Angioplasty and Stenting of the Carotid and Supra-Aortic Trunks

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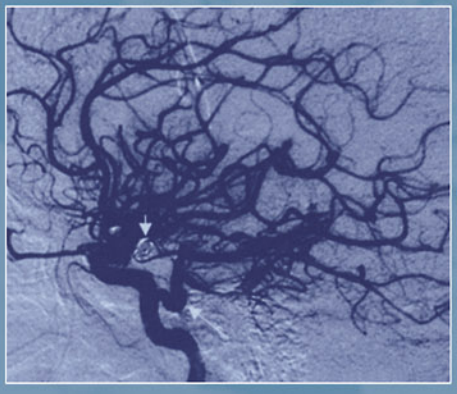

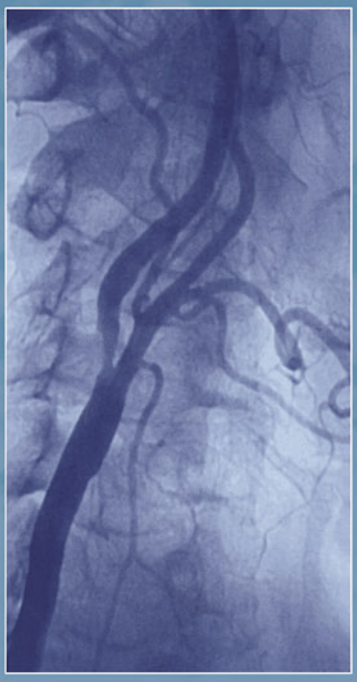

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Preface

In the USA there are more than 750 000 strokes every year, and in France there are more than 150 000. Stroke is currently the third leading cause of death and of severe neurological disability.

As the elderly proportion of the general population increases, the death rate from stroke has the potential to reach epidemic proportions.[1] Therefore, stroke is a growing major public health issue, Approximately one-third of all strokes are due to carotid bifurcation disease.

Patients are often screened with a carotid Doppler ultrasound. Although this test may be a good screening tool, it is not always accurate. Magnetic resonance angiography is also being increasingly employed as a screening tool but has yet to be validated. A four-vessel cerebral angiography with intracranial views remains indispensable before making the therapeutic decision.[2]

Up until now carotid endarterectomy (CEA) has been the accepted "gold standard" for carotid bifurcation stenosis. Four major randomized trials have shown the superiority of CEA over medical therapy in both symptomatic and asymptomatic patients who were eligible for these studies.[3–6] These results are impressive. However, there are several factors that are often overlooked.[2]

First there is a high rate of perioperative complications due to general anaesthesia and/or from dissection of the neck. The NASCET Study initiated in 1988 and completed in 1991 was a low-risk trial, considering that there was an extensive list of exclusion criteria. Despite this, the periprocedural stroke and death rate was 5.6% and the cumulative 2-year stroke rate of 9% in CEA patients versus 26% in medically managed patients. We also have to mention the 7.8% incidence of cranial nerve palsy 5.5% of significant neck haematomas that developed in the surgical group and a 14% of perioperative stroke and death rate in the high-risk subgroups of patients with contralateral carotid occlusion. The European Carotid Surgical Trial (ECST) described a perioperative stroke and death rate of 7.1 % and a rate of6.81 % for cranial nerve palsy

Second, studies in which unselected surgeons have performed CEA in the general population suggest that the perioperative complications rates may in fact be higher in the 'real world' compared with those reported in randomized trials.[7–9] Chaturvedi et al.[10] reported a 11 % stroke and death rate following the procedure describing those results as the real world of CEA.

Third, we have to point out that perioperative risks increase in certain subgroups, such as women, the elderly patients with hypertension, recent myocardial infarction and those with prior ipsilateral CEA and contralateral carotid artery occlusion.[5,11] Carotid angioplasty and stenting (CAS) has been proposed as an alternative to surgery and is rapidly becoming the preferred treatment for a carotid stenosis.

We now have class I scientific evidence from a pivotal randomized trial (CAVATAS) demonstrating that carotid angioplasty and bail-out stenting have equivalent short- and long-term neurological complication rates compared with CEA.[12] The 30-day mortality, stroke, myocardial infarction rate was 10% for carotid angioplasty versus 9.9% for CEA. There were more acute complications following CEA; cranial nerve palsy (0% vs 8.7% for PTA vs CEA, respectively, $P < 0.0001$) and hematoma (1.2% vs 6.7% for PTA vs CEA, $P < 0.00015$). Another randomized study recently published by Brooks and performed in a community hospital reported similar results comparing CAS and CEA.[13]

Both single- and multi-institutional studies utilizing CAS are now reporting encouraging short- and long-term results with a perioperative stroke and death rate comparable to surgical data and in some studies a complication rate less than 4%.

These studies included high-risk surgical patients and most of them would not have been eligible for enrolment in either the NASCET or the ACAS trials. Wholey[14] reported a series of 640 patients with CAS. Only 9% would have been eligible for these randomized studies. The perioperative stroke and death rate was 3.8%.

Over the past few years there have been many improvements in CAS techniques. The profile of the delivery system is better. Stents have improved from 7 Fr catheter systems to those of 5 Fr of new design and material that are being used currently. Catheter sheaths of 6 or 7 Fr can be used minimizing the risk of local and neurological complications. We also have better adjunct pharmaceutical therapies. But the most important improvement is certainly the use of protection devices and now we have to determine their role and whether CAS with their use can achieve even superior results to CEA.

Several series have been published showing that the neurological complication rate is reduced at least by 60% with protection devices. Roubin reported a 30-day neurological complication rate of 6.2% without protection (719 patients, 811 procedures) and of 2.6% with protection (432 patients, 465 procedures) with an important benefit for patients over 80 years (16.6% without protection vs 3.2% with protection) There was no difference between balloon occlusion and filters. Mathias[18] published a series of 1621 patients, 1799 carotid angioplasties with a death and stroke rate of 3.8% without protection and 1.7% with protection.

In our experience of 404 procedures performed with protection, we had a 30-day neurological complication rate of 2.2% with the Percusurge Guardwire (n=268) and 1.3% with filters. Without protection (186 patients) our neurological complication rate was 4.9%.

A multicentre carotid world registry has enrolled more than 1 1 000 patients from 42 centres in the USA, Europe, and South America. The results were reported by Wholey.[17] The 30-day neurological complication rate was 5.29% for unprotected CAS (n=6688), 6.07% for symptomatic patients (n=4223), 3.97% for asymptomatic patients (n = 2465) and 2.27% for protected CAS (n=4005), 2.82% for symptomatic patients (n=1949), 1.75% for asymptomatic patients (n=2056).

Although this world registry lacks the validation of a randomized trial, the neurological "event" rate in symptomatic and asymptomatic group of patients compares favourably with NASCET and ACAS studies. These data are even more significant when one considers that most of the patients who underwent CAS had significant high surgical risk criteria in contrast to those in the low-risk ACAS and NASCET studies.[14]

The most important report is perhaps the SAPPHIRE study,[18] the first randomized multicentre study comparing the safety and efficacy of CAS with ANGIOGUARD™ XP Emboli Protection Guardwire to CEA in high surgical risk patients. Of the 307 randomized patients, 156 received CORDIS PRECISE ™ Nitinol Self Expanding stent. At 30-day follow-up the major adverse event (MAE; defined as death, stroke or myocardial infarction) rate, for the randomized stented group was 5.8% vs 12.6% for the CEA treated patients, a statistically significant improvement.

This amount of data should be sufficient to overcome the resistance to CAS and allow full reimbursement for the procedure as a service-covered admission.[14] CAS is a safe and efficient procedure and should now be proposed to the majority of patients suffering from a carotid artery stenosis.

There are few absolute or relative contraindications for CAS (pedunculated thrombus, important tortuosities of the arteries, important calcifications and altherama of the aortic arch and carotid vessels, intracranial lesions, intolerance to antiplatelet therapy and so on). These patients are maybe better served by surgery.

The long-term follow-up of CAS is also encouraging, and comparable to CEA. Restenosis for endovascular stents is not an issue, less than 5% at 1 year, because the vessel is ideally suited to a high flow rate, excellent outflow and a low resistive system.[14]

After Roubin,[19] we can say that CAS under cerebral protection is now the standard of care, and as Wholey has shown with the world registry, there is now a new order and CAS is perhaps becoming the treatment of choice of a carotid stenosis.

CAS is ready for prime time.[14] There is little question that in the high surgical risk subset, it may be the preferred method. With the addition of cerebral protection and improved stent configuration, CAS is also a viable alternative in the presence of most other indications. An increasing number of patients are requesting CAS rather than CEA. But, the problem of reimbursement remains. Reimbursement is denied to a significant number of patients who are not surgical candidates. As pointed out by Wholey sending these patients home because the procedure is a non-service-covered admission is assigning them a stroke warrant.

We have to await the randomized studies that are programmed in different countries (USA, Europe and elsehwere), But by the time a randomized trial is completed, it would already be historically obsolete. The technology is not stabilized, the learning curve is important and will influence the results and case solution is frequently biased.

Carefully monitored, multicentre observational studies correlated closely with randomized clinical trials could enrol larger numbers in a more timely fashion, although this would be considerably expensive.[14]

We hope that very shortly health authorities, and physicians, particularly surgeons, will be convinced that CAS is at least as effective as CEA and is a major advance in the treatment of carotid stenosis.

This book reviews the present state of the art with CAS. Chapters from many of the pioneers in this field are included and these provide a broad overview of approaches, techniques, and particularly protection devices, pittfalls and methods to reduce potential complication, select and enlarge indications and improve short- and long-term results.

Although the picture in this rapidly evolving field may change considerably in the next few years, we have tried to provide all interventionists interested in CAS with this up-to-

date snapshot that summarizes current relevant knowledge about endovascular interventional therapy for carotid disease.

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Foreword: vascular surgery at the start of the twenty-first century

Vascular surgery started just before World War II with the monumental work of Professor Leriche and his school in Strasburg, France, with disciples developing vascular surgery in Europe and the USA thereafter. Among these disciples Michael E DeBakey in the USA, Rene Fontaine, Kunlin and others in France and J Cid Dos Santos in Portugal were the pioneers who introduced vascular techniques. DeBakey was instrumental in the development of plastic arterial prostheses. He also introduced his philosophy for the surgical treatment of arteriosclerosis by applying the surgical procedures of excision, thromboendarterectomy and by-pass on the affected arterial segments by occlusive or aneurysmatic changes.

In 1963 a breakthrough in vascular surgery was the introduction of the balloon catheter by Thomas Fogarty for the removal of occlusive material (embolic, thrombotic) from arteries and veins.

With the improvement of the quality of arterial prostheses, the extensive use of autologous veins for reconstructive arterial surgery and the refinement of vascular surgical techniques, vascular surgery had reached an impressive level of successful procedures in the 1980s.

It is interesting to point out that a revolutionary breakthrough in the interventional treatment of vascular diseases progressed during the 1970s and 1980s after the impressive accomplishments of Dotter, the US radiologist, in 1962–3 who had opened occluded arteries using the percutaneous insertion of solid catheters into the iliac and femoral arteries. This technique was popularized in Europe by Zeitler from Germany.

A few years later the introduction of the balloon catheter by Andreas Gruentzig in Zurich for the dilatation of the stenosed arteries (coronaries, splachnic and peripheral) resulted in an explosion in the field of the interventional therapy of vascular diseases. Percutaneous transluminal arterial dilatation has since then been used with increasing frequency worldwide. In the mean time, new percutaneously performed techniques and methods have emerged to open occluded arteries and for the dilatation of the stenosed arteries. Such techniques are various types of laser beams, atherotomes and the development of various stents and so on.

The introduction of a covered stent for the exclusion of an abdominal aortic aneurysm (endoluminal treatment) by Juan Parodi in Buenos Aires in 1990 not only opened the horizons of the treatment of the aneurysmatic disease but also stimulated the interest of the vascular surgical community in this field.

For many of the vascular surgeons of my generation who have been a part of the evolution of vascular surgery, it was obvious at the beginning of the 1990s that vascular surgery was at a standstill after the impressive progress of the endoluminal procedures

with a tendency to replace the classical surgical ones. After the initiatives of Edward B Diethrich, a small group of renowned vascular surgeons established the International Society of Endovascular Surgery in Phoenix, Arizona in 1992.

The new society was welcomed enthusiastically not only by vascular surgeons but also by cardiologists and radiologists, although a conflict of scientific and professional interest among the interventionists and the surgeons was obvious. For this reason the executive board of the society, in order to promote the close co-operation between the various specialists, decided to change the name of the society to the International Society of Endovascular Specialists.

Today more than half of peripheral vascular surgical procedures including aneurysmectomies at all the levels, can be replaced by endovascular procedures. These can be performed by interventionists, radiologists and cardiologists. However, in most cases the co-operation of vascular surgeons is important, not only for the follow-up but generally to cope with the complications. Also they are required in cases of endoluminal treatment of aneurysms to introduce the device surgically and to restore the arterial lumen, sometimes in combination with a vascular procedure. However, most vascular surgeons, except for a few who have expertise in the endoluminal procedures, have little operating experience. For this reason close co-operation of all the involved specialists in the interventional treatment of vascular diseases is absolutely essential. It is also important for vascular surgeons to learn the techniques of endovascular procedures. Also training in these techniques for vascular surgeons and vascular residents should be provided in major medical centres after proper arrangements between the various specialists.

It is now for all the parties concerned to realize that the endovascular procedures field is advancing very fast, thus making the procedures simpler, safer and transferable and in the near future more economical, limiting the necessity of classical surgical procedures. For this reason close co-operation among the various specialists is mandatory especially on the part of the vascular surgeons.

Time cannot wait
Thucydides—Greek philosopher

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PART I
Carotid artery stenosis: natural history, histopathology

1
Epidemiology and pathophysiology of carotid artery disease

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Introduction

Stroke remains a major complication of atherosclerotic cerebrovascular disease, with extracranial carotid occlusive disease accounting for nearly one-third of all events. Although dissection of the internal carotid artery, fibromuscular dysplasia, arteritis and trauma may result in cerebrovascular ischaemia, atherosclerosis is the most common aetiology of disease involving the extracranial internal carotid artery. Commonly associated risk factors for atherosclerosis play a role in carotid artery occlusive disease, although hypertension is by far the most important atherosclerotic risk factor for stroke. Increasing age, increasing number of risk factors and increasing severity of carotid stenosis all lead to an increased propensity for stroke.

One-third of all strokes that occur annually in the USA are due to extracranial carotid atherosclerosis, accounting for approximately 150 000 events per year. In the Minneapolis-St. Paul, Minnesota, metropolitan area, despite a decline in the incidence of stroke from 1970 to 1985, hospital discharges in 1985 with the diagnosis of acute stroke totalled 1792 patients (event rate of 828/100 000 population in men; 551/100 000 in women).[1] In addition to tobacco use, hypertension, hypercholesterolaemia and diabetes mellitus as direct risk factors for carotid arteriosclerosis, socioeconomic status appears to be inversely related to the development of carotid atherosclerosis.[2] This has been extended to men with demanding occupations and lower wages, who have greater progression of carotid atherosclerosis than men with higher salaries.[3]

There has been considerable controversy over the appropriate treatment of carotid artery stenosis in both symptomatic and asymptomatic patients. The major reasons for this include slowly evolving knowledge of the natural history of atherosclerotic carotid artery disease, along with a delay in definitive studies documenting proven benefit of surgical endarterectomy over standard medical therapy. Knowledge of the natural history and clinical course of mild, moderate and severe carotid stenosis allows physicians to make appropriate decisions regarding optimal strategies for treatment of this disorder.

Asymptomatic carotid bruit

Cervical bruits result from several causes (Table 1.1). Estimates of the prevalence of asymptomatic carotid bruits in adults range from 1 %[4] to 2.3% in patients aged 45–54

years and 8.2% in patients aged 75 years or older.[5] However, in a selected series of patients scheduled to undergo vascular surgical procedures, the incidence of cervical bruits ranged from 6%[6] to 16%,[7] with a mean prevalence of 10%.[8] The risk of developing a carotid bruit in patients over the age of 65 is approximately 1 % per year, nearly twice the rate found in patients aged 45–54 years.[9]

Table 1.1 Causes of cervical bruits (systolic, diastolic or both).

• Carotid atherosclerosis (systolic, diastolic or both)
• Thyrotoxicosis (systolic, diastolic or both)
• Transmitted cardiac murmur
– Aortic stenosis (systolic)
– Aortic insufficiency (diastolic)
• Arteriovenous fistula (systolic and diastolic)

Carotid bruits and risk of cardiovascular disease

The implications of an asymptomatic carotid bruit are vast. The incidence of subsequent stroke in the face of an asymptomatic carotid bruit ranges from 1.5% annually[10] to a 3 year risk of stroke (as demonstrated by the European Carotid Surgery Trialists) of 2.196. These investigators also noted that in 127 patients with severe (70–99%) carotid stenosis, the 3 year risk of stroke was 5.7%.[11]

This association of asymptomatic carotid bruits with subsequent stroke may not be as strong in the elderly population. In one study of 241 nursing home residents whose mean age was 86 years, 12% had asymptomatic carotid bruits. This ranged from 8% in patients aged 75–84 years, through 10% in those aged 85–94 years, to 13% in patients 95 years or older. The 3 year incidence of stroke was 10% in patients with a bruit, compared with 9% in those patients without a carotid bruit, demonstrating no increase in cerebrovascular events in patients with a bruit contrasted with those who had no bruit. In 60% of surviving residents the bruit had disappeared on follow-up without an interval stroke or cerebrovascular event.[12]

The presence of a carotid bruit does not adequately predict the severity of carotid stenosis. As a substudy of the North American Symptomatic Carotid Endarterectomy Trial (NASCET), 1268 patients with recent transient cerebral ischaemia or non-disabling stroke were examined for the presence of a carotid bruit. Of these patients, 58% had a bruit localized to the ipsilateral carotid artery, 31 % had a carotid bruit involving the contralateral vessel and 24% had bilateral carotid bruits. The sensitivity and specificity of a focal bruit to predict high-grade ipsilateral carotid stenosis were 63 and 61 %, respectively. The absence of a bruit lowered the pretest probability of a 70–99% carotid stenosis from 52 to 40% (Table 1.2).[13]

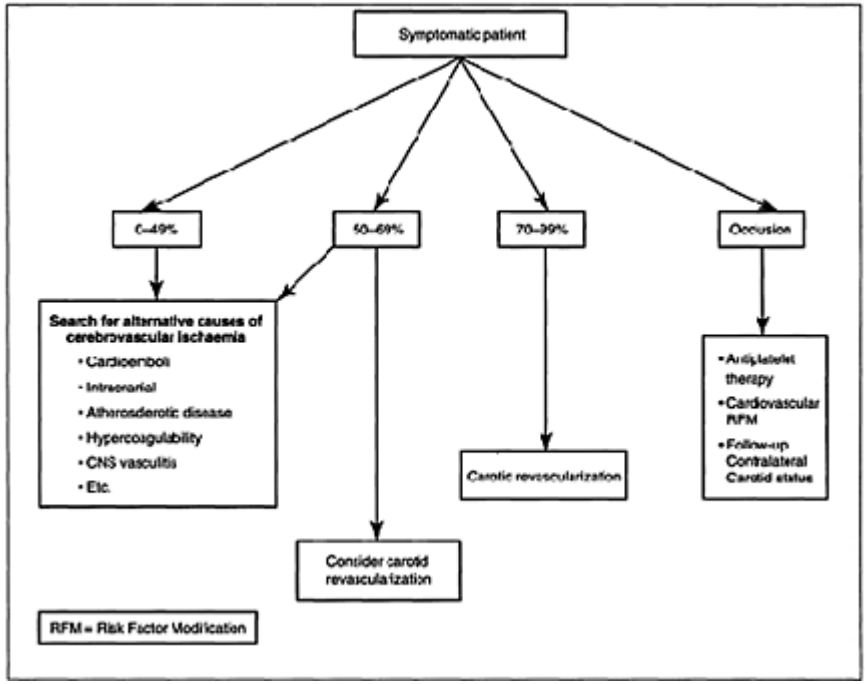

Figure 1.1 Algorithm for management of patients with symptomatic extracranial carotid stenosis.

Table 1.2 Indications for evalution of the carotid arteries.

• Symptoms suggestive of cerebral ischaemia
• Cervical bruit without symptoms
• Patient at high risk for carotid stenosis
– Concomitant coronary artery disease
– Concomitant peripheral artery disease
• Suspected carotid artery trauma or dissection

Other than the risk of subsequent cerebrovascular events in patients with asymptomatic carotid bruits, the incidence of coronary artery disease and coronary mortality in this patient group is much higher than that in the general population. The landmark study reported by Hertzer et al. demonstrated that in 506 patients with extracranial carotid artery disease and either symptoms or asymptomatic bruits, approximately 35% had severe coronary artery disease that required revascularization or had progressed to an inoperable status.[14]

This prevalence of severe coronary artery disease in patients with both symptomatic and asymptomatic carotid artery disease certainly translates into increased mortality. In one study of 444 male patients with asymptomatic carotid artery stenosis, the mortality rate was 37% at a mean of 4 years of follow-up, and 61 % of these deaths were due to coronary artery disease. Multivariate analysis revealed that diabetes mellitus, an abnormal electrocardiogram and the presence of intermittent claudication were all associated with an increased mortality risk (two or three risk factors revealed an annual mortality rate of 11.3 or 13%, respectively).[15]

Established extracranial carotid artery stenosis

The risk of stroke increases with the severity of the carotid stenosis. The stroke rate in patients with carotid stenosis of 75% or less is 1.3% per year, and 10.5% per year if the stenosis is greater than 75%.[16] In symptomatic patients with 70–99% carotid stenosis followed medically for 2 years, NASCET investigators demonstrated a 26% risk of ipsilateral stroke and 28% risk of any stroke.[17] It appears, however, that the initial cerebrovascular symptom conveys differing risks of subsequent stroke. A retinal transient ischaemic attack (TIA), such as amaurosis fugax, led to an annual stroke rate of 2%. Over 7 years of follow-up, the cumulative rate of cerebral infarction was 14% in patients with amaurosis, compared with 27% in patients with hemispheric TIAs as the initial cerebrovascular symptom.[18] The NASCET investigators demonstrated a 2 year risk of fatal and non-fatal stroke of 17% after transient monocular blindness, and 42% after hemispheric TIA.[17]

Progression of carotid stenosis

Established extracranial carotid artery stenosis demonstrates disease progression in approximately 20–40% of cases. In one prospective natural history study of 232 patients with mild (< 50%) and moderate (50–79%) carotid stenosis followed with annual carotid duplex ultrasonography for a mean of 7 years, 23% demonstrated disease progression. One-half of these patients progressed to severe stenosis (80–99%) or occlusion. Risk of progression to either 80–99% stenosis or occlusion was more likely in patients whose initial stenosis was categorized as 50–79% rather than < 50%.[19]

More recent data in 425 asymptomatic patients with 50–79% carotid stenosis followed for a mean of 38 months demonstrated progression of stenosis in 17% of 282 arteries with at least two serial carotid duplex ultrasound examinations. There was a low incidence of ipsilateral stroke, however, despite this rate of disease progression (0.85% at 1 year, 3.6% at 3 years, 5.4% at 5 years).[20] All natural history studies agree, nonetheless, that more severe stenoses carry increasing risks of disease progression and subsequent stroke. Of 242 asymptomatic patients with variable degrees of carotid stenosis, 35 patients suffered stroke or TIA. However, patients with 80–99% carotid stenosis had an annual neurological event rate of 20.6%.[21]

Internal carotid artery occlusion represents an unpredictable dilemma. In a

retrospective review of 167 patients with carotid occlusion followed for a mean of 39 months, 27% had no symptoms, 43% suffered stroke and 17% had a TIA. Over the course of follow-up, 18% had a stroke, with 67% ipsilateral to the occlusion. Consistent with other reports, heart disease was the cause of death in 41 % of the 54 patients who died during follow-up. The contralateral stroke event rate was 33%, with a lower 5 year stroke-free event rate in patients with stenoses of 50–99% (77%) compared with < 50% (94%) (p=0.08).[22]

Plaque ulceration clearly increases the risk of subsequent stroke. As in the coronary arterial bed, the pathophysiology of plaque rupture, foam cell infiltration and thinning of the fibrous cap occurs more often in patients with symptomatic than in those with asymptomatic carotid stenosis.[23] Plaque ulceration over 2 years of follow-up in the medically treated NASCET patients increased the risk of ipsilateral stroke from 26.3 to 73.2% as the degree of stenosis progressed from 75 to 99%. In patients without plaque ulceration, the 2 year stroke risk was 21.3% regardless of the degree of stenosis.[24]

Conclusion

Extracranial carotid artery disease represents an important cause of stroke. It is clear that the number of patients estimated to suffer from a first or subsequent stroke continues to rise. Patients with hypertension, hyperlipidaemia or diabetes mellitus, and those who continue to smoke, are at increased risk of stroke owing to a multitude of aetiologies, one of which is carotid artery disease.

Understanding these risks will allow physicians to identify patients who are eligible for either screening or diagnostic testing to identify the presence and severity of carotid artery disease.

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2
Histopathology of carotid stenosis: correlation between the types of plaque and the risks of neurological complications

R Virmani, AP Burke, FD Kolodgie and A Farb

Introduction

About 600 000 individuals each year suffer a new or recurrent ischaemic and haemorrhagic stroke.[1] Overall, ischaemic strokes account for about 85% of strokes (61% atherothrombotic, 24% embolic), and about 15% are haemorrhagic.[1] In symptomatic carotid disease, atheroembolism is considered the underlying cause of the majority of strokes, although the contribution of intracerebral atherosclerosis is unknown. This chapter characterizes atherosclerotic carotid disease in light of the knowledge of coronary atherosclerosis and relates carotid plaque morphology to cerebral ischaemic syndromes.

Pathological features of atherosclerosis

The earliest classification of atherosclerosis, although simple, had merit. Two well-accepted lesions were described: the fatty streak and the atheromatous plaque. The fatty streak was considered a precursor lesion to the advanced atheromatous plaque. The fatty streak consists of cellular elements (smooth muscle cells, macrophages and other inflammatory cells), the matrix (collagen, elastic fibres and proteoglycans), and intracellular and extracellular lipids. The atheromatous or fibrofatty plaque consists of a raised lesion having a lipid core (cholesterol and cholesterol esters) and a fibrous cap. The atheromatous plaque can result in complicated lesions, including calcification, ulceration, thrombosis and haemorrhage.

A more sophisticated numerical classification was put forth recently by the American Heart Association (AHA).[2,3] It implies an orderly, linear pattern of progression of lesions, which may or may not be valid, and is based on the assumption that all thrombosis occurs from plaque rupture, which is not the case in the coronary arteries.

A modification of the AHA classification, based on examination of over 200 cases of sudden coronary death, was published recently.[4] The early lesion classification is similar to that reported by Stary et al.[2,3] The more advanced lesions, or fibrous cap atheromas, can be further characterized by the nature of the fibrous cap. The thin-cap atheroma resembles plaque rupture and is its presumed precursor lesion; the thincap atheroma has also been termed the vulnerable plaque. Because plaque rupture accounts for the majority of thrombi in patients with sudden coronary death, identification of thincap atheroma is

critical. The precursor lesion for the less common type of coronary thrombosis, plaque erosion, differs from that of plaque rupture, and includes early lesions without a well-developed atheromatous core (pathological intimal thickening). A still rarer form of plaque disruption that may result in luminal thrombus, here called the 'calcified nodule', has yet to be characterized and represents 1–2% of coronary thrombi.[4]

Mechanisms of acute coronary syndromes

In sudden coronary death, at least 75–80% of coronary arteries show the presence of acute or organized thrombi, and only 20–25% of arteries show no thrombi except for ≥ 75% cross-sectional area luminal narrowing.[4] Stable plaque or fibrocalcific plaques, which lead to ≥ 75% luminal narrowing in the absence of a thrombus, are present in up to 26% of patients dying suddenly with severe coronary disease. The mechanisms of sudden death are more diverse than those of acute myocardial infarctions which, in the setting of atherosclerotic heart disease, are nearly always precipitated by acute coronary thrombosis.

Plaque rupture accounts for 62% of all thrombi in patients with sudden coronary death, and plaque erosion accounts for the remaining 38%. Subsequent morphological studies of patients dying with acute myocardial infarction have shown a similar breakdown of frequency of the type of coronary thrombus.[5,6] Plaque erosion is an infrequent cause of sudden coronary death in men and women over the age of 50 years, and the majority of coronary thrombi in women under the age of 50 are plaque erosions. The organization of acute thrombi, both healed plaque ruptures and erosions, may be one of the major mechanisms of plaque progression beyond 50% cross-sectional area stenosis. Chronic total occlusion of the coronary arteries can occur from rupture or erosion and is usually accompanied by a healed myocardial infarction (90%).

General features of carotid atherosclerosis

The earliest pathological studies described the occurrence of atherosclerosis near branch ostia, bifurcations and bends, suggesting that flow dynamics play an important role in its induction. It has been demonstrated that laminar flow is disturbed at bifurcations of the carotid artery. The greatest atherosclerotic plaque accumulation occurs on the outer wall of the proximal segment of the sinus of the internal carotid artery, in the region of the lowest wall shear stress. The intimal thickness is the least, on the flow divider side, where wall stress is the highest (Fig. 2.1).[7]

The mechanisms by which carotid atherosclerosis results in cerebrovascular symptoms are less well understood than those linking coronary disease and myocardial symptoms, because of fewer pathological studies at autopsy detailing carotid and aortic morphology with cerebral findings. In general, it is clear that occlusive thrombus triggered by plaque rupture is relatively uncommon in the carotid circulation, because of the high flow and tendency for ulceration and embolization of plaque contents and mural thrombus.

Only detailed morphological studies of carotid endarterectomy specimens will improve

the understanding of pathophysiological processes involved in the causation of symptoms in carotid stenosis. For example, what makes one patient with 60% diameter stenosis be symptomatic and

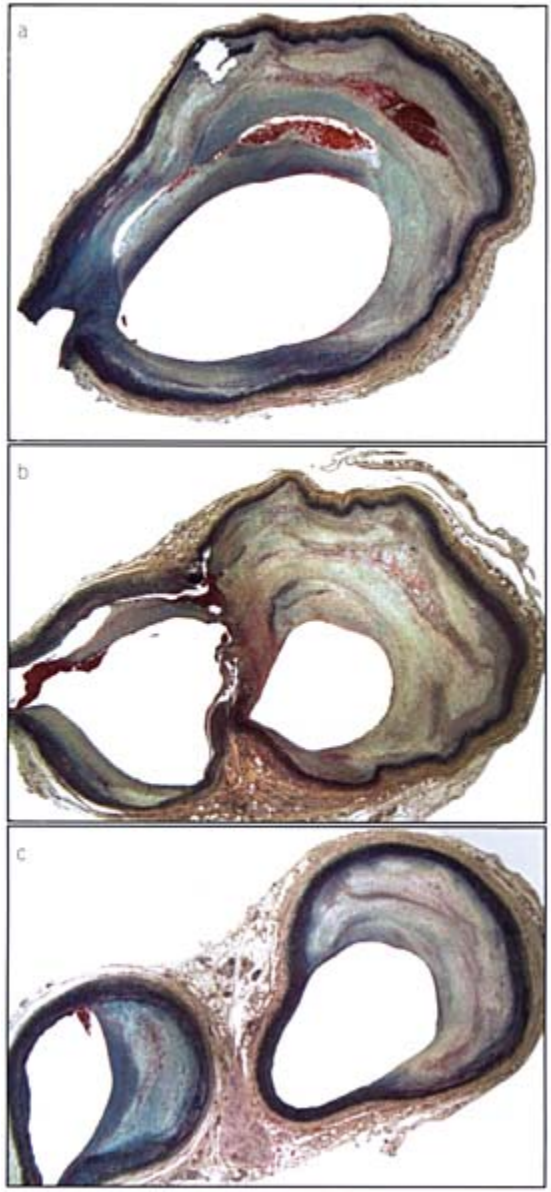

Figure 2.1 Carotid bifurcation, atherosclerotic disease. (a) The common carotid artery There is moderate narrowing by atherosclerotic plaque, with two haemorrhagic necrotic cores. This layering indicates repeated

surface disruption (rupture) and healing with smooth-muscle cells. (b) The bifurcation, with the flow divider illustrated in the centre. Note that the flow dividers on either side are relatively devoid of plaque, indicating that the high shear stress in this site is relatively protective of accumulation of atherosclerotic material. (c) The internal carotid artery (right), with the external carotid (left). Note the positive remodelling of the internal carotid artery at the site of the atherosclerotic plaque.

Table 2.1 Artherosclerotic plaque classifications.

		Virmani et al.	
	Stary et al.	*Initial*	*Progression*
Early plaques	Type 1: microscopic detection of lipid droplets in intima and small groups of macrophage foam cells	Intimal thickening	None
	Type II: fatty streaks visible on gross inspection, layers of foam cells, occasional lymphocytes and mast cells	Intimal xanthoma	None
	Type III (intermediate): extracellular lipid pools present among layers of smooth-muscle cells	Pathological intimal thickening	Thrombus (erosion)
Intermediate plaque	Type IV: well-defined lipid core; may develop surface disruption (fissure)	Fibrous cap atheroma	Thrombus (erosion)‡
Late lesions		Thin fibrous cap atheroma	Thrombus (rupture) haemorrhage/fibrin§
	Type Va: new fibrous tissue overlying lipid core (multilayered fibroatheroma) *	Healed plaque rupture, erosion	Repeated rupture or erosion with or without total occlusion
	Type Vb: calcification†	Fibrocalcific plaque (with or without necrotic core)	
	Type Vc: fibrotic lesion with minimal lipid (could be result of organized thrombi)		

Miscellaneous/ complicated features	Type Vla: surface disruption Type Vlb: intraplaque haemorrhage Type VIc: thrombosis		
		Calcified nodule	Thrombus (usually non-occlusive)

Adapted from data published in Refs 2–4.
*May overlap with healed plaque ruptures.
†Occasionally referred to as type VII lesion.
‡May further progress with healing (healed erosion).
§May further progress with healing (healed rupture).

another asymptomatic? Which patient will benefit from a surgical intervention?

Several recent reports indicate significant differences in frequency between symptomatic and asymptomatic patients of plaque rupture, thinning of the fibrous cap, and infiltration of the fibrous cap by macrophages and T-cells. Studies in the authors' laboratory showed that plaque rupture was present in 74% of carotid plaques removed for symptomatic carotid artery disease, as opposed to only 32% of plaques removed for asymptomatic disease.[8] These observations suggest differences in plaque morphology between patients with symptomatic and asymptomatic disease (Table 2.2).

In a study of carotid endarterectomy specimens from symptomatic high-grade stenosis lesions and asymptomatic autopsy specimens without high-grade carotid artery stenosis, Bassiouny et al. showed that high-grade carotid stenotic plaques were associated with a significantly higher incidence of ulceration (53%), thrombosis (49%) and lumen irregularity (78%) compared with non-stenotic asymptomatic plaques (6%, 0% and 17%, respectively, $p < 0.01$). Although these features were more prominent in lesions that produced symptoms, they were present in 80% of the stenotic bifurcations, and did not distinguish between symptomatic endarterectomy and asymptomatic autopsy lesions.[9]

Table 2.2 Gross and microscopic plaque characteristics in symptomatic and symptomatic patients undergoing carotid endarterectomy.

Gross morphology	*Symptomatic (n=25)*	*Asymptomatic (n=17)*	p-*Value*
% Stenosis (duplex)	74 ± 17	77 ± 15	ns
Ulceration	94	64	0.02
Plaque haemorrhage	47	52	ns
Microscopic characteristics (%)			
Plaque rupture	74	32	0.004

Thin fibrous cap	95	48	0.003
Cap foam cells	84	44	0.006
Intraplaque fibrin	100	68	0.008
Intraplaque haemorrhage	84	56	0.06
Necrotic core	84	72	ns
Ulceration	11	8	ns
Calcified nodule	7	7	ns
Thrombus	63	80	ns
SMC-rich area	5	0	ns
Eccentric shape	68	64	ns

Modified from Carr et al.[8]
SMC: smooth-muscle cell; ns: not significant.

The study by Bassiouny et al, did not show as clear-cut a difference between asymptomatic and symptomatic lesions as seen in the study by the present group. The reason for the discrepancies between these two studies are unclear. One explanation may be dependent on the degree of stenosis in the varying patient populations. Approximately half of symptomatic and asymptotic patients in this study had ≥ 80% stenosis, another 30–35% had 60–79% stenosis, and 21% of symptomatic versus 8% of asymptotic patients had < 60% stenosis. Further, the patient population was older than in Bassiouny's study (mean age 74 vs 61 years, respectively).

In a subsequent report from the same group, to identify further characteristics of the plaque that may predict symptomatic disease, examination of the necrotic core showed that it was twice as close to the lumen in symptomatic than in asymptomatic plaques (0.27 ± 0.3 mm vs 0.5 ± 0.5 mm, respectively, $p < 0.01$). The percentage area of necrotic core or calcification was similar for both groups (22 vs 26% and 7 the fibrous cap was three times greater in the symptomatic vs 6%, respectively). The number of macrophages infiltrating plaques than in the asymptomatic plaques (1114 ± 1104 vs 385 ± 622, respectively, $p < 0.009$).10 Finally, disruption or ulceration of the fibrous cap was more common in symptomatic than in asymptomatic plaques.

The mean fibrous cap thickness in carotid plaque rupture is nearly three times (72 ± 15 μm) that in coronary plaque ruptures (23 ± 17 μm). Carotid vulnerable plaques (necrotic core with overlying thin-cap and infiltration by macrophages, Fig. 2.2) have a mean cap thickness of 72 ± 24 μm, whereas the upper limit of a thin-cap fibroatheroma in the coronary artery is taken to be 65 μm. Quantification of the number of macrophages in the fibrous cap in the carotid and coronary arteries showed fewer macrophages in the fibrous cap of carotid plaques than of coronary plaque ruptures (13.5 ± 10.9% vs 26 ± 20%). Similarly, there were fewer macrophages in carotid vulnerable plaques than in coronary vulnerable plaques (10 ± 1.8% vs 14 ± 10%).[4]

Plaque vascularity has been shown to correlate with intraplaque haemorrhage and presence of symptomatic carotid disease.[11] The role of vasa vasorum in precipitation of

acute coronary syndromes and aortic plaque disruption is the object of intense study. Imaging techniques for the detection of vasa vasorum in carotid plaques may be important in future evaluation of carotid stenosis.

Carotid versus coronary disease: differences in plaque morphology

The classification of atherosclerotic plaque devised for coronary arteries and aortas is well suited for use in the carotid circulation. There are, however, unique features of carotid plaque morphology because of the high flow rates and the shear forces caused by the bifurcation of the common carotid artery into the internal and external carotids. Most importantly, the ulcerated plaque, which is rare in the coronary artery circulation, is relatively common in the carotid and other elastic arteries. Ulcerated plaque is a term used when the thrombus and a portion of the plaque have embolized, resulting in an excavation where the embolized components are missing (Fig. 2.3). Another feature of carotid disease is the

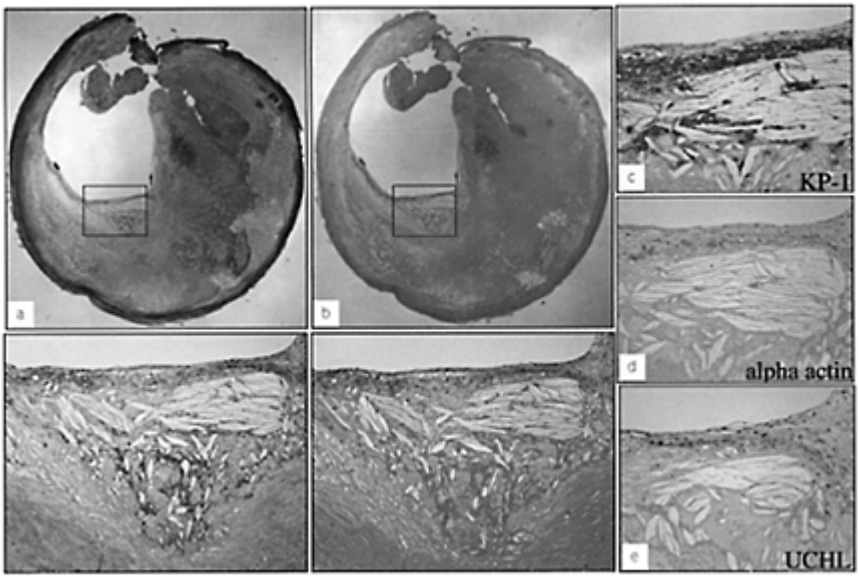

Figure 2.2 Thin fibrous cap, carotid plaque. (a, b) Carotid endarterectomy specimens with a thin fibrous cap (boxed areas, and insets below) (a: Movat; b: haematoxylin and eosin). (c-e) In the area of thinning of the cap, there are numerous macrophages, no smooth-muscle cells and a sprinkling of T-lymphocytes. (c: KP-1 for macrophages; d: α-actin for smooth-muscle cells; e: UCHL for T-cells).

infrequency of total occlusion relative to the coronary circulation. Again, the explanation for the low rate of total occlusions is likely to be related to high flow rates that prevent

the thrombotic occlusion unless repeated plaque ruptures have occurred at the same site. In patients dying suddenly, the incidence of chronic total occlusion in the presence or absence of an acute thrombus is 33%, whereas in the carotid artery the reported incidence in symptomatic carotid disease is 8%. than seen in the coronary and may be related to high flow Plaque haemorrhage in the carotid artery is far more frequent rates and pressures in the lumen and the vasa vasorum. Maximum frequency of haemorrhage is observed in arteries narrowed by 50–75% in cross-sectional area. The frequency of calcification is similar in coronary and carotid arteries, with maximum calcification seen in carotid arteries narrowed by greater than 70% in cross-sectional area. However, the frequency of calcified nodules (Fig. 2.4), a form a calcification that results in irregular nodules of calcium that possibly form on a nidus of intraplaque fibrin, is higher in carotid disease (approximately 6–7%) than in coronary artery disease (1–2%). In contrast, plaque erosion, while being common in the coronary circulation, is rare in the carotid artery. In carotid arteries the percentage stenosis was highest in healed plaque ruptures and was greater than that seen in thin-cap atheroma and in plaque rupture.

Risk factors contributing to symptomatic carotid disease

As is the case with coronary disease, the correlation of risk factors with stroke is complicated by the multiple aetiological categories of stroke, including carotid atherothrombosis, aortic arch plaque embolization, thromboembolism for ischaemic strokes and hypertensive haemorrhagic strokes. The major risk factor is elevated blood pressure. However, the risk factors show a similar spectrum to coronary disease, and include hypertension, atherogenic factors and thrombotic factors.

By far the most important risk factor for the development of all stroke, including cerebral infarction and intracerebral

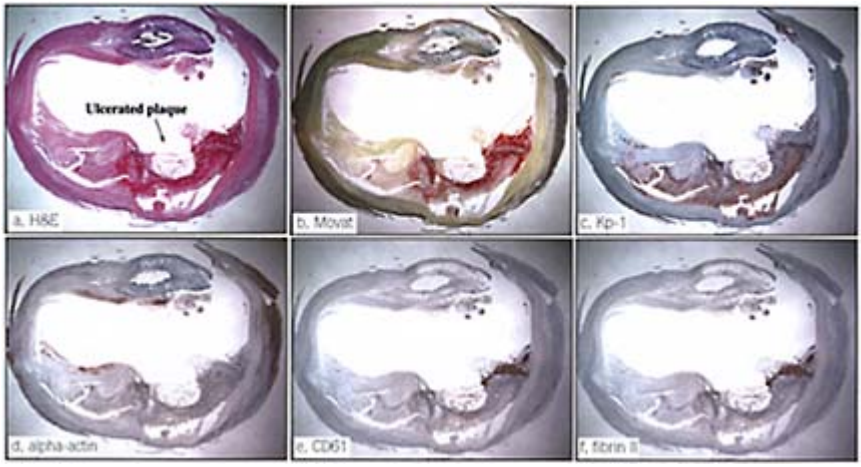

Figure 2.3 Carotid rupture with thrombosis and ulceration. Unlike coronary arteries, in which ulcers are unusual, plaque disruption in the carotid

artery frequently results in embolization and crater formation. (a) Routine haematoxylin and eosin section of a carotid artery with thrombus and ulcer. (b) Corresponding Movat pentachrome stain, which highlights collagen (yellow) and elastic tissue (black). (c-f) Immunohistochemical stains for macrophages (Kp-1), smooth-muscle cells (α-actin), platelets (CD61) and fibrin (fibrin II). Note that at the ulcer crater, there are abundant macrophages (c) with few smooth-muscle cells (d). The thrombus itself has largely embolized; there are residual platelets (e) and fibrin (f) at one edge of the crater.

haemorrhage, is hypertension, but other factors include impaired cardiac function, diabetes, non-valvular atrial fibrillation, migraine and family history. Modifiable risk factors are listed as cigarette smoking, low level of physical activity and abdominal obesity.[12,13] The incidence of stroke increases in proportion to both systolic and diastolic blood pressure, and is elevated in Blacks, who have a high rate of hypertension.[14]

Serum lipids have long been associated with coronary artery disease, but not with cerebrovascular disease. However, clinical trials using β-hydroxy-β-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors (statins) have shown a reduction of stroke risk in patients with coronary artery disease and elevated cholesterol levels. The relative risk of ischaemic stroke in diabetic patients ranges from 1.8-to 6-fold greater in case-control studies. More recently, attention has focused on inflammatory markers of atherosclerosis, and high C-reactive protein (CRP) has been shown to be predictive of the risk of future cardiovascular events. Similarly, independent of other cardiovascular risk factors, elevated plasma CRP levels significantly predict the risk of future ischaemic stroke and transient ischaemic attack in the asymptomatic elderly population.[15] High CRP at hospital discharge is a predictor of future cardiovascular events and death in patients admitted with ischaemic stroke.

Smoking is another independent risk factor for stroke, and in stroke patients is associated with an increase in arterial wall stiffness, increase in fibrinogen levels, increased platelet aggregation and haematocrit, and decreased high-density lipoprotein-cholesterol.[14] Hypercoagulable states associated with the development of stroke include antiphospholipid syndrome, factor V Leiden, prothrombin 20210 mutation, protein C and S deficiency, and high fibrinogen levels. Non-fasting total homocysteine levels are an independent risk factor for incident stroke in elderly people.[16]

Correlation of risk factors to plaque morphology

Several studies have correlated plaque morphology to risk factors in the carotid and coronary circulation. Spagnoli et al. showed that the fibrous carotid plaque was correlated with ageing and diabetes; the granulomatous plaque, rich in giant cells, with female gender and hypertension; and the xanthomatous plaque, rich in foam cells and with extensive alcianophilia, with hypercholesterolaemia. In smokers, the plaques were frequently complicated by mural thrombosis.[17]

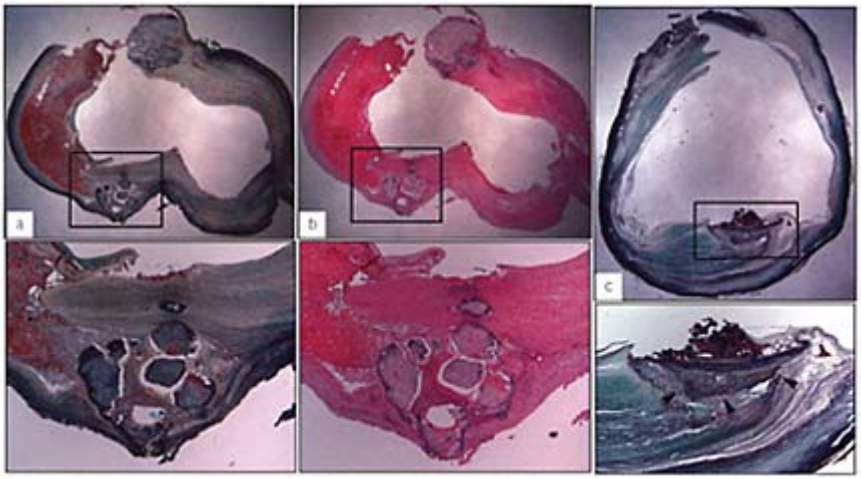

Figure 2.4 Nodular calcific disease. A form of thrombosis that is more common in the carotid artery than in the coronary is the nodular calcified plaque. (a, b) Carotid endarterectomy specimen with a nodular calcified area (boxes, and insets below) (a: Movat pentachrome; b: haematoxylin and eosin). (c) Area of surface thrombus (boxed area, and inset below) overlying the nodular calcification with underlying calcified plate (arrows).

Mauriello et al. studied carotid endarterectomy specimens removed at surgery and showed that patients with the highest tertile of fibrinogen (> 407 mg/dl) had a high incidence of thrombosis (67%) compared with plaques of subjects with the lower and middle tertile (22% and 29%, p= 0.002 and *p*= 0.009, respectively).[18] Plaque rupture was significantly associated with high fibrinogen level (54%, p= 0.003). Multivariate analysis showed that hyperfibrinogenaemia was an independent predictor of fibrous cap thickness (inverse correlation), macrophage foam cell infiltration of the cap and thrombosis. When accounting for the other risk factors, hyperfibrinogenaemia remained an independent predictor of carotid thrombosis.[18] More studies of the correlation of plaque morphology with risk factors are needed to improve the understanding of carotid disease and target risk factor modification as more detailed plaque composition is possible with imaging techniques.

Lipid-lowering therapy selectively depletes the lipid cores in carotid plaques. Zhao et al. studied carotid endarterectomy specimens from patients in the Familial Atherosclerosis Treatment Study (FATS), treated for 10 years with lipidlowering treatment. The lipid core was significantly smaller in the treated patients and there was a trend towards a higher calcified area than in non-treated controls, but the fibrous tissue content was the same.[19]

In the coronary circulation, in patients dying suddenly hypercholesterolaemia correlates with plaque rupture, and smoking is more frequent in men and women dying with acute thrombus, whether due to plaque erosion or to rupture.[20]

Hypercholesterolaemia also correlates with the number of thin-cap atheromas. Burke et al. also showed that CRP is significantly elevated in patients dying suddenly with severe coronary disease, both with and without coronary thrombosis, and correlates with plaque burden. Mean staining intensity of CRP of plaques (necrotic core and macrophage) was significantly higher in those with high serum CRP than in those with low CRP as was the mean number of thin-cap atheromas.[21]

Endarterectomy and stroke

The North American Symptomatic Carotid Endarterectomy trial showed that endarterectomy is efficacious in reducing the risk of stroke and death up to 2 years in patients with 70–99% stenosis of the ipsilateral carotid artery, who experience a transient ischaemic event or non-disabling stroke.[22] The benefit of carotid endarterectomy is reduced for those with 50–69% stenosis; however, for patients with less than 50% stenosis the failure rate was similar for endarterectomy or medical therapy.[23,24] Patients with asymptomatic carotid stenosis of60% or greater who are good surgical candidates will have areduced 5 year risk of ipsilateral stroke after carotidendarterectomy versus medical therapy.[25]

Recurrent carotid disease

The rate of recurrent carotid stenosis after carotid endarterectomy varies from 4 to 10% and usually occurs > 3 months following surgery.[26,27] In a series of 1726 endarterectomies performed at the Cleveland Clinic from 1997 to 1983, 65 (3.8%) patients were reoperated on for recurrent carotid stenosis occurring 16 months (mean 42 months) after the initial procedure. Of these patients, approximately half were symptomatic with neurological symptoms and half were asymptomatic. The recurrence interval was 57 months in specimens with atherosclerotic disease (n=37), whereas in specimens with myointimal hyperplasia (n=28) the recurrence interval was 21 months (p=0.0007). In recurrent disease, the myointimal hyperplasia consists of smooth-muscle cells in a proteoglycan matrix interspersed with fibrin; the collagen and elastin representing organization of the thrombus is sparse. Neovascularity may be present, but is usually not extensive, and surface thrombi tend to be platelet rich. Evidence of surface thrombosis is found in 77% of cases, but intraplaque thrombi are uncommon; only 15% are found in specimens collected < 36 months after the initial endarterectomy.

In the authors' experience, recurrent endarterectomy specimens collected up to 36 months typically contain myointimal hyperplasia, and beyond this interval atherosclerotic lesions are more common.[28] Seventy-four per cent of specimens with atherosclerotic lesions usually contain fibrinrich surface thrombi, which are in continuity with an intraplaque thrombus (Fig. 2.5). Extensive neovascularity in

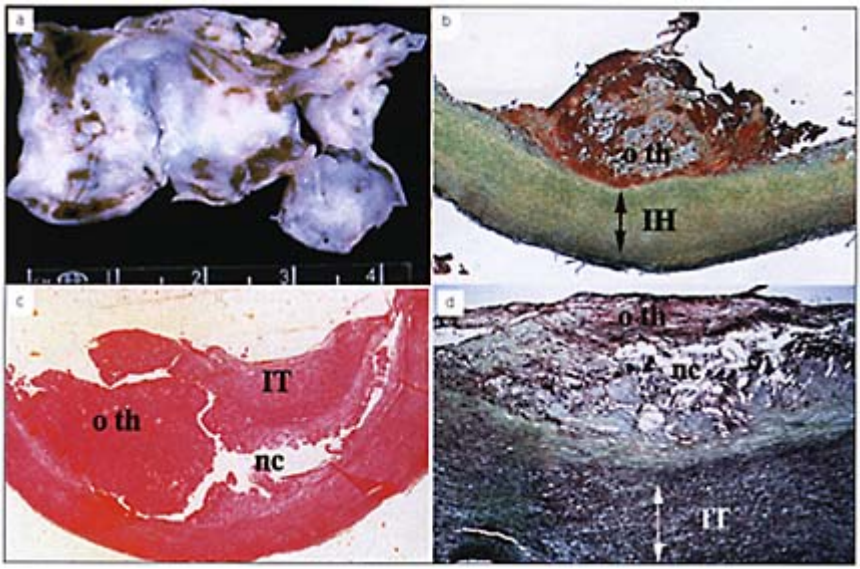

Figure 2.5 Recurrent carotid disease. (a) Recurrent carotid endarterectomy specimen with a mostly pearly white appearance from fibrointimal hyperplasia with focal thrombi. (b) Histological section of the same specimen showing organizing thrombus on the luminal surface with underlying fibrointimal hyperplasia (IH). (c) Low-power view of another specimen of a later recurrent lesion showing atherosclerotic change with necrotic core (nc) with fibrointimal thickening (IT) towards the lumen and organizing thrombus (o th) on the left. (d) High-power view of another atherosclerotic plaque: note fibrointimal thickening (IT) underneath the necrotic core (nc) and surface organizing fibrin thrombus (o th). Note the presence of cholesterol clefts with interspersed macrophages. (b, d: Movat stain; |c: haematoxylin and eosin stain). (Reproduced with permission from Virmani et al.[29] *Pathol Case Rev* 2001; 6:242, Fig. 5.)

lesions with atherosclerosis is common. The plaque components include foam cells, cholesterol clefts, and abundant collagen with focal areas of necrosis and calcification. Some cases may show myointimal hyperplasia in the deep intima, but it is usually interspersed with atherosclerotic plaque. Although all the components of atherosclerosis are present in primary and recurrent lesions, the atherosclerotic elements are arranged in a less orderly manner in the recurrent atherosclerosis. Primary plaques consist of a central necrotic core containing cholesterol clefts beneath a fibrous cap, whereas in recurrent lesions, the necrotic core is superficial and often unsupported by a dense layer of collagen. In recurrent lesions the thrombus is contained within the plaque, whereas in primary lesions it is usually associated with intraplaque haemorrhage, which is rarely observed in recurrent lesions.[27,28]

Atherosclerosis of the aortic arch as a risk factor for ischaemic stroke

Recently evidence has accumulated that atherosclerotic disease of the aortic arch may be a source of cerebral emboli.[30] Plaques located proximal to the ostium of the subclavian artery were seen in 60% of patients ≥ 60 years of age with ischaemic stroke and the association was strongest when the plaques were ≥ 4 mm in thickness.[31] In 1996, the French Study of Aortic Plaques in Stroke Group reported on patients 60 years and older admitted for brain infarction, followed with transoesophageal echocardiography to determine the presence of aortic atherosclerotic disease; the incidence of recurrent brain infarction was 11.9/100 person-years in patients with aortic wall thickness of ≥ 4 mm, compared with 2.8/100 person-years in patients with a wall thickness < 1 mm (p < 0.001).[32] It is not unusual no see plaque calcification in the aortic arch of sudden coronary death victims. Plaque ulceration and thrombosis of the aorta are not unusual findings at autopsy in patients > 60 years of age (Fig. 2.6).

Conclusion

Carotid atherosclerotic disease resembles coronary atherosclerosis but has distinct differences. Although small mural thrombi are common, occlusive luminal thrombosis is not a feature of carotid disease. Embolization of the atherothrombus results in an ulcerated plaque, a common feature of carotid disease, but infrequent in the coronary circulation.

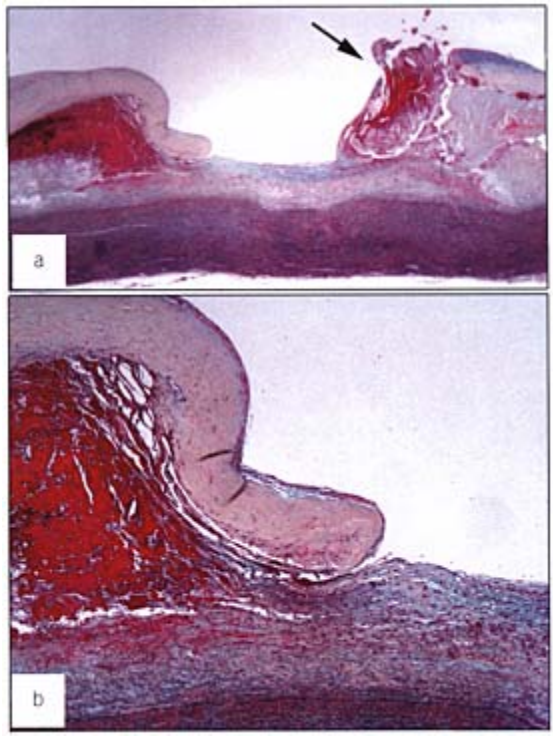

Figure 2.6 Aortic ulcer. Not all cerebrovascular ischaemia is the result of carotid disease. Aortic plaques in the area of the arch and great vessels may undergo rupture and ulceration, with embolization of the plaque and thrombus to the brain. (a) Low-magnification view of a healed rupture site, ulceration and loose necrotic core with haemorrhage and cholesterol clefts (arrow) in the aorta near the innominate artery ostium. (b) Higher magnification of the lip of the ulcer crater.

Similarly to coronary disease, carotid symptomatic disease is predominantly associated with plaque rupture, but plaque erosion, an important subset of coronary thrombosis, is rare in the carotid circulation. Calcified nodules are observed more commonly in calcified carotid than in coronary atherosclerosis. Although there is a higher incidence of plaque rupture in carotid symptomatic than in asymptomatic disease, the extent of lipid area, necrotic core size and calcification may not be different. The severity of luminal narrowing does not correlate with the presence of a vulnerable plaque. Not all cerebrovascular ischaemia originates from the carotid atherosclerotic plaque, but it may frequently arise from atherosclerotic aortic arch disease. Therefore, in any patient presenting with ischaemic stroke not only the carotid artery but also the aortic arch should be investigated.

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PART II
Supra-aortic arteries: anatomy, radiological anatomy

3
Anatomy and pathophysiology of supra-aortic vessels

NN Khanna

General anatomy of supra-aortic vessels

Knowledge of the aortic arch is mandatory for the specialist is performing interventions on vessels of the aortic arch. The aortic arch is slightly on the right, just behind the right sternoclavicular joint. The arch first ascends diagonally back and to the left, lying on the anterior surface of the trachea, then goes back across its left side and finally descends left of the fourth thoracic vertebral body to continue as the descending thoracic aorta. It is about 28 mm in diameter and tapers to a diameter of 20 mm after the origin of arch vessels. At the junction of aortic arch and descending thoracic aorta, a small stricture (the aortic isthmus) is present. In the foetus it lies between the origin of the left subclavian artery and the ductus arteriosus.

The aortic arch has two curvatures, one being convex upwards, and the second convex forwards and to the left. The aortic arch and the origin of its branches are ideally imaged as a left anterior oblique projection. The angle of the oblique projection increases with age, This view is also

Figure 3.1 Origin of the left internal carotid artery from the brachiocephalic trunk.

recommended for intubation of the supra-aortic vessels. The right anterior oblique projection is useful to delineate the bifurcation of the brachiocephalic trunk into the right common carotid artery and right subclavian artery.

Normally the aortic arch three main vessels: the innominate artery (brachiocephalic trunk), the left common carotid artery and the left subclavian artery, but variations are common. In an analysis of variations in the branching pattern of the aortic arch, Arisan et

al. showed that a normal branching pattern was seen in only 65%. The left common carotid artery originated from the innominate artery in 27% of cases (Fig. 3.1). In 2–5%, all four branches originated separately and in 3.5% two bronchiocephalic trunks were present. Rarely, external and internal carotid arteries may arise separately and the common carotid artery is absent. Sometimes both common carotid arteries arise from the common carotid trunk and both subclavian arteries arise from another common subclavian trunk (Fig. 3.2). In right-sided aortic arch, the arrangement of these three arch vessels is reversed.

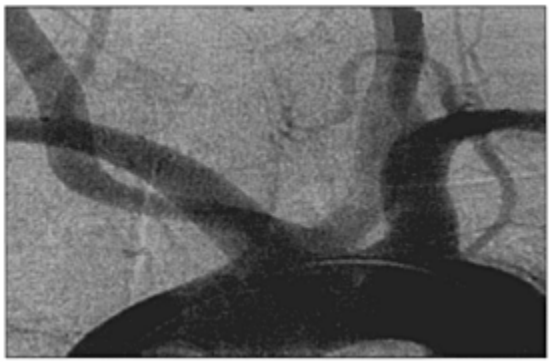

Figure 3.2 Bicarotid trunk.

Innominate artery

The normal innominate artery has a variable length, position and obliquity. When it is short and very oblique, selective catheterization of its branches is difficult and a sidewinder catheter may be needed for access. A marked right anterior oblique projection and the use of roadmapping facilitate catheterization of the right common carotid artery and right subclavian artery.

In 7–27% (depending on the authors), the left common carotid artery arises from the innominate artery.[1–4] The thyroidea ima artery can also sometimes branch from the innominate artery.

Common carotid artery

The brachiocephalic trunk bifurcates into the right subclavian and right common carotid artery at the level of the sternoclavicular joint. The latter is directed upwards in the neck towards the angle of the mandible. The left common carotid artery takes its origin directly from the aortic arch. Both common carotid arteries divide into external and internal carotid arteries at the level of the upper limit of the thyroid cartilage. In cases of anomalies of the aortic arch, variations in the common carotid arteries are seen.[4–7]

The common carotid artery has no branches. Its average length varies from 6 to 12 cm.

In general, it ends at the level of the C3 or C4 vertebra (upper border of thyroid cartilage). At its bifurcation the vessel has a dilation (carotid bulb). Here the tunica media is thin and the tunica adventitia is thick. It contains nerve endings of the glossopharangeal nerve, which functions as a baroreceptor (carotid sinus). The carotid body lies behind the common carotid bifurcation. This reddish brown structure acts as a chemoreceptor and senses any change in the oxygenation and pH of blood.

Subclavian artery

The right subclavian artery is a branch of the innominate artery. The left subclavian artery arises directly from the aortic arch. It is conventionally divided into three parts: the first part is from the origin to the medial border of scalenus anterior muscle, the second part lies behind the muscle and the third part extends up to the anterior border of the first rib. The branches of the subclavian artery are:

- vertebral artery
- internal thoracic artery
- thyrocervical trunk
- costocervical trunk
- dorsal scapular artery,

Aberrant right subclavian artery

The aberrant right subclavian artery is present in 0.2–1.7% of people. It originates from the arch distal to the origin of the left subclavian artery, sometimes by the way of the diverticulum of Kommerell.

Internal carotid artery

The internal carotid artery enters the cranium through the petrous inlet. It forms the carotid siphon (S-shaped part of the internal carotid artery) in the cavernous sinus and then divides into to the anterior and middle cerebral arteries. It also gives rise to the ophthalmic artery

External carotid arteries

The external central artery has no intracranial branches. It gives rise to six branches in the neck:

- superior thyroid artery
- lingual artery
- facial artery
- ascending pharyngeal artery
- occipital artery
- posterior auricular artery.

Vertebral arteries

The vertebral artery originates from the first part of subclavian artery and in most cases it enters the foramen transversarium of the C6 vertebra and ascends to the C2 vertebra. It is divided into four anatomical segments:

- VI: origin to the entrance of the foramen transversarium of C6
- V2: foramen transversarium of C6 to C2
- V3: passes through the transverse foramen and circles around the arch of the atlas to pierce the dura of the foramen magnum
- V4: courses upwards to join the other vertebral artery to form the basilar artery.

Only the V4 segment give rises to branches that supply the brainstem and cerebellum. Atherothrombotic lesions have a predilection for VI and V4 segments. Bony spurs in cervical spondylitis pinch the V2 segment. The symptoms of vertebral artery stenosis are solely dependent on the flow from contralateral vertebral artery, ascending cervical artery, thyrocervical trunk or occipital arteries.

The left vertebral artery is generally larger than the right. A direct origin from the aortic arch is reported in about 6% of cases, and which the left vertebral artery enters the foramen transversarium of C4 or C5 instead of C6. The left vertebral artery may also rarely originate from the left internal carotid artery.

The angiographer must be aware of a direct origin of the left vertebral artery from the aortic arch, since aggressive catheterization of this artery may lead to serious neurological complications.

Intracranial circulation

The internal carotid artery enters the cranium through the petrous inlet. It forms the carotid siphon (S-shaped part of the internal carotid artery) in the cavernous sinus and then divides into the anterior and middle cerebral arteries (Fig. 3.3). It also gives rise to the ophthalmic artery. The anterior cerebral artery is divided into the Al segment (preanterior communicating) and A2 segment (postanterior communicating). Atheromatosus deposits in the Al segment are usually asymptomatic because of good collateral circulation from the anterior communicating artery. Cerebral ischaemia only occurs if the anterior communicating artery is atretic. Occlusion of the A2 segment results in contralateral syndromes. The anterior cerebral artery rarely receives emboli.

The middle cerebral artery (MCA) supplies the lateral surface of the cerebral hemispheres. The proximal MCA (M1 segment) gives rise to penetrating branches termed the lenticulostriate arteries.

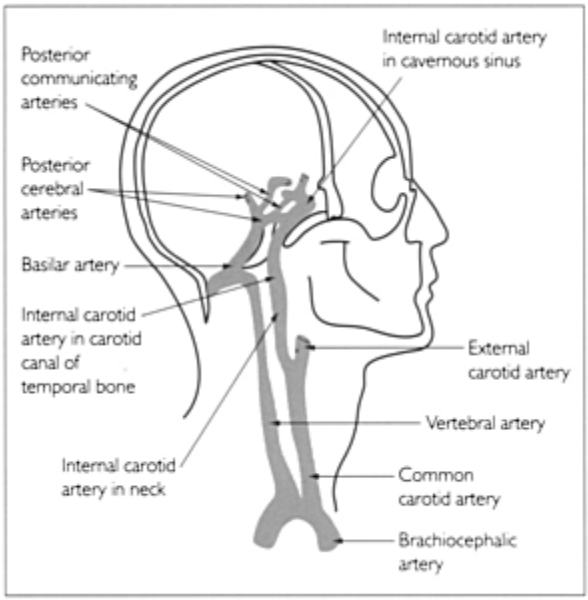

Figure 3.3 Right internal carotid and vertebral arteries.

They supply the putamen, globus pallidus, posterior limb of the internal capsule, adjacent corona radiata, and the body upper and lateral head of the caudate nucleus. In the sylvian fissure the MCA divides into the superior and inferior divisions (M2 branches). The MCA is prone to receiving emboli from plaques in the internal carotid artery or from cardiac sources, resulting in complete or partial MCA syndromes.

Intracranial arterial anastomosis (circle of Willis)

The two vertebral arteries give rise to the posterior inferior cerebellar arteries from the V4 segment before joining together to form the basilar artery. The basilar artery supplies the base of the brainstem and upper part of the cerebellum. The branches fall into three groups: paramedian (seven to ten in number), short circumferential (five to seven), superior cerebellar and anterior inferior cerebellar. The basilar artery bifurcates into two posterior cerebral arteries.

The circle of Willis lies (Fig. 3.4) between the base of skull and the anterior side of the brain. It joins the carotid and the vertebrobasilar arterial circulations.[3–6,8] The internal carotid arteries stand as anterior pillars, and the basilar trunk represents the posterior pillar of this arterial circle. These pillars are joined by a communicating system. The unique anterior communicating artery joins the two anterior pillars. The two posterior communicating arteries join each anterior pillar to the posterior pillar. All arteries of the brain arise from this circle.

This ideal shape is found in approximately 50% of patients. Lazorthes and Gauaze

studied 100 cases and described 22

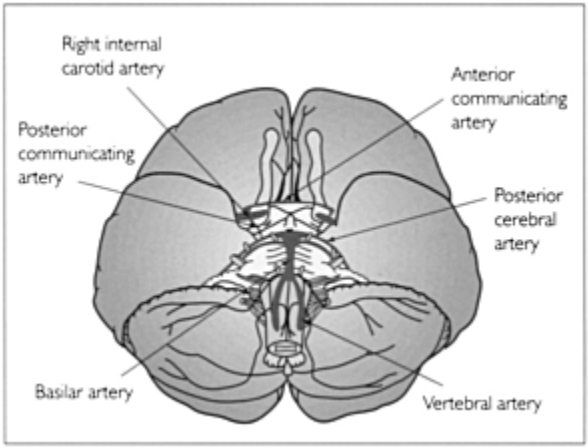

Figure 3.4 Intracranial anastomosis of vessels.

morphological types of circle. Perfect symmetry of pillars was seen in 59%.

An ideal circle of Willis shows equivalent parts of good size (type 1). If all parts are thin, the circle is termed foetal (type 2). In these two types, all the parts are functional and the three pillars give rise to two cerebral arteries each. If the communicating arteries are thin or hypoplasic, the pillars communicating arteries are non-functional, the circle is remain isolated and independent from each other. If the three named precarious (type 7).

Anastomoses of the extracranial vessels

ECA-ECA anastomoses

Anastomoses between the external carotid arteries are transversal and involve bilateral homologous arterial branches which join across the middle line.

ICA-ECA anastomoses

Anastomoses between the internal and external carotid arteries exist in the ear, nose, eye (the ophthalmic-facial anastomosis is important) and nasal fossae. There is no cervi cal anastomosis between the internal external carotid arteries, unless a variant occipital artery arises from the internal carotid artery or the ophthalmic artery has an anastomosis with the external carotid artery.

Pathophysiology of supra-aortic vessels

The hardening of arteries is known as arteriosclerosis and it includes three conditions:

atherosclerosis, arteriolosclerosis and Monckeberg's disease (Fig. 3.5). Atherosclerosis is the most common pathological lesion of supra-aortic vessels. Hypertension, diabetes mellitus, cigarette smoking, dyslipidaemia and genetic preponderance are common risk factors for developing atherosclerosis. Usually these risk factors have a multiplier effect (rather than an additive effect).

Although even severe atherosclerotic lesions of the internal carotid artery may be symptom free, they are the main cause of transient ischaemic attacks or cerebral infarctions. Arteriosclerosis develops principally in the region where shear stress is highest, that is, in the zone of flow separation and associated with whirlpools that form near the lateral wall of the bifurcation, away from the divider, extending into the carotid sinus.

Recently it has been demonstrated that atheromatous plaques removed from carotid arteries frequently harbour

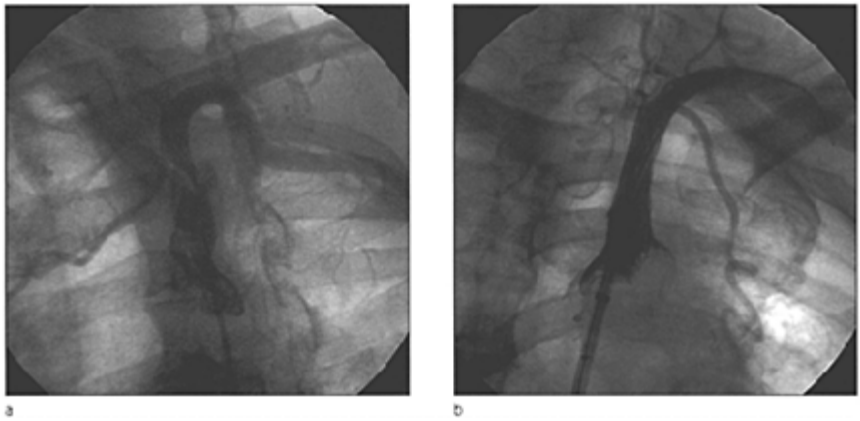

Figure 3.5 (a) Ostial stenosis of the left subclavian artery in a coronary artery bypass graft patient. (b) After stenting of the subclavian artery. Good flow in the vertebral and left internal mammary arteries is seen.

Chlamydia pneumoniae, even though the serology is negative.[9] The cause of this association remains uncertain.Epidemiological studies have also shown a link between foetalgrowth and cardiovascular disease. The risk of carotid arterystenosis is greater for people who had a weight of 3 kg or lessat birth.[10]

Pathogenesis of atherosclerotic lesions

Atherosclerosis begins as fatty streaks and progresses to the formation of fatty or fibrous plaques, which may become calcified and may also be complicated by ulceration or fissure to thrombus formation and antecedent complications of formation (Fig. 3.6). These unstable carotid plaques may lead thromboembolism.[1,11–13]

Atherosclerosis develops as intimal patches in the region of flow disturbances, especially near arterial bifurcations. The endothelial cells secrete vascular endothelium adhesion molecule-1. Lymphocytes and monocytes adhere to the intact endothelial surface at these sites. They migrate into the subendothelial space where they encounter lipid droplets in association with cholesterol esters (low-density lipoproteins). Here the monocytes are transformed into macrophages and phagocytose the lipid-forming foam cells.

Meanwhile, under the influence of endothelial-mediated cytokines, smooth-muscle cells from the media migrate to the intima. Here they are activated, undergo mitotic changes and secrete various growth factors. Under the influence of these growth factors, the plaque increases in size as collagen bundles, elastic fibres and other matrix proteins are laid down in the lesion.[2,7]

In early stage the lesion is referred to as a fatty plaque; as it is collagenized it becomes a fibrous plaque (Fig. 3.7). The plaque may be complicated by calcified deposits, areas of necrosis, thrombosis or haemorrhage, either alone or in variable combinations. Vascularization from the vasa vasorum may also occur.

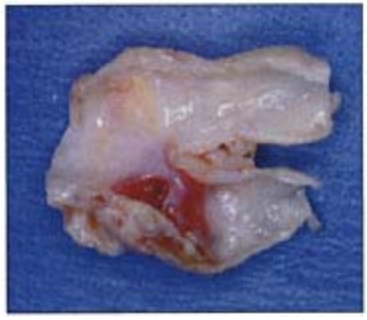

Figure 3.6 Complicated and ulcerated carotid plaque.

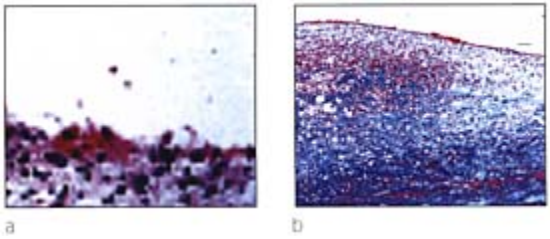

Figure 3.7 (a) Fatty streak showing subintimal monocytic and lymphocytic infiltration. (b) Collagenized fibrous plaque.

Other pathological lesions of the carotid arteries

Takayasu arteritis is a very common condition, especially in south-east Asia, affecting the vessels of aortic arch. It is a panarteritis, characteristically involving the vessels of the

aortic arch. It usually affects young females and presents as fatigue and loss of arterial pulsations in upper limb or episodes of transient ischaemic attacks, vertebrobasilar syndrome or stroke. There is some association with tuberculous infection elsewhere in the body. Corticosteroids may sometimes be helpful in the active stage of the disease. These lesions respond well to balloon angioplasty.

Traumatic lesions due to blunt injuries to the carotid artery are often asymptomatic. Their true incidence is therefore unknown. The majority is associated with motor vehicle accidents, athletic activities such as skiing, ice hockey, kickboxing or fighting, or chiropractic manipulations of the neck.[14,15] Rupture of the carotid arterial wall is the main pathological lesion. It may be intimal, parietal/paraparietal dissection or haematoma, or a combination of these. Long-standing lesions show evidence of healing with the presence of scar tissue. Sometimes partial or complete obstruction of the arterial lumen may be present.

Supravalvular aortic stenosis syndrome (SVAS) and Williams-Beuren syndrome (WBS) are closely linked genetic disorders. In both conditions the mutation is located on locus 7Q1 1.23, and is linked to the elastin gene. SVAS is inherited as an autosomal dominant disorder. WBS is the result of a submicroscopic deletion whose phenotypes affect the connective tissue of various organs. The aorta, pulmonary arteries, carotid arteries and branches show marked thickening due to intimal proliferation and fibrosis. There is marked stenosis or obliteration of the lumen of the carotid arteries.

Tumours of the carotid body (glomus tumour) are well documented. Primary tumours of the carotid arteries are extremely rare. Recently an intramural sarcoma of the carotid artery has been reported.[16]

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4
Angiographic anatomy of the craniocervical arterial system

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Introduction

This chapter discusses features of the craniocervical arterial system relevant to assessment and treatment with carotid stenting. Angiography of the craniocervical arteries begins with exploration of the aortic arch (AA).

Aortic arch

The AA presents three main segments: the ascending aorta, the aortic arch and the aortic isthmus. [1–3]

The thoracic aorta follows an oblique right anterior to the left posterior direction. Therefore, the aortic arch and its branches are studied by bolus injection into the ascending thoracic aorta with a left anterior oblique projection (Figs 4.1 and 4.2).

The three main craniocervical branches arise off the horizontal portion of the aorta: the innominate artery (IA), the left common carotid and the left subclavian. Usually the aortic arch is found above the carena at the T4 vertebral level.

With advanced age or the presence of anatomical variants, the typical disposition may vary. Visualization of the AA anatomy may be useful in choosing the appropriate catheter curve for selective angiographic exploration.

Innominate artery (brachiocephalic trunk)

The IA is the largest branch of the arch of the AA. The second right costal cartilage serves as a skeletal reference level. It extends for 2.5 cm until the origin of the right common carotid artery (CCA), which follows an anterior direction. The right subclavian artery (ScA) originates posteriorly from the brachiocephalic trunk. Its proximal branches are the vertebral artery (VA), about 4 cm from the AA (its origin is positioned at the postero-superior circumference of the ScA), the internal mammary artery and the thyrocervical trunk, including most of the time the deep and the ascending cervical branches (Fig. 4.3).

Origin of the left common carotid artery

The left CCA is longer and often larger than the right. The origin of the left CCA lies anterior to the line connecting the IA and left ScA origin, which may be useful to consider during angiographic exploration.

The CCA arises from the highest part of the arch of the AA, just next to the IA and at a small distance from the left ScA.

Left subclavian artery

The left ScA is the last craniocervical trunk of the AA. It gives origin to the same vessels as described above for the right ScA.

Frequent anatomical variants of craniocervical branches are: .[1–3]

- the right ScA, which may arise as a separate trunk off the aortic arch; in such cases it may be either the first, second, third or even the last craniocervical of the AA branch, or the ‘arteria lusoria’, found in 1% of the population (Fig. 4.4)

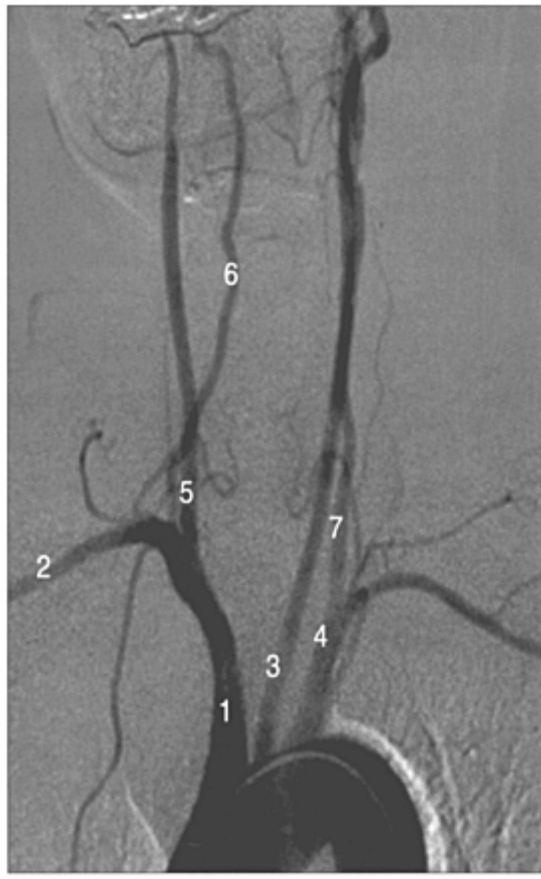

Figure 4.1 Selective injection into the aortic arch in left anterior oblique projection demonstrating its major branches. 1 Brachiocephalic trunk (innominate artery); 2: right subclavian artery (ScA); 3: left common carotid artery (CCA); 4: left ScA; 5: right CCA; 6: right vertebral artery (VA); 7: left VA.

- a common origin for the IA and the left CCA, in about 10% of the patients, called the 'bovine variant'. Selective angiography of the left CCA may require the use of more angulated catheter shapes, such as Bentson or Simmon curves, to gain access (Fig. 4.5)
- the left VA may arise directly off the aortic arch (4%), typically between the left CCA and ScA origins.

Vertebral artery and vertebrobasilar system

The VA is the first branch of the ScA on both sides, but it may arise from the

thyrocervical trunk. Typically the left VA is dominant. The cervical portions of the VA resemble each other so closely that one description will apply to both sides.

The VA may be divided into the following four parts.

- The first segment (VI) or pretransversal segment runs upward backward and medially. Its orientation may distinguish it from the right carotid artery.
- The second segment (V2) or transversal segment runs straight upwards through the foramina of the transverse process of the cervical vertebrae, usually entering at C6, but frequently seen between C4 and C7 (see Table 4.1). V2 first runs in a straight line until it reaches the transverse process of the atlas, where it becomes the third part (V3) or axoatloidal segment, circling around the transverse process of C1. V3 becomes the end segment (V4) intradural part of the VA at the dural penetration point, often accompanied by a slight reduction in the vessel's diameter. V4 then reaches medially to join the opposite VA at the vertebrobasilar junction (VBJ). Atherosclerotic lesions of the distal VA are typically found at the V4 segment.

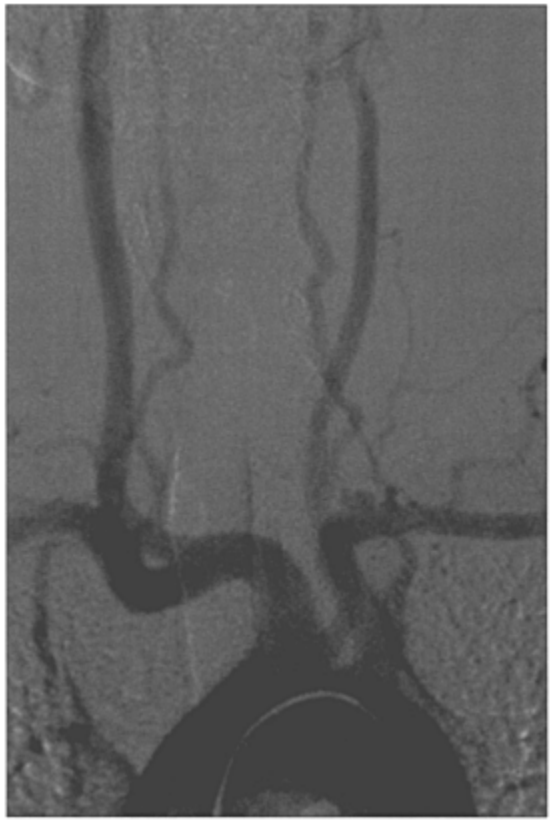

Figure 4.2 Aortic arch in left anterior oblique projection in a 78-year-old woman demonstrating modifications due to age with tortuous vessels.

Table 4.1 Vertebral artery penetration at cervical level.[1]

Level	*Frequency (96)*
C7	5
C6	87
C5	7
C4	1

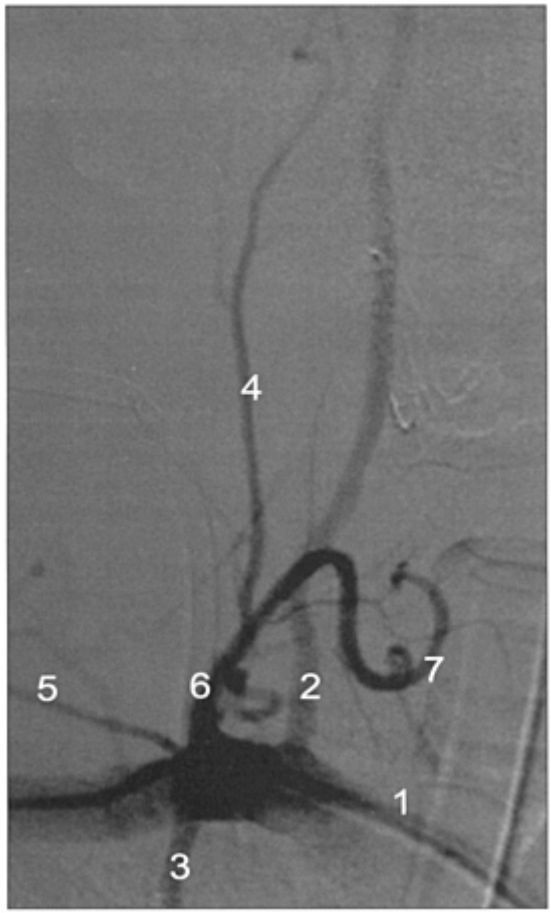

Figure 4.3 Selective injection into the right subclavian artery (ScA) demonstrating its main branches. 1: Right ScA; 2: right vertebral artery; 3: internal thoracic artery; 4: superficial cervical artery; 5: deep cervical artery; 6: thyrocervical trunk; 7: inferior thyroid artery

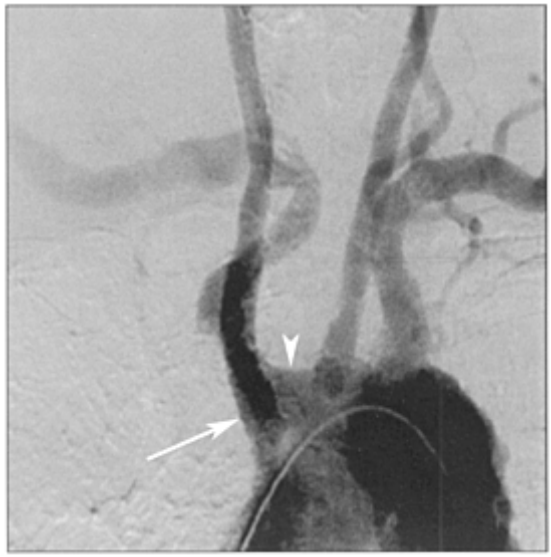

Figure 4.4 Aortic arch in left anterior oblique projection showing an arteria lusoria. The right subclavian artery (arrowhead) has its origin after the right common carotid artery (arrow).

- One branch of the VA is the posterior inferior cerebellar artery (PICA). It is the largest, typically arising from the middle portion of V4. Close to the VBJ, the spinal branches of the anterior spinal artery may arise from V4.
- The VA may end in a 'PICA form' (20%), which is more frequent on the right side (Fig. 4.6).

Vertebrobasilar system

This is shown in Fig. 4.7. The basilar artery is a single trunk formed by the medial fusion of both vertebral arteries. It ends by dividing into the two posterior cerebral arteries, which participate in the circle of Willis with the precommunicating segment (PI). The posterior cerebral artery may be divided in three segments: PI, the precommunicating segment, which receives the posterior communicating from the internal carotid; P2, the cisternal segment; and P3, the hemispheric segment. The transition point from P1 to P2 is at the level of the posterior communicating artery (PCoA).

Furthermore, the basilar artery gives rise to:

- the anterior inferior cerebellar artery (AICA), supplying the anteroinferior part of the cerebellum
- the superior cerebellar artery (SCA), supplying the anterosuperior part of the cerebellum.
- numerous perforating brainstem branches for both sides.

Note that there may exist a rich anastomotic collateral supply between the branches of the PICA, AICA and SCA.

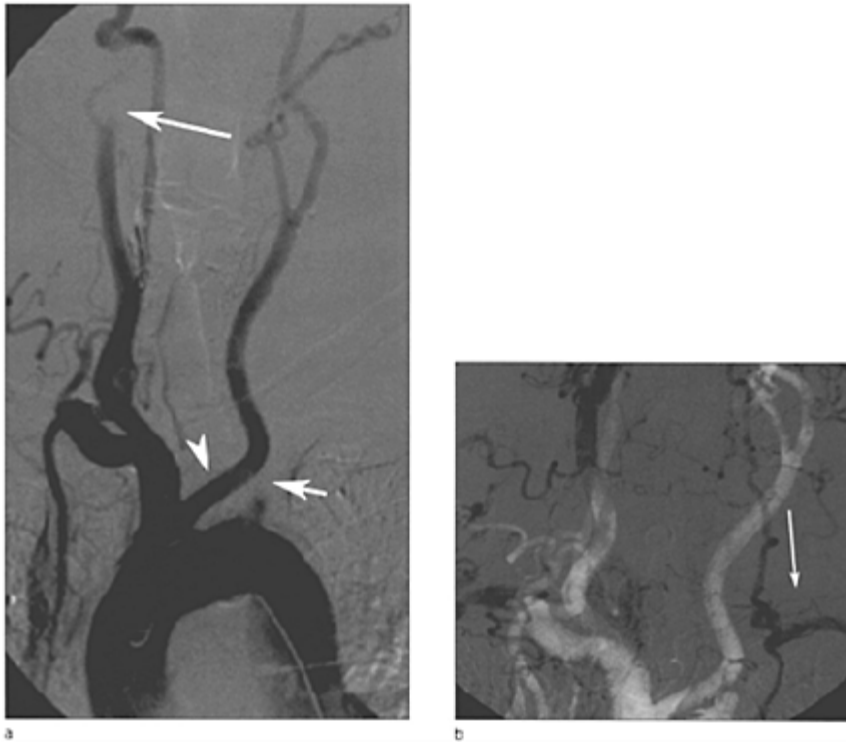

Figure 4.5

(a) Angiogram of the aortic arch in left anterior oblique projection showing a common trunk for both common carotid artery (arrowhead) and a thrombosis of the right internal carotid artery (long arrow) and the left subclavian artery (ScA) (short arrow).

(b) Late arterial phase demonstrating a reverse flow of the left vertebral artery (arrow): ScA steal syndrome.

Carotid system

Common carotid artery and bifurcation

The CCA divides into the external (ECA) and internal (ICA) carotid arteries. The mean diameter of the CCA varies from 7 to 9 mm.[1,3–5]

The carotid bifurcation is the preferential location of carotid atheromatoses. The development of the atheromatosis often begins at the posterolateral circumference of the

carotid bulb, evolving within an area 2 cm above and 1 cm below, on the CCA.

The CCA bifurcates at the level of C4, with a variation in level between C6 and C2 (Table 4.2)

Angiographically, oblique anterior and lateral projections provide the best views of the CCA bifurcation (Fig. 4.8).

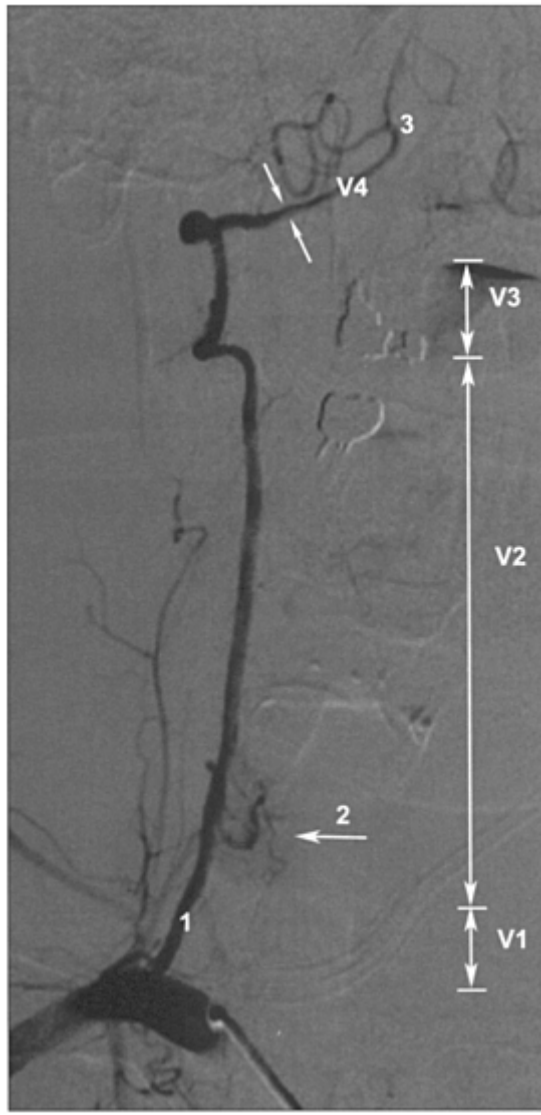

Figure 4.6 Selective injection into the right subclavian artery demonstrating the different segments of the right VA which terminates as a posterior inferior cerebellar artery (PICA). 1: Right vertebral artery (VA)

divided into four segments, V1-V4. Arrows show the dural junction between V3 and V4; 2: vascular blush at thyroid gland (arrow); 3: ending of VA as PICA.

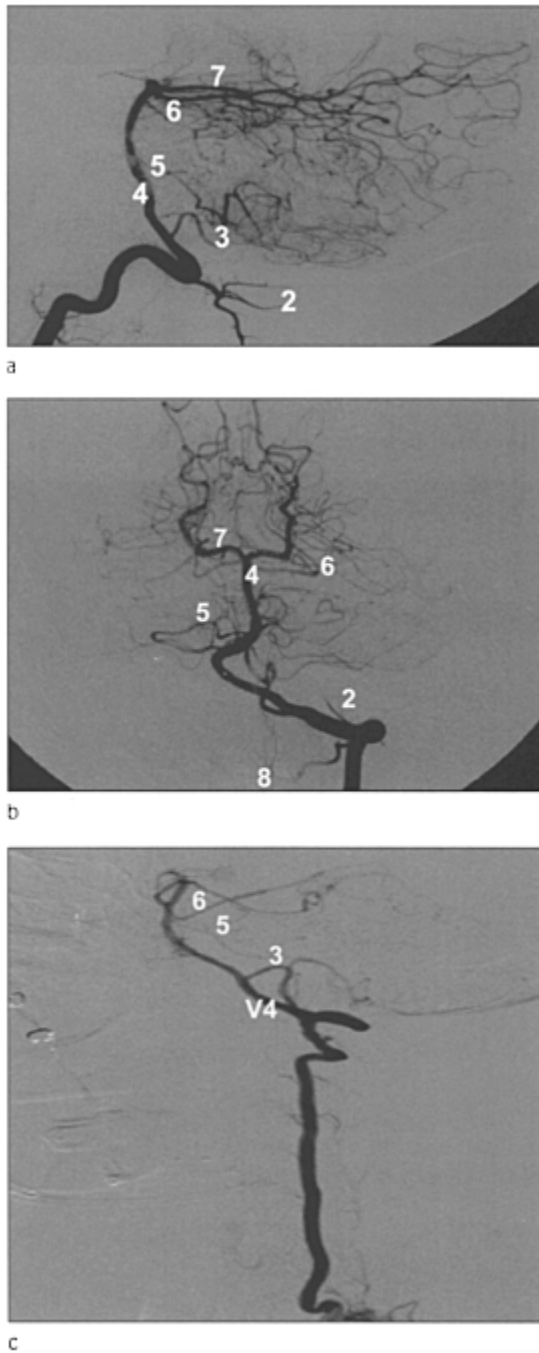

Figure 4.7 Angiograms of the left vertebral artery (VA): (a) lateral and (b)

anterior views. 1: Left VA; 2: posterior meningeal artery; 3: posterior inferior cerebellar artery; 4: basilar artery; 5: anterior inferior cerebellar artery; 6: superior cerebellar artery; 7: posterior cerebral artery; 8: anterior spinal artery.

Table 4.2 Carotid bifurcation level.[1]

Level	*Frequency (96)*
C1–2	0.3
C2–3	3.7
C3–4	34.2
C4–5	46.3
C5–6	13.0
C6–7	0.15

External carotid artery

The ECA divides into eight main branches (Fig. 4.9) and decreases in size over its course.[4,6,7] Three of them are anterior: the superior thyroid, lingual and facial arteries. Three of them posterior: the occipital artery, posterior auricular artery and ascending pharyngeal artery. The end branches of the ECA are the internal maxillary and superficial temporal arteries.

ECA branches that typically develop a collateral supply to cerebral vessels are branches from the internal maxillary artery (to the ICA system) or from the occipital artery (to the VA system)

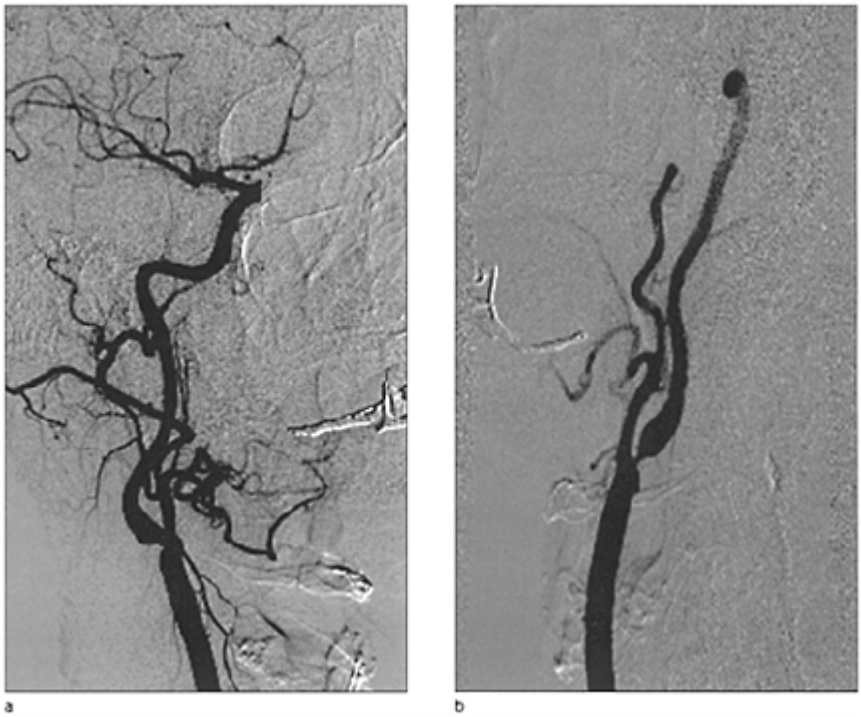

Figure 4.8 Selective injection into the right common carotid artery on (a) anterior oblique right and (b) lateral projections, showing a right internal carotid artery stenosis that can be better appreciated on the lateral view.

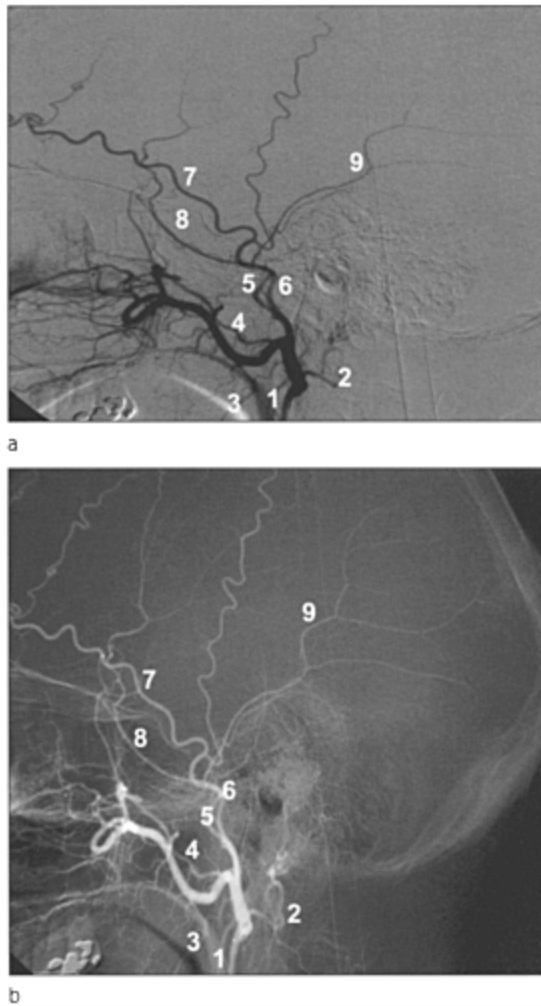

Figure 4.9 Selective injection of the right external carotid artery on lateral view (a) with and (b) without subtraction. 1: Superior thyroid artery; 2: occipital artery; 3: facial artery; 4: lingual artery; 5: posterior auricular artery; 6: ascending pharyngeal artery; 7: superficial temporal artery; 8: maxillary artery; 9: middle meningeal artery

Internal carotid artery

The ICA may be divided into four portions (Fig. 4.10).[1,2,4,5]

- Cervical portion: from its origin to the skull base. The average diameter of the cervical ICA is 6 mm. If the bulb is the preferential location of stenosis due to atheromatotic disease, the middle cervical portion is typically involved in fibromuscular dysplasia

and the upper cervical portion is often affected by dissection.

- Petrous portion: the ICA enters the osseous carotid canal in the petrous portion of the temporal bone.
- Cavernous portion: in this part the ICA is situated within the cavernous sinus. It is the second preferential location of ICA atheromatosis, often involving dilatation, forming a degenerative aneurysm.
- Intracranial portion: having perforated the dura mater on the medial side of the anterior clinoid process the ICA has an intradural course and divides at its bifurcation into the anterior cerebral artery (ACA) and the middle cerebral artery (MCA).

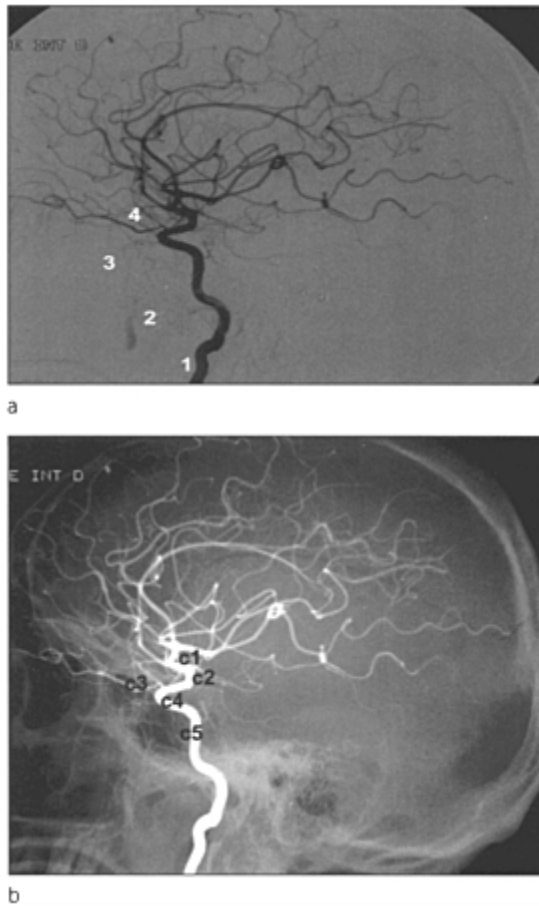

Figure 4.10 Selective injection of the right internal carotid artery (ICA) on lateral view. (a) Collaterals of the ICA. 1: internal carotid artery; 2: meningohypophyseal trunk; 3: ophthalmic artery (OA); 4: anterior choroidal artery; 5: posterior communicating artery (PCoA). (b) The five segments of the cavernous and cisternal ICA: C5 and C4 are separated by the meningohypophyseal trunk, C3 and C2 with the OA,

and C2 and C1 with the PCoA. 1: Cervical portion; 2: petrous portion; 3: cavernous portion; 4: cerebral portion;

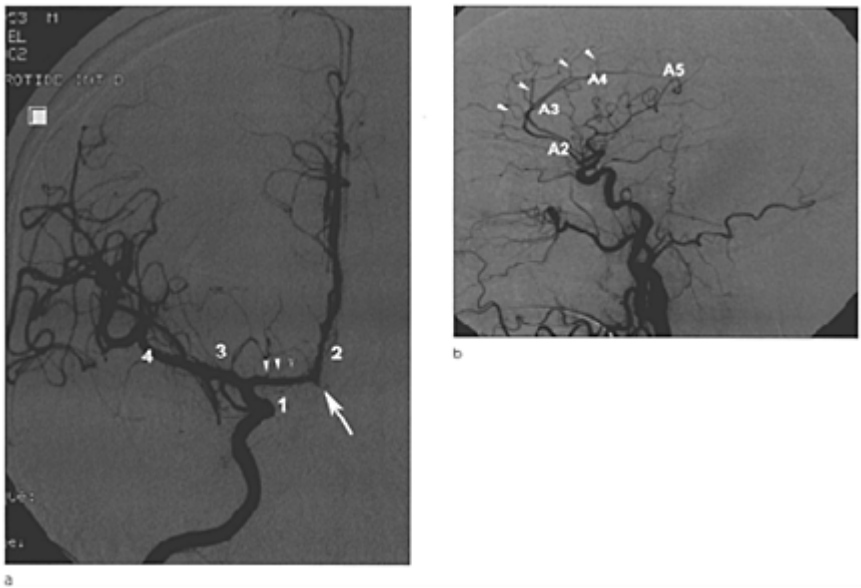

Figure 4.11 Intracranial right internal carotid artery. (a) Anteroposterior view. 1: Al or precommunicating segment; 2: A2 or pericallosal segment (arrow: anterior communicating artery; arrowhead: internal perforating artery); 3: Ml ; 4: M2 or insular segment. (b) Lateral view demonstrating anterior cerebral artery segments and callosomarginal arteries (arrowheads).

The cavernous and cerebral portions are divided into five segments from C5 to C1. Intradural atheromatosis is rare and, if present, concerns the distal ICA or MCA.

The three small but significant branches of the ICA are the ophthalmic artery (OA), the PCoA and the anterior choroidal artery (AchoA). The OA is the first intradural branch of the ICA and indicates the transition to the intradural ICA. ECA and ICA territories may be connected through ECA and OA. The AchoA provides the arterial supply to the internal capsule and cerebral peduncle and may lead to hemiplegia if occluded. It arises from the internal carotid (Cl segment), usually 3 mm above the PCoA (see Chapter 5) origin and 5 mm before the end of the ICA.[1–3,6,8]

The MCA is the largest branch of the ICA. It runs at first laterally in the lateral cerebral or sylvian fissure and then backwards and upwards on the insular surface, where it divides into a number of branches which are distributed to the lateral surface of the cerebral hemisphere. The ACA is the medial ending branch of the internal carotid. It passes forwards and medially across the anterior perforated substance (Fig. 4.11).

In cases of thromboembolism the flow conditions and the anatomy of the ICA

bifurcation favour emboli engaging the MCA.

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5
What you really need to know about the intracerebral circulation: the importance of collateral circulation assessment

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Introduction

The balance of cerebral blood flow haemodynamics in a patient with severe carotid artery disease depends not only on the circle of Willis but also on the state of the extracranial and intracranial collateral circulation.

Angiographic identification of the collateral supply to the cerebral circulation may be of interest when assessing the internal carotid artery (ICA) or vertebral artery (VA).

The presence of collaterals is associated with a lower risk of hemispheric stroke and transient ischaemic attack, calculated both in the long term and perioperatively.[1]

This chapter discusses angiographic collateral circulation analysis. The collateral networks between the interhemispheric circulation, the right and the left ICA, the ICA and vertebrobasilar system (VBS), the external carotid artery (ECA) and the ICA, the ECA and VBS, and in the intrahemispheric circulation the anterior cerebral artery (ACA)-middle cerebral artery (MCA), ACA-posterior cerebral artery (PCA) and MCA-PCA will be discussed (Fig. 5.1).[1–8]

The circle of Willis

The circle of Willis is regarded as the major source of collateral flow in patients with severe carotid artery disease. Many studies have demonstrated the importance of the circle of Willis in maintaining a low risk of low-flow infarct.[4,9–13]

It is composed of three major cerebral vessels (right ICA, left ICA and basilar trunk) and anastomotic branches: an anterior communicating artery (ACoA) and two posterior communicating arteries (PCoA) (Fig. 5.2).

An ACoA lumen of at least 1 mm in diameter may ensure sufficient continuous supply to the vascular territory.

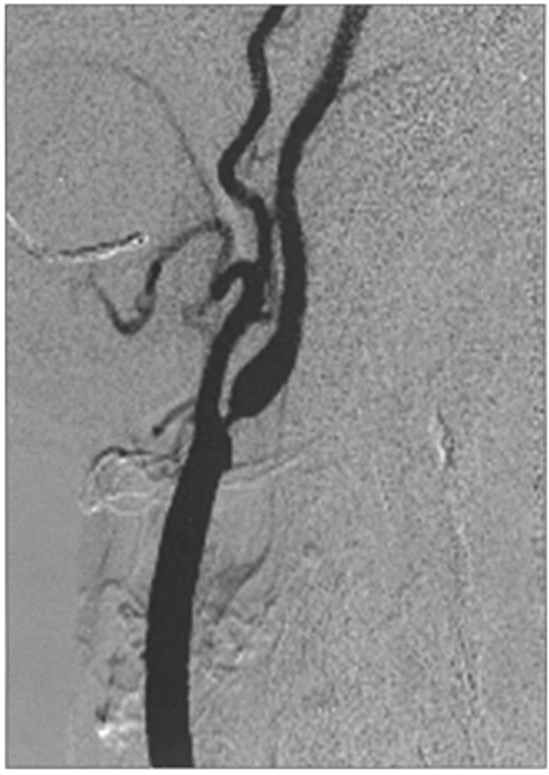
Anterior communicating artery

The ACoA forms the anterior part of the circle of Willis and connects the right and the

left ICA with two ACA with a single trunk (in 60% of the population), or it may be composed of two trunks (30%) (Fig. 5.3).

It is the most important location for intracranial cerebral aneurysms.

Absence of the ACoA is rare.



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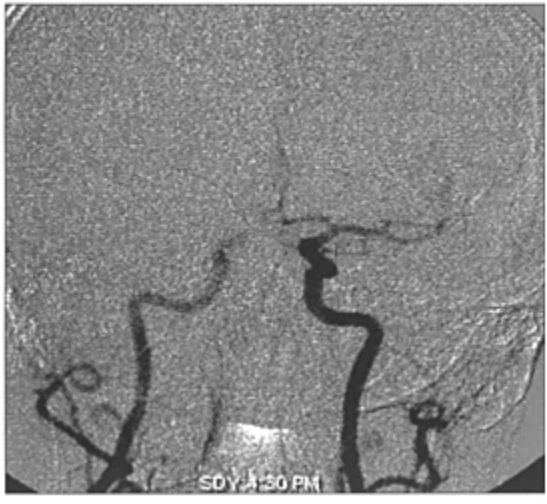

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Figure 5.1 (a) Injection into the aortic arch in a patient with a carotid stenosis: (b) delay of inflow on the right side.

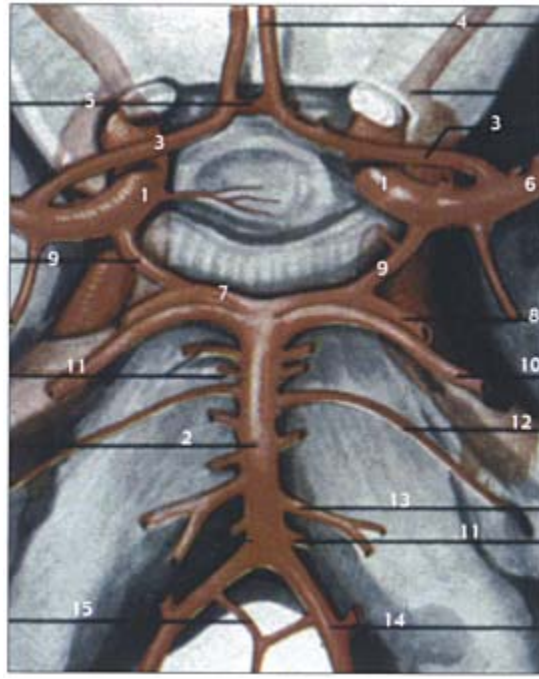


Figure 5.2 Scheme of the circle of Willis. 1: internal carotid artery termination; 2: basilar trunk; 3: Al segment; 4: A2 segment; 5: anterior communicating artery; 6: middle cerebral artery; 7: P1 segment; 8: P2 segment; 9: posterior communicating artery; 10: superior cerebellar artery; 11: perforating branches; 12: internal auditory artery; 13: anterior inferior cerebellar artery: 14: left vertebral artery and posterior inferior cerebellar artery; 15: anterior spinal artery.

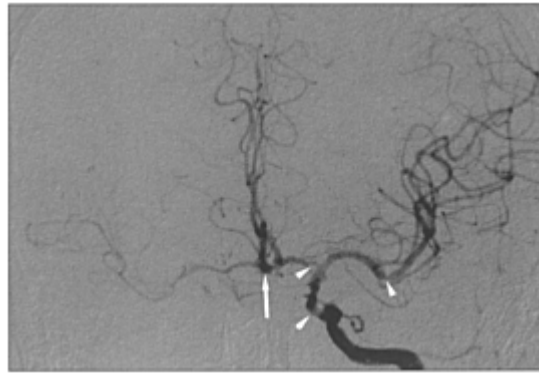
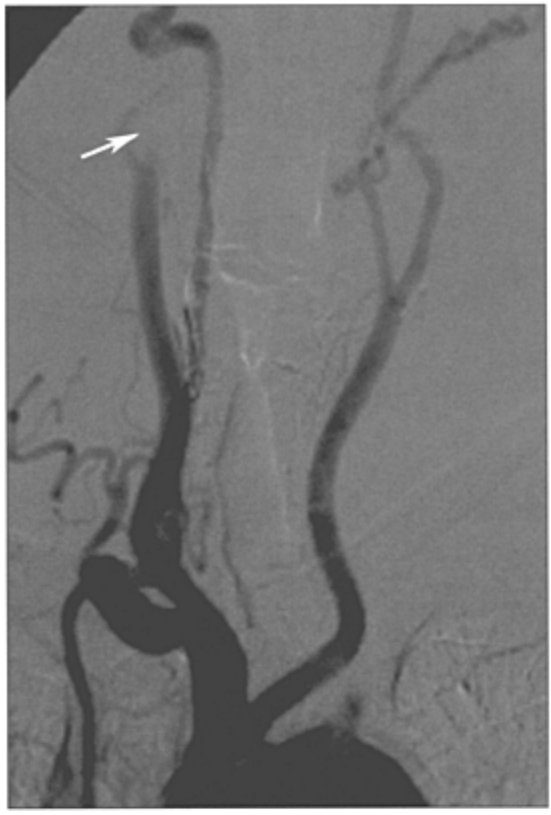


Figure 5.3 Selective injection of the left internal carotid artery (ICA) in a 68-year-old patient with right ICA thrombosis, showing cross-flow to the right middle cerebral artery and anterior cerebral artery using the anterior communicating artery (arrow). Note stenosis on the left ICA siphon, proximal A1 segment and M1 segment (arrowhead).



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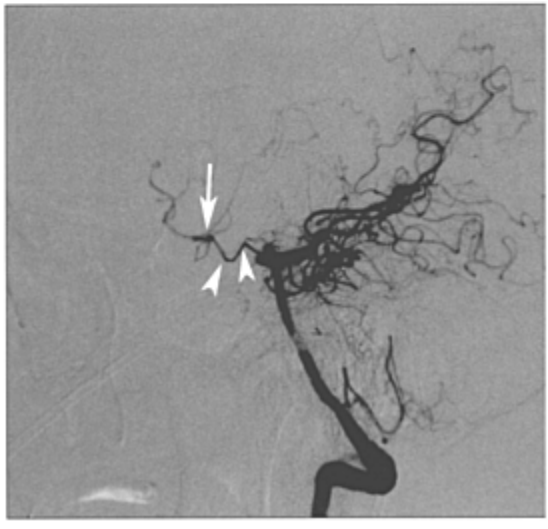

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Figure 5.4 (a) Selective injection in the aortic arch showing a thrombosis of the internal carotid artery (arrow). (b-d) Intracranial angiograms showing a right posterior communicating artery (arrow) which supplies the right carotid siphon (arrowhead). Global intracranial study showing right A1 and M1 segments (arrowhead).

Posterior communicating artery

The PCoA is a lateral branch of the circle of Willis and connects the anterior (ICA) to the posterior circulation (VBS). It arises off the internal carotid, and anastomoses with the PCA, distally to P1.

The PCoA runs posteriorly above the oculomotor nerve (IIIcn).

Variations in the size of the PCoA are found very frequently in the general population, possibly because of its evolution. The 'foetal form' of PCoA (20% of population) presents as a large PCoA with an associated hypoplastic PI segment (Fig. 5.4). Absence of the PCoA is exceptional.

Abnormal anastomoses

Rarely, below the circle of Willis, some abnormal anastomoses may directly connect the carotid to the vertebrobasilar system. These derive from the persistence of embryonic arteries. There are four such anastomoses: the trigeminal, acoustic, hypoglossal and proatlantal intersegmental arteries (Fig. 5.5).[11]

ICA-ECA anastomosis

This kind of anastomosis depends on the meningeal arteries (Fig. 5.6).[5,10,11,14] The ophthalmic artery (OA) anastomoses the ECA and ICA by the way of the meningeal branch of the internal maxillary or ascending superficial temporal artery. The OA may arise from the middle meningeal artery. The presence of an OA arising from the middle meningeal artery is a frequently encountered variant.

The others meningeal arteries connecting the ICA and ECA are:

- carotid-tympanic artery (in the petrous bone)
- pterygoid and vidian arteries (in the cavernous sinus)
- meningohypophyseal trunk (sella)
- anterior and posterior ethmoidal arteries (in the nasal fossae).

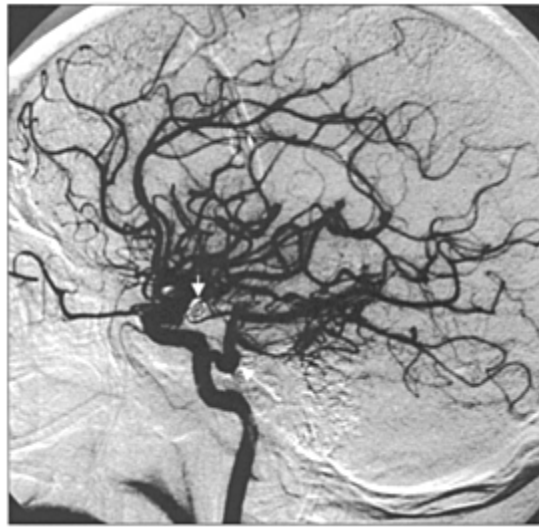

Figure 5.5 Selective injection into the left internal carotid artery (lateral view projection) showing a persistent trigeminal artery (arrow) representing the most common primitive carotid-basilar anastomosis. The anomaly was discovered incidentally in this 45-year-old man treated for a posterior communicating artery aneurysm (arrowhead).

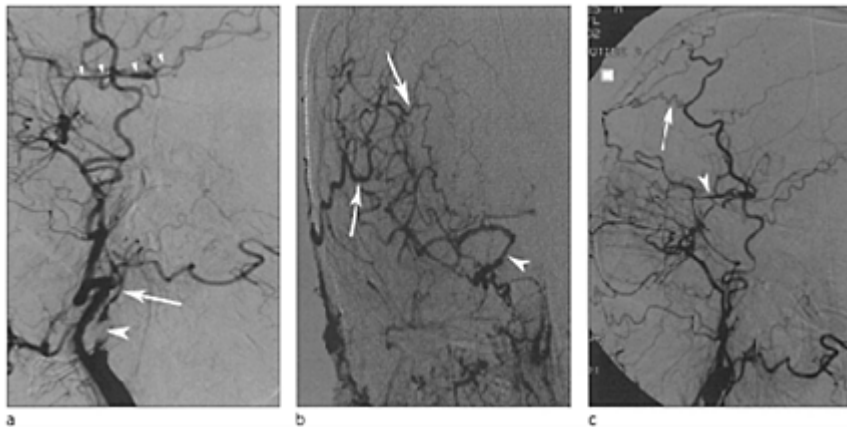

Figure 5.6 Right carotid angiogram showing (a) a severe stenosis of the internal carotid artery (ICA) (arrowhead); little flow is seen in the ICA (arrow); the carotid siphon is filled by reverse flow via the ophthalmic artery (OA) (arrowhead). (b, c) Intracranial angiograms showing meningeal anastomoses (arrow) and the OA (arrowhead) supply both contributing to the vascularization of the right hemisphere.

ECA-VBS anastomosis

The muscular branch of the occipital artery anastomoses the deep and ascending cervical muscular branches to the occipital artery or the ascending pharyngeal artery branches of the ECA, thus creating the Bosniak node, which may supply the intracranial circulation if there is an occlusion of the ICA or VBS.[10,11,14]

VBS-VBS anastomosis

Radicular branches may supply the VBS if there is thrombosis of the VA. Another collateral pathway in the VBS is between the posterior inferior cerebellar artery (PICA) and superior cerebellar artery (SCA), as may be seen in cases of basilar artery stenosis.[10,11]

Intraterritorial anastomoses

These anastomoses between two different vascular territories are ACA-PCA with the pericallosal artery and occipital artery, the anterior and posterior choroidal arteries, and MCA-PCA with branches of the temporal arteries.[10,11,14]

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PART III
Imaging techniques

6
Geometric effects of carotid stents

J Berkefeld, JB Martin, N Tanaka, J Théron, S Rohde and DA Rüfenacht

Introduction

Metallic stents are being increasingly used for the endovascular reconstruction of stenotic carotid arteries. Compared with conventional balloon angioplasty, stents provide a more reliable expansion of the vascular lumen and fixation of the plaque material, and larger case series have demonstrated the technical feasibility and safety of carotid stenting.[1–5] To avoid stent deformations due to external compression, the use of self-expanding stents with radial shape memory and elasticity is mandatory at the superficially located carotid bifurcation.[5] Self-expanding stents show chronic radial forces to expand to their nominal diameter. Most of the atheromatous carotid stenoses involve the extracranial bifurcation, and carotid stents frequently have to bridge the bifurcation ranging from the common carotid artery (CCA) through the carotid bulb into the internal carotid artery (ICA).[2,6,7] Bridging the bulb and the bifurcation requires apposition of the stent to changing vessel diameters.[7] Beneath these radial effects interactions between the artificial geometry of the prosthesis and the longitudinal course of the biological vessels have to be expected. Therefore, the purpose of this work was to compare the geometric effects of different types of self-expanding carotid stents.

According to cardiological concepts, radial stent-assisted expansion of the stenotic vessel lumen was described as luminal gain.[7] The measurement of the changes in the percentage of stenosis was supplemented by qualitative assessment of the apposition between the stent struts and the vessel wall.

To quantify the bifurcational and stent geometry, a measurement system was developed, adapted to digital subtraction angiographic projection images of the carotid bifurcation.[8] The angulation between the CCA and ICA was described as the CCA-ICA angle between the extended CCA midaxis and the midaxis of the initial ICA segment. The intersection of both axes was located at the level of the external carotid artery origin. In addition, tortuosity of the ICA course was measured as the maximal deviation of the ICA (offset) perpendicular to the extended CCA axis (Fig. 6.1). The evaluation system was applied to clinical carotid stent cases as well as to experiments with simplified silicone models of the carotid bifurcation.

The Wallstent was the first self-expanding stent that was widely used for endovascular treatment of carotid stenoses. A braided meshwork of filaments from a cobalt-chrome alloy extends through the whole length of the prosthesis (Fig. 6.2). The moderate radial expansion forces depend on the crossing angle of adjacent filaments. The stent foreshortens markedly during deployment, which is achieved by retrieval of a covering sheath. The unsegmented longitudinal course of the filaments limits the longitudinal

flexibility of the Wallstent and provides certain longitudinal forces. To enhance radial force and longitudinal flexibility, several manufacturers have developed self-expanding carotid stents that are laser-cut from a nickel-titanium alloy (nitinol) tube. The stents are composed of ring-like zigzag segments that are only partly connected with each other (Fig. 6.2). To achieve longitudinal flexibility the unconnected parts of the stent modules can separate at the outer and overlap partly at the inner circumference of a vascular curve. Similarly to the Wallstents, self-expanding nitinol stents are folded on a catheter and retained by a covering sheath. Owing to thermic memory effects they gain their original size and shape after retrieval of the sheath at body temperature. Nitinol stents show only minimal foreshortening during deployment and can be placed exactly on the point.

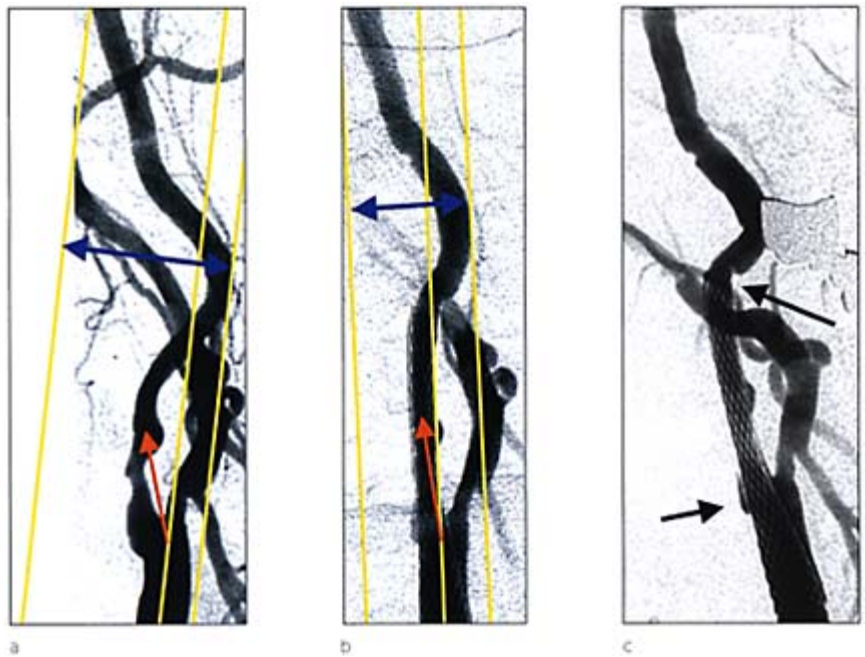

Figure 6.1 Clinical application of a measurement system for quantitative description of the geometry at the carotid bifurcation. (a) Digital subtraction angiographic projection image of a carotid bifurcation with a 70% atheromatous stenosis at the internal carotid artery (ICA) origin and a moderately tortuous course of the vessel. The common carotid artery (CCA)-ICA angle (red arrow) is measured between the extended midaxis of the CCA and the axis of the initial ICA segment, which is terminated by the upper limit of the stenosis. The ICA offset indicates the maximal deviation of ICA tortuosities perpendicular to the extended CCA midaxis in a range between the bifurcation and the skull base (blue arrow). (b) Implantation of a Carotid Wallstent straightens the bifurcation and ICA tortuosity, and reduces angle and offset. (c) Longitudinal stent forces induce slight kinking above the

distal stent termination (upper arrow) which resolve spontaneously Note also minimal stent recoil at the former maximum of the fibrous plaque and a lack of apposition between stent struts and the bulging of the carotid bulb (lower arrow).

Geometric effects in clinical Wallstent cases

In 40 cases with high-grade atherosclerotic carotid stenoses, the same angiographic projection of the carotid bifurcation was evaluated with the measurement system before and after implantation of a self-expanding Carotid Wallstent (Schneider-Boston Scientific, Galway, Ireland). All stents bridged the bifurcation. Stenting reduced the mean degree of stenosis from 86 to 6%, measured according to the NASCET (North American Symptomatic Carotid Endarterectomy Trial) criteria, and no residuals above 30% were observed. However, on looking at the details, suboptimal recanalization results occurred frequently with the use of Wallstents.[6] Stent recoil despite adequate postdilatation was observed in 16 cases with residual reduction of the vessel lumen between 5 and 29%. Areas with a lack of apposition between stent struts and the arterial wall were observed in 21 out of 40 bifurcations (52%), especially at the bulging of the carotid bulb (Figs 6.1a, c).

The angulation of the ICA origin in relationship to the CCA axis decreased statistically significantly, from a mean of 18° before to 7° after stenting. The maximal deviation of ICA tortuosity perpendicular to the CCA axis (offset) was also significantly reduced from a mean of 24 mm to a mean of 18 mm (Wilcoxon test, $p < 0.01$). Images obtained after stenting showed a straightening of the bifurcation and ICA tortuosity (Fig. 6.1a, b). In six cases longitudinal stent forces induced kinks in the artery above the distal stent termination, which resolved spontaneously after retrieval of the guiding catheter or over time, within the next few days (Fig. 6.1c). Despite of a limited compliance between Wallstents and vessel geometry, clinical outcome analysis proved no evidence of increased neurological event or restenosis rates.[6] The 30 day stroke and death rate was 2.5%. During follow-up periods of between 12 and 37 months (mean 24 months) only one patient developed a 70% in-stent restenosis by marked intimal hyperplasia at the former maximum of the plaque, which was successfully redilated and has since been stable. Another patient with normal patency of the stent had an ipsilateral major stroke, probably due to a cardiac embolus.

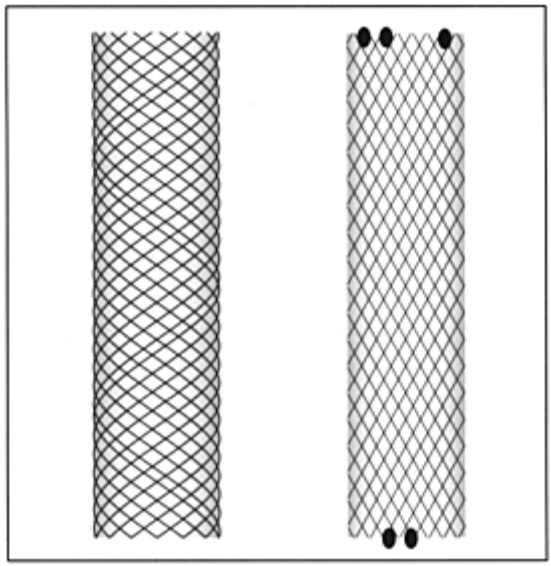

Figure 6.2 Two types of carotid stent design (digital radiograph). The Wallstent (left) is braided from continuous helical filaments. The Zilver stent as a prototype of a segmented Nitinol stent (right) shows partly unconnected, ring-like zigzag segments cut from a nickel-titanium tube.

Model experiments: Wallstent versus segmented Nitinol stent

After clinical experiences with the geometric effects of Wallstents, the geometric effects of different stent types were studied, with silicone models of the carotid bifurcation (Elastrat, Geneva, Switzerland) exhibiting elastic wall properties similar to human arteries.[9] The models were fixed in an acrylic box and connected to an artificial circulation to make the silicone arteries suitable for angiographic examination. The bifurcation was constructed with a defined CCA-ICA angle and ICA offset, and a circumscribed curve counterbalanced the initial angulation to continue the course of the CCA up to a level of 6 cm (corresponding to the entrance into the skull base), where the ICA was fixed within a burr hole of a Plexiglas sheath. Behind the burr hole the ICA deviated with a 90° curve. After resolution of the fixation at that level, longitudinal extensions of the ICA above the level of the Plexiglas sheath could be demonstrated (Fig. 6.3). The luminal diameter of the ICA was 6 mm and the diameter of the CCA was 8 mm. Self-expanding stents with a nominal diameter of 9 mm and a length of 8 cm were implanted into the model and should cover the bifurcation as well as the curve. In a first set of experiments, Easy Wallstents and Carotid Wallstents (Schneider-Boston Scientific, Galway, Ireland) were implanted as examples for braided stent designs with different longitudinal elasticity. The Wallstents were compared with a Zilver Stent (William.

Cook, Bjaeverskov, Denmark), which was used as a prototype of a segmented Nitinol stent. The Easy Wallstent showed a marked straightening of the bifurcation with clear reductions in angle and ICA tortuosity. The Carotid Wallstent transformed the circumscribed curve of the model into a larger C-shaped bend (Figs 6.3 and 6.4).

In a second set of experiments a thin wall 60° model made of two-layer silicone with a wall elasticity similar to an atherosclerotic human artery was used. Zilver stents (9–80 mm) were implanted and compared with Carotid Wallstents of the same size. To increase reliability each experiment was repeated three times, using the same model. The compliance between stent and ICA tortuosity was evaluated visually and with the use of the above-mentioned angle and offset measurements, which were compared between the status before and after stenting as well as between different stent types.

Compared with the braided Wallstent, the segmented Nitinol stent complied better with the given curve and showed an improved apposition with only minimal dehiscences between stent and vessel wall. The Wallstent straightened the circumscribed tortuosity into a C-shaped curve. After loosening of the distal fixation, longitudinal forces extended the ICA upwards, which was not observed with the use of the nitinol stents (Fig. 6.3). The Nitinol stent reduced the mean CCA-ICA angle (n=4) from 59° (range 56–62°, SD 2.5°) to 47° (range 45–49°, SD 1.7°) and the mean ICA offset from 20 mm (range 19–21 mm, SD 0.8 mm) to 16 mm (range 14–18 mm, SD 1.7 mm). Longitudinal extension of the stented model was minimal (3 mm). The Carotid Wallstent implanted into the same model reduced the mean angle form 59 to 32° (range 30–35°, SD 2.2°) and the mean offset from 20 to 12 mm (range 11–14 mm, SD 1.4 mm). After loosening of the fixation at the skull base, longitudinal extension of the ICA was 6 mm (Fig. 6.4). Larger dehiscences between Wallstent filaments and the vessel wall were observed at the peak of the curve (Fig. 6.3). Both stent types complied well with the two-diameter situation at the bifurcation. Incomplete stent expansion or abrupt changes in the vessel diameter may induce separation and protrusion of parts of a Nitinol stent segment (Fig. 6.5), whereas the Wallstent tends to bridge these irregularities, leaving pouches without stent apposition (Fig. 6.3).

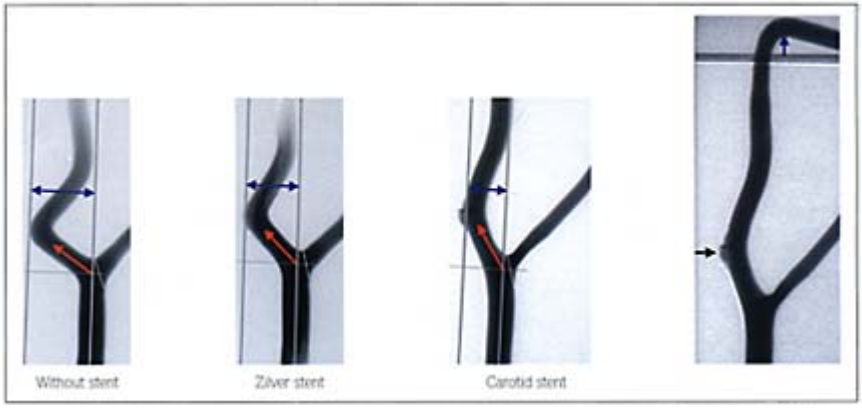

Figure 6.3 Angiograms of a simplified silicone model of the carotid bifurcation

exhibiting elastic properties similar to a human artery The model is fixed in a box and shows a 60° angulation between initial internal carotid artery (ICA) segment and common carotid artery (CCA) axis (red arrow). The deviation of a circumscribed curve (offset) perpendicular to the CCA axis is 20 mm (blue arrow). Implantation of a segmented nitinol stent shows only moderate geometric effects with slight reduction of angle and offset. The Carotid Wallstent induces straightening of the bifurcation and ICA tortuosity which is transformed into a larger C-shaped curve. Angle and offset are reduced more markedly. After loosening of the fixation at the virtual skull base longitudinal forces of the Wallstent induce an upward displacement of the distal ICA. Note the dehiscence between the Wallstent filaments and the vessel wall at the bulging of the ICA curve (black arrow). The segmented nitinol stent shows an improved apposition to the vessel wall.

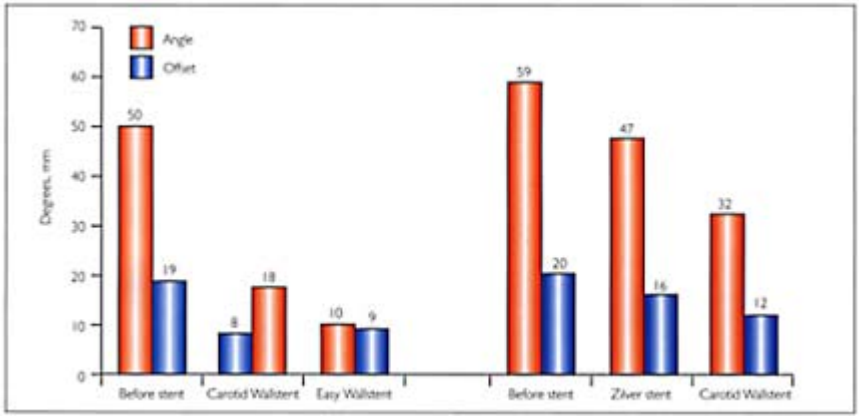

Figure 6.4 Diagrammatic representation of angle and offset measurements in silicone models to compare geometric effects of different tent types. The red bars show the changes in the common carotid artery (CCA)-internal carotid artery (ICA) angle induced by the Carotid Wallstent and the former type of less shortening Easy Wallstent. as well as by a segmented Nitinol stent (Zilver stent) compared with a Carotid Wallstent in a second set of experiments (right diagram). The blue bars show the corresponding changes in the ICA offset. The mean values of four measurements are indicated.

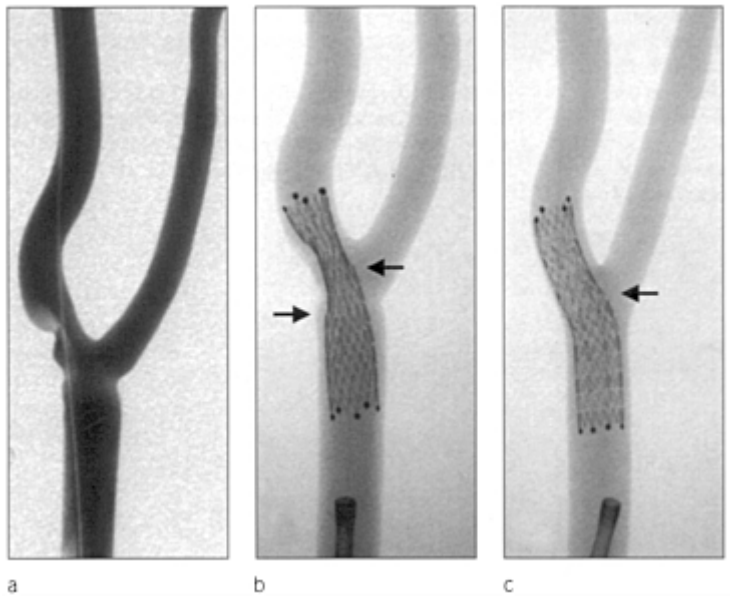

Figure 6.5

(a) Digital subtraction angiographic image of a silicone model with an eccentric stenosis at the bifurcation. (b) Slight separation and protrusion of stent segments (arrows) after deployment and inadequate postdilatation of a segmented nitinol stent. (c) Harmonic configuration of the stent after adequate postdilatation with normal separation of segments at the outer circumference of a curve at the external carotid origin.

Conclusion

Wallstents, with their braided continuous filaments, show marked reconstructive effects on vessel geometry, mainly in the longitudinal direction.[8] This results in straightening of the bifurcation angle and of ICA tortuosity. Clinical cases and model experiments proved longitudinal extension of the ICA distal to the stent, which may induce kinks, especially in patients with generalized atherosclerotic disease and loss of elastic components in the vessel wall.[8,10] Segmented nitinol stents show only moderate reconstructive effects and a good adaptation to the angulation and tortuosity of the vessel.[11] Owing to the segmental design, stent apposition to the bulges in the vessel is improved compared with the Wallstent, which showed larger dehiscences between stent filaments and the vessel wall. However, the continuity of the Wallstent struts provides a smooth inner stent surface, and protrusions or separations of single segments are not observed. Despite suboptimal recanalization results, Wallstent studies showed a good clinical and angiological outcome for the patients.[6,7] So far, there are insufficient data concerning the complication and

restenosis rate of the new-generation Nitinol carotid stents; more will hopefully be published soon, with the widespread use of these prostheses. Future carotid stent designs may be modified to optimize radial apposition and longitudinal flexibility without the disadvantage of separation of unconnected segments. The aim of optimization of carotid stent geometry is to improve haemodynamics and endothelial coverage to avoid late lumen loss by neointimal hyperplasia.

Acknowledgements

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7
Imaging of the carotid arteries

A Al-Kutoubi

Introduction

Imaging of the carotid arteries is carried out to define the level of disease and establish its nature and severity. Once the underlying pathology has been established, treatment plans can be defined and imaging may then be used to guide treatment

Pathology

Atheromatous stenosis of the carotids is the most commonpathology in clinical practice. However, other types of diseaseinvolving the neck as well as the vessels may be encountered.These include:

- fibromuscular dysplasia (FMD)
- arteritis
- dissection (traumatic or spontaneous)
- atheromatous and non-atheromatous aneurysms (FMD, traumatic false aneurysms)
- chemodectomas (carotid body, glomus vagale and glomus jugulare) and other neck tumours (lymph nodes, carcinomas, etc.)
- radiation-induced changes.

Ultrasound

This totally non-invasive and widely available modality is the first investigation in carotid disease. The degree of stenosis is estimated by establishing the peak systolic velocity (PSV) and end-diastolic velocity (EDV) at the area of interest. The ratio between the measurements in the internal carotid and common carotid arteries (PSV_{ICA}/PSV_{CCA}) is often added to improve the specificity by reducing variations related to cardiac output. The haemodynamic evaluation of stenosis with colour and power Doppler and the detection of flow patterns in the neck arteries are now well established,[1,2] as are many of the pitfalls related to changing flow patterns, for example in contralateral occlusion.[3] Ultrasound is able to evaluate wall thickness and characteristics of the plaques at the site of disease.[4–7] Digital analysis of the plaque morphology[8] can be used to predict outcome and justify treatment. Many practitioners argue that this method should be the gold standard and are willing to make surgical management decisions based on ultrasound

alone.[9,10] As this modality is operator dependent it is imperative that quality assurance programmes against international standards are established within individual units so that the results are validated.[11] Weaknesses of ultrasound include the inability to assess of the origins of the neck vessels from the arch as well as the distal circulation. Difficulties arise when there is dense calcification at the carotid bifurcation, when the arteries are tortuous or when there is unusual anatomy, and when the stenosis is so tight that it could be mistaken for a total occlusion. In these cases it is necessary to supplement the ultrasound with another imaging method to evaluate and confirm the findings. There is an estimated annual stroke risk of 5% if a 99% stenosis is ignored.[12] Criteria for grading carotid stenoses are summarized in Table 7.1. Figures 7.1–7.5 illustrate typical findings.

Ultrasound-specific contrast media have been used to enhance the ability of this method to detect disease and particularly to distinguish between total occlusion and severe stenosis,[13,14] but this has not abolished the necessity for further imaging in difficult cases.

Table 7.1 Ultrasound criteria for carotid artery stenosis.

Diameter reduction (96)	*PSV*	*EDV*	*PSVICA/ PSVCCA*
50	155	52	1.8
60	210	72	2.6
70	285	100	3.75
80	385	140	5.25
90	525	200	7.5
100	No flow		

After Polak.[1]
PSV: peak systolic velocity; EDV: end diastolic velocity;
CCA: common carotid artery; ICA: internal carotid artery;
PSVICA/PSVCCA: ratio of velocities. Velocity measurements are in cm/second. Confidence levels are not included; refer to original article for more detail.

Transcranial Doppler (TCD) ultrasound studies in conjunction with vasodilation are extremely useful in the assessment of perfusion of the brain and detection of occlusion or anatomical variation of the branches of the circle of Willis.[15,16] Repeated studies at regular intervals provide important information for the management of patients after ischaemic strokes.

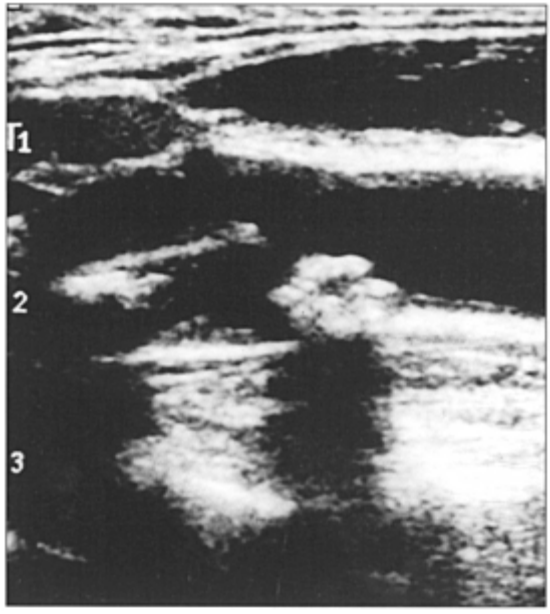


Figure 7.1 Hyperechoic plaque at origin of internal carotid and atheroma in the common carotid.

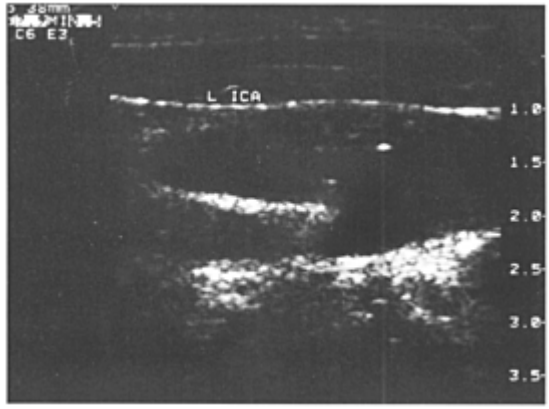
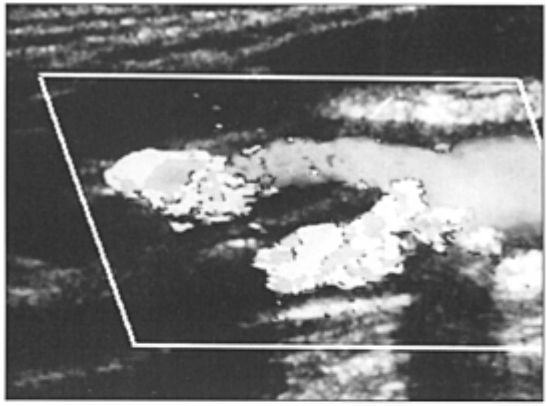


Figure 7.2 Tight carotid stenosis and hypoechoic plaque.



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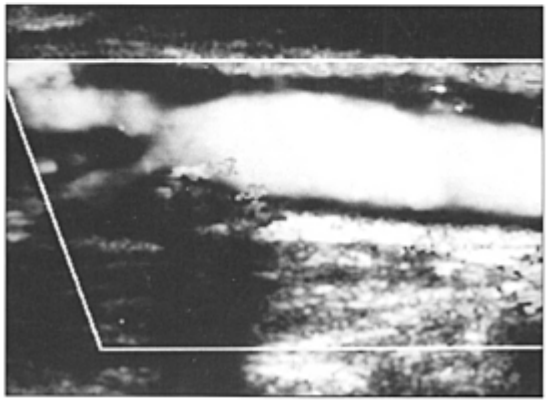

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Figure 7.3 (a) Colour Doppler and (b) power Doppler studies showing a moderate carotid stenosis.

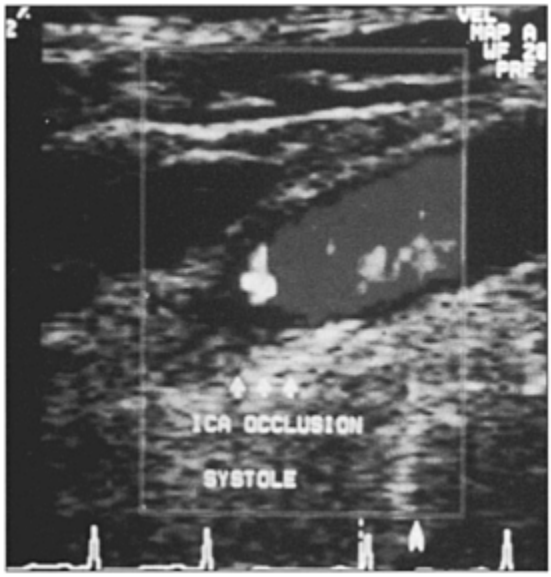


Figure 7.4 Complete occlusion of the internal carotid.

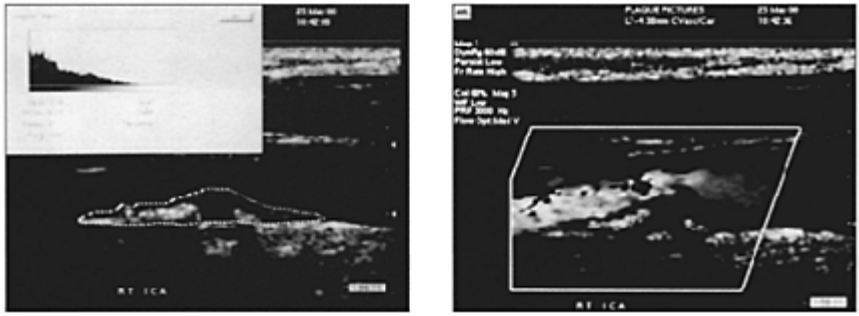
Angiography

Angiography remains the established gold standard for evaluation of all the arterial branches, but it is invasive and is associated with a documented incidence of cerebrovascular events of 0.5–1%,[17] which increases as the length and complexity of catheter manipulation in the arch and neck vessels increase. Other complications occur mainly at the entry site and range from haematoma formation to arterial dissection and thrombosis. Many of the patients who present with carotid artery disease suffer from atheromatous involvement of other parts of the vascular tree, particularly the aortoiliac segment, such as occlusion or aneurysm formation, which may limit access. Access routes other than the femoral artery may be used, such as brachial and radial arteries, but the risk of cerebrovascular events remains the same irrespective of the route of catheter placement.

Angiography is the ideal imaging method to show the origins of the major vessels from the arch, and to demonstrate anatomical variants, significant tandem lesions in intracranial disease[18] and abnormalities of the neck vessels. Collateral circulation and patterns of flow are also documented on angiography. Ideally any suspicion of unusual pathology, such as fibromuscular dysplasia or trauma, should lead to evaluation by

angiography.

One of the indications for angiography is distinguishing between total occlusion and severe stenosis (99%); however, the main weakness of angiographic grading of stenoses stems from the fact that only the lumen of the vessel is demonstrated. Grading the severity of stenosis at the origin of the internal carotid varies depending on whether the estimation is proportional to the lumen of the distal internal



a b

Figure 7.5 Digital analysis of carotid plaque: predominantly echogenic plaque with a grey-scale median histogram.

carotid [North American Symptomatic Carotid Surgery Trial (NASCET)][19] or to the assumed diameter of the bulb [European Carotid Surgery Trial (ECST)].[20] Correlation with the haemodynamic groups as decided by ultrasound should be routinely used in the clinical context.[21] Figures 7.6–7.18 show examples of angiographically illustrated pathology.

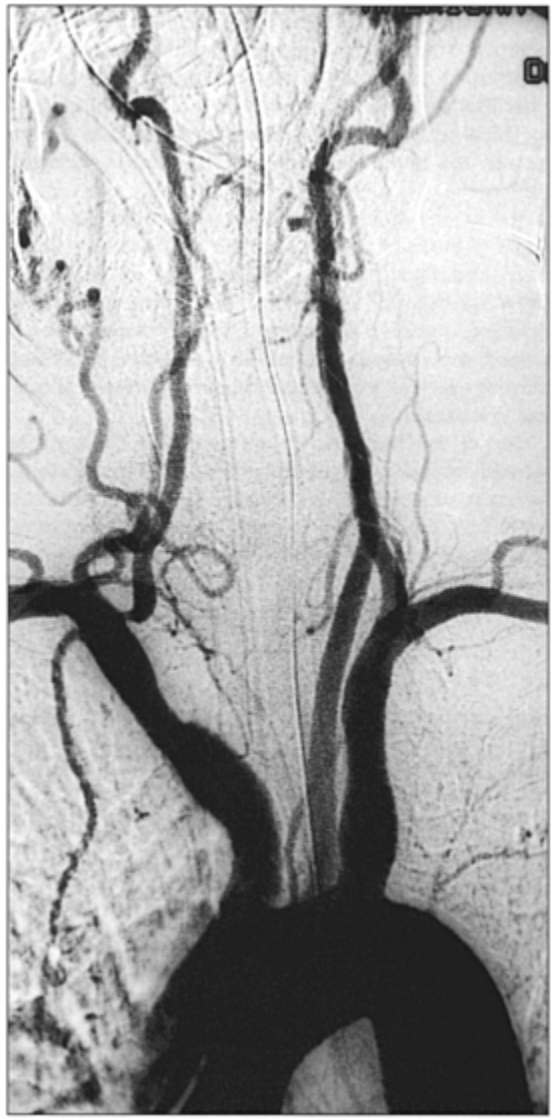
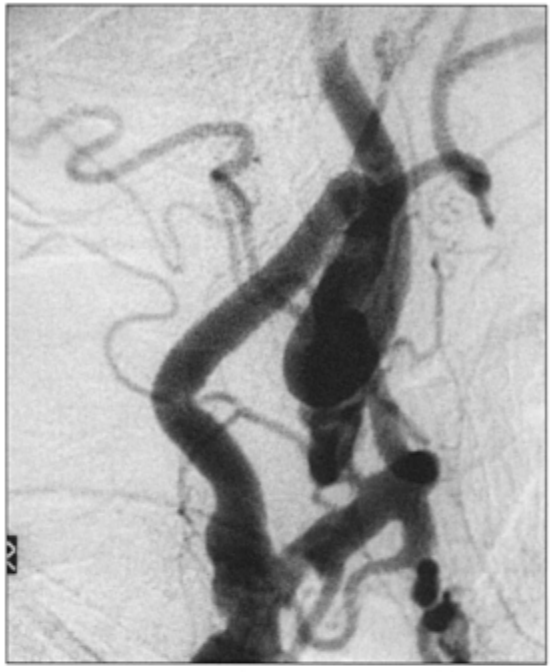


Figure 7.6 Arch aortogram: occlusion of the right common carotid. Slow filling of the left carotid indicating distal stenosis. Stenosis at the origin of the right vertebral artery



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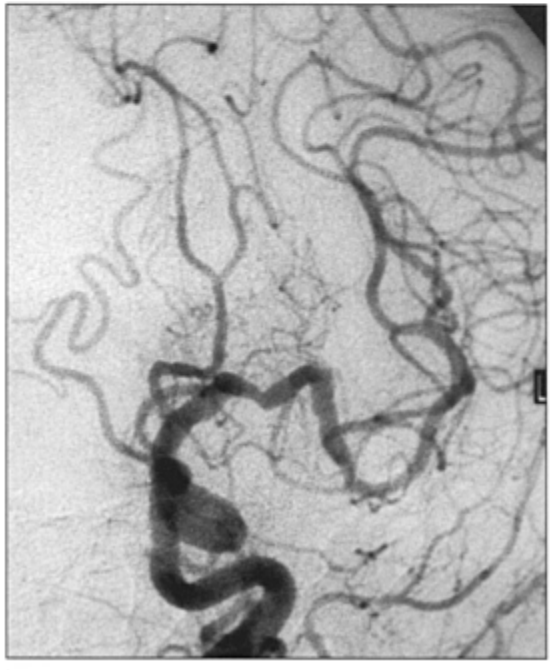

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