Vertebrobasilar Ischemia

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Etiology

1. Atherosclerosis
2. Extrinsic compression (e.g. osteophyte, cervical spine dislocation) – esp. V2 segment
3. Repetitive trauma (e.g. chiropractic manipulation)
4. Fibromuscular lesions
5. Aneurysms
6. Dissections
7. Takayasu disease

N.B. most serious vertebrobasilar disease is ***intracranial***, where surgery is not feasible.

(vs. carotid disease – ***extracranial***, with good surgery results)

Pathophysiology

1. **Hemodynamic ischemia** (low risk of infarction):
	1. significant disease must be present in both VAs + incomplete compensation via circle of Willis.
	2. subclavian-vertebral steal phenomenon. *see below*
		* repetitive, short-lived symptoms, which are more of nuisance than danger (but *risk of traumatic injuries* resulting from loss of balance).
		* symptoms are reproducible with ***postural* - *positional***\* ***changes***.

\*neck rotation or extension

* + - symptoms are aggravated by cardiac insufficiency.
1. **Embolic ischemia** (≈ 30%) from atherosclerotic ostial stenosis of subclavian, vertebral, or basilar arteries.
	* + likely to cause dangerous infarcts that leave permanent and debilitating strokes.
2. **Thrombotic ischemia**

N.B it is very rare for VA to be completely occluded at the origin!

Clinical Features

- ischemia of "end-organ" of posterior circulation (brainstem, cerebellum, occipital lobes).

[see p. Vas3 >>](http://www.neurosurgeryresident.net/Vas.%20Vascular%5CVas3.%20Ischemic%20Stroke%2C%20TIA.pdf#VA_BA_distribution)

Diagnosis

Only few studies clearly ascertain VA anatomy:

1. **Systolic BP difference** > 15 mmHg between arms (suggests subclavian stenosis).
2. **Duplex ultrasound** - significant limitations (vs. carotid artery); used mainly to detect:
	1. *flow reversal* within VA
	2. *flow velocity changes* (consistent with proximal stenosis).
3. **MRI & MRA** - visualization of VA and BA, posterior fossa infarcts.
4. **Arteriography** (diagnostic test of choice; mandatory prior to any operative intervention)
* most common site of VA disease is at its origin - requires special projections.
* suspected VA ***compression*** → dynamic angiography (incorporates provocative positioning).
* *delayed imaging* - reconstitution of extracranial VA through cervical collaterals.

Medical Treatment

**Dual antiplatelet therapy** (long-term) - for patients not amenable to surgery or endoluminal therapy.

N.B. role of dual antiplatelet therapy is proven (vs. unclear for carotid stenosis)

**Cerebroselective calcium antagonists**:

Cinnarizine

Mechanism of action:

* 1. blocks Ca2+ channels → vasodilatation (cerebral, coronary, peripheral).
		+ does not affect BP and heart rate.
	2. blocks H1 receptors → vestibular suppression
	3. improves blood rheology.

Administration: well absorbed per os; T1/2 ≈ 3-6 hrs; max dose – 225 mg/d.

Indications:

1. chronic cerebrovascular insufficiency 25-75 mg × 3/d
2. labyrinth disorders (vertigo, tinnitus, motion sickness, Ménière disease) 50 mg × 1-2/d.
3. migraine prophylaxis
4. peripheral circulation disorders 50 mg × 3/d.

Adverse effects: CNS sedation, dry mouth, constipation, epigastric distress (H: use with food), extrapyramidal side effects (e.g. hand tremor), hypotension.

Flunarizine – bifluor derivative of cinnarizine – can be administered ×1/d (T1/2 ≈ 18 days).

10-15 mg × 1/d at bedtime

Surgical Vertebral artery reconstruction

- fewer ischemic complications (than carotid surgery) and durable long-term results.

Indications

N.B. unilateral VA stenosis in asymptomatic patient is not indication for surgery!

Single normal-caliber VA can supply sufficient blood flow into BA regardless of status of contralateral VA.

1. Bilateral severe (> 70%) stenosis
2. Symptomatic cases:

***Hemodynamic symptoms*** + ***insufficient blood flow to basilar artery*** (i.e. severely stenotic [> 75%] VA and equally diseased or occluded contralateral VA)

* asymptomatic status indicates good compensation from carotid circulation (via PComA).

***Embolic*** ***symptoms*** regardless of condition of contralateral VA.

Procedure

- disease location dictates type of surgical reconstruction:

1. **Ostial (V1 segment)** - two main options:
	1. **balloon angioplasty & stenting**; stent has to protrude into subclavian lumen (VA ostium is very muscular and has to be stented)
	2. **transposition** of proximal VA onto common carotid artery
	3. VA **bypass**:
		* origin - common carotid artery or subclavian artery.
		* conduit - saphenous vein or prosthetic material (polytetrafluoroethylene or Dacron).
	4. subclavian-vertebral endarterectomy is less commonly performed.
2. **V2 segment** (surgical reconstruction is very rarely undertaken) – **ligation** (at C1-2 level) and **bypass** to V3 segment.
* extrinsic lesions can be corrected to relieve kinking or compression of artery (e.g. cervical alignment or surgically opening foramina transversaria).
* most common indication for V2 segment exposure - control of hemorrhage in traumatic injuries to V2 segment (H: proximal & distal ligation of artery or coil embolization).
1. **V3 segment**:
	1. saphenous vein **bypass** from CCA, subclavian, or proximal VA.
	2. **transposition** of ECA or hypertrophied occipital artery to distal VA.
	3. **transposition** of distal VA to side of distal ICA.
2. **V4 segment** – treat only if maximal medical therapy fails!
* **balloon angioplasty & stenting**
* saphenous vein **bypass** from distal ICA - surgical exposure requires resection of C1 transverse process and part of its posterior arch.

Postoperative

* long-term antiplatelet therapy (e.g. aspirin).

Complications

Combined morbidity/mortality rates:

***Proximal reconstructions*** (technically easier) - 0.9%.

***Distal reconstructions*** - 3-4%.

1. **Stroke**, **hematoma**, **thrombosis**, **lymphocele**.
2. **Nerve injury** (e.g. Horner syndrome, spinal accessory nerve, vagus nerve).
* ***ptosis on operative side*** (known complication of proximal VA reconstructions - traction injury of lower cervical sympathetic nerves) - usually temporary.

Subclavian Steal

- occlusive disease in proximal subclavian artery (or innomin­ate artery) → blood flow reversal down VA\* on affected side to ischemic limb (i.e. blood siphoning away from brain):

\*VA fills from contralateral VA



Clinical Features

- often precipitated by limb exercise:

* vertebrobasilar TIAs (but rarely causes stroke).

posterior circulation symptoms with arm exercise

* **bruit** may be heard over subclavian artery.
* **BP diminished** in affected limb (systolic BP difference > 15-20 mmHg between arms supports diagnosis).
* ***pulse beat in radial artery*** is diminished and asynchronously follows that of other side.
* ***limb claudication***; limb may become cyanotic if held above heart level.

N.B. majority of persons with subclavian steal detected by noninvasive techniques have no neurological symptoms (i.e. merely radiographic curiosity).

Diagnosis

|  |  |
| --- | --- |
| Oblique **MRA** of aortic arch - high-grade stenosis of proximal left subclavian artery (*arrow*):D:\Viktoro\Neuroscience\Vas. Vascular\00. Pictures\Subclavian steal (MRA 1).jpg | Oblique 3D contrast-enhanced **MRA** of aortic arch - high-grade stenosis of proximal left subclavian artery (*white arrow*); left common carotid artery (LCCA); left subclavian artery (LSCA) (*blue arrow*):D:\Viktoro\Neuroscience\Vas. Vascular\00. Pictures\Subclavian steal (MRA 2).jpg |
| Aortic arch **aortogram**:A. Left anterior oblique projection, arterial phase: proximal occlusion of left subclavian artery (*arrow*). Note irregularity and tortuosity of right VA - related to degenerative changes in cervical vertebrae (*small arrow*).B. Right anterior oblique projection late phase of aortogram; distal segment of left subclavian artery (*arrow*) fills via retrograde flow in left VA, despite this vessel being almost completely obstructed at its origin (*small arrow*): | D:\Viktoro\Neuroscience\Vas. Vascular\00. Pictures\Subclavian steal (arteriogram).jpg |

Treatment

Surgery is almost never indicated.

Bibliography for ch. “Neurovascular Disorders” → follow this [link >>](http://www.neurosurgeryresident.net/Vas.%20Vascular%5CVas.%20Bibliography.pdf)

[Viktor’s Notes℠ for the Neurosurgery Resident](http://www.neurosurgeryresident.net/)

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