stroke (symptomatic stenoses <50%, asymptomatic stenoses <70%), who carry a high perioperative or peri-interventional risk due to comorbid conditions, or who have a limited life expectancy.

7.2 Endovascular Treatment

The aim of carotid stenting is to prevent stroke due to carotid artery stenosis. Typical candidates are symptomatic patients (TIA or stroke within 6 months) with >50% stenosis of the carotid artery, or asymptomatic patients with >70% stenosis of the carotid artery. Endovascular treatment is the preferred therapy in patients who are at increased risk for carotid surgery. High-risk features include medical comorbidities (severe heart disease, heart failure, severe lung disease, etc.) and anatomic features (radiation therapy of the neck, prior ipsilateral carotid artery surgery, concomitant intra-thoracic or intracranial carotid disease).

7.2.1 Patient Preparation

- Cranial CT or MRI examination
- Duplex ultrasound
- Aspirin 100–300 mg/day and clopidogrel 75 mg/day, starting at least 5 days before a planned intervention, or bolus administration

(aspirin 500 mg, clopidogrel 600 mg) on the day before the procedure.

7.3 Surgical Treatment

There are basically two techniques that can be used for plaque removal and reconstruction of the internal carotid artery or carotid bifurcation:

- Thromboendarterectomy (TEA) with a patch graft
- Eversion endarterectomy (EEA)

The operation can be performed under general or local anesthesia and with or without intraoperative shunting to decrease the risk of cerebral ischemia during the operation.

8 Intervention

8.1 Peri-interventional therapy

- Heparin (70–100 IU/kg) with an activated clotting time (ACT) of 250–300 s
- Electrocardiographic (ECG) monitoring due to potential bradycardia
- Blood pressure monitoring for possible hypotension related to carotid sinus stimulation by balloon inflation
- Intravenous administration of 1 mg atropine 2–3 min before implantation of the carotid stent, to prevent possible bradycardia or asystole (to be used with caution in patients with narrow-angle glaucoma)
- Infusions for hypotension

8.2 Access Route

Establishing a safe vascular access route in order to minimize complications during carotid stent implantation is essential. The access via the femoral artery is the approach most often employed. The common

femoral artery is punctured using a Seldinger needle and then a 5–6 F sheath is placed. Afterward, this initial sheath is exchanged during the procedure for a 90 cm long sheath. If a guiding catheter is to be used, a 8–9 F sheath is needed. In patients in whom the iliac arteries are occluded or who have high-grade stenosis in these arteries, or in situations in which the access route via the femoral artery is unavailable for other reasons, access via the brachial or radial artery can be obtained. The right brachial artery is preferable for interventions in both the right internal carotid artery and the left internal carotid artery. If neither access route is possible, direct cervical common carotid access (percutaneous or open surgical) might be considered.

8.3 Engaging the Supra-Aortic Arteries

Angiography of the aortic arch is often performed prior to selective carotid angiography in order to identify possible difficult anatomic conditions that might make it necessary to exchange the typically employed diagnostic catheters (e.g., Berenstein, Judkins Right, Head Hunter, IMS, JB-1) for an alternative one (e.g., Simmons or Vitek catheter). To engage the common carotid artery, the 5 F diagnostic catheter is positioned over a 0.035 in. hydrophillic guidewire in the ascending aorta with the catheter tip pointing downward. This technique reduces the likehood of embolization of aortic plaque or traumatic injury to the intima of the aortic arch and prevents the catheter from becoming caught in a vascular ostium. As soon as the catheter reaches the ascending aorta, it is rotated 180°. This places the tip of the catheter in a vertical, upright position on fluoroscopy. The catheter is then advanced over this wire into the common carotid artery.

To intubate the left common carotid artery, the catheter is slowly withdrawn from the ostium of the brachiocephalic trunk. It should be rotated 20° counterclockwise, so that the catheter tip points slightly anteriorly. When the aortic arch becomes increasingly kinked with advancing age, the ostium of the left common carotid artery is located slightly further posterior. In these cases, it may be necessary to rotate the catheter posteriorly instead.

Once the left common carotid artery has been entered, the catheter should be rotated back 20° clockwise, so that the tip is pointing vertically or slightly posteriorly. The catheter position is checked by administering a small amount of contrast. This can exclude subintimal

contrast flow or reduced blood flow. The hydrophilic wire is advanced to the distal common carotid artery, followed by the catheter.

If engagement of the common carotid artery is unsuccessful with the standard catheter, then a switch to a Simmons catheter is usually made. This type of catheter has a large reverse curve, which must be re-shaped in the aorta after wire removal, usually in the ascending aorta. Moving the catheter backward slightly guides the tip into the brachiocephalic trunk, then into the left common carotid artery, and finally into the left subclavian artery. In contrast, Vitek or Mani catheters have smaller pre-formed curves and do not require shaping. Therefore, these catheters are advanced forward, rather than withdrawn from, the distal aortic arch selecting the left subclavian artery first, and so on. Once the desired vessel is engaged, the wire is advanced followed by the catheter. Advancement of the catheter is carried out slowly with assistance from the pulsating blood flow. At the same time, the wire is withdrawn slightly, so that its position is maintained proximal to the carotid bifurcation. Advancement of the catheter and withdrawal of the guidewire are performed several times alternately until the catheter is safely positioned in the targeted vessel (push-and-pull technique).

8.4 Visualizing the Intracranial Vessels

Injections of contrast medium should be carried out manually or with a small amount of automated contrast administration (a maximum of 6 ml per injection). Larger amounts would lead to mixing of the arterial, intermediate, and venous phases, potentially leading to masking of early venous filling or other types of pathology. Some operators conduct four-vessel angiography to show the status of the collateral arteries. However, as this presents an additional procedural risk, the need for it is questionable, particularly in patients in whom MRA has previously been performed. During balloon dilatation, the absence of collaterals may cause short periods of cerebral ischemia due to brief occlusion of the internal carotid artery. However, this reaction is reversible after deflation of the balloon and has no influence on the completion of the procedure. Once the anatomy of the target vessel has been identified, a hydrophillic guidewire is advanced into the external carotid artery so that the diagnostic catheter can be exchanged for a sheath or a guiding sheath. Bony landmarks can be used for

guidance instead of road mapping to mark the origin of the external carotid artery during wire placement.

8.5 Vascular Kinking

If the vessel is very tortuous, it can be straightened using a wire. It is also helpful to ask the patient to inhale deeply and hold the breath. An acute vessel angle can be negotiated by careful rotation and advancement of the catheter until it has reached the desired position. If it is still not possible to advance the catheter, a Simmons III catheter should be used to introduce the guidewire into the external carotid artery. The Simmons III catheter can than be exchanged for a 4 F multipurpose catheter. After this, the hydrophillic wire is exchanged for a 0.035 in. Amplatz wire or a softer wire. Finally, the 4 F catheter is exchanged for a 5 F catheter.

8.6 Placing of the Guiding Catheter

An 8 F guiding catheter (e.g., right coronary or hockey-stick guiding catheter) is introduced into the ascending aorta via a hydrophillic 0.035 in. wire. It is advisable to use a 5 F catheter inside of the guiding catheter in order to avoid a step-up from the wire to the guiding catheter. In case of difficult or abnormal anatomy, aortography of the aortic arch can be used to assist in selective exploration. Following angiography of the aortic arch and assessment of the anatomy, the guiding catheter is introduced into the common carotid artery. This should be carried out by careful aspiration and flushing with saline to clear any possible atherosclerotic particles out of the catheter.

8.7 Placement of the Long Sheath

Engagement of the common carotid artery is carried out with a 5 F diagnostic catheter. Access to the external carotid artery is obtained with an angled hydrophilic guidewire and the diagnostic catheter introduced into the external carotid artery as described above.

The wire is then exchanged for a 0.035 in. wire, typically a stiff Amplatz wire. Afterward, the diagnostic catheter is removed and a 6 F 90 cm long sheath is placed using the over-the-wire technique into the common carotid artery below the carotid bifurcation. The sheath should be handled very carefully, as trauma to the common carotid artery ostium or release of atherosclerotic deposits may subsequently lead to neurological disorders. The sheath should be meticulously aspirated and flushed to eliminate possible air or atherosclerotic debris.

When the external carotid artery is occluded, when there is significant stenosis below the bifurcation, or when there is a stenosis at the ostium of the common carotid artery, placing the 6 F 90 cm port in the common carotid artery may represent a considerable challenge. If possible, crossing the stenosis with a stiff wire should be avoided, as this may dislodge plaque material and cause distal embolization. If necessary, the 5 F diagnostic catheter is advanced over a 0.035 or 0.038 in. guidewire for placement further distally, slightly proximal to the stenosis. It can then be exchanged over a 0.035 in. Amplatz wire (extra stiff). If there is an ostial/proximal stenosis of the common carotid artery, it may be necessary to treat this stenosis first in order to obtain access to the distal stenosis. However, if this stenosis is not severe, the bifurcation stenosis should be treated first, then the proximal stenosis on the way back.

8.8 Predilation

Some operators predilate the stenosis using a small angioplasty balloon and a short inflation time of 5–10 s. This provides for better passage and positioning of the stent. The present authors would only recommend predilation if primary stent placement has failed. In our view, primary stent implantation has a protective effect against distal embolization by fixing deposits on the vascular wall.

8.9 Embolic Protection

The possibility of peri-procedural cerebral embolization is an important concern in carotid angioplasty. Balloon dilatation, stent implantation, and manipulation of the vessels by catheters and wires

can easily release emboli, which if large enough, can cause severe cerebral damage. For this reason, embolic protection systems are routinely used in most centers. There are currently three different underlying principles on which protection is based: distal occlusion balloons, filter systems, and proximal occlusion balloons.

Distal occlusion balloons were the first embolic protection systems to become available, and were widely used in the initial carotid stent experience. It consists of a 0.014 in. guidewire with an occlusion balloon in the distal section, which is inflated and deflated through a very small channel in the guiding catheter (Guardwire® Temporary Occlusion and Aspiration System, Medtronic Vascular; TriActiv® ProGuard™ Embolic Protection System, Kensey Nash). After the guiding catheter is placed, the occlusion balloon is positioned distal to the stenosis and the balloon inflated until blood flow into the internal carotid artery stops. Stent implantation then follows. After the intervention, an aspiration catheter is introduced up to the occlusion balloon, and the blood in the occluded artery is aspirated. Any particles released during the intervention are thus removed. The advantages of the distal occlusion systems are the low profile, flexibility, and good steerability. Disadvantages include the fact that balloon occlusion is not tolerated in 6–10% of patients, and that the vascular segment distal to the occlusion balloon cannot be imaged during the balloon occlusion procedure.

Most *filter systems* consist of a metal framework that is covered with a polyethylene membrane or a nitinol mesh. The pore size can vary between 80 and 180 μm in diameter depending on the specific device. Filters are usually attached to the distal section of a 0.014 in. guidewire. In their closed state, filters are sheathed by an introducer catheter, and are introduced into the vascular segment distal to the stenosis. Once the stenosis has been crossed, the filter of choice is opened by withdrawing it into a recovery catheter, and then removed from the vessel. A wide range of second- and third-generation filter systems are currently available. The technical characteristics of a good filter consist of:

1. Low profile (less than 3 F)
2. Adequate steerability for maneuvering through highly tortuous vessels
3. When the filter is opened—good wall apposition to allow the best possible protection against emboli.

All distal protection systems, occlusion balloons, and filters have the potential disadvantage that the stenosis has to be crossed before the system can be deployed and protection established. This unavoidable step carries a risk of distal embolization during the initial unprotected phase of the procedure. *Proximal protection systems*, such as the Gore Neuro Protection System (Gore) and the MO.MA System (Invatec), provide protection against cerebral embolism even before crossing the stenosis. This is particularly important in the case of stenoses in which fresh thrombi have been identified. Here, the risk of embolization with a distally placed system may be raised. The use of a proximal occlusion device allows the operator to choose any wire of choice to negotiate difficult stenoses. These systems consist of a long main sheath with a balloon on its distal end that is inflated in the common carotid artery to occlude forward carotid flow. A second balloon, which is inflated in the external carotid artery, prevents retrograde external flow, thus establishing complete arrest of antegrade flow into the internal carotid artery. This principle takes advantage of the cerebral collateral system of the circle of Willis. Following balloon occlusion of the external and common carotid arteries, collateral flow via the circle of Willis produces what is known as back pressure. This prevents antegrade flow into the internal carotid artery. After stent implantation and before deflation of the occlusion balloon, blood in the internal carotid artery, which might contain released particles, is aspirated and removed. One disadvantage of proximal protection systems is that a small percentage of patients are unable to tolerate balloon occlusion due to incomplete intracranial collateralization.

8.10 Stent Implantation

Usually self-expandable stents are implanted in carotid stenting. Balloon-expandable stents are recommended in ostial stenoses of the common carotid artery, stenoses located in the distal internal carotid artery, and sometimes in severely calcified stenoses. The disadvantages of balloon-expandable stents, however, are the repeated balloon dilations that are needed to implant the stent adequately and stent compression that can occur during the long-term follow-up in areas vulnerable to external manipulation.

In vessels which carry the risk to bend or to be manipulated, self-expandable nitinol stents are generally the best choice. They are designed to adapt to the shape of the vessel and therefore have only little tendency to straighten it. Stent-induced straightening of the vessel can give rise to a new stenosis distal to the stent due to kinking or folding of the vessel. Stents with a strong radial force are recommended for treatment of severely calcified stenoses. Closed-cell carotid stents typically have stronger radial force. Their cell structure may also provide better plaque coverage, which may theoretically be advantageous in stenoses with high embolic risk. The clinical value of closed-cell designs versus open-cell designs is currently still unclear. Likewise, the importance of the stent cell size is subject to further research. In case of soft or large plaques and visible thrombus, the combination of a proximal occlusion device and a stent system with small struts is probably favorable to manipulate the plaque as little as possible during the intervention.

The authors recommend a stent diameter 1–2 mm larger than the largest vascular diameter to be stented. Carotid stents with a diameter of 6–8 mm are usually used if the stent is being implanted exclusively in the internal carotid artery, or with a diameter of 8–10 mm if the stent is to cross the bifurcation. Stenting across the external carotid artery does not pose a problem and priority should be given to the stent covering the entire stenosis, which in most cases will mean crossing the bifurcation to cover the distal common carotid artery.

8.11 Post-Dilation

Post-dilation is usually carried out using a balloon with a diameter of 4–5 mm, but not larger than the diameter of the internal carotid artery. A balloon with an unnecessarily large diameter might force particles through the stent cells and cause distal embolization. To prevent dissections, postdilatation should be carried out at nominal pressure, and within the stent borders. A residual stenosis of <30% is acceptable, since an adequate blood flow is established and the potentially embogligenic atherosclerotic deposits are compressed sufficiently to induce neo-intimal formation and eliminate the embolic potential of the lesion. The stent expands further during the following few hours. If contrast-enhancing ulcerations occur outside the stent edges, they do not need to be obliterated and can be left without any untoward

effects. Post-dilation of the stent segment in the common carotid artery is not necessary. If significant stenosis or occlusion of the external carotid artery develops subsequently to postdilation, it does not require treatment.

Following post-dilation of the stent, angiography of the carotid arteries and intracranial vessels is carried out. Imaging of the intracerebral vessels should always include the venous phase. This allows comparison of conditions before and after stent implantation. For assessment of the intracerebral vessels and in preparation for a possible intracranial emergency intervention in case of cerebral embolism, angiography should be carried out with a lateral and anteroposterior 30° cranial projection.

9 Post-Procedure Management and Follow-up

Following the intervention, blood pressure has to be checked closely for at least 6 h. It should be as low as possible. The post-interventional medication consists of lifelong aspirin treatment (100 mg/day) and clopidogrel (75 mg/day) during the first month after stent placement. Color duplex ultrasound scan should be performed 1 month after the intervention to verify the acute result of the procedure, and thereafter at an interval of 6 months as routine follow-up examination.

10 Acute Complications and Management of Treatment

10.1 Ischemic Events (TIA, Stroke)

Ischemic events are the main complication of carotid stent implantations. Possible predictors of complications and potential sources for thrombotic embolization are:

– Insufficient pre-treatment: aspirin and clopidogrel should be started a week before to avoid fresh thrombus.

- Heavily calcified or ulcerated lesions: In case of large plaque volume or a visible thrombus use a proximal occlusion device and a stent with small struts and a small cell area to manipulate the stenosis as little as possible and to secure the plaque as good as possible.
- Malposition of distal filter devices: Use angiography to verify stabile filter position.
- Malposition of proximal occlusion devices: Contrast dye disappears as soon as the occlusion balloon is not occlusive anymore, concurrently the measured blood pressure rises. Flow arrest has to be re-established as soon as possible because malposition immediately results in zero protection.

Cerebral ischemia can present with a large variety of symptoms. Depending on the occluded vessel, the embolization may bring a sudden change of patient's neurological status. Yet sometimes, the ischemia causes only a subtle change of the neurological status or may even only cause a change in vital signs.

In case of sudden symptom onset due to cerebral ischemia, it is most important to act systematically. The operator should finish the current maneuver, then aspirate and re-establish blood flow (if proximal occlusion is used). After a neurological evaluation of the patient's situation, fluids should be administered and a cerebral angiography has to be performed to compare actual findings with baseline. Depending on the type of occluded artery and the inventory of the cathlab, the operator has different treatment options to restore cerebral blood flow. After advancing a microcatheter to the clot interface, lytic agents and glycoprotein IIb/IIIa inhibitors can be administered distally, proximally and into the clot. If available, intracranial retrieval devices (e.g., Phenox Clot Retriever, Concentric Clot Retriever, Merci Retrieval Device, Penumbra) can be used to try to disrupt and retrieve the thrombus or debris. If clot retrieval fails, placement of an intracranial stent (e.g., Wingspan, Solitaire, Enterprise, Neuroform) may be an option to re-establish cerebral perfusion.

In case of very small vessel occlusions, or asymptomatic post-procedural filling defects that may evolve into significant stroke, the administration of glycoprotein IIb/IIIa inhibitors leads to an increased platelet blockade to prevent further clot formation. However, they also increase the risk of bleeding.

10.2 Stent Thrombosis

Despite combined antiplatelet therapy with aspirin and clopidogrel, as well as peri-interventional use of heparin, in some cases a sudden formation of thrombotic material can be seen after stent-implantation. In these cases the ACT has to be raised again by administration of heparin. The administration of glycoprotein IIb/IIIa inhibitors is effective to prevent further clot-formation which may cause distal embolization and stroke. The application of a bolus of thrombolytics in to the clot, followed by continuous intra-arterial infusion may resolve the thrombus. An additional stent implantation may be indicated to fix the formed clot at its position.

10.3 Spasm

In case of vasospasm during carotid interventions, in most of the cases it is attributed to mechanical irritation of the vessel wall caused by movement of distal filter devices. The most crucial point in preventing spasm is to avoid any unnecessary movement of the filter. If spasm occurs during the intervention, it will be of highest importance that the interventionalist stays calm. In most of the cases vasospasm is self-limitating and does not need any further treatment. In symptomatic spasms, intra-arterial nitroglycerin can be given.

10.4 Bradycardia and Hypotension

Bradycardia and hypotension may occur due to compression of baroreceptors in the carotid bulb during post-dilation. These symptoms may occur either during balloon inflation or after the intervention, or even may reoccur and can last up to 48 h. The administration of Atropine 3–5 min before inflating the balloon is most effective to prevent circulatory disorders. In case of bradycardia and hypotension despite of the application, 0.5–1 mg of Atropine can be administered repeatedly. In rare cases, catecholamines are necessary, occasionally for up to 2 days.

11 Inventory for Carotid Artery Stenting

Basic Inventory
- Sheaths
- Guiding catheters
- Guidewires
- Diagnostic catheters (e.g., Berenstein, Judkins Right, Head Hunter, IMS, JB-1, Simmons, Vittek)
- Embolic protection devices (Table 9.1)
- Carotid artery stent systems (Table 9.2)
- Dilation balloons (different diameters and different lengths)
- Neuro wires (e.g., Taper, Dasher)
- Micro catheters (e.g., Fast Tracker, Prowler)

Extended Inventory
- Covered stents (e.g., Hemobahn)
- Intracranial stents (e.g., Neuroform, Wingspan, Enterprise, Solitaire)
- Clot retrieval devices (e.g., Concentric, Merci, Catch, Phenox)
- Aspiration devices (e.g., Penumbra)
- Coils

Table 9.1 Embolic protection devices

Embolic protection devices			
Type	Product	FDA approval	CE mark
Distal occlusion balloon	PercuSurge (Medtronic)	X	X
	TwinOne (Minvasys)		X
Distal filter	Angioguard (Cordis)	X	X
	Accunet (Guidant)	X	X
	Spider (ev3)	X	X
	FilterWire (Boston)	X	X
	Emboshield (Abbott)	X	X
	Fibernet (Medtronic Invatec)	X	X
Proximal occluion devices	Gore Flow Reversal Device (GORE)	X	X
	MO.MA (Medtronic Invatec)	X	X

Table 9.2 Carotid artery stent systems

Carotid artery stents			
Stent design	Product	FDA approval	CE mark
Closed cell—woven	Carotid Wallstent (Boston)	X	X
Closed cell	Xact (Abbott)	X	X
	Adapt (Boston)		X
Open cell	Acculink (Guidant/Abbott)	X	X
	Precise (Cordis)	X	X
	Protégé (ev3)	X	X
	ViVEXX (Bard)		X
	Zilver (Cook)		X
	Sinus (Optimed)		X
Hybrid	Cristallo Ideale (Medtronic Invatec)		X

12 Outcomes

In 2003, Wholey et al. published a summary of the results of 12,392 carotid stent implantations in a total of 11,243 patients from 53 centers worldwide. The complications during the first 30 days included: TIA (3.1%), minor stroke (2.1%), major stroke (1.2%), and death (0.6%) [3]. In the same year, Cremonesi et al. published a series of 442 consecutive patients treated with carotid stent implantation with embolic protection. Stroke or death occurred within the first month after the procedure in 1.1% of these patients [4]. The German Association for Angiology and Radiology has developed a prospective registry for carotid stent implantations. The results for the first 48 months, from a total of 38 participating centers, were published in 2004. Carotid stent implantation was carried out in 3,267 patients. The procedure was successful in 98% of the interventions. The periinterventional mortality was 0.6%, the major stroke rate was 1.2%, and the minor stroke rate was 1.3%. In 2005, Bosiers et al. published the ELOCAS Registry, compiled retrospectively and prospectively from the results of four high-volume centers. A total of 2,172 consecutive patients were treated and 99.7% of the procedures were technically successful. The stroke/death rate was 4.1% after 1 year, 10.1% after 3 years, and 15.5% after 5 years [5]. The CaRESS study

was a nonrandomized multicenter study including 143 patients treated with carotid stent implantation and 254 patients who underwent carotid endarterectomy. No significant differences were observed with regard to the stroke/death rates either after 30 days (2.1% stent, 3.6% surgery) or after 1 year (10% stent, 13.6% surgery) (CaRESS 2005). The ARCHeR study was published by Gray et al. in 2006 and consisted of three sequential multicenter studies. In ARCHeR 1, only the use of the Acculink carotid stent was evaluated. In the two subsequent studies (ARCHeR 2 and 3), adjuvant use of the Accunet embolic protection system was also tested. A total of 581 patients with high surgical risk from 48 centers were included between 2000 and 2003. The combined stroke/death/myocardial infarction rate was 8.3% after 30 days. The ipsilateral stroke death rate after the first month and up to 1 year was 1.8%. The repeat stenosis rate was 2.2% within the first year [6]. The CAPTURE Registry/Carotid Acculink/Accunet Post-Approval Trial to Uncover Unanticipated or Rare Events) was published in 2007. A total of 3,500 patients with high surgical risk and a stenosis grade>50% (symptomatic) or>80% (asymptomatic) were included. The major stroke/death rate after 30 days was 2.9%. In 2008, Stabile et al. presented the results of the European Registry of Carotid Artery Stenting (ERCAS)—a retrospective analysis of 1,611 consecutive neuro-protected carotid interventions in eight European high-volume centers. All procedures were technically successful and the combined stroke and death rate at 30 days was 1.36%. The results of the EPIC trial (Evaluating the Use of the FiberNet Embolic Protection System in Carotid Artery Stenting) were presented by Myla et al. in 2008. Two hundred thirty-seven patients at high risk for carotid surgery were treated in 26 centers using the FiberNet device for embolic protection. The combined 30-day stroke/death/myocardial infarction rate was 3.0%. In the same year, Hopkins et al. presented the results of the EMPiRE trial (Embolic Protection with Flow Reversal using the GORE Flow Reversal System), which enrolled 245 patients at 28 US sites. The 30-day any stroke or death rate was 2.9% and the primary endpoint including TIA and myocardial infarction was reached in 4.5% of all cases. In 2009, the results of the ARMOUR trial were announced by Hopkins et al. This trial evaluated proximal occlusion with the MO.MA device during carotid stenting in patients at high risk for surgery. The primary endpoint of the study, the rate of major adverse cardiac and cerebrovascular events at 30 days, was 2.7%.

In 2010, *CREST (The Carotid Revascularization Endarterectomy* vs. *Stenting Trial)*, the largest randomized clinical trial comparing the efficacy of carotid stenting to carotid endarterectomy, could show equivalence between interventional and surgical treatment of carotid stenoses. This trial enrolled 2,522 patients across North America and is the most important data set in a long series of trials that have shown over the last 15 years that carotid stenting has gotten better over time. In CREST, the primary endpoint was composite occurrence of stroke, myocardial infarction, or death from any cause during the 30-day periprocedural period or any postprocedural ipsilateral stroke within 4 years of randomization. The combined primary endpoint demonstrated equivalence between CAS and CEA (7.2% vs. 6.8%; $p=0.51$ for stroke, death, MI, or long-term [4 years] ipsilateral stroke event). The rates of major stroke for CAS and CEA were approximately equal (0.9% vs 0.6%; $p=0.52$), the rate of minor stroke for CAS exceeded that for CEA (total 4.1% vs. total 2.3%; $p=0.01$). CAS was superior to CEA with respect to the incidence of periprocedural MI (1.1% vs 2.3%; $p=0.03$). In addition, cranial nerve palsies were less frequent during the periprocedural period with CAS (0.3% vs 4.7% with CEA; hazard ratio, 0.07; 95% confidence interval, 0.02–0.18). There was no differential treatment effect with regard to the primary endpoint according to symptomatic status.

13 Differential Diagnosis of Atherosclerotic Carotid Disease

Although the following diseases are not frequent, it is important to know which etiological differential diagnosis of atherosclerotic carotid disease exist:

– Fibromuscular dysplasia: The most common non-traumatic, non-atherosclerotic, non-inflammatory lesion of the internal carotid artery is estimated to be 0.02%. The preferred treatment for refractory cases is percutaneous balloon angioplasty. If the arterial wall is damaged or weakened, then stenting of the affected artery may be chosen. Besides high blood pressure control, anti-platelet drugs and blood thinner drugs may be used. Bypass surgery is a considered treatment.

- Vasulitis (e.g., Takayasu's disease): The mainstay of therapy are glucocorticoids. Angioplasty or bypass grafts may be necessary once irreversible arterial stenosis has occurred.
- Thoracic outlet syndrome: Should be treated initially with physical therapy, but may require interventional stent angioplasty or even surgical decompression, including first rib excision and scalenectomy.
- Traumatic injuries: Interventional or surgical treatment is indicated in patients with traumatic carotid artery dissection and hemodynamic significant hemispheric hypoperfusion, or in whom anticoagulant therapy was either contraindicated or failed clinically.
- Vasospastic disorders: Since vasospasms can contribute to the severity of ischemia, percutaneous interventions and stent angioplasty may be indicated.

14 Carotid Artery Aneurysm

Aneurysms of the extracranial carotid arteries are rare. They may cause cerebral embolism. The location of the aneurysm often makes surgical correction difficult and leads to perioperative lower cranial nerve injuries and neurological deficits. Endovascular interventions using uncovered stents are effective in treating dissecting aneurysm with intimal flap or small defects in the arterial wall as the stent mesh impedes flow into the aneurysmal sac, inducing thrombosis, or relocating the intimal flap to occlude the aneurysm. However, flow into wide-necked aneurysms is difficult to exclude with an uncovered stent, so that coil embolization of the sac through the stent is often required. Wide-necked saccular aneurysms or pseudoaneurysms can be more appropriately treated with a covered stent (e.g., Hemobahn), leading to immediate and definitive reconstruction of the arterial wall.

Covered stents have been used in the subclavian and common carotid location for pseudoaneuryms related to trauma with excellent long-term results. A bare stent does not pose a good alternative, as it cannot completely eliminate the risk of embolization since the aneurysm would still communicate with the true lumen through the stent struts. Potential limitations of a covered stent include larger delivery systems compared to bare stents and the need for technical expertise for appropriate delivery.

15 Carotid Artery Dissection

The incidence of spontaneous carotid dissection has been reported to be up to 2.6 per 100,000 and is responsible for up to 20% of strokes in the younger population. Advantages of an endovascular therapy of carotid dissections are that it enables the identification of the true and false lumens by superselective catheterization and angiography, and further allows the recanalization of completely occluded vessels by use of microcatheter techniques, provided the thrombus burden is not prohibitive. Additionally, simultaneous treatment of any coexistent pseudoaneurysmal dilatation by the coil-through-stent technique is possible.

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