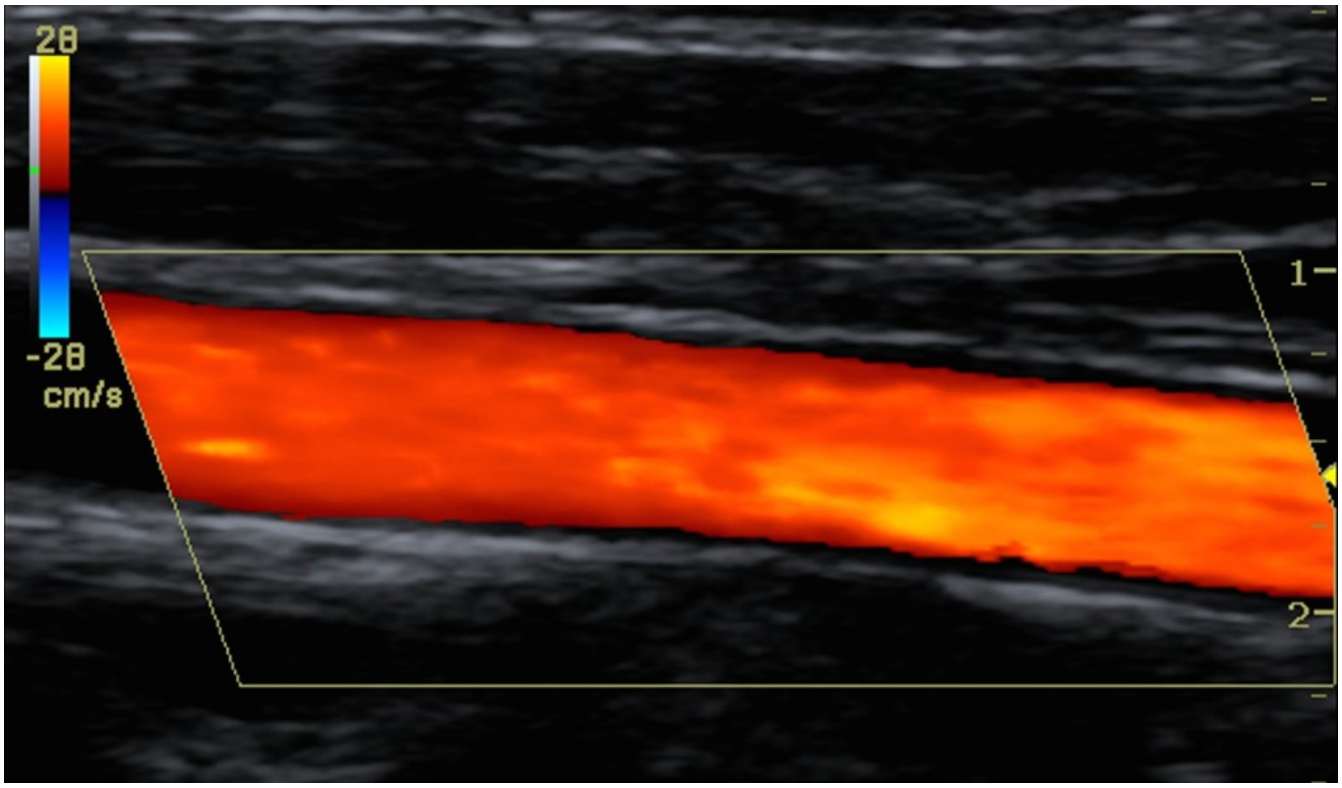
Study guide to

Vascular Ultrasound Registry Review



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www.myultrasoundtutor.com
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Vascular Registry Review

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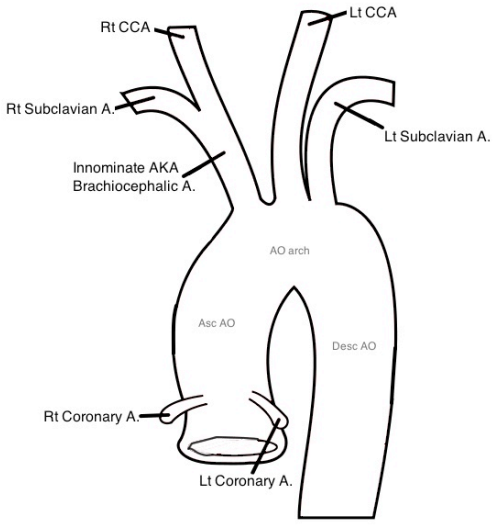
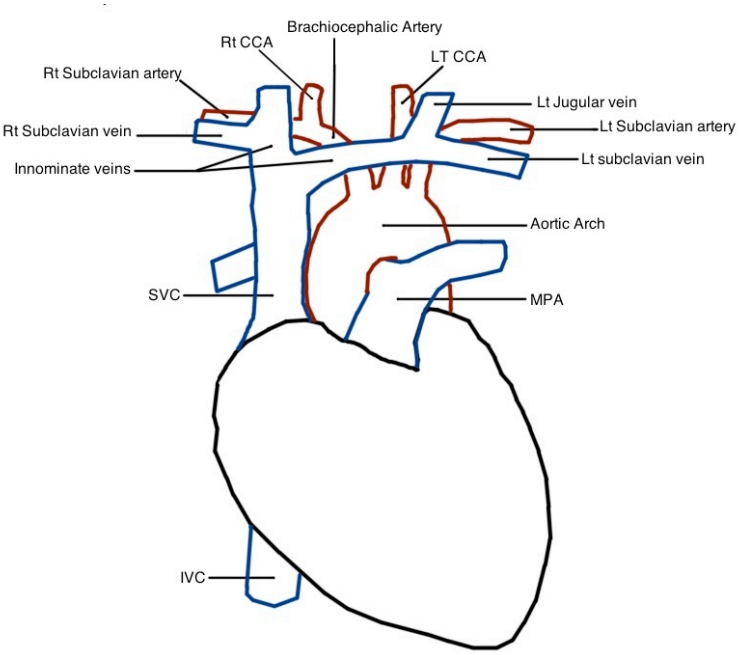
ARTERIAL

Peripheral Arterial Anatomy

Originates at aortic arch

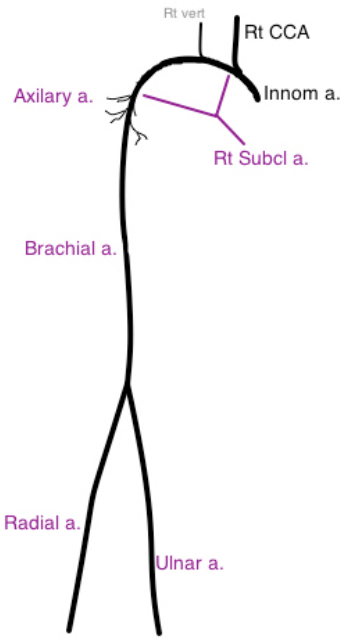
- 1st - Innominate/Brachiocephalic a,..bifurcates into Rt Subclavian and Rt CCA
- 2nd - Lt CCA
- 3rd - Lt Subclavian a



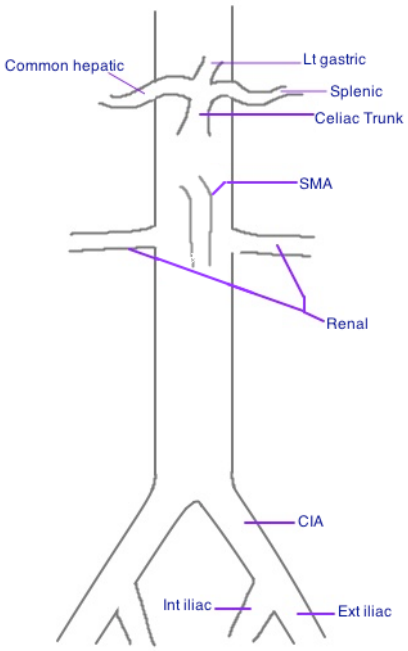


Upper Extremities

Rt subclavian originates at innominate. *Lt subclavian* originates directly from aortic arch.
Subclavian terminates at outer/lateral border of first rib and becomes *axillary*.
Axillary gives off 8 branches then becomes *brachial*.
Brachial terminates at the bifurcation of *radial* and *ulnar* arteries.
Radial term at Deep palmar arch and Ulnar at Superficial

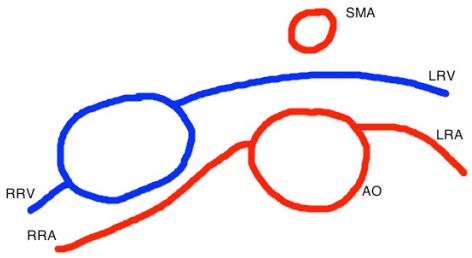


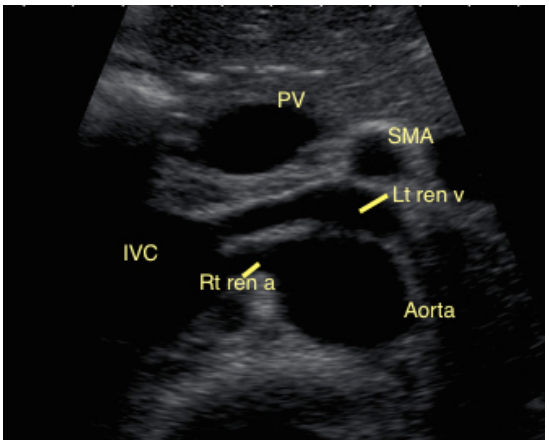
Abdominal Aorta

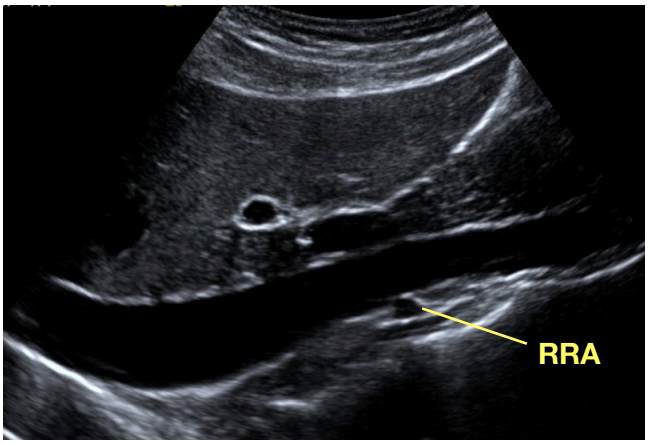


Originates at crus of the diaphragm.
Superior to Inferior branches from aorta
1st branch: *Celiac* trunk. Celiac gives rise to *common hepatic, lt gastric, splenic a.*
In transverse view (seagull sign) = only see common hep and splenic vis.
2nd: *Superior mesenteric a.* (*SMA*)
3rd: *Renal arteries* arise approx 2cm distal to take-off of the SMA on anterolateral surface of aorta.
Aorta terminates at bifurcation of RT/LT CIA

Renal vessel anatomy in cross section:
LRV posterior to SMA and anterior to AO
RRA posterior to IVC
Renal veins run anterior to renal arteries
Best landmark for the Lt renal artery is the Lt renal vein as it crosses posterior to SMA and anterior to AO.

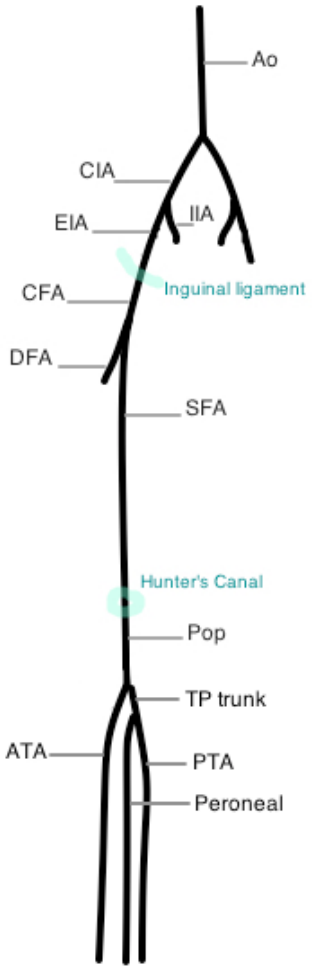


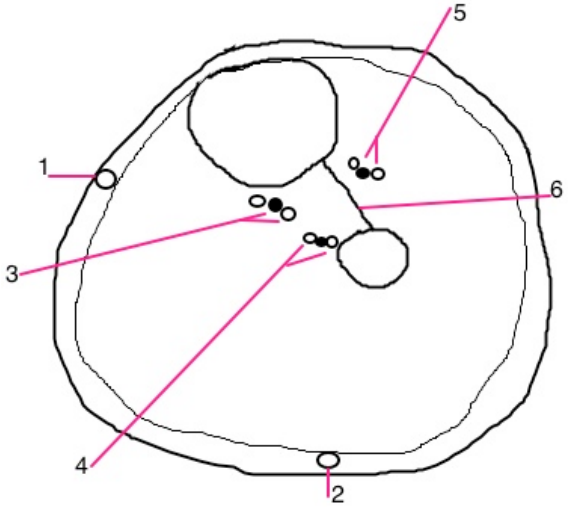




Lower Extremities

Aorta terminates at the branches of *Common Iliac a.* CIA divide into *External and Internal Iliac a.*
IIA (hypogastric) provide blood to pelvis, pelvic muscles and organs.
EIA provides blood to lower extremities. Terminates at inguinal ligament and becomes *Common femoral a.*
CFA then bifurcates into *Superficial femoral a.* and *Deep femoral a.* SFA travels anteromedial to the DFA (deeper/ posterolateral course).
SFA terminate at level of Hunter's canal (AKA adductor canal) when it becomes the *Popliteal a.*
"Trifurcation" vessels = Not really a trifurcation!!
** The Pop bifurcates first into *Anterior tibial a.* and *Tibeoperoneal trunk.* TP trunk then bifurcates again into *Posterior tibial a.* and *peroneal a.*
PTA courses anteromedial and Peroneal posterolateral.
ATA course anterolateral.





Key to cross-section diagram:

What level? Must be level of calf. 2 bones and 3 sets of vessels with 2 veins each.

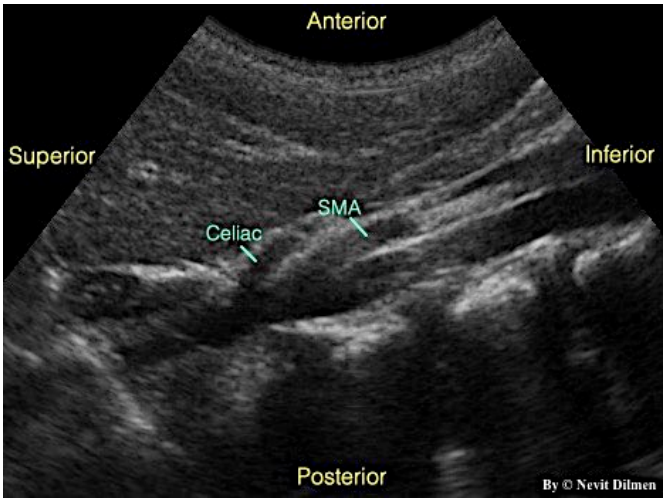
1. GSV
2. SSV
3. Post tibials
4. Peroneals
5. Ant tibials

✳ Relational anatomy

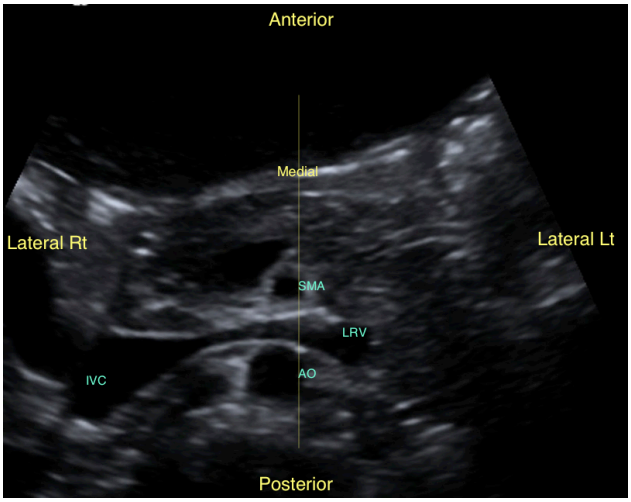
Anterior- Closer to the front of body AKA superficial
Posterior- Closer to back AKA deep
Superior - Towards the head AKA Cephalad
Inferior - Towards the feet AKA Caudal
Medial - Closer to middle
Lateral - Towards the side
Proximal - Closer to heart, flow comes from
Distal - Away from heart, flows goes to

Example: SMA originates inferior to celiac artery and runs caudally anterior to the aorta. The celiac is superior to SMA.

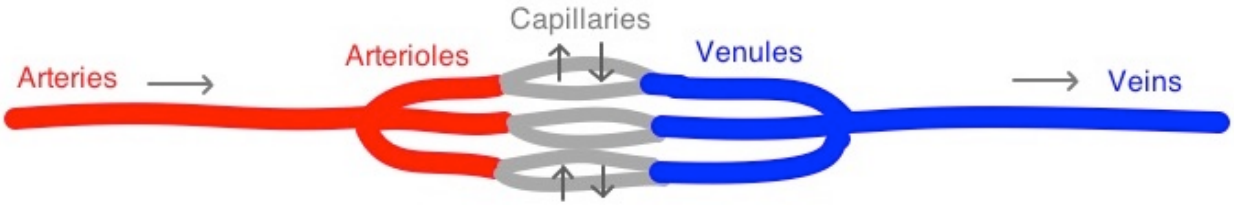
Sagittal



Transverse



Functional level of circulation



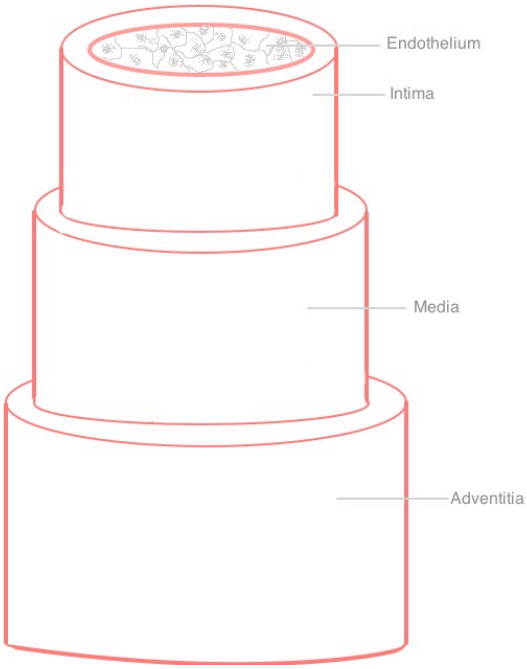
Arteries >> Arterioles >> Capillaries >> Venules >> Veins

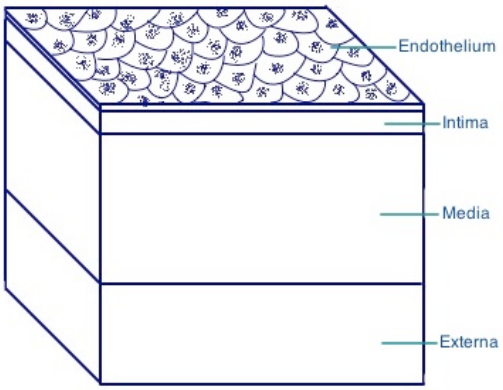
Arterioles : Smallest arteries. Vessels of resistance

Capillaries : Vessels of microcirculation. Walls are 1 cell layer thick
*** most vital part of circulatory system

Venules : Smallest veins.

Microscopic Anatomy





Intima: Thin innermost, covered by endothelium

Media: Thickest layer, smooth muscle and connective tissue

Externa/Adventitia: Outer, fibrous connective tissue. Contains *vasa vasorum*, tiny vessels that supply blood to vessel walls.

Hemodynamics

The study of blood moving through the circulatory system

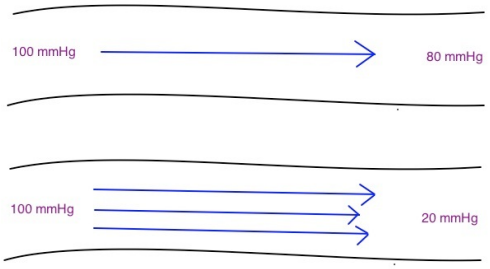
Blood flow depends on 2 main things
Pressure gradient and Resistance

Greater pressure gradient = greater flow (directly related)
Increased resistance = decreases flow (inversely related)

Pressure gradient The driving force behind flow

Pressure gradient is the difference of pressure from high to low

Greater the difference from hi to lo = more flow



Arterial flow is *pulsatile* because it is driven by the pressure gradient from cardiac cycle.

Systole LV contracts (hi pressure) creating pressure energy that pushes blood to lower pressure regions (vessels). Systole creates the greatest pressure gradient and therefore the greatest amount of volume flow ejected = *STROKE VOLUME*
During cardiac contraction = *potential or pressure energy*

Diastole Heart relaxes. Blood continues to flow through arterial tree because it is already in motion *(kinetic energy)*. How much flow will continue during diastole will depend on resistance

Total sum of energy

Pressure (potential) energy - created by pumping action of heart

Kinetic energy - energy of something already in motion. Potential turns to kinetic as blood moves along system.

Hydrostatic (gravitational) energy - weight of column of blood.
Supine = 0mmHg at ankle (all same level of heart)
Standing =100mmHg at ankle (-50mmHg at raised hand)

Resistance

Increased resistance will decrease volume flow

Resistance is determined by
- Vessel size length and diameter
- Thickness of blood (viscosity)
- Outside forces upon vessel (elasticity of walls)
- Organization of vascular network (tortuous vessels)

Biggest effects to resistance occur when there is a change of vessel diameter or radius

Poiseuille's Law describes relationship of *resistance*, *pressure gradient* and *flow*

$$Q = \frac{\Delta P \pi r^4}{8 \eta l}$$

Q = volume flow
ΔP = pressure gradient
r = radius
η = viscosity
l = length

Increase pressure gradient = Increase volume flow

Increase resistance = Decrease volume flow

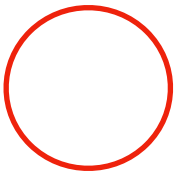
Decreasing diameter would increase resistance and decrease flow

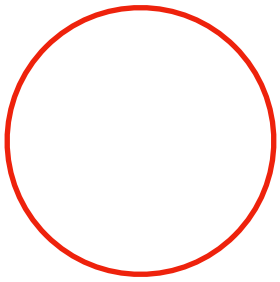
Flow volume directly proportional to diameter. Notice how radius is to the 4th power.
That means small changes in radius result in big changes to flow.
Decreased radius = increased resistance = decreased volume
Flow is inversely related to length and viscosity

Resistance and US

Resistance is determined by *where the flow is traveling to*. Volume flow can be adjusted by the body by changing the distal resistance. Arterioles have vaso-motor tone, which means they can change their size to meet the demands of the vascular bed. Change resistance = change volume flow

If an organ needs constant forward flow = vaso-dilated vascular bed. That means the arterioles are bigger. Bigger means lower resistance. (ie - ICA)
If it does not require constant perfusion = vascular bed will be vaso-constricted. That means smaller and smaller means higher resistance. (ie - ECA)





VASO-CONSTRICTED
Smaller radius
Higher resistance
Less volume
Less DIASTOLE

VASO-DILATED
Larger radius
Less resistance
More volume
More DIASTOLE

ECA
Fasting SMA
Infrarenal aorta
Peripheral arteries:
CFA, SFA, Subcl a, Brach a, etc

ICA
Post prandial SMA
Suprarenal aorta
Organ arteries:
Celiac, Hepatic, Splenic, Renal

We can tell resistance by how much diastolic flow there is.
Hi resistance = little or no diastolic flow, dias flow reversal "stop and go"
Lo resistance = more diastolic flow. Constant forward flow, non-stop

Causes of vaso-dilation and vaso-constriction

Purpose = to meet the demands of blood supply

Vaso-constriction	Vaso-dilation
cold	heat
stress	exercise
smoking	stenosis / distal ischemia

Vaso-dilation will occur when the body wants to get more blood

In the case of obstruction/stenosis

Arterial flow volume comes from cardiac output (how much heart pumps) so it cannot change. We can't tell the blood to slow down or stop when we have obstruction.

** a stenosis is NOT same as vaso-constriction

$V = \frac{Q}{A}$

This is described by the Law of Conservation of Mass

When vessel size decreases and volume is constant = *Velocity must increase*

Bernoulli Effect describes relationship of pressure and velocity when radius changes.
Pressure and velocity are inversely related

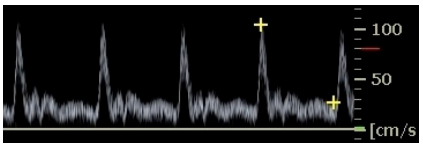
↑ **Velocity** ↓ **Pressure**

When there is a narrowing (stenosis), the velocities increase and the pressure drops

Bernoulli effect is also what's responsible for flow separations
Due to a larger vessel size, the velocity decreases. When velocity decreases, pressure rises. The increase in pressure at this point causes a momentary flow direction change. Most often occurs at the carotid bulb or bypass anastomosis.

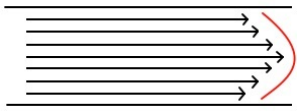
Types of blood flow

Laminar
Normal flow that moves in concentric streamlines or layers. Organized. Laminar flow seen by the quality of the spectral waveform.

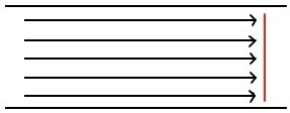


Spectral window indicates laminar flow. The PW is sampling one, neat layer which moves at its own speed. So the waveform displays a neat, organized flow pattern

Parabolic: most common type of flow. Highest velocities found in the center of vessel and lowest next to wall. Parabolic shaped flow



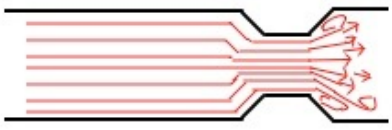
Plug: Found at origin of vessels. All layers move at the same velocity



Turbulent
Abnormal, disorganized flow. Flow patterns become disturbed and form eddies or swirling patterns. Occurs when we have a sudden change in resistance and elevated velocities. Often seen distal to stenosis or tortuous vessels

Reynold's #
Predicts when flow becomes disorganized or turbulent

CRITICAL VALUE = >2000



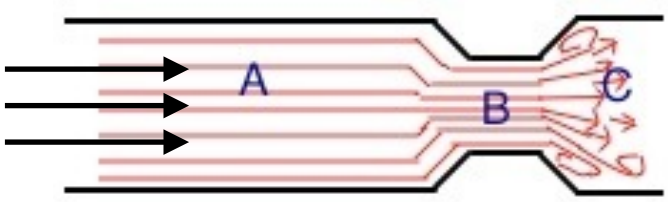
The 2 main factors are radius and velocity. Both DIRECTLY RELATED

The larger the vessel and higher the velocity will increase the Reynold's # and more likely there will be turbulent flow

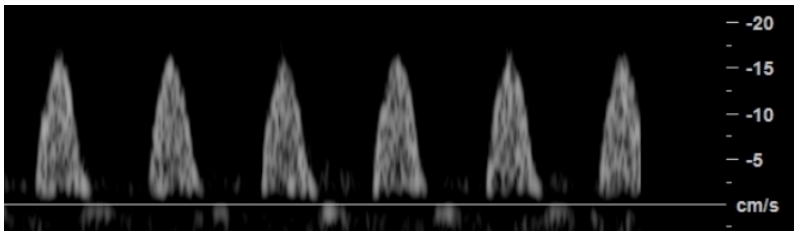
"Post-stenotic turbulence" = AFTER stenosis when vessel diameter is larger but velocity is increased. *Spectral broadening* indicates turbulence.

Stenosis profile

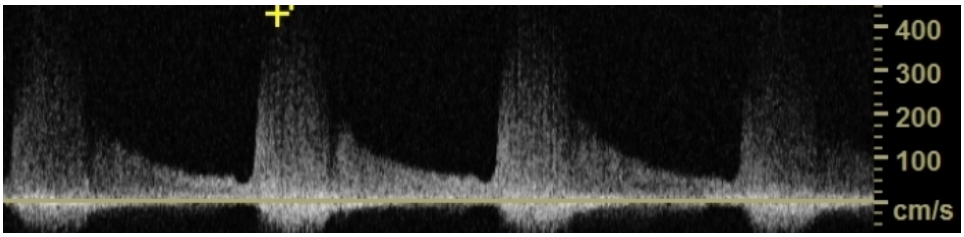
How stenosis affects blood flow. Hemodynamically significant means the stenosis has reached at least 50% diameter reduction and has altered flow patterns.
50% diameter = 75% area reduction



A. **Proximal to stenosis**. Decrease in diameter upstream = increase in resistance. Going towards the obstruction, like going towards traffic jam. "stop and go" pattern
Hi resistance waveform (less diastolic) or absent diastolic component



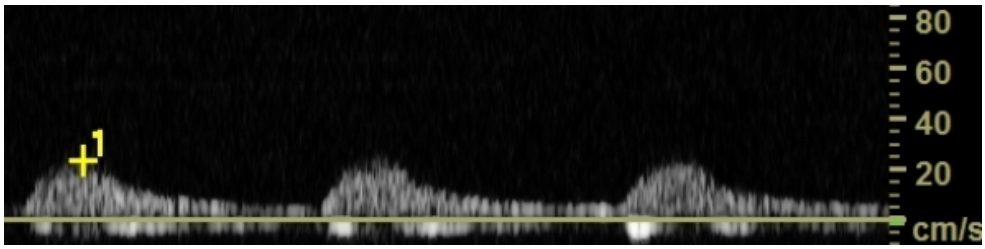
B. **At the stenosis.** Elevated PSV and EDV through the narrowed section. Velocity must increase when area decreases to maintain volume flow. Increase to velocity = decrease in pressure (Bernoulli). Highest velocity



C. **Distal to stenosis.** Turbulent flow patterns. Larger radius with the elevated velocities will increase the likelihood of turbulent flow patterns (Reynolds). 'post-stenotic turbulence'. Lo resistance waveforms since vessel widened.

Flow may also be dampened distal to severe disease

Tardus parvus = monophasic, continuous with rounded peak and slow upstroke >> flow change is further distal to obstruction.
Indicates the presence of proximal disease

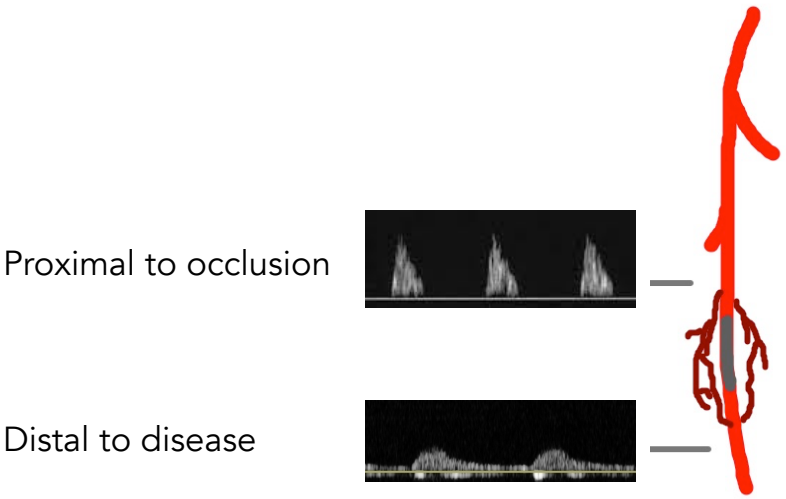


Proximal or Distal???

Depends on if we're talking about where the disease is or where the flow change/waveform is found.
Where's the disease?? *OR* Where's the waveform??

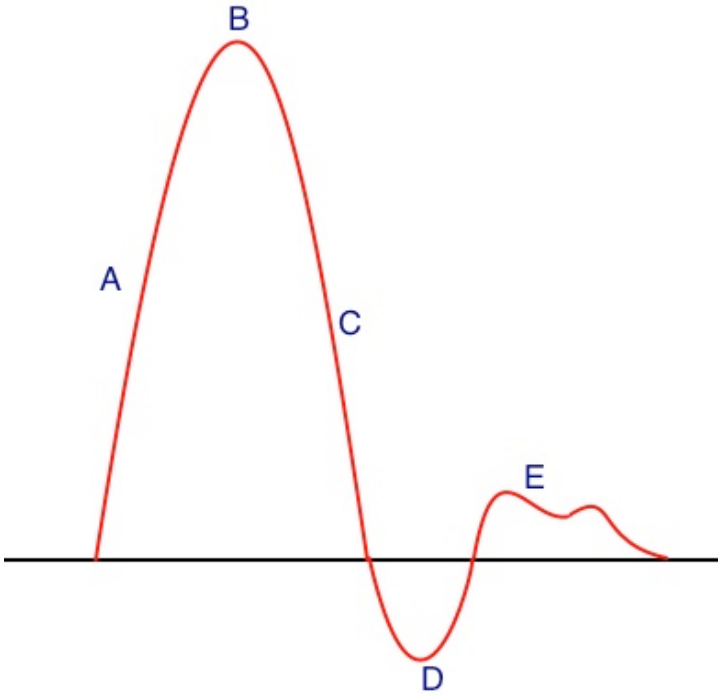
Proximal to stenosis = Distal obstruction

Distal to stenosis = Proximal obstruction



Cardiac effects on peripheral flow

Peripheral flow reflects the pulsatility of the cardiac cycle



A - Early systole

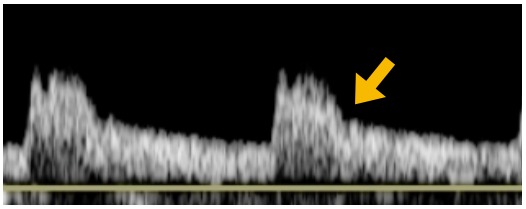
B - Peak systole - Maximum forward flow. Aortic walls distend = potential energy

C - Late systole - Aortic valve closure. Temporary flow reversal due to high peripheral resistance. (like ocean wave hitting shore = bounces back)

D - Early diastole - Flow moves forward again.

E - Late diastole - Potential to kinetic energy, flow keeps moving forward

Low resistance flow shows continuous flow (no bouncing back) because there is little resisting it. Instead, in late systole, there will be dicrotic notch to represent aortic valve closure.



Additional cardiac effects

Bilateral flow alterations will be seen in conditions that affect the left side of heart or aorta (ex- poor cardiac output or aortic stenosis)

High cardiac output - Elevated velocities with normal waveform contour BILAT

Poor cardiac output - Low velocity, dampened waves with round peaks BILAT

Aortic regurgitation/insufficiency - Pulses bisferiens (double peak pulse)

Intra-aortic ballon pumps and ventricular assist devices - all arterial waveforms systemically will be affected

Arterial Disease and Clinical History

Chronic Peripheral Arterial Disease

Atherosclerosis - hardening and thickening of arterial wall (intima and media), eventually forming plaque and stenosis.

Most common type of arterial disease
Most common location: Ds SFA in general population / Tibial vessels in diabetics

Risk Factors:
- Diabetes
- Hypertension
- Hyperlipidemia
- Smoking **most contributing factor

Age, family history, male gender

Symptoms:

- Claudication - MODERATE disease. Pain and muscle fatigue with activity. Pt must stop to rest. Pain is relieved upon rest

  Symptom location is constant and distal to level of disease
  Example - Pop disease = calf claudication

  Pseudo-claudication = Not vascular related. MSK or neurogenic. Patient will have similar pain in extremity but without muscle fatigue or needing to stop.

- Rest pain - SEVERE disease. Pain in feet and heels at night in bed or when limb is same level as heart. Relieved upon dependency.

- Tissue loss/Necrosis - MOST SEVERE. Ulcers and gangrene in most distal part of limb like toes or bony regions like top of feet.

Physical signs:

Trophic changes: dry skin, loss of hair, thick toenails
Pale skin, cool to touch
Ulcers - dry, deep, painful, toes, tops of feet

Diminished or absent pulses
Dependent Rubor - Pale when elevated, red when dependent (SEVERE)
Delayed capillary filling - >3sec after pressing pulp of digit (SEVERE)
Bruits - sound you can hear (auscultate) when there is high velocity, turbulent flow. Does not definitely mean they have disease. Absent bruit does not mean they do not have disease.

Acute Arterial Occlusion

Acute = sudden onset.

6 P's

Pain
Pallor
Polar
Pulselessness
Paresthesia
Paralysis

- Thrombus - caused by trauma or dissection

  Penetrating trauma (gunshot or stabbing) causes dissection. Dissection leads to the thrombosis of vessel

- Embolism - air, liquid, solid that travels and lodges distally

  Most common source is the heart.
  Pt known to have aneurysm (thrombus can break off) or PAD (plaque) also at risk of embolism.

  Example: Blue toe syndrome caused by embolism, usually big toe.

Cold Sensitivity/Raynauds phenomenon

Pt experiences symptoms of ischemia in hands or feet when exposed to cold. Symptoms include: blue or white discoloration, pain, tingling, numbness

- Primary Raynauds:

Young women with bilateral symptoms. Experience vasospasm when cold or emotionally stressed. Functional disease

- Secondary Raynauds:

Secondary = caused by something else (like side effect). Pt has existing fixed arterial disease. May not have ischemia symptoms at rest. When cold, vasoconstriction worsens ischemia. Most likely unilateral and pt fits chronic PAD history.

Arteritis

Inflammation of vessel wall.

- Takayasu: young asian women. Affects larger vessels such as aorta. "Pulseless disease"
- Buerger's disease AKA thromboangitis obliterans. **Most common arteritis**

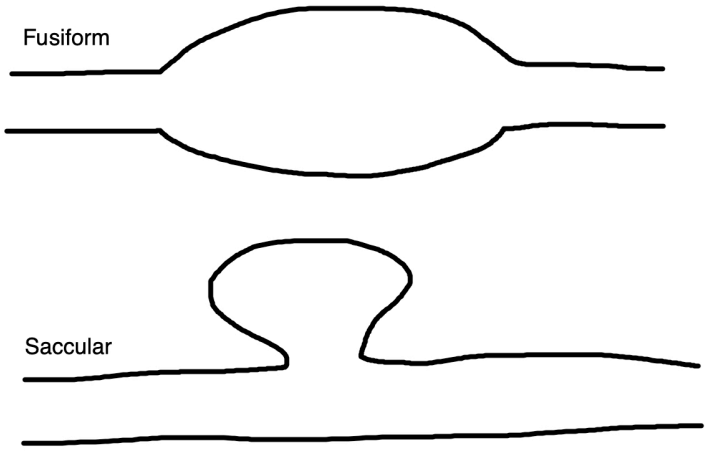
  Young men (<40yo) that are heavy smokers. Affects the smallest most distal vessels (digits).

  Symptoms: rest pain, gangrene, ulcers
  NOT claudicators (larger arteries going to muscle are not affected)

Aneurysm

True: dilatation of all 3 layers of the wall.

Symptoms: bounding pulse. AAA: abdominal/back pain



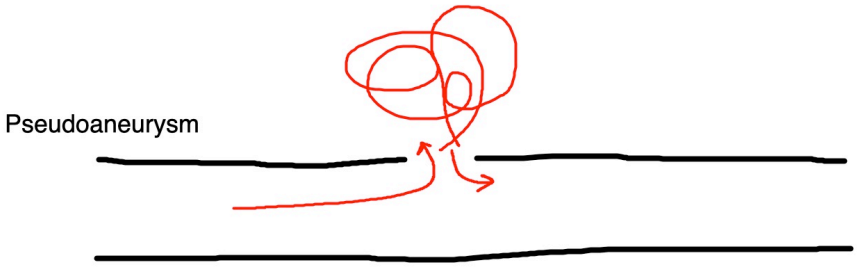
Most common location: Infrarenal aorta
Most common cause: Atherosclerosis
Most common type: Fusiform

Most likely complications:
AAA: Rupture (especially >5cm)
Peripheral: Embolization

False: not all 3 layers are dilated.

Weakening of layers causing intimal flap or tear. Flow goes into both "lumens". Very dangerous- high risk of rupture. Most often in thoracic aorta in hypertensive pt with severe back / chest pain

Pseudoaneurysm: AKA pulsating hematoma. Puncture of all 3 layers most often post procedure such as catheterization or angio. Must have communicating neck or channel to confirm diagnosis.



MISC

- Coarctation: congenital narrowing of aortic arch. Diminished pulses and hypertension in younger people
- Popliteal entrapment: compression of pop by gastrocnemius muscle. Found in younger athletic men experiencing intermittent claudication
- Thoracic outlet syndrome: compression of nerves or blood vessels by shoulder, ribs, and muscles
- Compartment syndrome: compression of artery by swelling trapped inside fascia. Most often anterior tibial region

Arterial Testing

For all types of testing, need to know the following:

Capabilities and limitations : What it's for and what it's not for

Physical principles : How it works

Technique : How to do it

Interpretation : How to read it, what it means

The interpretative criteria will either be qualitative or quantitative, or both

Qualitative VS Quantitative

Qualitative "Quality"	Quantitative "Quantity"
How it looks	How it measures
NOT measurable	Velocities, ratios, indices
Waveform contour	% Diameter reduction

Non-imaging testing includes CW, pressure testing, and plethysmographic studies.

CW doppler analysis

Capabilities and limitations

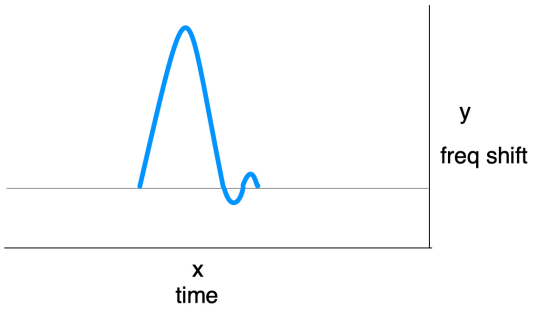
Presence/absence of arterial occlusive disease
Approximate level of disease, NOT exact location or severity
Tech dependent

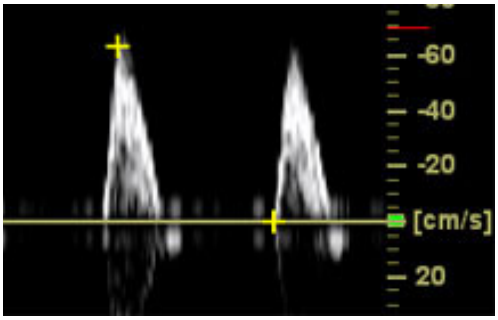
Physical principles

CW = requires 2 crystals. 1 transmitting/1 receiving
Non-imaging, only shows waveform
No depth specificity AKA range resolution, unable to choose location of signal

Types of recording

- Audio - doppler shifts are within audible range
- Analog - zero-crossing frequency meter on strip chart recorder
  ESTIMATES freq shifts, not very sensitive
- Spectral analysis - displays TRUE or INDIVIDUAL freq shifts according to time. More sensitive.





Technique

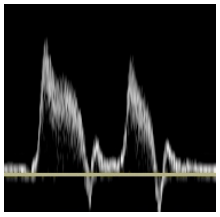
Supine - limbs same as heart
8-10 MHz CW probe
Probe angle 45-60 degrees to skin surface

Segmental evaluation - groin (CFA), thigh (SFA), pop, ankle (PTA/DP)

Interpretation

Mainly qualitative

Normal: Triphasic or Biphasic



Abnormal: Monophasic or monophasic, continuous.

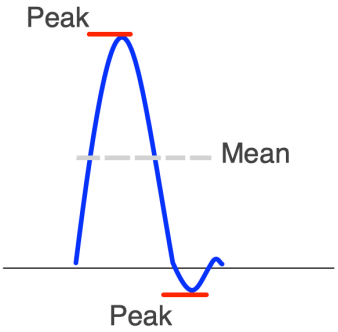
** Deterioration in signal contour from one level to the next =

Disease between those levels

Quantitative

Pulsatility index = measure resistance

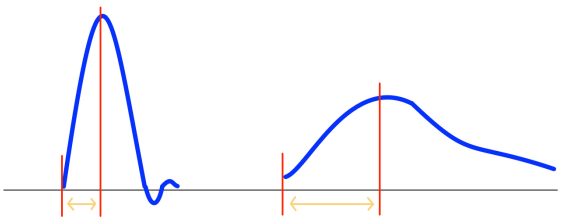
$$\frac{\text{Peak to peak freq}}{\text{Mean freq}}$$



Normal PI increases as you go down the limb.

Any drop in PI between levels = Disease between those levels

Acceleration time = time from beginning of systole to peak AKA upstroke



Inc accel time >133 msec

= Proximal (inflow) disease

When accel time is not affected, there is no proximal disease

Segmental Pressure Testing - Lower Extremities

Capabilities and Limitations

Presence/absence of disease
Approximate level and severity
Not exact location or stenosis vs occlusion

Medial calcinosis = incompressible vessels. Often seen in diabetics and end stage renal disease. Pressures will be inaccurately high

Pt with dialysis fistula, stents, DVT, or hx of lymphedema cannot have this exam

Any surgical modifications = must consult vascular surgeon before proceeding

Technique

Pt in basal state (resting for at least 20 minutes)
Pt must be supine (no hydrostatic pressure)

CUFFS
- Bilat brachial cuffs
- 4 cuff method = 2 thighs, calf, ankle
- 3 cuff method = 1 large thigh, calf, ankle

Width of cuff = 1.2x greater than diameter of limb
If too wide = falsely low pressure
If too narrow = falsely high pressure

Obtaining systolic pressure:
- Use CW doppler to locate arterial pulse distally (ex-PTA or DPA)
- Inflate cuff to suprasystolic pressure (30 higher than last audible signal)
- Slowly deflate
- Record pressure when pulsation returns

Pressures must be taken distal to proximal = ankle > calf > thigh
**** use higher of 2 ankle pressures on each leg to complete rest of study*

Interpretation

Calculate Ankle/Brachial Index ABI

Ankle pressure divided by HIGHEST brachial pressure.

Normal ankle pressures should be about the same as the brachial.

In presence of arterial disease = pressures drop.
Ankle < Brachial indicates there is disease. ABI would be <1.0
Ankle > Brachial would give an ABI >1.0 = No disease proximally

ABI >1.3 indicates the vessels are likely incompressible = *NOT accurate*

ABI criteria

1.0	Normal
0.9-1.0	WNL/Minimal disease
0.8-0.9	Mild disease
0.5-0.8	Moderate disease (claudication)
<0.5	Severe disease (rest pain)

Segmental criteria

Pressures DROP abnormally distal to disease

1. Compare thigh pressure to highest brachial.
   NORMAL thigh pressures depending on study type…
   3 cuff : thigh should same or higher than brachial
   4 cuff : high thigh should be 30 or more higher than highest brachial

2. Look for drops of 30 or more between consecutive vertical levels.
   <30 difference between levels is NORMAL. 30 or more drop means disease between those levels.

An increase in pressure is not significant! Arterial obstructive disease will always **decrease** the pressures distally.

Exercise Testing

Purpose: *Produces physiologic stress on circulation and reproduce ischemia.* Allows to distinguish true vs pseudo claudication

Contraindications: difficulty walking or breathing, severe HTN, cardiac

Technique: constant load treadmill (unchanging)
<12% grade and 1.5 mph for a max of 5 min

Leave ankle cuffs on. After exercise, repeat ankle pressures

NORMAL: No change or increase in ankle pressures

ABNORMAL: Decrease in ankle pressure

If abnormal: repeat ankle pressures every 2 min until return to resting

If returns <6 min = single level disease

If returns >6 min = multi level disease

Reactive hyperemia

For pts that cannot do treadmill

Purpose: *Produces ischemia and distal vasodilation*. Cutting off the flow forces the body to vasodilate. Then when the cuff is released we see how the 'hyperemia' (increase in flow) reacts = how quick it gets down

Technique: Inflate thigh cuff to suprasystolic pressures 3-5min
Release cuff and record ankle pressures

NORMAL: transient drop in pressure that returns to resting within 1 min (<34%)

ABNORMAL: Big drop in pressures

<50% drop = single level

>50% drop = multi level

***hyperemia is prolonged in the obstructed limb
= takes longer to get down to the bottom because of the blockage

Segmental Pressure Testing - Upper Extremities

Capabilities and Limitations

Same as lower extremities

Technique

CUFFS

Brachial cuff (obtain brachial pressure using brachial at antecubital fossa)
Forearm cuff (obtain radial and ulnar pressure from same cuff)
Same principles as legs.

Interpretation

Pressures DROP abnormally distal to disease

1. Compare both brachial to each other
   Normally they should be within 20mmHg of each other . If greater than 20 diff, proximal disease on arm with lower pressure

2. Look for drops of 20 or more between consecutive vertical levels.
   20 or more drop means disease between those levels. Always compare up. Radial to brachial. Ulnar to brachial

- Remember: Arterial obstructive disease will **decrease** the pressures. Disease is PROXIMAL to the lower pressure.

Allen test

Purpose: evaluate patency of the palmar arch

50% = incomplete palmar arch. May be used to confirm usability of radial artery in possible surgical modifications (harvesting or hemodialysis access graft)

Technique

Apply pressure to radial artery to occlude it
Make fist. Hand will turn pale as blood is drained from hand
Open hand maintaining pressure on artery

Interpretation

Standard : color returns to normal
Modified : PPG waveforms are maintained throughout compression
Repeat with ulnar artery compression

Plethysmography

Capabilities and Limitations

Usually performed with segmental pressure study
Evaluates functional aspects of disease
**Not vessel specific. Can underestimate disease

Physical principles

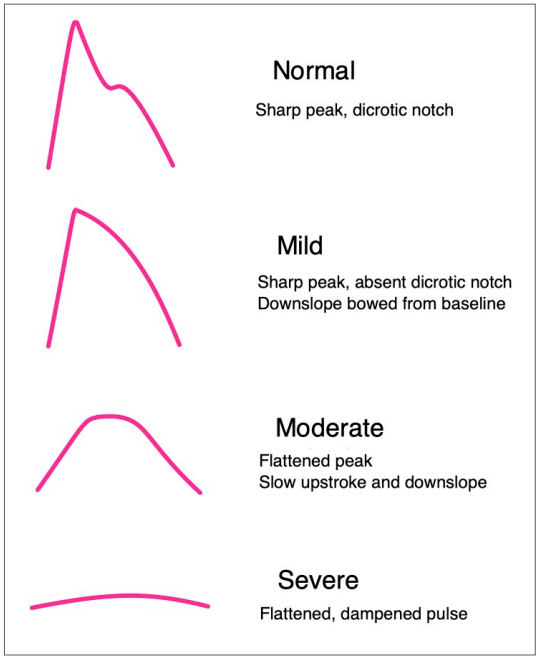
pneumo/true/air plethysmography = uses pneumatic cuffs (BP cuffs with special sensor) to detect changes in volume

When there is a volume increase in systole, sensor detects increase in volume and demonstrates a peak on PVR (pulse volume recording). When the volume drops in diastole, there will be less pressure on the cuff so a downslope will be recorded on PVR

PPG - photoplethysmography. Not TRUE
Small sensor used on digit sends infrared light and the capillary pulsations reflect the light.

Technique

Use same cuffs as segmental pressure study.
Cuff inflated to 40-65mmHg (not to occlude vessel)



Interpretation

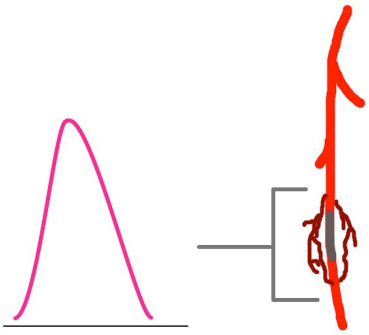
Qualitative only. Based on waveform contour!

CHANGE IN CONTOUR BETWEEN LEVELS = DISEASE

All plethysmography is interpreted with this criteria. No matter what extremity or body part. It is all the same.

We do not use terms like triphasic, biphasic, or monophasic. Cannot be described as high resistance/ low resistance

Since PVR is not vessel specific, it can underestimate the severity of disease in the presence of collaterals. It detects the overall volume of blood in the limb at that level. If there are collaterals, the volume will be normal.



A high amplitude "fair" PVR contour in the presence of abnormal pressures = severe disease but presence of collateral development.

Digital Pressure and PPG

Capabilities and Limitations

Same as plethysmography
Useful in patients with cold sensitivity and medial calcinosis

Physical principles

Same as pressure studies/plethysmography

Technique

Obtain bilat brachial pressures
Use PPG to obtain digital pressures (when pulsation returns record pressure)

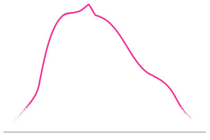
For Raynauds - *Cold stress:*
1st perform at rest
2nd soak in ice water for 3min or to tolerance
Obtain pressures and waveforms

Interpretation

Finger FBI	Normal >0.8
Toes TBI	Normal >0.6

Same criteria as above to evaluate for obstructive disease.
Dropped pressures and dampened waves = fixed arterial disease (Secondary Raynauds)

Primary Raynauds = normal resting waves and pressures.
After cold stress, "Peaked pulse" = Functional disease



Penile Pressure and Testing

Capabilities and Limitations

Evaluate for vasculogenic impotence (erectile dysfunction caused by vascular inflow or outflow)

Example: Leriche syndrome

Leriche syndrome

Terminal aorta obstruction
Impotence, hip and thigh claudication

Risk factors of chronic PAD. May have additional PAD clinical findings

Physical principles and Technique

Pressures and PPG waveforms (same as digits)
Obtain ankle and brachial pressures

Interpretation

Penile/Brachial Index *Normal ≥ 0.65*

Obtaining ABI helps to locate disease.
Normal ABI with abnormal PBI = disease isolated to internal iliac vessels
Leriche syndrome = abnormal ABI bilaterally and abnormal PBI

Post injection duplex > injection causes erection. Velocities documented to look for abnormal changes

Dorsal venous vel >4cm/s = venous leak

Transcutaneous Oximetry $TcPO_2$

Evaluates wound healing potential and for determining amputation level
Special sensors measure oxygen tension

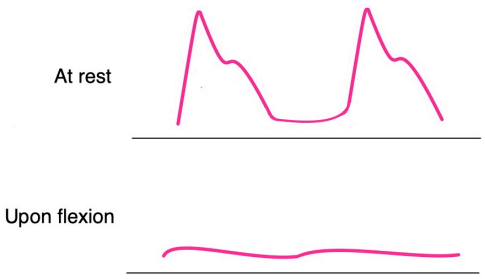
Normal >50mmHg
Poor <40mmHg (impaired wound healing)
Critical <30mmHg (non-healing)

Compression disorders

- Popliteal entrapment syndrome
  Compression of the popliteal artery by the medial head of the gastrocnemius muscle or fibrous bands. Usually young male pt complaining of claudication in calf with exercise.

  Eval: PPG on toes. Plantar flexion and dorsiflexion.

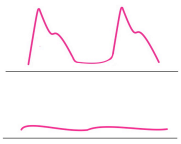
  Findings: *Normal at rest.* Only compressed when muscle is activated. *Waveforms will diminish or flatten upon flexions.*



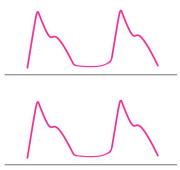
- Thoracic outlet syndrome
  Compression of neurovascular bundle by shoulder structures (ribs, ligaments, muscles). Pt complain pain, tingling, weakness in arm when in certain positions. *Most common cause is neurogenic (nerves).* Must also tested when replicating compression

  PPG sensors on index finger. Tested in the following positions:
  ~ rest with hand in lap
  ~ arm raised at 90 degrees to body
  ~ 180 degrees
  ~ exaggerated military stance (shoulders back)
  ~ Adson maneuver (exaggerated military stance with head turned sharply toward arm being tested and then switched
  ~ causative position

If arterial compression = Normal at rest, attenuated/flattened in position



If only neurogenic (**most likely finding**) = All normal with and without causative positions.



Duplex Upper Extremities

Capabilities and Limitations

Localize stenosis/occlusion/aneurysm
Hemodialysis access graft surveillance

Hemodialysis Access Graft Surgical AVF (fistula graft)
Connection between artery and vein Allows for greater volume flow as needed with hemodialysis
Types: Autologous = Brescia-Cimino. Radial a to Cephalic v Synthetic = straight or looped. (ie-brach a to ax v)

Physical principles

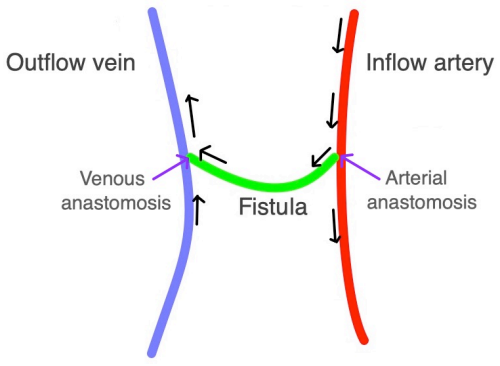
Duplex uses PW doppler (doppler physics covered in physics review)

Technique

7-9 MHz Linear array transducer
Arm extended laterally and 45 deg to body = pledge position
Check for thrill if evaluating for hemo access. Thrill is vibration felt under skin due to high velocity, turbulent flow

Native arteries B-mode, color, doppler waveforms from prox to distal

Hemodialysis access graft:
- inflow artery
- arterial anastomosis
- graft body
- venous anastomosis
- outflow vein

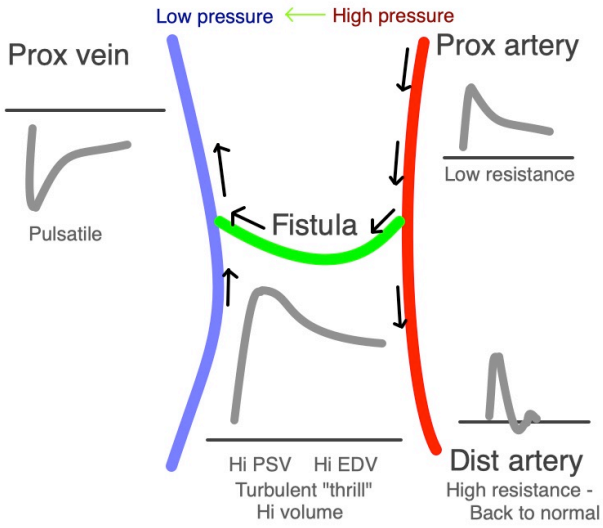


Interpretation

Native arteries: Normal high resistance = triphasic or biphasic

Stenosis: *No velocity criteria! Qualitative only*

Only basis for criteria is stenosis profile and knowledge of normal vs obstructive waveform contours. FOCALLY ELEVATED VEL AND PST = STENOSIS



Normal hemodialysis waveforms:

Prox art (inflow) - low resistance / inc EDV

Dist art - back to normal hi resistance (triphasic)

Fistula - low resistance / hi PSV and hi EDV

Prox vein (outflow) - pulsatile

Normal flow in graft: high velocity & turbulent

Abnormal findings for fistula graft:

- Thumping high resistance in prox artery or graft = graft occlusion
- Low velocity dampened, continuous = proximal inflow problem
- Focal elevated velocity = graft stenosis
  ***most common location is outflow vein
- Perigraft fluid = infection (clinical: red, warm, tender)
- Steal syndrome = reversal of flow in the distal artery
  ** ex - retrograde distal radial artery in brescia-cimino

Duplex Lower Extremities

Capabilities and Limitations

Eval location and severity of stenosis vs occlusion / % DR / Aneurysm
Bypass graft and stent surveillance

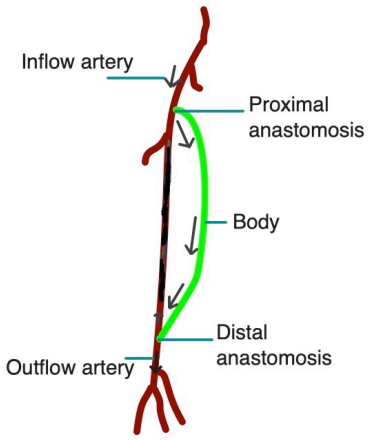
Technique

Native arteries prox to distal - B-mode, color, doppler

Bypass graft: Connect artery to artery to reroute blood flow in presence of significant extensive arterial obstruction.

Important areas to be eval

inflow artery
proximal anastomosis
graft body
distal anastomosis
outflow artery



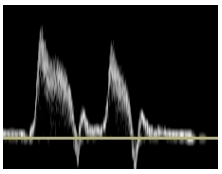
All successful bypasses must start above disease (patent inflow), have a patent conduit (graft), and end after any disease (patent outflow)

Arterial Bypass Grafts

Types:

- Synthetic = PTFE. Eval: anastomosis sites for possible leakage
- In situ saphenous vein graft (autologous) = vein is kept in place. Removed from venous and connected to arterial. Branches must be ligated and valves removed (Tool=Valvutome). Eval: Body due to risk of AVF from branches or stenosis from residual valves
- Reversed saphenous vein graft (autologous) = vein is removed and flipped upside down. Branches ligated. Valves kept in place since now allow for blood to move in right direction. Eval: prox anastomosis due to small size

Interpretation



Normal - triphasic or biphasic. **See CW doppler analysis

Stenosis velocity criteria - Prestenotic to stenotic ratio
(proximal PSV is compared to highest stenotic PSV)

2:1 ratio **≥50% diameter reduction** PSV doubles at stenosis

4:1 ratio **≥75% diameter reduction** PSV quadruples at stenosis

Any velocity >400cm/s = ≥75% DR

This criteria applies to both NATIVE arteries and BYPASSES

Additional bypass surveillance criteria:
Look for the following changes from past study

- Decrease of 30cm/s in the same graft segment from one study to next

- Change or deterioration in waveform quality

- Decrease in ABI >0.15

Retrograde flow in native artery at distal anastomosis is NORMAL. Flow moves into lower pressure of native artery

Additional findings
Aneurysm: increase in diameter >50%. Peripherally most likely seen in pop

Pseudoaneurysm: post procedure. Connecting channel or neck must be documented. To and fro flow (bidirectional)

Intraoperative US: (12-15MHz)
Main focus is check patency of anastomosis sites, intimal flaps, platelet aggregation
Based on type of bypass, attention focused in following areas:

Synthetic: anastomosis
Reversed: proximal connection (small prox size)
In situ: residual valves and branches

Duplex Abdomen

Abdominal aortic aneurysm / aortoiliac stenosis
Renal artery stenosis and kidney perfusion
Mesenteric ischemia
Organ transplants

Abdominal imaging

Fasting
3-5 MHz curvilinear transducer
Deeper/large body habitus = Lower freq
Superficial/thinner patient = Higher freq
Variable patient position >> "window"

AORTA

Technique

Scanning plane must be along axis or perpendicular to aorta. Measure in sagittal (parallel to aorta) = max AP. Transverse view (perpendicular) = AP/width
Outer to outer = true lumen
Coronal plane may be utilized if bowel gas obstructs view in supine.
(**may see renal arteries coming off aorta)

Interpretation

AAA >3cm
Dissection - visualization of intimal flap
Stenosis: same criteria as LE

RENAL ARTERY

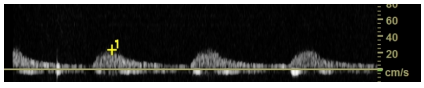
Clinical: hypertension
Renal artery stenosis > renal ischemia > renin > angiotensinogen > angiotensin

Technique

Prox, mid, dist renal artery
Segmental artery
Aorta (just prox to renal artery)

Interpretation

Renal/Aorta ratio ≥ 3.5 = ≥60% DR
***Tardus Parvus distally = segmental artery



Cannot use RAR if aorta is <40cm/s or >90cm/s. In that case, base criteria on renal artery velocities and presence of stenotic flow changes

KIDNEY PERFUSION

Renal disease = nephrosclerosis

Technique

Segmental artery
Interlobar artery
Arcuate artery

Interpretation

Normal Low resistance

- EDR end diastolic ratio >0.2
- RI (Poucelot's resistivity index) <0.7

Abnormal Increased resistance = nephrosclerosis

MESENTERIC ISCHEMIA

Clinical: Abd pain and cramping 15-30 min after eating. Most patients present malnourished, underweight.

Technique

Celiac, SMA, IMA "splanchnic vessels"
High freq curvilinear (5-6MHz). Fasting and post-prandial

Interpretation

	Waveform		PSV	% DR
Celiac	Low resistance	High EDV	≥200cm/s	≥70%
SMA	Fasting: High resistance Post-prandial: Low resistance	Low EDV Increased EDV	≥275cm/s	≥70%
IMA	Not easily seen. If easily imaged and high vel = abnormal = COLLATERAL			

2 out of 3 Abnormal = Chronic Mesenteric Ischemia

Celiac band syndrome: Compression of celiac by median arcuate ligament of the diaphragm. Most often seen in young athletic women. Only compressed during expiration, normalizes with inspiration

ORGAN TRANSPLANTS (allografts)

Technique

Liver: Hepatic artery, portal vein, hepatic veins
Renal: Renal artery and vein (donor vessels connected to External iliac vessels)

Interpretation

Normal transplants should have normal organ flow patterns = LOW resistance a.
Liver - Hepatopedal flow PV

Rejection: *Feeding artery HIGH resistance (dec or loss of EDV)*
Organ also will be increased size, altered echogenicity, and fluid. Possible thrombosis of veins

Liver- hi RI hepatic artery. PV thrombosis or hepatofugal flow
Renal- hi RI renal artery. Renal vein thrombosis

Preoperative mapping

Locate, check for usability/size, and map if OK
Very high freq linear array - all are superficial

- Epigastric artery
  TRAM flap - Transverse rectus abdominis myocutaneous flap when used in breast reconstruction. Section of tissue in anterior abdomen harvested along with epigastric a to reconstruct breast.

- Internal mammary artery
  AKA internal thoracic a. Used as recipient site for TRAM flap.
  Or can be used as coronary artery bypass. Min 2mm

- Radial artery
  Used as coronary artery bypass graft or hemodialysis access
  Must first perform modified Allen test to eval patency of palmar arch. Must have normal Doppler signals, free of wall abnormalities. Min of 2mm

- Vein mapping
  Superficial veins used in grafts - bypass (coronary/arterial or hemodialysis).
  Only GSV, SSV, cephalic, or basilic
  Eval for patency, compressibility, diameter, and continuity. Min 2mm

Atypical disorders

- Arteriovenous fistula = communication between artery and vein
  Traumatic - acquired (ex- post procedure) and usually 1 connection
  Congenital - born with it "malformation" usually numerous connections

  Complications: close to heart and large >> risk of CHF
  peripheral >> distal ischemia 'steals blood'

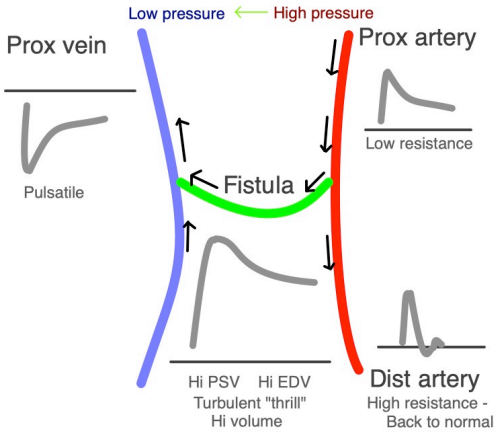
  Same hemodynamics as hemodialysis access graft = AVF!

  Proximal artery to AVF = Increased diastolic flow

  Distal artery to AVF = Normal arterial flow

  Fistula = Turbulent high velocity

  Proximal vein to AVF = Pulsatile



- Compartment syndromes
  Swelling within fascial compartments causing compression and increased pressure to capillary vascular beds. Anterior compartment syndrome compresses tibial artery >>> decreased arterial perfusion = paresthesia, pain, muscle weakness and foot drop.
  Treatment = fasciotomy

- Trauma
  Blunt force or penetrating trauma
  Intimal injury / flap >> dissection >> thrombosis >> acute arterial occlusion

Alternative Testing

Angiography

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:

NOT functional. Only anatomic/morphologic. 2 dimensional only
Only physically what fills with dye.

FILLING DEFECTS

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

MRI/MRA

Radio frequency energy and magnetic fields, produces multiplanar views.
MRI can eval anatomic. MRA quantifies blood flow and creates angiogram like images. NO CONTRAST needed.

Good for: AAA and dissection. Peripheral arterial disease

Contraindications: metallic objects, pacemakers.

CT/CTA

Ionizing radiation produces cross sectional images. CTA requires contrast.

Good for: AAA and dissection. Not good for peripheral.

Treatment

Medical management = lifestyle changes and drug therapy

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

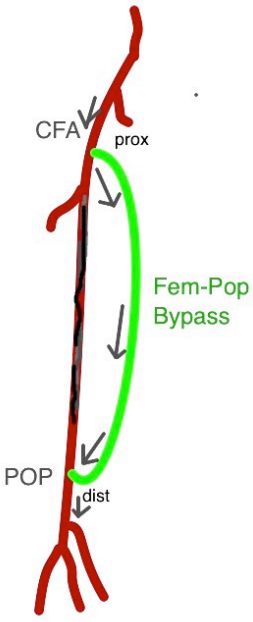
Exercise - promotes collateral formation

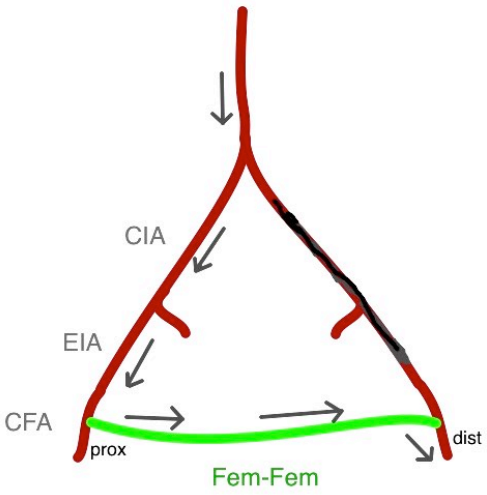
Aspirin : anti-platelet drug, reduces thrombotic activity

Surgical therapy

Endarterectomy - removal of plaque and intimal lining (not common)

Bypass grafts - alternate pathway for significant stenosis and occlusions. Needs good inflow, patent conduit, and good outflow
1st part of name - proximal anastomosis
2nd part of name - distal anastomosis





Endovascular therapy

Done via catheter under angiography

Angioplasty - Widens vessels from inside. Does not remove plaque. Balloon opens vessel and then stent inserted.
*** focal stenotic lesions in larger vessels

Stent grafts - AKA endografts = AAA repair.
Covered stent to block off flow to aneurysm. Important to look for endoleaks. No flow should be going to aneurysm. Size should not change or may decrease.
If leakage = aneurysm may grow and may rupture

Type 1 - Attachment sites
Type 2 - Branch leaks (most common)
Type 3 - Modular connect
Type 4 - Transgraft (tears)

Pseudoaneurysm treatment

- Nothing - may thrombus
- Manual compression.
  Need to fully compress the neck. If unable to uniformly compress the neck or if there are multiple communicating channels = cannot be done
  Firmly compress with transducer for periods of 10min, rest, repeat. Up to hour until thromboses
- Thrombin injection
  US guided thrombin injection into body of pseudo as blood flows into it. NOT neck. Cannot be done if patient has allergy, skin ischemia or infection, if there is wide or short neck. If successful, thrombose within seconds
- Surgery - if none of the above works

CEREBROVASCULAR

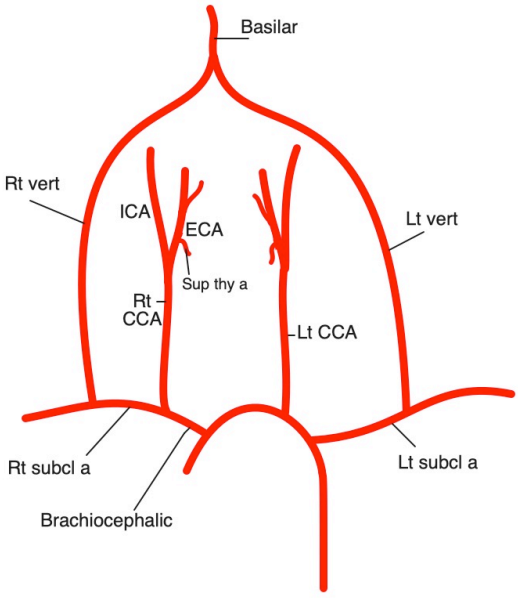
Anterior circulation

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)

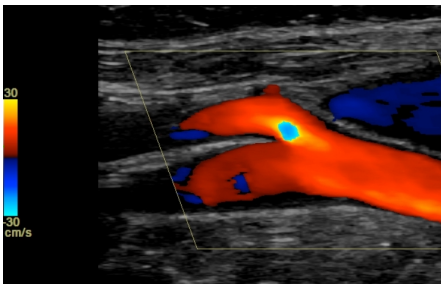
ICA has no extracranial branches. 1st branch at syphon: *Ophthalmic a.* (intracranial)

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



ICA vs ECA

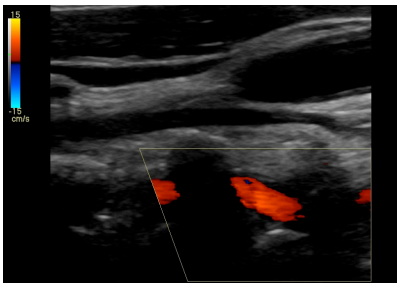
Most accurate way of differentiating ICA from ECA: Visualization of ECA branches. ICA has NO branches



Posterior circulation

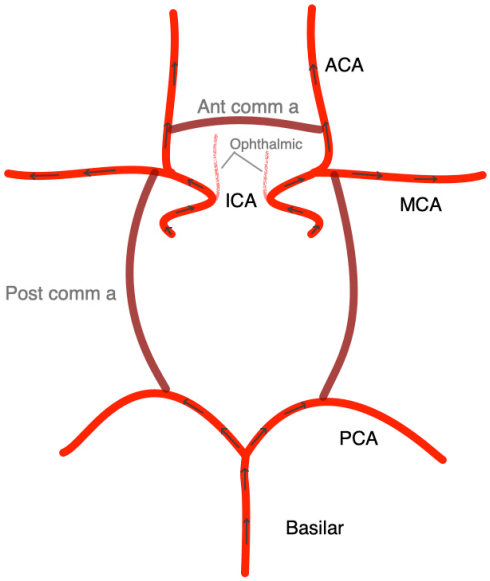
Vertebral a originate at subclavian arteries and travel through vertebral processes. Unite to form *Basilar artery*

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*.

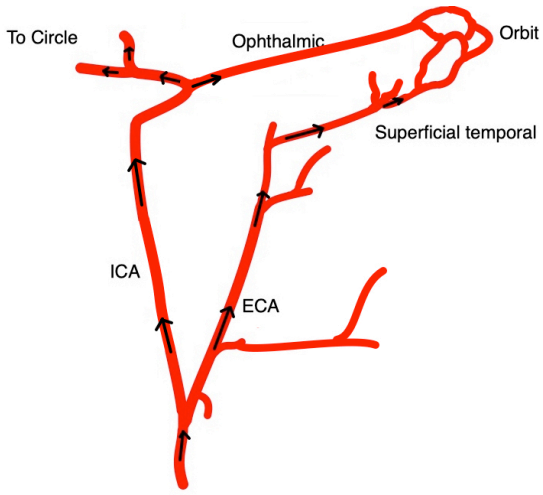
Anterior communicating a - connects Rt and Lt ACA
Posterior communicating a - connect posterior to anterior
These are only used when needed as collateral

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

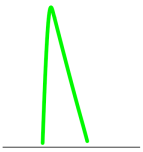
Same principles as arterial

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

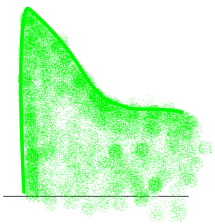
MUST BE AWARE OF THIS. Carotid stenosis criteria is based on PSV. Other conditions may cause elevated PSV without stenosis. If no PST = NO stenosis

Hemodynamic stenosis flow changes:

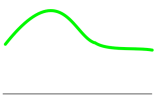
Prox to severe obstruction : Increased resistance. Decreased EDV. May also have dec PSV.



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

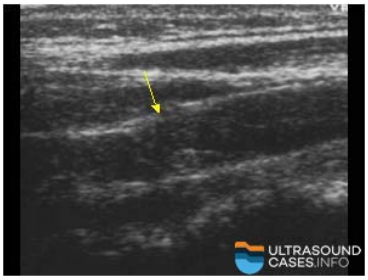
Age, family history, male gender

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

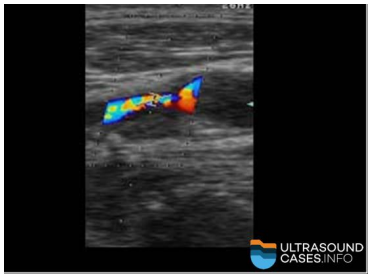
Type of plaque

- Fatty streak - thin hypoechoic layer

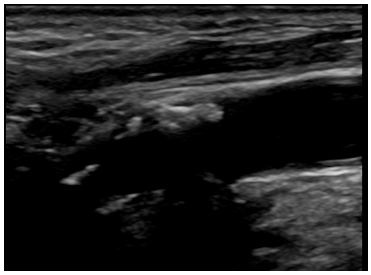
- Fibrous plaque - lipids with collagen and fibrous material. AKA smooth or homogeneous.
  Soft fibrous = hypoechoic
  Hard/dense fibrous = echogenic



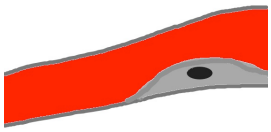
- Complicated lesion - complex or heterogeneous. Fibrous mixed with calcium and debris. Mixed appearance = hypo, echogenic, calcified



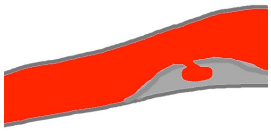
- Calcified - Completely hyperechoic with posterior shadowing. May limit visualization and evaluation of high velocity flow.



- 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

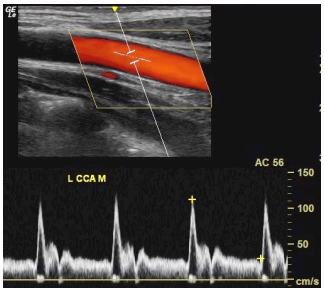
PW Doppler

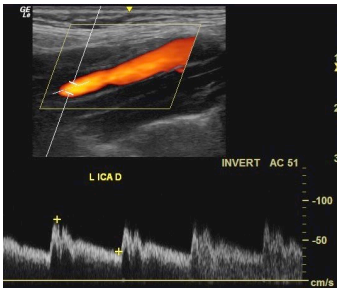
Normal waveform patterns

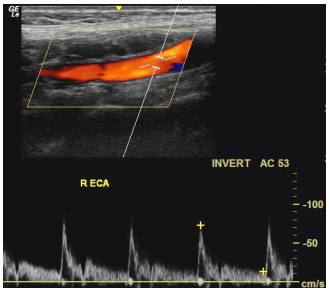
ICA - low resistance

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

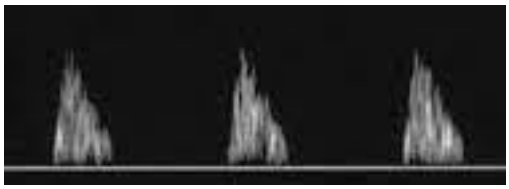
Reasons for over or underestimating disease

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

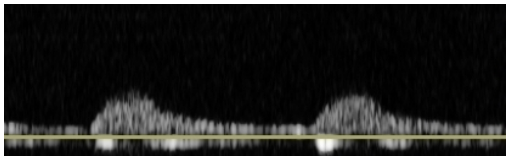
- Abnormally high resistance, absent diastolic



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



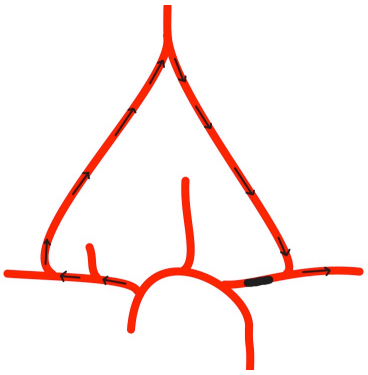
Proximal disease if unilateral

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

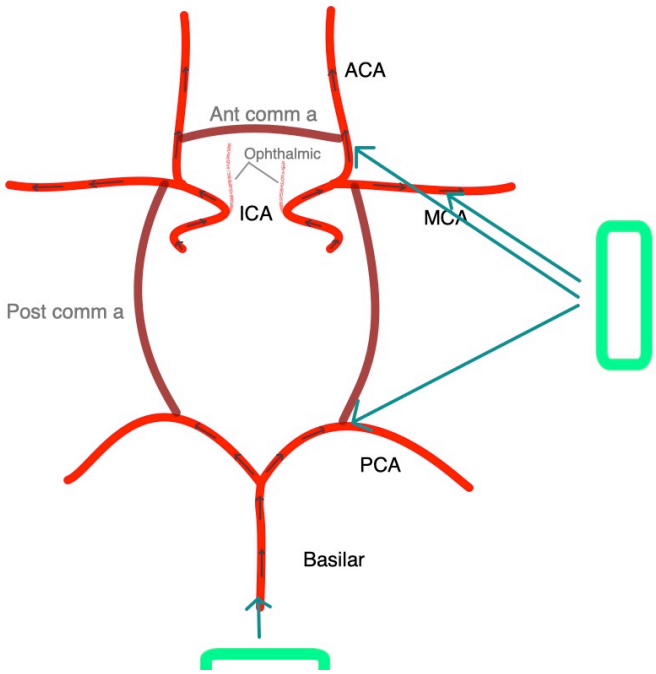
Technique

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.

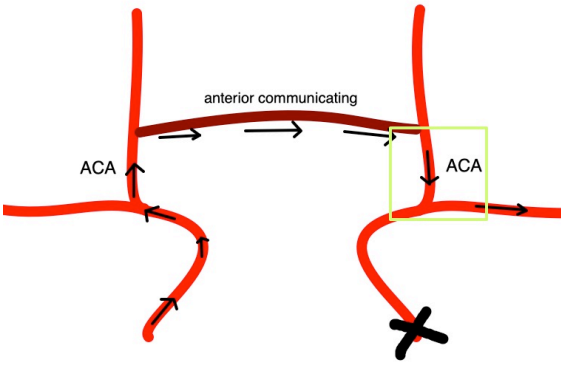
Collateral circulation

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*

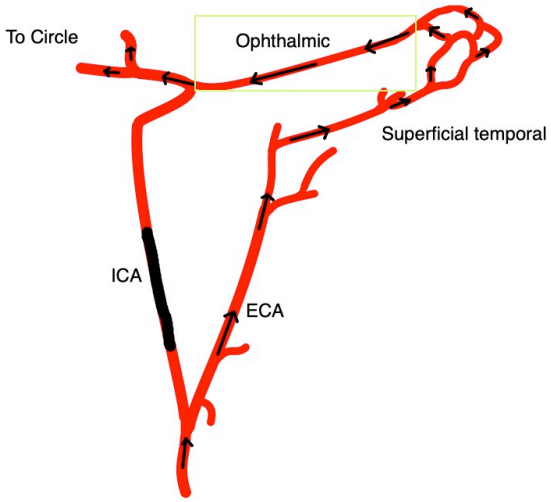


TCD findings: Antegrade flow in the ipsilateral ACA

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



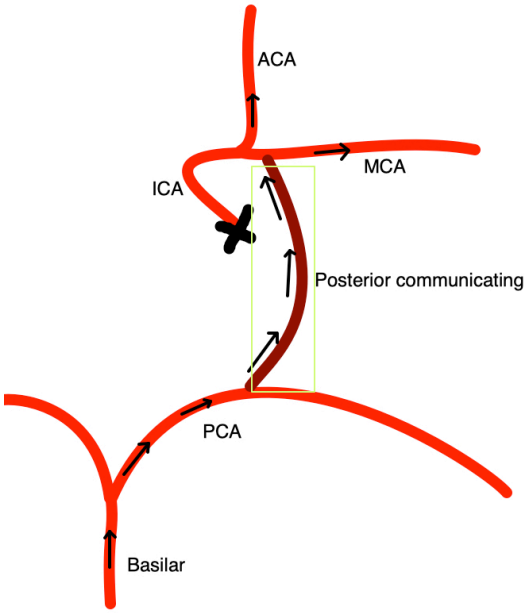
TCD findings: Retrograde flow in ipsilateral Ophthalmic

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

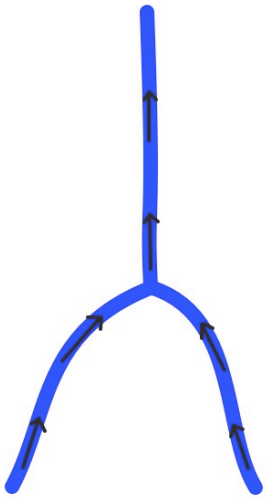
- Stent - during angioplasty. For neointimal hyperplasia or atherosclerotic stenosis. Plaque stays in place, just widens vessel and holds it open
  *** Does not use same criteria for stenosis as stented vessels have higher velocity <225cm/s may be within normal limits

- Total occlusion - nothing. Generally collaterals already in place and functional

- Bypasses - very rare!

VENOUS

Peripheral Venous Anatomy



Think direction of flow!!

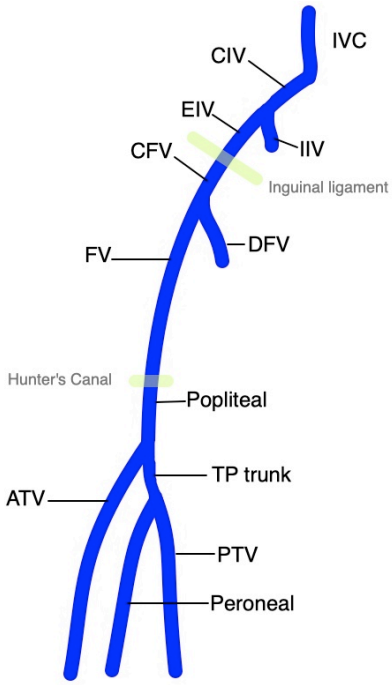
Proximal is still closer to heart and distal away. BUT … veins flow distal to proximal. That means they start at the bottom and end at the top. Anatomy is based on direction of flow. Veins never bifurcate, they form confluences.

Empty blood from = Where blood is coming from
*** example : hepatic veins empty blood from the liver

Drain/empty blood *into* = Where blood is going
*** example : hepatic veins drain blood into the IVC

Veins always flow up into the next vessel. When 2 veins join each other and form a new vein, they both flow into the new vein. They do not empty into each other. The junction is ALWAYS the new vein

Lower Extremities



Deep veins accompany arteries.
Venae comitantes = corresponding veins. Paired deep veins. In LE = all calf veins PTV, ATV, PER

PTV and PER drain into tibeoperoneal trunk. TP trunk and ATVs join to form the POP v.
Popliteal becomes the FV at Hunters Canal.
FV and DFV form the CFV. CFV terminates when it drains into the EIV at the inguinal canal.
EIV and IIV empty into CIV.

IVC is formed by the confluence of the RT and LT CIVs.

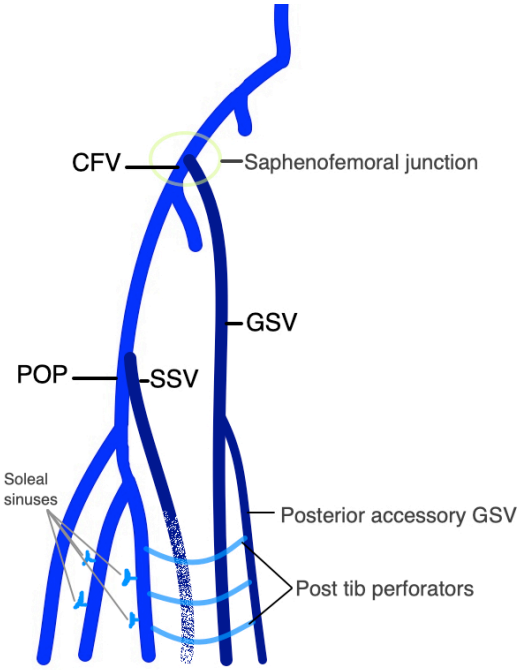
Superficial veins

Do not have an artery. Located between the fascial layers within the subcutaneous fat

Greater saphenous vein = longest vein in body. Medial leg. Drains into the CFV at the saphenofemoral junction.

Small saphenous vein = posterior calf. Drains into POP.

Perforatoring veins = Connect superficial to deep. Used in connection with the calf muscle pump. Most are found below the calf, superior to medial malleolus (connecting post accessory GSV to post tibs)



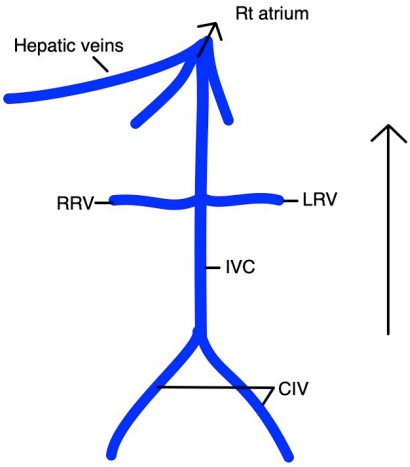
Communicating vein = Connect veins of same system.

Venous sinuses = Potential reservoirs or channels that allow blood to accumulate in the calf and during calf contraction > ejected into peroneals and PTVs. Important role in calf muscle pump.

<u>Abdominal venous system</u>

IVC courses superiorly. Renal veins drain into IVC

Hepatic veins drain the liver (Hepatofugal) and empty into IVC. All drains into Rt atrium of heart.

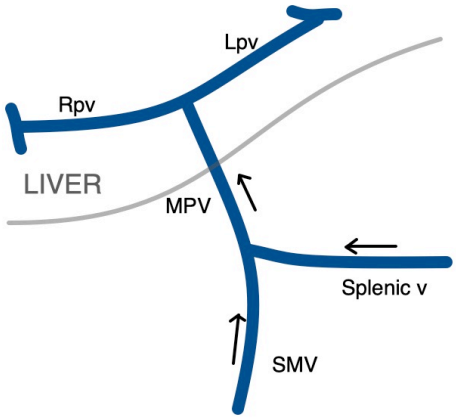


Portal venous system

Not related to IVC or its veins

Drains blood from digestive tract and spleen into liver.

SMV and splenic form Portal confluence. Both drain into MPV.
MPV drains into liver (supplies 80% of liver's blood) = Hepatopedal flow

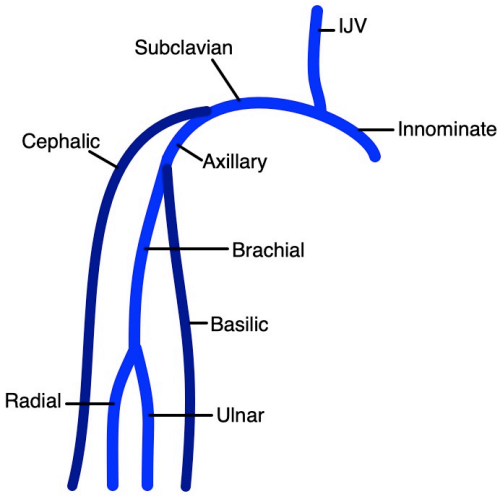


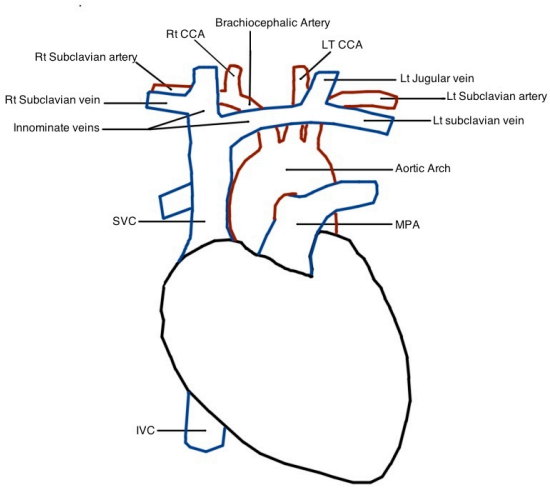
Upper Extremities

Venae comitantes = Radial, Ulnar, Brachial

Superficial veins = Cephalic and Basilic

Radial and Ulnar v drain into Brachial. Brachial and basilic (superficial vein that courses medial) join together to form the axillary v. Both drain into axillary.
Axillary and cephalic (superficial vein that courses lateral) join to become the subclavian vein. Both drain up into the subclavian.





<u>Venous return to heart</u>

Subclavian and IJV join to form the brachiocephalic or innominate.

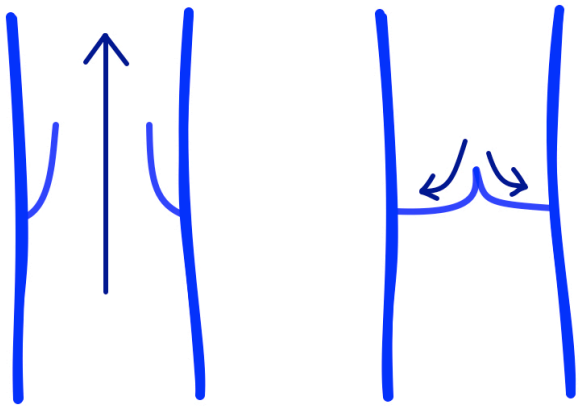
Rt and Lt innominate drain into the SVC

SVC carries blood to Rt atrium of heart

Venous structure

Same 3 layers as arteries = intima, media, adventitia
*** difference = medial layer is thinner in veins. Collapsible

Venous valves - bicuspid extensions of intimal layer. Purpose is to prevent backflow and maintain unidirectional flow. Venous blood has greatest hemodynamic challenge to overcome = gravity. Needs valves to assist



Due to hydrostatic pressure greatest closest to ankle….

Most # valves = veins in calf. Decrease in # closer to abdomen.

NO valves = veins in chest and abdomen. Ex- IVC, SVC, iliac veins, etc

Arms have very few valves, 1 per vein.

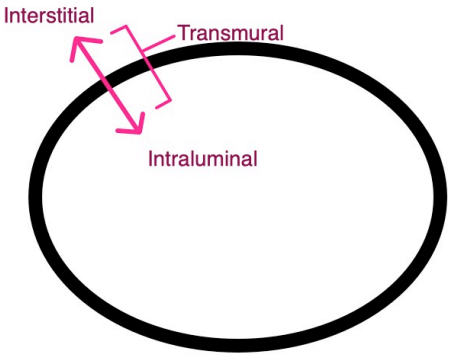
Perforators - must have at least 1 valve

Venous Hemodynamics

Venous compliance

Ability to handle large changes in volume without greatly affecting the pressure.

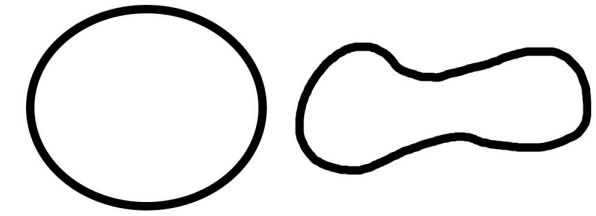
Veins can expand to accommodate more volume



Transmural pressure - pressure on the wall.

Difference in pressure from outside (interstitial) and pressure from the inside (intraluminal).

***Depends on volume



Circular: Full and more volume. Has less resistance which allows for emptying

Elliptical: Less volume but higher resistance. Allows for venous filling

<u>Respiration "Phasic flow"</u>

Big role in venous return to heart. Creates pressure gradient for flow into abdomen (for LE) or chest (for UE)

Inspiration: Inc abdominal pressure / Dec thoracic pressure.

*** flow needs pressure gradient (hi to low pressure)

If abd pressure is high = flow from legs STOP

If chest pressure is low = flow from arms FLOWS into chest

When veins are not flowing/emptying = they are filling.

Filling = Inc volume = Inc pressure

Expiration: Dec abdominal pressure / Inc thoracic pressure

*** LOW abd pressure and HIGH venous pressure in legs = FLOW

LE venous flows into abdomen

UE venous flow halts and fills

Valsalva: Take deep breath in, hold it, and bear down (tightening abdomen as if having a bowel movement). Increases both abdomen and chest pressures

>> ALL VENOUS FLOW HALTS

After release of valsalva = venous flow should augment

If there is augmentation of flow DURING manuever = reflux flow

Hydrostatic pressure

Has same influence on venous as arterial. Makes it more difficult for venous flow to go up

*** Supine = 0mmHg at ankle

*** Standing = 100mmHg at ankle

Calf muscle pump

Venous heart. To propel blood up towards the heart. Only works if valves are competent. Venous sinuses fill during relaxation. Perforators allow blood to flow from superficial to deep systems. During contraction, muscle squeezes sinuses and deep veins to empty up. Valves in superficial and perforators normally should close during contraction.

Muscle contraction = Empties Relaxation = Filling

Effective Calf Pump	Ineffective Calf Pump
>60% blood ejected from calf	Incompetent valves
Increased venous return to heart	Increased venous pooling
Decreases venous pooling	Increases venous volume
Decreases venous volume	Increases venous pressure
Decreases venous pressure	Venous hypertension

Venous hypertension

Any abnormal increase in venous pressure due to high venous volume

Venous Disease and Clinical History

2 types of disease - Acute obstruction (thrombosis) and Chronic insufficiency

ACUTE DVT

Thrombosis - RBCs trapped in fibrin web. Most frequently originate at venous valves or sinuses (stasis).
Greatest clinical danger: pulmonary embolism
Most likely complication: venous insufficiency (damage to valves)

Symptoms: **Pain**, **swelling**, redness, warmth

Virchow's triad : risk factors and contributing to formation of clot

1. Trauma
   Intrinsic- damage to inside. Catheters, PICC lines, IV
   Extrinsic- from outside. Accident, fall

2. Stasis
   Bed rest
   Obesity
   Pregnancy
   Paralytics
   Congestive heart failure
   Surgery related

3. Hypercoagulability
   Increased clotting of blood
   Pregnancy
   Cancer
   Oral contraceptives
   Inherited states (factor V Leiden, protein C, protein S)

Other sources of thrombus

- Paget-Schroetter
  Stress or effort thrombosis of subclavian or axillary due to repetitive trauma to vessel. (Ex-Baseball pitcher with unilateral UE pain and swelling)
  Venous component of TOS
- Lt CIV compression AKA May-Thurner syndrome
  Compression of LT CIV by RT CIA as artery crosses over it. Stasis >thrombosis
- Nutcracker syndrome
  Compression of Lt renal vein by SMA and aorta. LRV may thrombose
- SVC syndrome
  Thrombosis or compression of SVC by mass. Causes bilateral facial swelling, bilateral UE swelling, and dyspnea

Limb threatening acute venous disease

Severe acute extensive ileofemoral DVT. So obstructive it affects the arterial circulations. Combo of DVT and arterial symptoms

- Phlegmasia alba dolens — PAIN, SWELLING, *WHITE*
  Triggers arterial spasms
- Phlegmasia cerulea dolens — PAIN, SWELLING, *BLUE*
  Reduces arterial inflow

VENOUS INSUFFICIENCY

Valvular incompetence. Valves leak, no longer maintain unidirectional flow. Flow refluxes caudally (back down). Causes VENOUS HYPERTENSION

Causes

Congenital
Avalvular veins
Inherited incompetent valves
Klippel-Trenauny (hypoplastic or absent deep veins)

Post-thrombotic / post-phlebitic syndrome
Pt had previous DVT. Damaged valves lead to chronic insufficiency

Other acquired causes for *increased venous pressure*: obesity or pregnancy

Venous walls dilate and stretch, collaterals (varicose veins) develop. Dilated tortuous superficial venous collaterals. Caused by chronic venous hypertension

Primary: hereditary

Secondary: due to obstructive process

High venous pressure to back up into capillaries. Under pressure allow leakage of fluids into tissue = edema and brawny discoloration
Brawny: leakage of fibrin, RBCs into tissue. RBCs die and create hemosiderin = brown. Most common location is superior to medial malleolus

Symptoms of venous insufficiency are the same whatever the cause

- Edema
- Heaviness
- Varicose veins
- Brawny
- Lipodermatosclerosis (severe and chronic= hardening of skin due to constant irritation of tissue. Bottle neck appearance)
- Venous ulcers

Venous	Arterial
Medial malleolus/calf	Toes/bony regions
Shallow	Deep
Oozy	Dry
Less painful	Very painful
Irregular	Regular
Other: brawny, swelling	Other: Dry skin, thick toenails

<u>Edema</u>

Fluid enters tissues. May be caused by venous hypertension. Increased capillary pressure

Pitting = manual pressure applied and leaves dent in skin. Fluid retention, renal dysfunction, inc venous pressure

Non-pitting = tissue so engorged with fluid, no indent. Caused by lymphedema

Venous PPG

Capabilities and limitations

Presence/absence of venous insufficiency
Determine superficial vs deep insufficiency

Not quantify insufficiency / severity

Physical principles

Documents capillary blood volume

Same technology as arterial, except setting is DC (direct current)
AC (alternating current) > arterial
DC (direct current) > venous (detects slower flow changes)

Technique

Pt sitting with legs dangling

PPG placed above medial malleolus (not directly on varicose veins)

*** Activate calf muscle pump to empty blood from calf
Exaggerated flexions and dorsiflexions
See how long it takes to fill back up = Venous Refill Time (VRT)

Interpretation

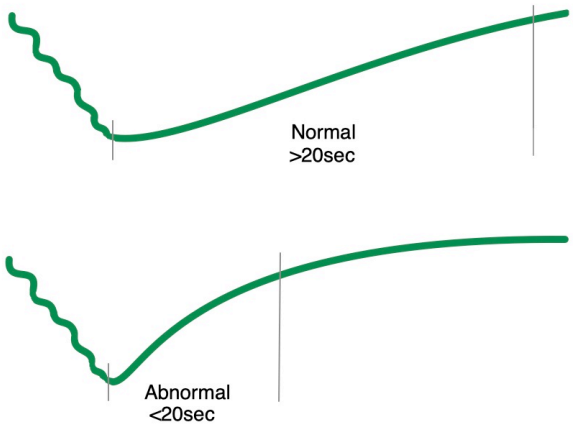
Venous Refill Time VRT

Normal = Gradual venous filling	>20sec
Incompetent valves = Fills up quickly	<20sec

1. If initial reading is normal = No venous insufficiency
2. If initial reading is abnormal = repeat with tourniquet. Purpose of the tourniquet is to cut off influence of the superficial system. Helps to know if insufficiency is coming from the deep or superficial.
   - Normalizes with the tourniquet = Superficial insufficiency
   - Never normalizes = Deep insufficiency

If initial reading is abnormal and tourniquet needs to be placed, 1st place the tourniquet above the knee = rule out GSV

If still abnormal, place below the knee = rule out SSV



Venous PPG reading	
>20 sec VRT	Normal
<20 sec VRT	Insufficiency
Tourniquet normal AK	GSV insuff
Tourniquet normal BK	SSV insuff
Never normalizes	Deep insuff

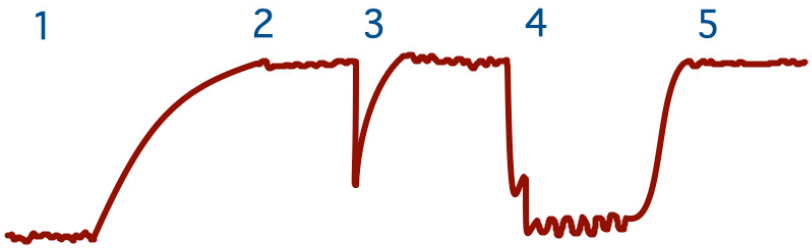
Venous Air Plethysmography

Same purpose as Venous PPG.

Can quantify venous reflux and measure venous function

- Pt has large cuff on calf and completes various maneuvers

  1: Leg elevated 2: Standing 3: 1 toe-up 4: 10 toe-ups 5: Resting



- Ejection Fraction >60% is normal
- If initially abnormal, can be repeated with tourniquet to distinguish superficial from deep insuff (same as PPG)

CW Doppler

Capabilities and limitations

Presence/absence of venous insufficiency
Presence of DVT

Cannot specifically choose vessel or sampling depth (CW)
No imaging, only waveform contour reading

More likely to have false negatives or false positives

- False Negative = Test seems negative but there really is disease
  Partial non-occlusive DVT
  Venous collaterals
  Bifid system (duplicate deep veins)

- False Positive = Test has abnormal reading but really is normal
  Extrinsic compression (pregnancy, tight clothing, tumors, ascites)
  Severe PAD (reduced inflow=reduced outflow)
  COPD
  Doppler angle/tech error

Technique

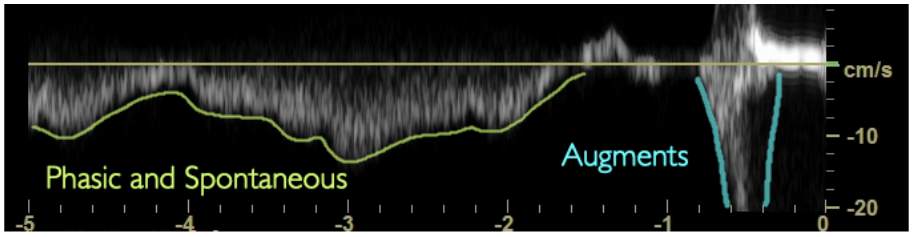
Similar to CW in arterial
45-60 degree angle to skin surface

Pt position = reverse trendelenburg (limb lower than heart). Increased hydrostatic pressure encourages venous filling and allows for easier evaluation of the LE veins
LE externally rotated with knee slightly bent

Done in segments (groin, thigh, behind knee, ankle)

Interpretation

NORMAL peripheral venous waveform



- Spontaneity - naturally occurring. Most larger deep veins should be spontaneous. Non-spontaneous flow may be seen in tibials, superficial veins, and radial/ulnar

  Abnormally non-spontaneous = location of obstruction

- Phasicity - flows with respiration. Normally is seen in peripheral veins

  Non-phasic or continuous flow = proximal obstruction
  Pulsatile =Normal for veins close to heart (IVC, subclavian veins, IJV). In peripheral veins = CHF, tricuspid regurgitation, or fluid overload.

- Augmentation with distal compression or proximal release - Normal flow should augment or increase when compressed distally or after the release of the proximal compression

  No augmentation = obstruction between probe and compression/release

- Proximal compression or Valsalva - both increase the proximal pressure and are done to test competence of venous valves. Adds stress or pressure on valves. During maneuvers, venous flow should halt or stop. Normal result after release is to augment.

  Augments DURING = Valvular incompetence

Venous Duplex

Capabilities and limitations

Location of disease (DVT or reflux)
Acute vs Chronic DVT
Partial vs complete

Less limitations

Technique

Patient position: reverse trendelenburg (limb lower than heart)

B-mode : perform with and without compressions

Color: show full filling of vessel and directionality

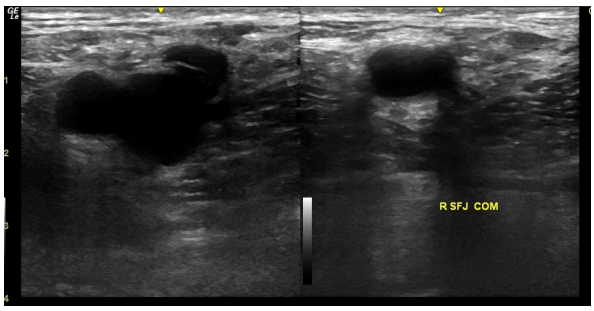
PW doppler: waveform morphology

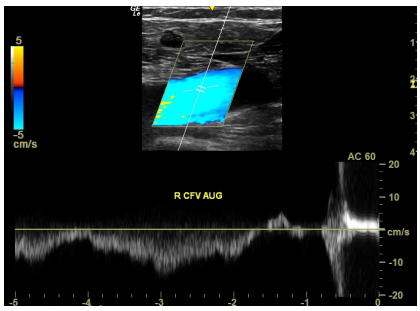
Insufficiency testing: Pt in reverse trendelenburg or standing. Add valsalva maneuver or proximal compressions to stress valves.

Interpretation

Normal

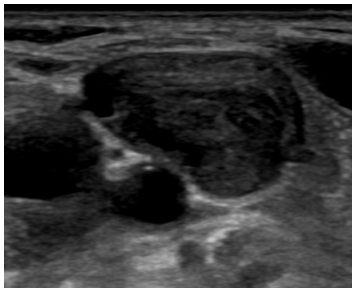
- Normal veins fully compress = Coaptation. No thrombus
- Fully fill with color
- Phasic, spontaneous and augmentation
- No reflux when standing or during valsalva/prox comp





Abnormal

- Acute DVT



Dark, dilated, incompressible
Larger vein size, spongy texture, poorly attached the walls
Acute - anechoic, hypoechoic. Subacute - possible hypo to echogenic. Still dilated and spongy looking
** If superficial vein is thrombosed = NOT DVT.
"Superficial thrombophlebitis"

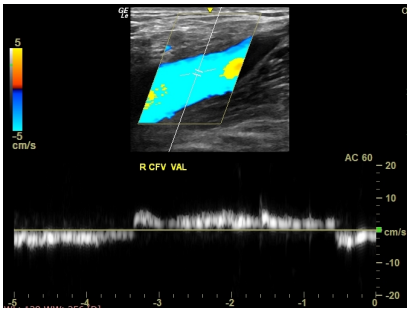
Color - no signal or trace amounts along walls
Continuous doppler = proximal obstruction
No augmentations = indicate proximal or distal DVT

- Chronic DVT

Smaller vessel size, thicker walls.
Hyperechoic striations, linear bands scattered within vessel.
Flow recanalizes throughout vessel, patchy color flow. And partially compressible vessel. Collateral veins may also be seen

Doppler - may be normal. May have evidence of reflux

- Venous insufficiency



Flow augments DURING valsalva or proximal compression.
Flow is retrograde through valves during the maneuvers.

Reflux lasting >0.5sec = valvular incompetence.
(Normal valve closure time is about 0.5sec)

Varicose veins should be documented. Should be compressible. Will demonstrate augmented flow during valsalva or proximal compression
Perforating veins: If easily visible and >3mm = insufficiency. Normal flow direction is superficial to deep (blue or negative). During valsalva, turns red when abnormal

- Additional findings

  Lymph nodes - usually in groin. Hypoechoic with echogenic hilum. Abn = >2cm

  Edema - fluid found infiltrating tissues

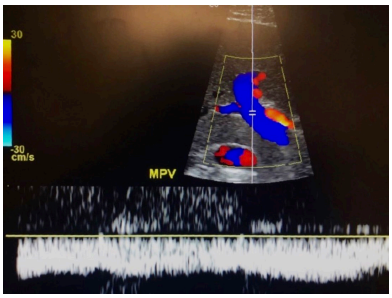
  Joint effusion - fluid at joint, anterior to bone

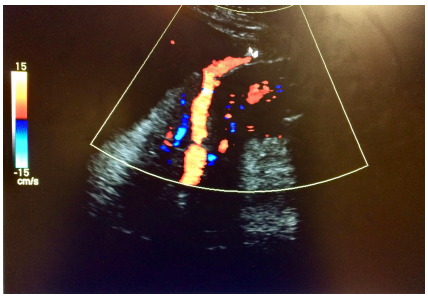
  Bakers cyst - cyst filled with synovial fluid in medial pop fossa

<u>Abdominal venous duplex</u>

- Portal vein. Hepatopedal flow (towards the liver) and minimally phasic, almost continuous. All veins related to this system should reflect similar flow patterns.

  Portal hypertension: Increased pressure in portal system most likely caused by cirrhosis of liver or other chronic liver disease. Increased resistance of liver cause flow reversal = hepatofugal PV flow and portosystemic collaterals. Abdominal varices caused by portal hypertensions may be found near spleen, stomach, and esophagus. Dilated coronary vein (drains stomach). Recanalized paraumbilical vein may also be present.





- IVC and hepatic veins. HV hepatofugal and triphasic pattern. All veins draining into this system also will be similar

  Budd-Chiari: occlusion of hepatic veins and possible IVC

  Enlargement of hepatic veins and IVC: Caused by rt sided heart failure

  IVC tumor: Invasion from Renal cell carcinoma. Check kidneys

Alternate testing

D-dimer

Serum blood test for thrombolytic activity in body. May indicate presence of DVT if positive. May have false positives

Contrast venogram

2 types: ascending and descending

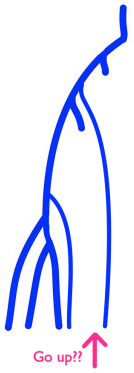
Same idea as angio. Dye is injected into vein and x-rays show the flow of dye material

Ascending: Goes up R/O DVT

Dye injected into distal superficial vein on dorsum of foot

Full filling = No obstruction

No filling/partial filling/railroad tracks = DVT



Descending: Goes down R/O Valvular incompetence

Dye injected into CFV

No filling = No reflux

Filling = Reflux

Pulmonary embolism testing

In patients with +DVT indications and shortness of breath

VQ scan - ventilation quotient. Measures air and blood flow in lungs

Most sensitive = CT pulmonary angiography

Treatment

Controlling of risk factors

Reduce Virchow's Triad:

- reduce injury
- decrease venous stasis.
  Compression stockings, leg elevation, pneumatic compression devices
- therapy for hypercoagulable states

Anticoagulant therapy for prophylaxis

Prophylaxis = preventative
Decreases risk of DVT in high risk pt perioperatively
Unfractionated heparin, low molecular weight

Anticoagulant therapy for Acute DVT and PE

Weight based, loading dose of unfractionated heparin, administered intravenously. Pt can ambulate 30min after initial dose. 4-5days then oral anticoagulant

Heparin is an anticoagulant (prevents propagation of clot). NOT break down clot (thrombolytic). The body always has thrombolytic abilities.

Thrombolytic treatment is for complicated cases or limb threatening DVT (phlegmasia dolens)

Thrombectomy/Embolectomy

Last resort with limb or life threatening diseases. If pt does not respond to thrombolytic therapy (phlegmasia)

Vena Caval filter

Placed infrarenal IVC in patients that are high risk of pulmonary embolism.

Varicose veins/ Insufficiency

Deep venous insufficiency cannot be corrected. Only symptoms minimized and reduced progression of disease = Palliative to reduce venous pooling

Superficial insuff can be treated by removing or closing the GSV or SSV to stop supplying the varicose veins.

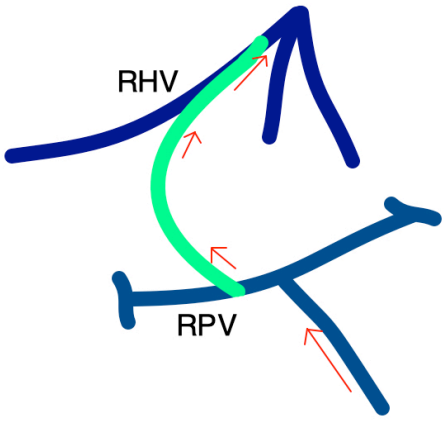
Ablation procedures: Radio freq and laser
1. Catheter placed into distal superficial vein
2. US guided to place tip distal to SFJ. ***landmark superficial epigastric vein
3. US guided injection of tumescent anesthesia surrounding vein down the leg. Creates halo around vein- helps in protecting surrounding tissues from burning and to compress vein to ensure good contact with catheter tip
4. Ablation heats inside of vein, damaging it, and cause thrombus. Vein permanently sealed
5. Varicose veins can be surgically removed - phlebectomy. Or left to disappear on own

Sclerotherapy: Injections for the removal/closure of spider veins

Venous ulcers: Unna boot, hyperbaric oxygen chamber

Portal hypertension

TIPSS
transjugular intrahepatic portosystemic shunt



Provides communication or shunt between portal vein and hepatic veins to decompress the portal vein and normalize flow direction.

Rt portal vein > Rt hepatic vein. If successful, flow will be hepatopedal at proximal anastomosis (portal) and hepatofugal at distal anastomosis (hepatic)

Quality Assurance

Gold standard testing: Comparing the "truth" (invasive exam) with the duplex

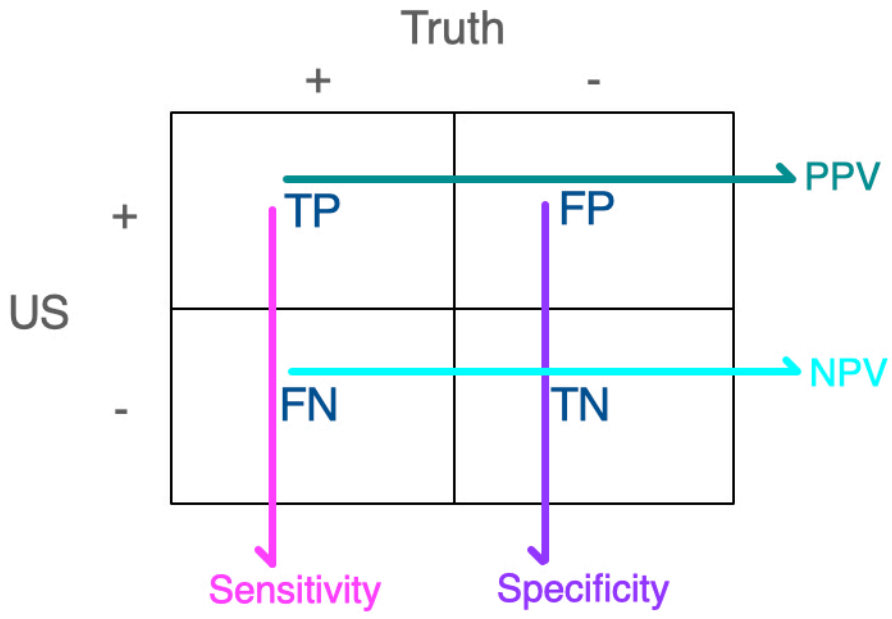
True positive- our test found disease and there really was disease

True negative- our test was normal and pt really is normal

False positive- our test said it was abnormal, but pt really is normal. False alarm

False negative- our test said no disease, but it really was abnormal. We missed it

- Sensitivity
  Ability to DETECT disease
- Specificity
  Ability to DETECT or IDENTIFY normal
- Positive predictive value
  How good at predicting disease
- Negative predictive value
  How good at predicting normal
- Accuracy - between all values above



Practice:

125 patients received LE venous duplex studies and venogram for venous insufficiency

64 patients had reflux by both the duplex study and the venogram

53 patients demonstrated competent valves by both methods

6 patients were thought to be normal on ultrasound but abnormal findings were found on venogram

2 patients had documented reflux on duplex but venogram demonstrated no abnormality

	+	-
+	64	2
-	6	53

Sensitivity	64/70	91%
Specificity	53/55	96%
PPV	64/66	96%
NPV	53/59	89%
Accuracy	117/125	93%

**Accuracy will fall between sensitivity and specificity and also between PPV and NPV

Physics Review

** basic physics coverage as applied to Vascular

Transducer choice and principles of frequency

Operating or transducer frequency is chosen based on *scanning depth* and depends on 2 main factors

- Scan type
- Body habitus

Increase frequency = better axial resolution / poorer penetration

Decrease frequency = deeper penetration / degrades axial resolution

Peripheral duplex exams

7-12 MHz linear array

More superficial = higher frequency to improve resolution

Larger body habitus or deeper vessels = lower end of frequency range

Abdominal duplex exams

2-6 MHz curved linear array

Need larger field of view/sector

In general deeper structures, overall lower frequencies

Thinner patient = higher 6 MHz

Obese patient = lower 2 or 3 MHz

Intraoperative

12-15 MHz array

Very high frequency because probe placed directly on vessel

Image Optimization

Incident angles

Imaging B-mode surfaces/vessel walls

- 90 degrees AKA perpendicular

Improving resolutions

- Axial: depends on transducer frequency
  Increase frequency to improve vis of vessel wall thickness or plaque
- Lateral: depends on beam width / narrow beam
  Focal position = at or below area of interest
  Increase # focal zones
  Increase line density
  Decrease sector angle
- Temporal: frame rate / work faster
  Decrease line density / sector angle
  Decrease # focal zones
  Decrease color box size

Artifacts

- Reverberation / Comet tail / Ring down
  Several bright false echoes deep to real reflector
  Examples: microcalcifications, gas bubbles, syringe needle, catheter
- Posterior shadowing
  Severe attenuation. Dark band deep to highly reflecting object
  Examples: bony structures, calcified plaque
- Posterior enhancement
  Lack of attenuation from fluid-filled structures
  Examples: cysts, hematoma
- Mirror image
  Copy of echoes deep to real anatomy/specular reflector. May be seen in color
  Example: liver/diaphragm, larger artery

The DOPPLER EFFECT

Object in motion will produce echoes that come back at a slightly different frequency than what was sent out.

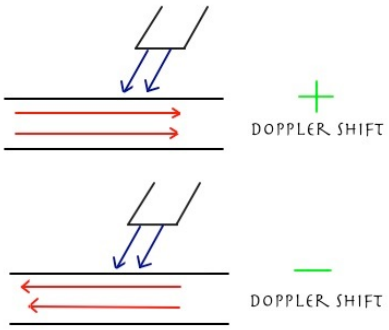
Moving TOWARDS the sound source = HIGHER received frequency
Moving AWAY from sound source = LOWER received frequency

The difference between the reflected and transmitted frequencies is called the Frequency Shift or Doppler Shift.

If the received freq is greater than the transmitted, it's a positive shift.

If the received freq is less than the transmitted, it's a negative shift

Doppler shift tells us presence, direction, and magnitude



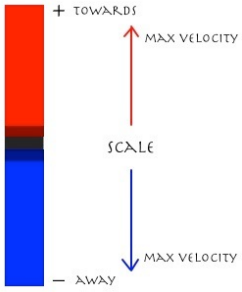
$$\text{DOPPLER SHIFT} = \frac{2 \times \text{FREQ} \times V \times \cos\measuredangle}{C}$$

3 things that affect the shift:

- Velocity Directly related to shift
  Increase blood velocity = Increase doppler shift
- Frequency Directly related to shift
  Increase transducer frequency = Increase doppler shift
- Angle Inversely related to shift
  Increase angle (closer to 90) = Decrease doppler shift
  At 90 degrees/perpendicular = NO shift
  At 0 degrees/parallel or 180 degrees = Greatest shift

COLOR

Doppler shift info colorized and superimposed or 'pasted' onto B-Mode image (duplex). Only displays direction and average velocities. *Qualitative*

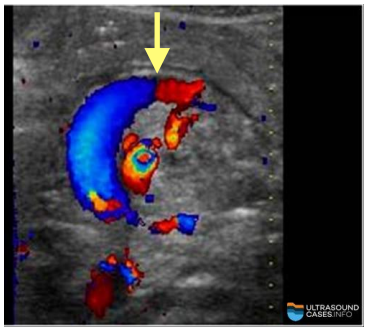


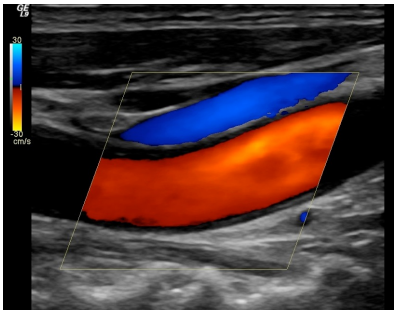
Color scale display
What color is assigned for positive and for negative.
PRF (displayed doppler shift freq) and the range it's able to detect (maximum velocity)

The scale should be set to the type of flow you are evaluating in order to be displayed accurately
Slow flow = Low scale Fast flow = High scale

How angle affects brightness and color

Closer to parallel = higher doppler shift and therefore, brighter color. Perpendicular (as shown by arrow) = NO doppler shift and so black. Flow separations and any vessel that loops or curves will show both red and blue as part of the flow moves towards the transducer and then away.





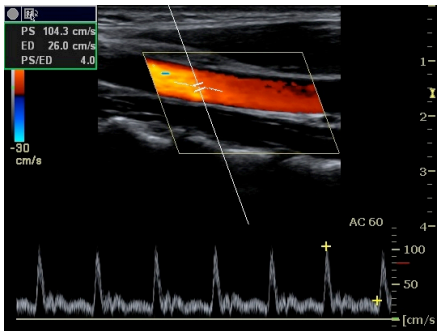
Determining flow direction

First, notice the scale. The red is negative because it's on the bottom.
Negative means away or downhill. Box is steered to left, so flow is going away from us (downhill) to the LEFT. The negative color is always going towards the side the box is steered. The positive color is the opposite.

How to correctly use Color Doppler

Steer the box in the direction of the vessel angle.
The size of the box should just cover area of interest.
Adjust the scale to fit the type of flow you are evaluating.
Adjust color gain so color fills in vessel but does not 'bleed' out of the vessel walls.

Pulsed Doppler



Evaluate flow over one small area called our sample volume or range gating.

Benefits: We decide position (where to sample) and size (how much to sample). Information will be specific and velocities can be measured. *Quantitative*

SPECTRAL ANALYSIS which breaks down the signal into separate components according to velocity(magnitude of shift) and time(location).

How to correctly use PW doppler

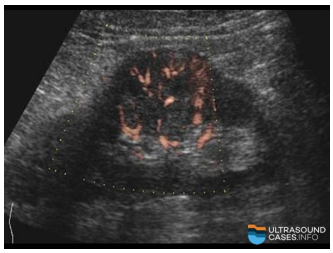
Sample at center of flow/vessel with sample angle steered in direction of vessel and parallel to vessel walls.

If the angle is not parallel, velocity measurement will be inaccurate.

Generally, ideal angles are 45-60 degrees.

Sample gate should be approximately 1/3 the size of the vessel

Power Doppler



AKA energy doppler, amplitude doppler

Only one color, usually yellow or orange.

** Maps magnitude/power/amplitude of doppler signal.

Only detects and displays the presence of flow. NO direction info

Benefit: Very sensitive to slow flow

Continuous Wave Doppler (non-imaging)

Requires 2 separate crystals side by side: 1 transmitting & 1 receiving

Only used as flow detector or to measure severely elevated velocities.

Range ambiguous: unable to determine depth or location. *Cannot choose a location.*

Doppler Optimization

Scale
Needs to match the type of flow you are evaluating

- Decrease the scale = Not sensitive enough
- Increase the scale = Aliasing

Wall filters and *High Pass filters*
Filters LOW FREQUENCY/HIGH AMPLITUDE.

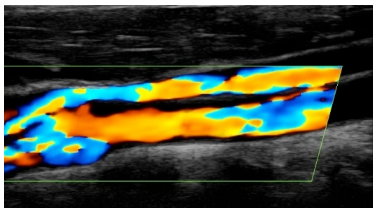
- Decrease WF = Not sensitive enough

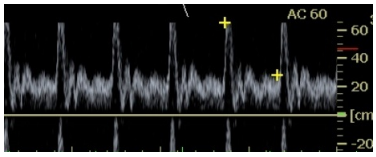
Gain
Fine tuning

- Increase gain to enhance the strength of the doppler signal
- Decrease if bleeding out of vessel

Problem: ALIASING

Shift exceeds the Nyquist limit (1/2 PRF)

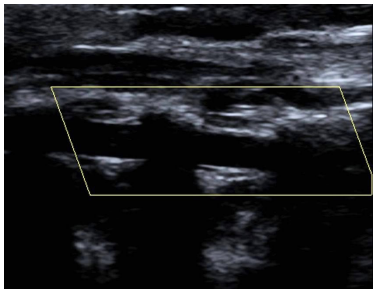




- Increase scale or PRF
- Lower baseline
- Decrease transducer frequency
- Increase doppler angle

Problem: SLOW FLOW

Low velocity flow will produce a small doppler shift. The opposite to aliasing. Simply the problem is the shift is too small to see.



- LOW scale
- LOW wall filter
- Increase frequency
- Decrease angle (closer to parallel)
- Increase color gain
- Increase packet size
- Use power doppler