The Vascular System

9

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9.1 Stenosis

9.1.1 Intracranial Stenosis

Intracranial atherosclerosis is a major cause of stroke, especially in blacks, Asians, and Hispanics. In the Warfarin-Aspirin Symptomatic Intracranial Disease Trial, the 1-year ischemic stroke rate in the territory of a symptomatic intracranial stenosis (50–99 %) was 11 % in patients treated with warfarin and 12 % in patients treated with aspirin (WASID Trial Investigators 2005). Data on asymptomatic intracranial arterial stenosis are limited. Ischemic stroke rates for asymptomatic stenosis have only been reported for the middle cerebral artery, where the rate is 0–1.4 % per year (Kern et al. 2005). Intracranial atherosclerosis is thought to account for approx. 8–10 % of all ischemic strokes in whites (Arenillas et al. 2004). Nevertheless, its true importance in whites tends to be underestimated. A French autopsy study of 339 patients who died of ischemic or hemorrhagic stroke revealed a strikingly high prevalence of intracranial plaque and intracranial stenosis (Mazighi et al. 2008). It is probably safe to assume that intracranial stenosis represents merely the most advanced stage of intracranial atherosclerosis and that nonstenotic disease is far more common (Arenillas 2011).

Three-dimensional time-of-flight magnetic resonance angiography (TOF-MRA) allows good morphologic assessment of stenotic lesions in the proximal intracranial arteries (80–100 % sensitivity, 80–99 % specificity). One must be aware, however, that loss of signal due to turbulent flow or loss of laminar flow in the carotid bulb can simulate narrowing or lead to overestimation of stenosis.

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Table 9.1 Stenosis of extracranial cerebral arteries

Prevalence	Approx. 0.5 % below age 60, approx. 10 % over age 80; 28 % in patients with PAOD
Location	Extracranial carotid stenosis
Signs and symptoms	Approx. 2 % stroke rate per year when asymptomatic and detected incidentally

Clinical Management
All subjects with asymptomatic stenosis of the carotid artery, the vertebral arteries, or the intracranial circulation should see a neurologist to determine whether other risk factors for cerebrovascular events exist and whether drug therapy or interventional/surgical management is necessary.

9.1.2 Stenosis of Extracranial Cerebral Arteries

The prevalence of extracranial carotid stenosis is approx. 0.5 % in individuals under 60 and increases to approx. 10 % in those over 80. In patients with peripheral arterial occlusive disease, the prevalence may be as high as 28 % (Table 9.1). Risk factors for carotid stenosis include male sex, hypertension, hypercholesterolemia, and smoking. The risk of stroke in subjects with asymptomatic carotid stenosis is low (approx. 2 % per year) but correlates with the degree of stenosis and increases with stenosis progression (Hennerici et al. 1987). Patients with symptomatic internal carotid artery (ICA) stenosis have a markedly higher risk (Anonymous 1998).

Clinical Management
All subjects with asymptomatic stenosis of the carotid artery, the vertebral arteries, or the intracranial circulation should see a neurologist to determine whether other risk factors for cerebrovascular events exist and whether drug therapy or interventional/surgical management is necessary.

9.1.3 Stenosis of Lower Extremity Arteries

Peripheral arterial occlusive disease (PAOD) is an umbrella term for all forms of impaired arterial circulation caused by stenotic or obstructive vascular processes including lesions of the aorta and arteries supplying the extremities (Spengel et al. 2001). Chronic arterial occlusive disease, unlike acute disease, is characterized by slow development of single or multiple stenotic lesions, which in over 90 % of cases affect the arteries of the pelvic region and legs. Steno-occlusive disease of the leg arteries leads to intermittent claudication due to muscle ischemia and increasingly limits the pain-free walking distance. About 20 % of patients with poor circulation of the legs have PAOD (Alexander 1992).

Table 9.2 Stenosis of lower extremity arteries

Prevalence	Age related 2.5 % < 60 years 18.8 % > 70 years
Location	1. Aortoiliac PAOD 2. Femoropopliteal PAOD 3. Calf and foot (anterior tibial artery > posterior tibial artery > fibular artery)
Signs and symptoms	Asymptomatic incidental finding, reduced walking distance, rest pain, necrosis, gangrene

Three types of occlusive disease of the lower extremities can be distinguished by level of involvement: (1) aortoiliac disease with steno-occlusive lesions in the aorta and/or iliac arteries, (2) femoropopliteal PAOD, and (3) PAOD of more distal arteries (calf and foot). Only about one third of PAOD patients have ischemic symptoms (McDermott et al. 2002). Many patients limit their activities to reduce PAOD-related symptoms. Initially, they report feeling well but when questioned admit that they can only walk a short distance (Table 9.2).

The prevalence of stenotic and occlusive lesions of the distal leg varies with age and sex and also depends on the presence of risk factors (Kannel et al. 1970). The leading risk factors are smoking, diabetes mellitus, arterial hypertension, hyperlipoproteinemia, hypercholesterolemia, and hyperfibrinogenemia. The prevalence is 2.5 % before age 60 and as high as 18.8 % in those over 70 (Kannel 1996). Half of patients with lower leg occlusion have diabetes mellitus (Albers et al. 2005; Hirsch et al. 2005; Schneider et al. 1993).

PAOD patients have an increased risk of cerebrovascular and cardiovascular events (Criqui et al. 1997; Ness and Aronow 1999); the risk is the same regardless of whether they are symptomatic or asymptomatic (Hooi et al. 1998, 2001). Hence, the traditional concept of asymptomatic PAOD seems to downplay the risk. Patients with chronic PAOD have a mortality rate of over 20 % in the first year after diagnosis (Hirsch et al. 2005). Atherosclerotic occlusion most commonly affects the anterior tibial artery, followed by the posterior tibial artery and the fibular artery. Occlusion of a single calf artery with patency of the other branches is clinically silent. MR angiography with administration of gadolinium-based contrast medium is comparable to digital subtraction angiography (DSA) in terms of specificity and sensitivity for anatomic localization and grading of stenosis severity (Fig. 9.1).

Evaluation of vascular disease of the upper extremities was not part of the whole-body MRI protocol used.

Clinical Management

Multiple occlusions of lower leg arteries can be treated medically, interventionally, or surgically. The best treatment should be determined by interdisciplinary consensus on the basis of clinical parameters.

When no symptoms are present or when the subject feels well, management focuses on identifying and reducing risk factors such as smoking, diabetes

mellitus, hypertension, and hyperlipoproteinemia. These risk factors should be screened for in a thorough history and brought to the subject's attention.

An inexpensive and simple test for evaluating arterial insufficiency is to determine the ratio of ankle systolic pressure to brachial systolic pressure, the ankle-brachial index (ABI). An ABI of 1.00–1.29 is considered normal, while values between 0.5 and 1.0 indicate moderate PAOD. Values below 0.4 suggest an increased risk of developing rest pain and ischemic necrosis (Hirsch et al. 2005). Another measure of disease severity is the distance an individual can walk without pain.

Treatment of Fontaine stage 2 disease is primarily physical and medical and aims at reducing or eliminating risk factors.

Chronic stage 3 and 4 occlusions require interventional or surgical revascularization but are unlikely to be encountered as incidental findings in the screening situation or in epidemiologic studies of healthy subjects.

Fig. 9.1 Stenosis (*arrow*) of the left superficial femoral artery on MIP image

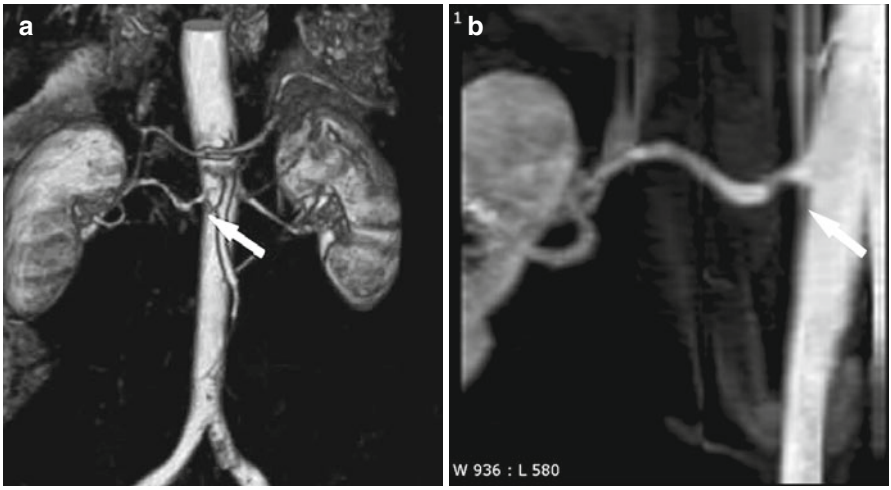

Fig. 9.2 Right-sided RAS (*arrow*) on VRT (**a**) and MIP (**b**) images

9.1.4 Renal Artery Stenosis

A prevalence of renal artery stenosis (RAS) of 6.8 % has been reported by Hansen et al. (2002), but in high-risk groups, it may be as high as 50–70 % (Schwartz and White 1964; Harding et al. 1992; Weber-Mzell et al. 2002; Jean et al. 1994). Roughly two thirds of renal artery stenoses are due to atherosclerotic plaques, and one third of these are bilateral. Fibromuscular dysplasia accounts for the other third. Uni- or bilateral RAS underlies the development of arterial hypertension in 1–5 % of cases. Fibromuscular dysplasia is the predominant cause of RAS in young patients, usually women aged 20–40, and tends to affect the distal renal arteries. New-onset hypertension or progression of known hypertension in older individuals is mostly attributable to atherosclerotic stenosis, which typically involves the proximal renal arteries (Fig. 9.2).

Clinically, RAS may be asymptomatic or may present with renovascular hypertension or renal dysfunction. Mixed forms are also possible (Balk et al. 2006; Safian and Textor 2001). RAS greater than 70 % is considered hemodynamically relevant.

Clinical Management

In the routine diagnostic setting, mild hypocalcemia, which indicates activation of the renin-angiotensin-aldosterone system, can alert the physician to the diagnosis of renal artery stenosis. Several tests are available to assess renin release. The sensitivity and specificity of plasma renin activity measurement can be improved by furosemide stimulation. When renin in renal vein blood is determined separately for each side, a ratio of renin levels ≥1.5 (stenotic versus nonstenotic side) is considered abnormal. An abnormal ratio and an absolute increase in plasma renin activity reliably predict RAS as the underlying cause of hypertension – a condition that is potentially curable by surgery or angioplasty.

9.1.5 Mesenteric Artery Stenosis

An acute ischemic event is not likely to be encountered in the classic screening situation. It is more likely to incidentally detect chronic mesenteric stenosis promoting or maintaining ischemia of upper abdominal organs and of the small and large intestines (abdominal angina).

The incidence of chronic occlusive disease of a mesenteric artery is approx. 5 % in patients presenting with abdominal pain of unknown cause. Because chronic occlusion progresses slowly and collaterals provide compensatory blood supply, the imaging appearance or autopsy findings do not tend to correlate with the clinical picture. Mesenteric stenosis is present in 80 % of individuals over 60 but rarely becomes clinically apparent. Symptoms do not develop unless there is appreciable ischemia in a target organ, which in turn requires a high-grade stenosis of at least 70 % (Luther 2001).

Clinical Management

There are four clinical stages of chronic mesenteric ischemia:

1. Asymptomatic stage
2. Intermittent abdominal complaints (abdominal angina, recurrent organ insufficiency)
3. Abdominal pain at rest
4. Ischemic organ lesion (infarct)

A typical manifestation is postprandial abdominal pain persisting for 3–4 h after eating. Chronic visceral artery occlusion is characterized by the classic triad of abdominal pain, weight loss, and paraumbilical murmur.

An asymptomatic individual (stage 1) needs no treatment. Stage 2 and 3 disease is an absolute indication for treatment, and stage 4 is an emergency.

9.2 Aneurysm

9.2.1 Intracranial Aneurysm

Time-of-flight MR angiography (TOF-MRA) of the intracranial arteries is a suitable screening tool for intracranial aneurysms (Raaymakers et al. 1999) (Fig. 9.3). The wide range of sensitivities (74–100 %) reported for aneurysm detection by MRA is in part attributable to the investigation of different patient populations (ruptured versus nonruptured aneurysm) and technical advances. The TOF-MRA technique used here offers better resolution than contrast-enhanced MRA (CE-MRA). Unlike CE-MRA, TOF-MRA is not limited by a narrow imaging window (which requires careful bolus timing) and thus avoids the problem of venous overlay in aneurysm imaging (Ozsarlak et al. 2004).

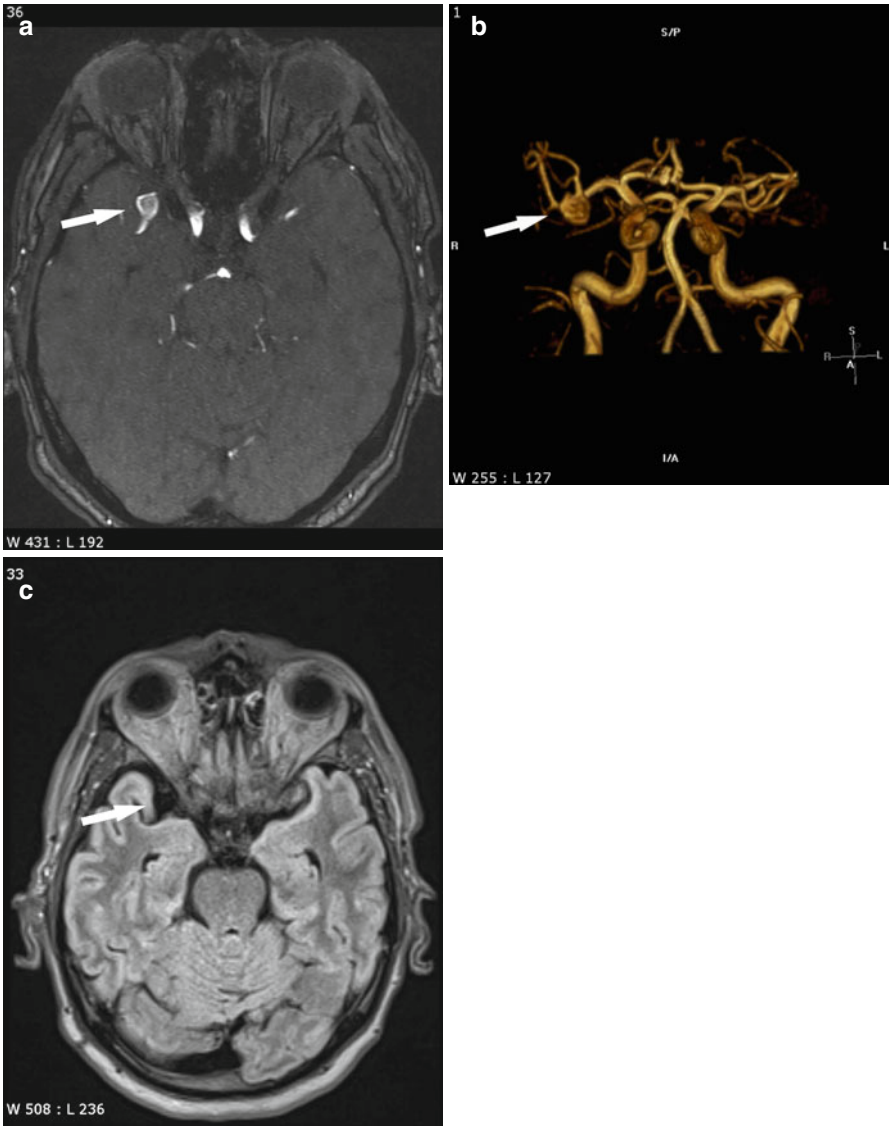

Fig. 9.3 Right-sided middle cerebral artery bifurcation aneurysm (*arrow*) on TOF (**a**) and VRT (**b**) images. The FLAIR image (**c**) shows normal flow void

Intracranial aneurysm is the most common cause of acute subarachnoid hemorrhage (SAH) and/or intracerebral bleeding, accounting for about 75 % of cases. Aneurysmal bleeding is estimated to occur in approx. 5–10 cases/100,000 population per year (Rinkel et al. 1998). The estimated prevalence of cerebral aneurysm is approx. 2.3 % in the adult population (Rinkel 2008). Most cerebral aneurysms are

Table 9.3 Intracranial aneurysm

Frequency	2.3 % An estimated 1.5–2 mill. Germans harbor aneurysms
Location	Carotid territory (85–95 %): anterior communicating and anterior cerebral arteries (approx. 40 %), posterior communicating and internal carotid arteries (approx. 30 %), middle cerebral artery (approx. 20 %) Vertebrobasilar territory (approx. 5–15 %): basilar artery (approx. 10 %), vertebral artery (approx. 5 %) Multiple aneurysms (approx. 20–30 %)
Signs and symptoms	Most common cause of subarachnoid hemorrhage (SAH) and/or intracerebral hemorrhage (75 %) Frequency of aneurysmal hemorrhage: 5–10/100,000 population/year

small and are located in the anterior circulation. In Germany, it is assumed that approx. 1.5–2 million individuals have aneurysms, among them 30 % with multiple aneurysms (Table 9.3).

Clinical Management
When imaging detects an incidental intracranial aneurysm, the possible complications of a preventive neurosurgical or endovascular intervention must be weighed against the risks of death or disability from future aneurysm rupture. Patient age is the most important factor in decision-making: the benefits of preventive treatment are high in young individuals, while the risks related to treatment are relatively low. With increasing age and decreasing life expectancy, however, the benefits of prophylactic intervention decrease, and the risk of complications increases (Rinkel 2008). Other factors to be considered in the decision include the presence of additional aneurysms and comorbidity.

9.2.2 Aortic Aneurysm

Contrast-enhanced whole-body MR angiography allows excellent evaluation, especially of the great arteries. An aneurysm is defined as a circumscribed fusiform or saccular dilation with a diameter of at least 3 cm in the abdominal aorta and at least 2 cm in the pelvic aorta (which corresponds to twice the normal width of these aortic segments). Accounting for over 60 % of all aneurysms, infrarenal aneurysm is the most common type involving the great vessels. The male-to-female ratio is 6:1. The prevalence of abdominal aortic aneurysm (AAA) is about 1 % after age 50 and can be as high as 10 % in hypertensive men over 65. The annual rupture rate in individuals harboring an aneurysm is 2.2 % (Brown and Powell 1999). The main risk factors are male sex, smoking, and age over 65 (Table 9.4).

Isolated pelvic artery aneurysms are rare, constituting only 1.3 % of all aneurysms. Over 50 % of pelvic artery aneurysms occur in individuals with a concomitant infrarenal aneurysm. Thoracic aortic aneurysms are less common than infrarenal aortic aneurysms. Their incidence is 6 per 100,000 population per year but is on the rise (Olsson et al. 2006).

Table 9.4 Aortic aneurysm

Frequency	Infrarenal aneurysms account for 60 % of all aneurysms
Prevalence	Approx. 1 % over age 50 10 % over age 65
Sex predilection	M:F ratio of 6:1

Table 9.5 Aneurysm of lower extremity arteries

Frequency	3–7 % of all patients with AAA
Signs and symptoms	Thromboembolism and thrombosis

Clinical Management
The aim of treating an asymptomatic aneurysm is to prevent rupture or rare embolic events. The UK Small Aneurysm Trial has revealed that, for abdominal aortic aneurysms (AAA) <5.5 cm, early open surgery offers no survival advantage over regular sonographic follow-up (The UK Small Aneurysm Trial Participants 1998). Hence, current recommendations in Europe suggest elective treatment for AAA ≥5.5 cm in transverse diameter (Hirsch et al. 2005). Definitive specific data on the best time for endovascular intervention are still lacking, which is why the recommendations are based on the existing indications for open surgery (Diehm 2009). Patients with untreated AAA are recommended to undergo regular sonographic monitoring. Morphologic risk factors for AAA rupture include diameter, shape, and growth rate. The rupture risk is approx. 3 % for aneurysms with diameters of 4.0–4.9 cm, 10 % for AAA diameters of 5.0–5.9 cm, and more than 60 % for diameters >7 cm. While fusiform aneurysms rarely rupture, the risk is much higher for saccular aneurysms and penetrating aortic ulcers.

9.2.3 Aneurysm of Lower Extremity Arteries

Concomitant aneurysms of femoral or popliteal arteries are present in 3–7 % of patients with AAA. Popliteal aneurysms, with an estimated incidence of 0.1–2.8 %, account for approx. 70 % of all lower extremity aneurysms. Unlike aortic aneurysms, which tend to grow and rupture, the natural history of popliteal aneurysms is associated with the risk of thromboembolism and thrombosis (Table 9.5).

Clinical Management
A popliteal aneurysm with a size of 2 cm or more should be operated on due to the risk of thromboembolic complications with possible limb loss. Subjects with asymptomatic dilation of the popliteal artery to twice its normal diameter are recommended to undergo an annual ultrasound examination. Individuals harboring a femoral or popliteal artery aneurysm benefit from antiplatelet therapy.

Table 9.6 Aneurysm of visceral and renal arteries

Frequency	60 % of visceral aneurysms occur in the splenic artery, 20 % in the hepatic artery; renal artery aneurysms are rare (incidence of 0.09 %)
Sex predilection	Most common in multiparous women
Signs and symptoms	Chronic development with upper abdominal pain, acute risk of rupture

9.2.4 Aneurysm of Visceral and Renal Arteries

Visceral and renal artery aneurysms are rare. They are most commonly found in multiparous women (Trastek et al. 1982; Cohen and Shamash 1987). Approx. 60 % of all visceral aneurysms occur in the splenic arteries, followed by aneurysms of the hepatic artery (20 %) (Table 9.6). Partial thrombosis is not uncommon and may obscure the aneurysm on MR angiograms; a thrombotic aneurysm may be easier to detect as an inhomogeneous mass related to an artery on T1-weighted and T2-weighted images (typically high T1 signal intensity and low T2 signal intensity).

Clinical Management
Approx. 20 % of patients with a splenic artery aneurysm either develop chronic upper abdominal pain or present with acute rupture. Open surgery or endovascular treatment of visceral aneurysms is probably indicated in women beyond reproductive age and in men when the diameter exceeds 2 cm. With definitive data still lacking, decisions on the treatment of visceral and renal artery aneurysms must be made on a case-by-case basis taking an interdisciplinary approach.

9.3 Cervical Artery Dissection

The incidence of carotid artery dissection is low with an estimated incidence of 2.6 per 100,000 inhabitants per year in a population-based study conducted in North America (Lee et al. 2006). A dissection may, however, go unnoticed when clinical symptoms are mild or absent, meaning that the true incidence is probably higher. Vertebral artery dissection is less common compared with other localizations (1/100,000/year). The typical imaging findings include long, tapering luminal narrowing, vascular occlusion, and pseudoaneurysm. Wall hematoma may be apparent on MR angiography due to high signal intensity resulting from T1 shortening. The typical appearance combines dilation of the dissected vessel with sickle-shaped wall hematoma and reduced residual lumen. The intimal flap is usually visualized when there is flow in both the true and false lumens.

Clinical Management

Asymptomatic carotid dissection is a very rare event, accounting for only approx. 6 % of all cases of carotid dissection (Lee et al. 2006). Hence, there are no definitive guidelines for the management of such lesions. In line with the recommendations for symptomatic cervical dissection, a limited course of antithrombotic therapy has been proposed to prevent ischemic events.

References

Albers M, Romiti M, Brochado-Neto FC, Braganca Perira CA (2005) Meta-analysis of alternate autologous vein bypass to infrapopliteal arteries. J Vasc Surg 42:449–455

Alexander K (1992) Krankheiten der Arterien. In: Siegenthaler W, Kaufmann W, Hornbostel H, Waller HD (eds) Lehrbuch der inneren Medizin, 3rd edn. Thieme, Stuttgard/New York

Anonymous (1998) Randomised trial of endarterectomy for recently symptomatic carotid stenosis: final results of the MRC European Carotid Surgery Trial (ECST). Lancet 351:1379–1387

Arenillas JF (2011) Intracranial atherosclerosis: current concepts. Stroke 42(1 Suppl):S20–S23

Arenillas JF, Molina CA, Chacón P, Rovira A, Montaner J, Coscojuela P, Sanchez E, Quintana M, Alvarez-Sabín J (2004) High lipoprotein (a), diabetes, and the extent of symptomatic intracranial atherosclerosis. Neurology 63:27–32

Balk E, Raman G, Chung M, Ip S, Tatsioni A, Alonso A, Chew P, Gilbert SJ, Lau J (2006) Effectiveness of management strategies for renal artery stenosis: a systematic review. Ann Intern Med 145:901–912

Brown LC, Powell JT (1999) Risk factors for aneurysm rupture in patients kept under ultrasound surveillance. UK Small Aneurysm Trial Participants. Ann Surg 230:289–296, discussion 296–297

Cohen JR, Shamash FS (1987) Ruptured renal artery aneurysms during pregnancy. J Vasc Surg 6:51–59

Criqui MH, Denenberg JO, Langer RD et al (1997) The epidemiology of peripheral arterial disease: importance of identifying the population at risk. Vasc Med 2:221–226

Diehm N (2009) Abdominal aortic aneurysm. Internist (Berl) 50:972–978

Hansen KJ, Edwards MS, Craven TE et al (2002) Prevalence of renovascular disease in the elderly: a population-based study. J Vasc Surg 36:443–451

Harding MB, Smith LR, Himmelstein SI et al (1992) Renal artery stenosis: prevalence and associated risk factors in patients undergoing routine cardiac catheterization. J Am Soc Nephrol 2:1608–1616

Hennerici MG, Hülsbömer HB, Hefter H et al (1987) Natural history of asymptomatic extracranial arterial disease. Results of a long-term prospective study. Brain 110:777–791

Hirsch AT, Haskal ZJ, Hertzer NR, Bakal CW, Craeger MA et al (2005) ACC/AHA 2005 practice guidelines for the management of patients with peripheral arterial disease. Circulation 113:e463–e654

Hooi JD, Stoffers HE, Kester AD et al (1998) Risk factors and cardiovascular diseases associated with asymptomatic peripheral arterial occlusive disease. The Limburg PAOD Study. Peripheral Arterial Occlusive Disease. Scand J Prim Health Care 16:177–182

Hooi JD, Kester AD, Stoffers HE et al (2001) Incidence of and risk factors for asymptomatic peripheral arterial occlusive disease: a longitudinal study. Am J Epidemiol 153:666–672

Jean WJ, al-Bitar I, Zwicke DL et al (1994) High incidence of renal artery stenosis in patients with coronary artery disease. Cathet Cardiovasc Diagn 32:8–10

Kannel WB (1996) The demographics of claudication and the aging of the American population. Vasc Med 1:60–64
Kannel WB, Skinner JJ Jr, Schwartz MJ et al (1970) Intermittent claudication: incidence in the Framingham Study. Circulation 41:875–883
Kern R, Steinke W, Daffertshofer M, Prager R, Hennerici M (2005) Stroke recurrences in patients with symptomatic vs asymptomatic middle cerebral artery disease. Neurology 65:859–864
Lee VH, Brown RD, Mandrekar JN, Mokri B (2006) Incidence and outcome of cervical artery dissection: a population-based study. Neurology 67:1809–1812
Luther BLP (2001) Intestinale Durchblutungsstörungen, Mesenterialinfarkt, Angina abdominalis. Therapieoptionen, Prognosen. Steinkopff, Darmstadt
Mazighi M, Labreuche J, Gongora-Rivera F, Duyckaerts C, Hauw JJ, Amarenco P (2008) Autopsy prevalence of intracranial atherosclerosis in patients with fatal stroke. Stroke 39:1142–1147
McDermott MM, Ferrucci L, Simonsick EM et al (2002) The ankle brachial index and change in lower extremity functioning over time: the Women's Health and Aging Study. J Am Geriatr Soc 50:238–246
Ness J, Aronow WS (1999) Prevalence of coexistence of coronary artery disease, ischemic stroke, and peripheral arterial disease in older persons, mean age 80 years, in an academic hospital-based geriatrics practice. J Am Geriatr Soc 47:1255–1256
Olsson C, Thelin S, Stähle E et al (2006) Thoracic aortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14,000 cases from 1987 to 2002. Circulation 114:2611–2618
Ozsarlak Ö, von Goethem JW, Maes M, Parizel PM (2004) MR angiography of the intracranial vessels: technical aspects and clinical applications. Neuroradiology 46:955–972
Raaymakers TW, Buys PC, Jr Verbeeten B et al (1999) MR angiography as a screening tool for intracranial aneurysms: feasibility, test characteristics, and interobserver agreement. AJR Am J Roentgenol 173:1469–1475
Rinkel GJ (2008) Natural history, epidemiology and screening of unruptured intracranial aneurysms. J Neuroradiol 35:99–103
Rinkel GJ, Djibuti M, Algra A, van Gijn J (1998) Prevalence and risk of rupture of intracranial aneurysms: a systematic review. Stroke 29:251–256
Safian RD, Textor SC (2001) Renal-artery stenosis. N Engl J Med 44:431–442
Schneider JR, Walsh DB, McDaniel MD, Zwolak RM, Besso SR, Cronenwett JL (1993) Pedal bypass versus tibial bypass with autogenous vein: a comparison of outcome and hemodynamic results. J Vasc Surg 17:1029–1040
Schwartz CJ, White TA (1964) Stenosis of renal artery: an unselected necropsy study. Br Med J 5422:1415–1421
Spengel, FA, Diehm C, Heidrich H, Schulte KL, Theiss W (2001) Diagnostik und Therapie der arteriellen Verschlusskrankheit der Becken-Beinarterien. VASA 30(Suppl 57):1–20
The UK Small Aneurysm Trial Participants (1998) Mortality results for randomised controlled trial of early elective surgery or ultrasonographic surveillance for small abdominal aortic aneurysms. Lancet 352:1649–1655
Trastek VF, Pairolero PC, Joyce JW et al (1982) Splenic artery aneurysms. Surgery 91:694–699
WASID Trial Investigators (2005) Comparison of warfarin and aspirin for symptomatic intracranial arterial stenosis. N Engl J Med 352:1305–1316
Weber-Mzell D, Kotanko P, Schumacher M et al (2002) Coronary anatomy predicts presence or absence of renal artery stenosis: a prospective study in patients undergoing cardiac catheterization for suspected coronary artery disease. Eur Heart J 23:1684–1691