CEREBROVASCULAR

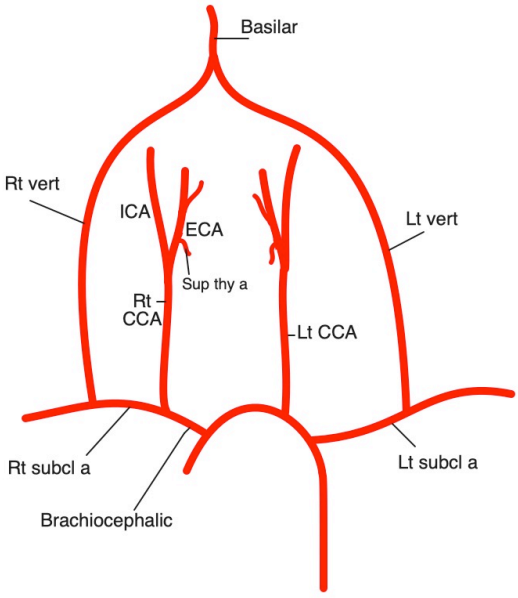
Anterior circulation

Rt common carotid artery originates at brachiocephalic a.
Lt CCA originates directly off arch.
CCA bifurcates into *internal carotid and external carotid a.*
70% of CCA blood goes to ICA

ECA gives off 8 branches to feed face, neck, scalp. 1st branch: *Superior thyroid a.* (seen traveling caudally after branching off ECA)

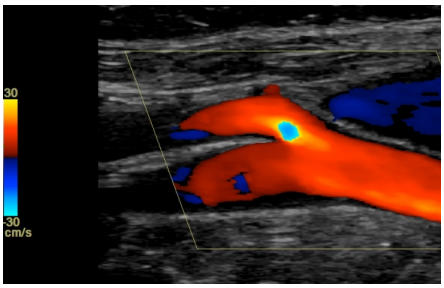
ICA has no extracranial branches. 1st branch at syphon: *Ophthalmic a.* (intracranial)

ICA terminates at circle of willis when it bifurcates into ACA and MCA



ICA vs ECA

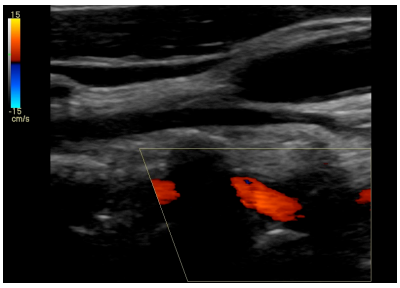
Most accurate way of differentiating ICA from ECA: Visualization of ECA branches. ICA has NO branches



Posterior circulation

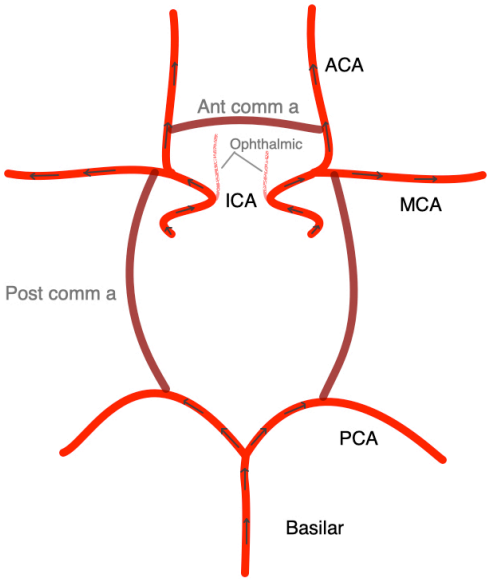
Vertebral a originate at subclavian arteries and travel through vertebral processes. Unite to form *Basilar artery*

Basilar then bifurcates to PCA



Circle of Willis

Intracranial pathway for collateral flow.
50% have incomplete circle.



Supplies the circle : ICAs and basilar
Supplied BY the circle: ACA, MCA, PCA
ICA bifurcates into *Anterior Cerebral a and Middle Cerebral a.*
Basilar bifurcates into *Posterior Cerebral a*.

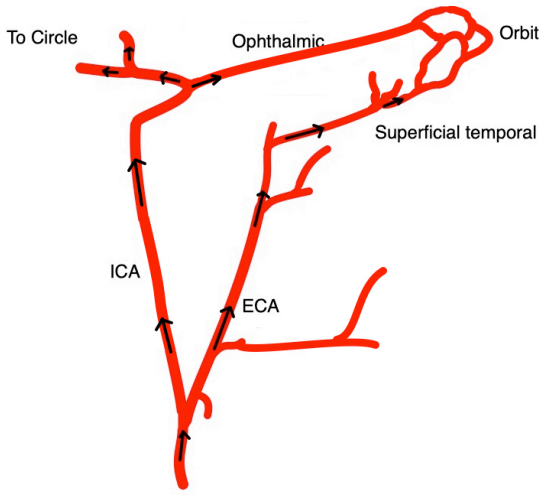
Anterior communicating a - connects Rt and Lt ACA
Posterior communicating a - connect posterior to anterior
These are only used when needed as collateral

Periorbital circulation

Network of vessels near the eye connecting the external and internal systems.

Supraorbital, superficial temporal, nasal, facial
>> ophthalmic

May be used as collateral network



Hemodynamics

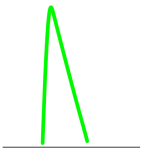
Same principles as arterial

Cerebro system since it's closer to heart, it is affected more greatly by cardiac state and other factors = Bilateral flow changes
High cardiac output = increased PSV bilat
Poor cardiac output = decreased PSV and dampened wave profile
Changes in viscosity = low hemoglobin > less viscous. Leads to decrease in resistance and overall high velocities throughout

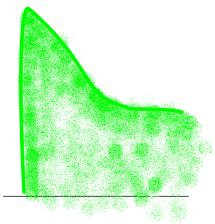
MUST BE AWARE OF THIS. Carotid stenosis criteria is based on PSV. Other conditions may cause elevated PSV without stenosis. If no PST = NO stenosis

Hemodynamic stenosis flow changes:

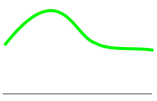
Prox to severe obstruction : Increased resistance. Decreased EDV. May also have dec PSV.



Within stenosis: Focal elevation is PSV and EDV. Spectral broadening



Distal to obstruction: Post stenotic turbulence. Rounding of waveform. Slow upstroke



Cerebrovascular Disease and Clinical History

Insufficiency symptoms

- TIA transient ischemic attack
  Neurologic deficit with no lasting effects. Complete recovery within 24 hours.
  *** Must be past event and completely back to normal.
  Usually embolic is (most common from heart)

- RIND reversible ischemic neurologic deficit
  Last longer than TIA but complete recovery within 72 hours.
  *** Must be past event

- CVA cerebrovascular accident
  Permanent neurologic deficit

- VBI vertebrobasilar insufficiency
  Ischemia affecting posterior circulation

Mechanism of disease : Ischemia and hemorrhage

Ischemia : blockages… *most common reason for cerebrovascular insufficiency*.
Can be atherothrombotic, cardiogenic = stenosis, occlusion, thrombus/ embolism

Hemorrhage : Bleeds from hypertension, ruptured aneurysm, trauma

Since ischemia is #1. Most common arterial disease is atherosclerosis. Risk is same as arterial

Diabetes
Hypertension
Hyperlipidemia
Smoking **most contributing factor

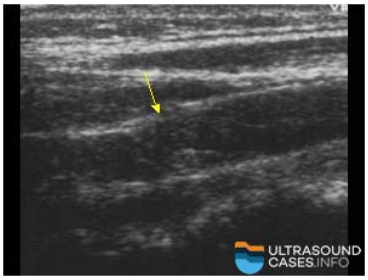
Age, family history, male gender

Atherosclerosis
Thickening and hardening of intima and medial layers. Most commonly found at bifurcations and proximal vessels

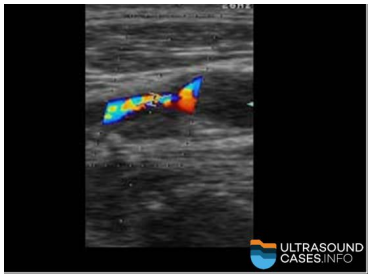
Type of plaque

- Fatty streak - thin hypoechoic layer

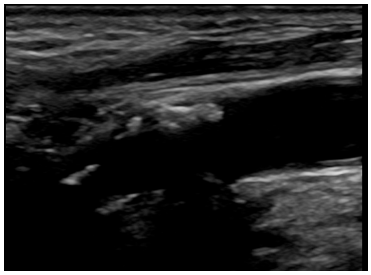
- Fibrous plaque - lipids with collagen and fibrous material. AKA smooth or homogeneous.
  Soft fibrous = hypoechoic
  Hard/dense fibrous = echogenic



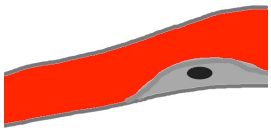
- Complicated lesion - complex or heterogeneous. Fibrous mixed with calcium and debris. Mixed appearance = hypo, echogenic, calcified



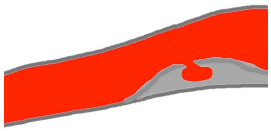
- Calcified - Completely hyperechoic with posterior shadowing. May limit visualization and evaluation of high velocity flow.



- Intraplaque hemorrhage - oval sonolucent area within the plaque. Fibrous cap is maintained. No flow seen inside. Unstable lesion, may rupture



- Ulcerative - crater-like deterioration of the cap. Flow seen inside. Unstable, may embolize or rupture



Embolism
Heart is most common source
Hollenhorst plaque - cholesterol crystals from the carotid that travelled to retina in the eye.

Thrombosis
May come from plaque, trauma, dissection. Acute, total occlusion = stroke. Stroke patients with a history of trauma (gunshot, stabbing, etc) would have had a dissection, then thrombosis.

Misc diseases

- Aneurysm : Rare in carotid system
- Tortuous vessels : *Most common cause of pulsatile neck mass*
- Carotid body tumor : Found between ICA and ECA, spreading them apart. Fed by vessels of ECA
- Dissection : Most commonly found by trauma. Danger = thrombosis > stroke
- Fibromuscular Dysplasia : Overgrowth of collagen in medial layer. Usually younger women, mid to distal ICA. Most likely to be found on angiogram.
- Neointimal hyperplasia : Intimal thickening caused by rapid reproduction of smooth muscle. 6-24 months POST endarterectomy. May cause restenosis. Seen as hypoechoic lining inside of vessel

Physical signs of cerebrovascular insufficiency

Anterior circulation

Lateralizing = indicate which side or hemisphere is affected. Anterior circulation feeds either right or left hemisphere. RT ICA goes to RT hemisphere and LT ICA goes to LT hemisphere. Symptoms will appear depending on which hemisphere is affected and based on the symptom we know what hemisphere and therefore what vessel would be the cause. If the symptoms are seen in a side of the body, it means the problem is on the other side.

RT side vessel feeds RT hemisphere. RT hemisphere controls LT side of body

LT hemispheric stroke would have RT side body symptoms. LT ICA cause

Vessel to brain = SAME
Vessel to body = OPPOSITE

Symptoms:

- Unilateral paresis - weakness or paralysis. Contralateral vessels
- Unilateral paresthesia/anesthesia - tingling, numbness. Contralateral vessels
- Dysphasia - impaired speech. Aphasia (unable to speak). Dominant hemisphere is affected. Depends on rt or lt handed.
  RT handed = LT hemisphere dominant
- Amaurosis fugax - temporary blindness in one eye. Comes from ophthalmic. Ipsilateral ICA
- Behavior changes - RT MCA
- Homonymous hemianopia - Loss of vision is half of visual field in both eyes. Stroke of cerebral cortex

Vessel specific signs

ICA - entire side of body, amaurosis fugax, speech

MCA - face and arm, speech, behavior

ACA - leg, incontinence, loss of coordination

*** Most common location for disease causing stroke = MCA

Posterior Circulation

Non-lateralizing. No sides known. Vertebrobasilar insufficiency VBI

- Vertigo - Sensation of moving around. Spinning sensation
- Ataxia - Lack of coordination. Poor gait
- Bilateral blurry vision
- Diplopia - Double vision
- Bilateral paresthesia
- Drop attack - Falling to ground without losing consciousness

PCA : dyslexia and coma

Non-localizing: Location unknown. Tells us nothing

Dizziness
Syncope
Headache
Confusion

Carotid Duplex

Capabilities and Limitations

Same as all duplex. Locating disease and surface characteristics. % DR

Technique

High freq linear array 7-9MHz
Sample volume small 1-1.5mm
Doppler angle 45-60 degrees. NEVER above 60.
*** must be parallel to flow and midstream

B-mode : sag and trans demonstrating presence/absence/severity plaque

Color : turbulent patterns/aliasing

PW : spectral analysis = quantitative assessment

Prox, mid, dist CCA and bulb
ECA (identify with branch)
Prox, mid, dist extracranial ICA
Vertebral (posterolateral to CCA. Runs between transverse processes)

Interpretation

B-mode

IMT (intima-media thickness) predictor for cardiovascular disease
Intimal thickening >0.9mm

Plaque classification

- Fatty - hypoechoic along wall
- Fibrous/homogeneous - low to medium level echoes. Overall smooth, even
- Complex/heterogeneous - mixed. Low/medium/high level echoes all within
- Calcified - all hyperechoic with posterior shadowing. Limits evaluation
- Ulcerative - crater like
- Intraplaque hemorrhage - oval anechoic area within the plaque

Thrombosis - acute will be anechoic or hypoechoic and large caliber

Chronic occlusion - decreased vessel size. Hypoechoic to echogenic. Thumping pattern, piston like horizontal motion

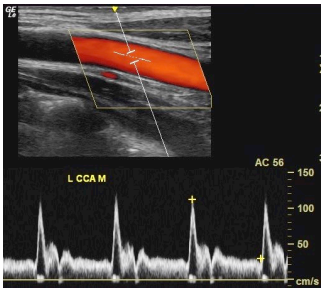
PW Doppler

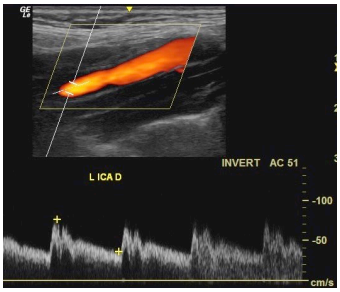
Normal waveform patterns

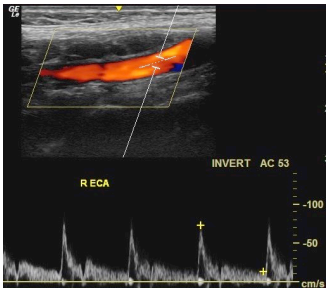
ICA - low resistance

ECA - high resistance

CCA - mixed between the 2







Interpretation

PSV >125cm/s may indicate hemodynamic significant stenosis

*** must also demonstrate all features of stenosis

Main criteria: % diameter reduction is determined by **EDV**

PSV > 125cm/s	Maybe stenosis
EDV <140cm/s	50-79% DR
EDV >140cm/s	80-99% DR

NASCET North America Symptomatic Carotid Endarterectomy Trials

* highest PSV ICA divided by distal CCA

- ICA/CCA ratio ≥ 4.0 ≥ 70% DR

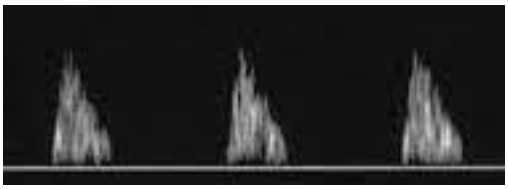
Reasons for over or underestimating disease

Overestimates stenosis - accelerated flow not related to disease
- High cardiac output > bilat and throughout
- Decreases blood viscosity > bilat and throughout
- Tortuous vessels
- Compensatory flow changes

Underestimates stenosis - dampened flow patterns
- Proximal stenosis
- Poor cardiac output > bilat and throughout
- Congestive heart failure

Indirect indications of the presence of disease

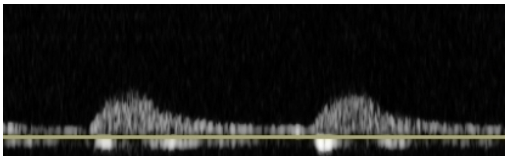
- Abnormally high resistance, absent diastolic



Distal occlusion. If in CCA = ICA occlusion

If possible occlusion found > use power doppler to eval for string sign = near total occlusion

- Dampened tardus parvus like pattern



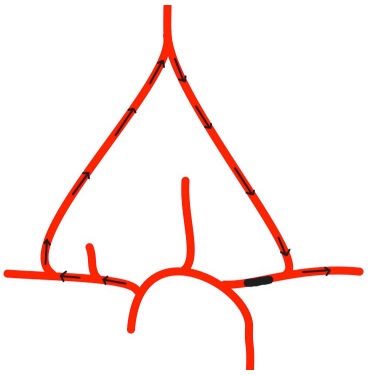
Proximal disease if unilateral

Poor cardiac output if bilateral

- Oscillating or helical pattern (low velocity and to-and-fro)

Brain death

- Subclavian Steal: Proximal subclavian/brachiocephalic obstruction or occlusion causing "steal" of blood from vertebrals. Pt may have VBI symptoms with UE ischemia symptoms and brachial pressures with >20mmHg diff. Lower pressure on same side as disease
  *** most common on left

  Findings
  Retrograde (caudal) flow in vertebral artery = Indicates occlusion of *ipsilateral* prox subclavian or innominate

  Bidirectional vertebral flow (pre-steal) = stenosis of *ipsilateral* prox subclavian or innominate

- Takayasu arteritis
  Usually younger women. Affects larger vessels (may be CCA) "pulseless disease"
  Finding: "donut" vessel

- Temporal arteritis
  Inflammation of superficial temporal artery. Patient % headache
  Findings: "halo" inside vessels

Intraoperative

Very high frequency 12-15MHz "hockey stick" probe

During carotid endarterectomy - acute defects
- Stricture of suture line
- Intimal flaps
- Platelet aggregation
- Residual plaque

Post procedure surveillance

Immediately post op - thrombosis / dissections

6-24 months - neointimal hyperplasia

Post-stenting : stented vessels have normally higher velocities. Cannot use same criteria as regular vessels. Up to 225cm/s is considered WNL.
* Look for signs of stenosis profile

Transcranial Doppler

Capabilities and Limitations

Intracranial stenosis and occlusions
Assess for collateral circulation
Evidence of vasospasm due to hemorrhage

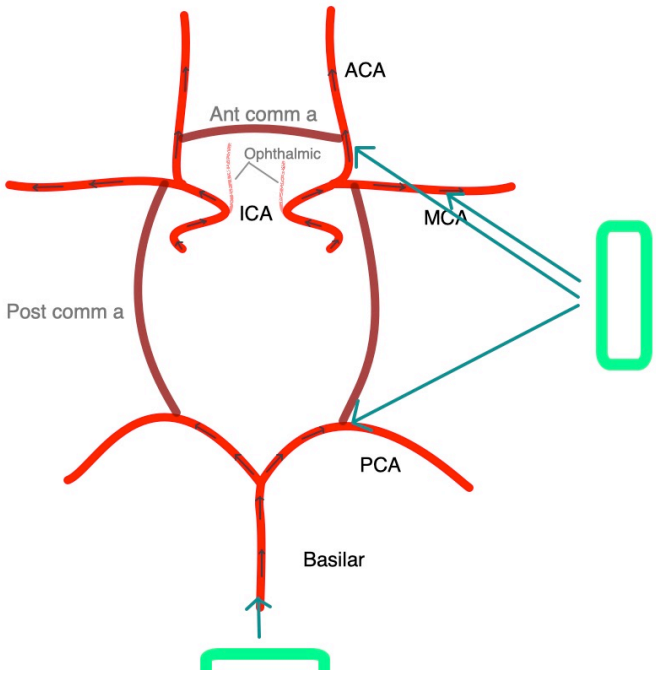
Technique

2MHz non-imaging PW doppler unit
Must be PW for range resolution (depth or location)
0 angle is assumed

Angulation and depth of signal used for vessel identification

Windows:

- Transorbital - through closed eyelid (each side)
- Transtemporal - through temporal (each side)
- Transforamenal - aka suboccipital. Through foramen magnum
- Submandibular (optional)



Criteria

Vessel	Window	Depth	Angle	Direction
ACA	Transtemporal	>60mm	Anterior/Superior	Retro / Away
MCA	Transtemporal	<60mm	Anterior/Superior	Ante / Towards
Terminal ICA	Transtemporal	+/- 60mm	Anterior/Inferior	Bidirectional
PCA	Transtemporal	>60mm	Posterior/Inferior	Ante / Towards
Ophthalmic	Transorbital	<60mm	————	Ante / Towards
ICA syphon	Transorbital	>60mm	————	Bidirectional
Basilar	Transforamenal	70-120mm	Midline	Retro / Away
Vertebral	Transforamenal	60-90mm	RT and LT	Retro / Away

**Normally retrograde means wrong way. In TCD, it means AWAY from the transducer or negative doppler shift.

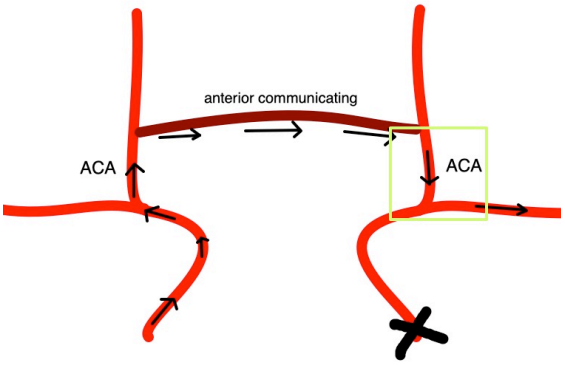
Collateral circulation

In the presence of proximal pressure drop due to stenosis or occlusion of the carotid arteries, collateral pathways flowing through the circle may open. The following may happen when the vessels proximal to circle are occluded and blood is redirected to the necessary vessels supplied by the circle. Includes the communicating arteries (anterior and posterior) and periorbital circulation. Goal: redirect blood towards the ACA/MCA

Supplying vessels will show compensatory flow changes = elevated velocities and low resistance.

- Cross Over

  Contralateral ICA to ACA to ipsilateral ACA *VIA anterior communicating artery*

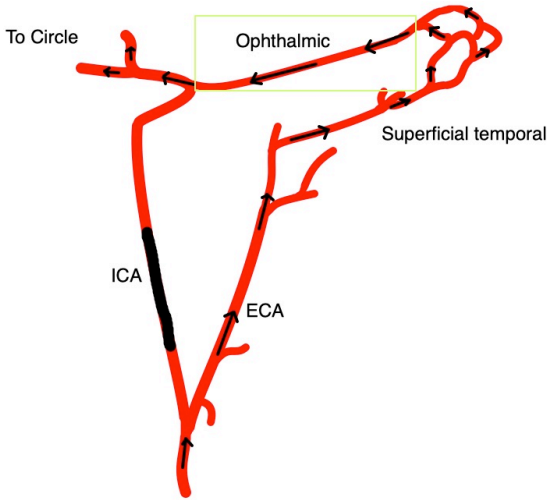


TCD findings: Antegrade flow in the ipsilateral ACA

Carotid duplex findings: evidence of ICA disease/ occlusion. Or abnormally high resistance pattern in ICA (thumping)
Contralateral ICA is the feeding vessel = compensatory flow changes. Higher PSV and low resistance

- External to Internal

  Ipsilateral ECA to periorbital branches. Superficial temporal to *ophthalmic* to ICA



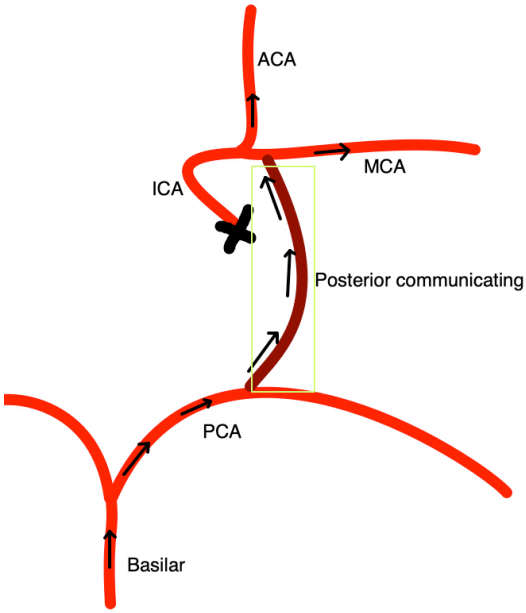
TCD findings: Retrograde flow in ipsilateral Ophthalmic

Carotid duplex findings: evidence of ICA disease. Ipsilateral ECA is feeding vessel = elevated PSV and decreased resistance. "internalized ECA"

Possible scenario:
Distal ICA occlusion > high resistance extracranial ICA
Same side ECA > low resistance

- Posterior to Anterior

  Basilar to PCA to MCA/ACA *via posterior communicating artery*



TCD findings: elevated velocities in the ipsilateral PCA compared to MCA. PSV of PCA 125% greater than MCA
NO flow direction changes

Normal PCA flow = lower velocity than ICA branches. Now will show compensatory flow changes of supplying vessel

Carotid duplex findings: evidence of ICA disease
Vertebrals are feeding vessels = higher PSV and low resistance

<u>Additional findings</u>

Occlusion: absent signals or absent EDV (distal occlusion)

Vasospasms: complication of hemorrhage. High velocity flow in MCA >120cm/s

<u>Intraoperative</u>

MCA monitoring during vascular procedures/surgeries
Changes in flow velocity or patterns indicate micro embolic event. Surgeon may need to modify technique. *** Continuous doppler on MCA at transtemporal window

Alternative Testing

Angiography - same as arterial!

Ionizing radiation to vis contrast material (radiopaque dye) injected into vessels. Catheter inserted in artery - most common CFA. Positioned proximal to vessels to be eval. Dye released and imaged as it goes through arterial tree.

Contraindications: allergy to contrast, poor renal function
Complications: hematomas, nerve damage, pseudoaneurysm, AVF

Interpretation:

FILLING DEFECTS
Stenosis - pinching, narrowing. string sign = near total occlusion
Occlusion - missing vessels. Blunted loss of dye
Fibromuscular dysplasia (FMD) - string of beads

Calculating % diameter reduction

d = residual lumen
D = normal lumen

1. d/D = what is left over
2. turn into %
3. Subtract from 100

ex- 2mm residual lumen and 8mm true lumen

2/8 = 0.25 or 25%

100-25 = 75% Diameter reduction

MRI/MRA

Radio frequency energy and magnetic fields, produces multiplanar views.
MRI : shows cerebral infarction
MRA : in cerebro, requires contrast

Contraindications: metallic objects, pacemakers.

CT/CTA

Ionizing radiation produces cross sectional images. CTA requires contrast.
CT : cerebral infarctions, hemorrhages, tumors, masses, etc
** Gold standard for acute stroke patients

Treatment

Acute stroke standard treatment: IV tPA Tissue Plasminogen Activator.
Thrombolytic (breaks down the clot)

Chronic occlusive disease treatment:

Medical management = lifestyle changes and drug therapy

Control of risk factors
- Stop smoking
- Reduce HTN
- Reduce cholesterol (meds/diet)
- Control diabetes

Aspirin : anti-platelet drug, reduces thrombotic activity

Depending on severity of disease and symptoms, may treat surgically…

Surgical therapy

- Endarterectomy - removal of plaque and intimal lining. More common method of treatment
  Possible future complication is neointimal hyperplasia 6-24months. If causing stenosis = may regress over time. If does not improve and becomes hemodynamically significant, may need to be stented
- Stent - during angioplasty. For neointimal hyperplasia or atherosclerotic stenosis. Plaque stays in place, just widens vessel and holds it open
  *** Does not use same criteria for stenosis as stented vessels have higher velocity <225cm/s may be within normal limits
- Total occlusion - nothing. Generally collaterals already in place and functional
- Bypasses - very rare!