

INTRODUCTION

Extracranial cerebrovascular disease is an important cause of stroke and transient cerebral ischaemic attack.

Stroke is the third most common cause of death after heart disease and cancer in developed nations and is the leading cause of severe disability.

Predominantly affects middle aged/elderly Caucasian male patients. Incidence increases with age and more than half of all strokes occur in patients over the age of 75yrs.

ANATOMY AND BLOOD SUPPLY OF BRAIN

There are four major arteries that supply the brain

- **Posterior Circulation**
Two vertebral arteries originate from the first part of the subclavian arteries. These join intra-cranially to form the basilar artery and supply the posterior cerebral circulation. This is the main blood supply to the brain stem, cerebellum and posterior cerebrum.
- **Anterior Circulation**
Both internal carotid arteries arise from the carotid bifurcation in the neck. Intra-cranially, the internal carotid arteries form the middle cerebral arteries, the most important branches in the anterior cerebral circulation.

The Circle of Willis provides communication between the anterior and posterior cerebral circulations but may be incomplete in about 20% of individuals.

DEFINITIONS

Stroke

- Acute focal **ischaemia or infarction** of the brain secondary to vascular insufficiency.
- Symptoms exceeding more than 24hrs or lead to death.

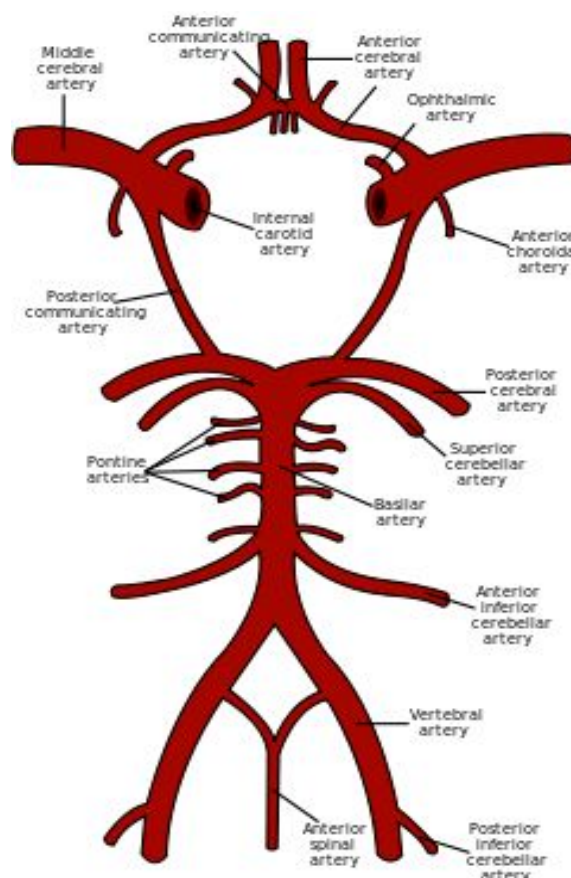
Transient Ischaemic Attack (TIA)

- Temporary focal **ischaemia** of the brain secondary to vascular insufficiency with symptoms lasting less than 24 hours.
- Majority of TIA's last less than 10 minutes

Amaurosis Fugax

- "Fleeting blindness" Temporary retinal artery ischaemia.
- Symptoms described as curtain coming down over the eye.

PATHOLOGY AND PATHOGENESIS



There are two main types of strokes:

Ischaemic (80% of all strokes)

- Embolisation or occlusion of an artery that supplies the brain
- About 75% of ischaemic strokes are due to atherosclerosis of large arteries (predominately the carotid arteries)
- Cardiac embolism accounts for 15% of ischaemic strokes and the remaining 10% is due to small vessel occlusion (normally secondary to hypertension)

Hemorrhagic (15-20% of all strokes)

- Primary intracranial hemorrhage (10-15%)
- Subarachnoid hemorrhage (5%)

EXTRACRANIAL ARTERIAL PATHOLOGY

Atherosclerosis is the pathological basis of extracranial arterial disease in 90% of cases. The remaining 10% of conditions include fibromuscular dysplasia, dissection, aneurysm, arteritis (Takayasu's and Giant cell arteritis) and carotid body tumours.

Atherosclerosis affecting the extra cranial arteries tends to occur at the carotid bifurcation and particularly at the origin of the internal carotid artery (ICA). This is due to the turbulent flow in this region.

There are two main theories of how atherosclerotic disease at the bifurcation can lead to strokes and TIAs:

• **Emboic Theory**

- Particularly with tight/high grade complicated atherosclerotic plaques (>70%) at the bifurcation and the turbulent flow in this region, there is embolization of atherosclerotic or thrombotic material to the brain.

- Likelihood of embolization or occlusion of ICA from atherosclerotic plaque increases with degree of stenosis

• **Haemodynamic Theory**

- With very tight stenosis or occlusion of the ICA and incomplete intracranial collateral circulation there is a risk of cerebral hypoperfusion that can result in a TIA or stroke.

CLINICAL PRESENTATION

Extracranial cerebrovascular disease can either be asymptomatic or symptomatic.

Asymptomatic

- Bruits in the neck may indicate carotid artery disease.
 - Need to distinguish bruits that originate from carotids and those radiating from the heart or great vessels
 - Only one third of patients with significant ICA stenosis will have a bruit
- Incidental finding on carotid duplex ultrasound scanning

Symptomatic

Symptoms may be transient (TIA or amaurosis fugax) or more permanent (stroke); focal or global (usually associated a large hemorrhagic stroke); and may affect the anterior or posterior circulation

Motor and sensory symptoms and signs are usually focal and involve the contralateral limbs. Dysphasia and contralateral cranial nerve palsies are common.

When the vertebrobasilar system is involved, symptoms may be less specific. Symptoms may include limb weakness, bilateral visual disturbance, ataxia, imbalance, dizziness, vertigo or bradycardia.

Headache and loss of consciousness is more commonly associated with intracranial haemorrhage or space-occupying lesions.

INVESTIGATIONS FOR EXTRACRANIAL CAROTID ARTERY DISEASE

All patients who present with focal neurological symptoms require imaging to determine the extent of damage and to ascertain the cause. It is particularly important to differentiate between cerebral infarction and intracranial haemorrhage.

General medical evaluation

- **Cardiovascular evaluation**
 - Blood pressure measurement to detect hypertension
 - Electrocardiography (ECG) to assess any cardiac rhythm disturbance like atrial fibrillation or evidence of co-existent coronary artery disease.
- **Haematological investigations**
 - Renal function, lipid and glucose levels are measured to exclude renal failure, hyperlipidaemia and diabetes respectively
 - Coagulopathy screening when appropriate

Imaging

- **CT Angiogram or MRI** (uncontrasted/contrasted)

Early CT or MRI scanning can identify intracranial haemorrhage or space-occupying lesions. This is important as the acute management and prognosis of these two pathologies is different from ischaemic strokes.

Both, CT and MRI, are also useful for the imaging of the extracranial vasculature but are not the first line investigations to assess for disease in the carotid artery.

- **Duplex ultrasound**

Is most commonly used as the primary diagnostic test to evaluate the carotid arteries and used by many surgeons

as the definitive investigation when planning a carotid endarterectomy. Duplex ultrasound is operator dependent but in experienced hands it can show the morphology of the carotid bifurcation and can accurately identify the degree of stenosis.

Ideally all patients who have had a focal neurological event should have a carotid duplex ultrasound as soon as possible to identify a possible vulnerable atherosclerotic carotid plaque that could potentially cause further neurological events. However in reality we do not possess the resources to perform a duplex ultrasound on every patient acutely after a TIA or stroke. Thus we have developed a scoring system looking at five clinical features to help us predict to likelihood of a secondary neurological event after having suffered a TIA - ABCD² Stroke Risk Score

ABCD² Score

- **Age:** greater than or equal to 60 (1pt)
- **Blood pressure:** SBP \geq 140 or DBP \geq 90 (1 pt)
- **Clinical Features:**
 - focal weakness (2pt) or
 - speech impairment without focal weakness (1pt)
- **Duration of symptoms:**
 - \geq 60minutes (2pt) or
 - \leq 59 minutes (1pt)
- **Diabetes** (1pt)

Risk of CVA at 2 days

- 0-3 points = 1% risk
- 4-5 points = 4.1% risk
- 6-7 points = 8.1% risk

Johnson SC, et al. Lancet 2007;369:283-92.

Patients who score 6-7 points have the highest risk of an early recurrent TIA or stroke and thus will receive urgent carotid duplex and possible intervention if warranted.

- **Digital subtraction angiography (DSA)**

DSA of the carotid arteries was historically considered the gold standard of extracranial arterial investigations. However, with improvement in duplex ultrasound, CT

scanning and MRI technology and the risks associated with direct angiography, DSA is now only used selectively. DSA tends to be reserved for situations where ultrasound findings are uncertain or the aortic arch and other branch vessels need to be assessed for the planning of carotid artery stenting.

TREATMENT OF EXTRACRANIAL CAROTID ARTERY DISEASE

The aim of treatment after a stroke or TIA is to initially stabilize and support the patient and then to institute steps to prevent or reduce the risk of further neurological events. (Secondary prevention)

Optimal Medical therapy

Controlling the risk factors that precipitate atherosclerosis has been demonstrated to reduce the risk of developing future strokes. Thus we use a combination of counseling and pharmaceutical means to control patients' hypertension, diabetes and cholesterol and assist with smoking cessation. Antiplatelet aggregation therapy, Aspirin, is also administered to reduce the risk of future TIAs.

Surgical or Endovascular Intervention of the Extracranial Carotid Artery

There are however defined subgroups of patients who may benefit from surgical or endovascular intervention to remove an identified carotid embolic lesion. These subgroups are defined by the degree of internal carotid artery stenosis and whether the patient is symptomatic or asymptomatic.

Symptomatic carotid artery stenosis

Two large randomized control studies; North American Symptomatic Carotid Endarterectomy Trial (NASCET) and European Carotid Stenosis Trial (ECST), have clearly demonstrated that in patients who have had a focal neurological event within the last 6 months with a residual ICA stenosis of

more than 70% (high grade), benefit from carotid endarterectomy (CEA). The relative stroke risk reduction at two years is around 30% with CEA compared to medical therapy alone.

Asymptomatic carotid artery stenosis

The benefit of CEA in an asymptomatic (never had a focal neurological event or had an event more than six months before presenting to hospital) patient compared to a symptomatic patient, with high grade stenosis of the ICA, is significantly less. The reason for this decreased benefit is that we are unable to predict which one of these asymptomatic plaques may actually become symptomatic with time and thus may over treat patients unnecessarily. Thus, when deciding to offer these asymptomatic patients with high grade ICA stenosis surgery, one needs to take into account the patient's fitness and life expectancy (>5yrs) and the surgical complication risks (<3%).

Surgery: Carotid Endarterectomy (CEA)

The carotid bifurcation is the commonest site for extra-cranial carotid artery disease and this is surgically accessible in most cases.

CEA can be performed under local/regional or general anaesthesia with similar outcomes. The benefit of local/regional anaesthesia is that one is able to evaluate any neurological fall-out during the procedure instantly, however the patient may be more anxious and uncomfortable throughout the procedure.

To expose the carotid bifurcation a longitudinal incision is made along the medial border of the sternocleidomastoid muscle in the neck. The internal jugular vein is mobilized laterally to enter the carotid sheath. The common carotid artery and vagus nerve run in this sheath.

The dissection is continued proximally to expose the carotid bulb, internal and external carotid arteries. Be sure to identify and preserve the hypoglossal nerve that normally crosses over both the internal and external carotid arteries just above the carotid bifurcation.

Once systemic heparin has been given we assess the stump pressure. To perform this test we clamp the common carotid and external carotid artery and evaluate the retrograde perfusion across the ICA stenosis. The stump pressure should be more than 50mmHg, with a pulsatile trace. Then the carotid vessels may be clamped, and the surgeon may proceed with the endarterectomy without the need for a shunt. However, if the stump pressure measurement do not fulfill these requirements a shunt is used to maintain inline cerebral flow during the endarterectomy.

A longitudinal arteriotomy is made extending from the common carotid artery to internal carotid artery across the stenotic lesion. The diseased intima and proximal media are then very carefully removed from the remainder of the vessel wall. Meticulous care is taken to ensure there are no loose fibers or flaps left behind as these may lead to a dissection of the artery at a later stage. The arteriotomy is then sutured closed using a prosthetic Dacron or vein patch to ensure a wide patent lumen and reduce the chances of late restenosis.

The platysma muscle and skin are then closed after haemostasis has been achieved.

Endovascular: Carotid Angioplasty and Stenting

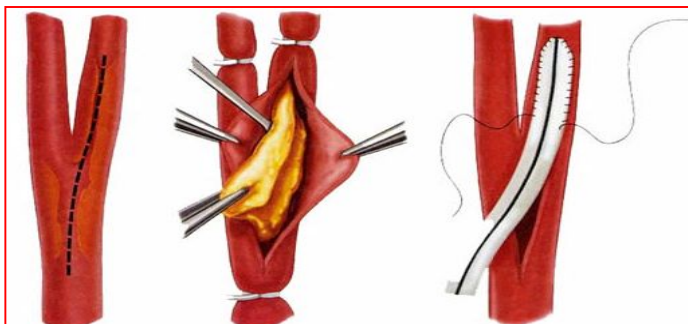
The role of carotid angioplasty is limited to patients with symptomatic fibromuscular dysplasia of the carotid artery.



In patients with atherosclerotic carotid artery disease the role of angioplasty with stenting is less defined. Current evidence demonstrates that despite advances in stent technology and cerebral protection devices endovascular intervention is associated with higher procedural strokes than CEA. Thus we reserve carotid angioplasty with stents for a select group of symptomatic patients with high grade ICA stenosis. These are patients with multiple comorbidities that will not tolerate surgery, patients with previous neck surgery or radiation and patients with late restenosis of the ICA post-CEA.

Procedure-related complications

- Ipsilateral Ischaemic Stroke from embolization during procedure
- Labile blood pressure and hypertension
- Acute Coronary Syndrome
- Death



- Local neck haemorrhage/haematoma
- Nerve injuries – vagus, recurrent laryngeal, hypoglossal, glossopharyngeal

VERTEBROBASILAR INSUFFICIENCY

The vertebrobasilar arteries supply blood and oxygen to the occipital cortex, the cerebellum, thalamus and brain stem. When there is significant disease and insufficient perfusion of these vessels an array of atypical symptoms may present.

Vertebrobasilar disease is twice as common in men as in women, and typically occurs in the elderly.

Pathology

Atherosclerosis is by far the commonest cause of disease in the vertebral and basilar arteries. Very rarely arterial damage can occur secondary to vertebral artery compression

Clinical Presentation

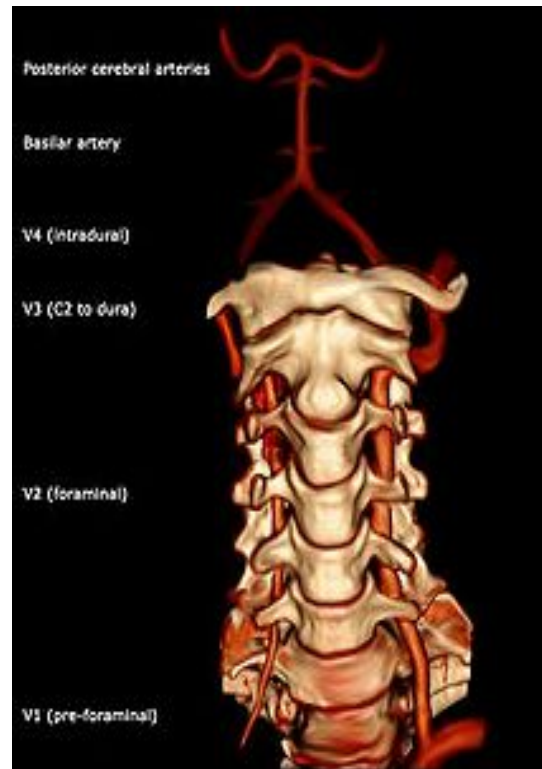
The symptoms of vertebrobasilar syndrome can include: vertigo (dizziness), visual disturbances (blurring, graying, double vision), drop attacks (sudden falls), numbness or tingling and slurred or lost speech. These symptoms can last a few minutes (TIA) or be permanent (stroke).

Diagnostic Appraisal

Due to the variation and specificity of symptoms clinical assessment and investigation tend to be extensive. Thorough cardiac, ENT, ophthalmologic, musculoskeletal and neurological assessments need to be performed.

Haematological tests

- Should include electrolytes and an assessment for risk factors for atherosclerosis.



Imaging

- Imaging all the parts of the vertebral artery may prove difficult. Duplex ultrasound may be useful in assessing the first and second part of the vertebral artery, however it cannot visualize the whole artery and thus is not as sensitive as MRA or CTA. Both MR and CT angiogram are capable of demonstrating the entire vertebrobasilar vasculature and pathologies. However, they are not without problems involving subjecting patients to radiation (CTA), and a potentially nephrotoxic contrast agent as well as being inaccurate for heavily calcified stenosis (MRA and CTA).

Treatment

Once again like with carotid artery disease, the aim of treatment after a stroke or TIA is to initially stabilize and support the patient and then to institute steps to prevent or reduce the risk of further neurological events (Secondary prevention).

Medical management and Lifestyle changes

As with carotid artery disease, the mainstay of treatment is the pharmaceutical and lifestyle modification of atherosclerotic risk factors.

Surgical or Endovascular Intervention of the Vertebral Artery

In certain situations, depending on the location of the disease there may be surgical or endovascular interventions to improve posterior intra-cranial circulation.

Surgery

- **Endarterectomy**
 - Mainly performed for focal, ostial or para-ostial vertebral artery lesion
- **Bypass grafting or direct arterial transposition**
 - Performed for longer lesions particularly diseased V2 segment of vertebral artery

Endovascular

Newer long and low profile endovascular devices have lent themselves to angioplasty and stenting of vertebral artery lesions. Commonly reserved for shorter lesions, unfit patients or where the lesion is difficult or not accessible to open surgery.



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