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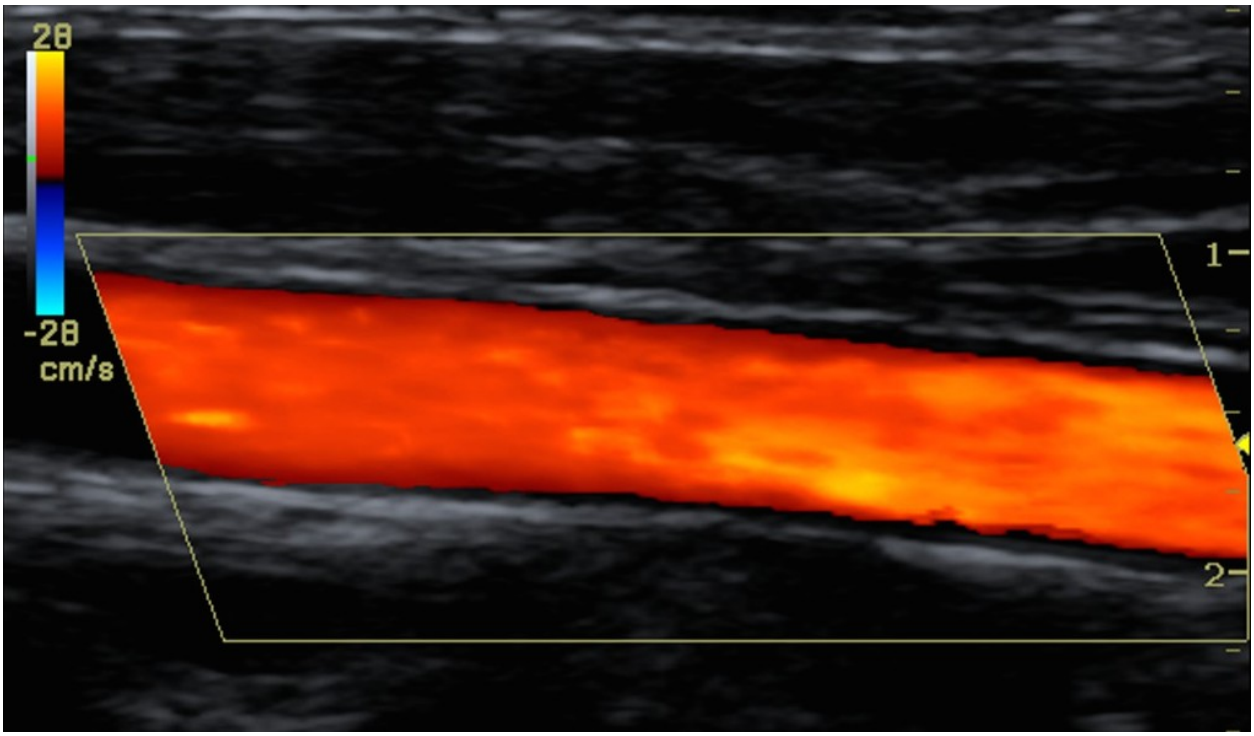
Vascular Ultrasound Registry Review



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2019 edition

Vascular Registry Review

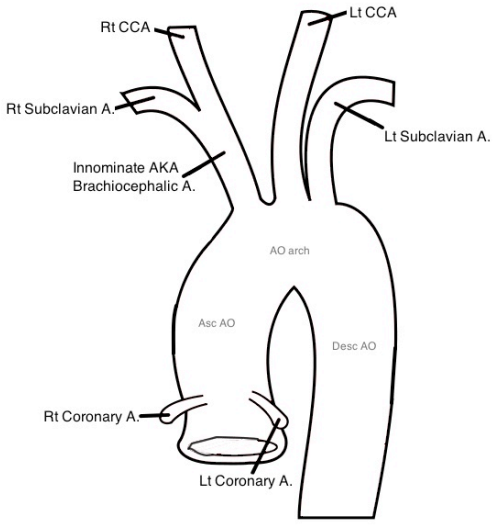
Table of Contents

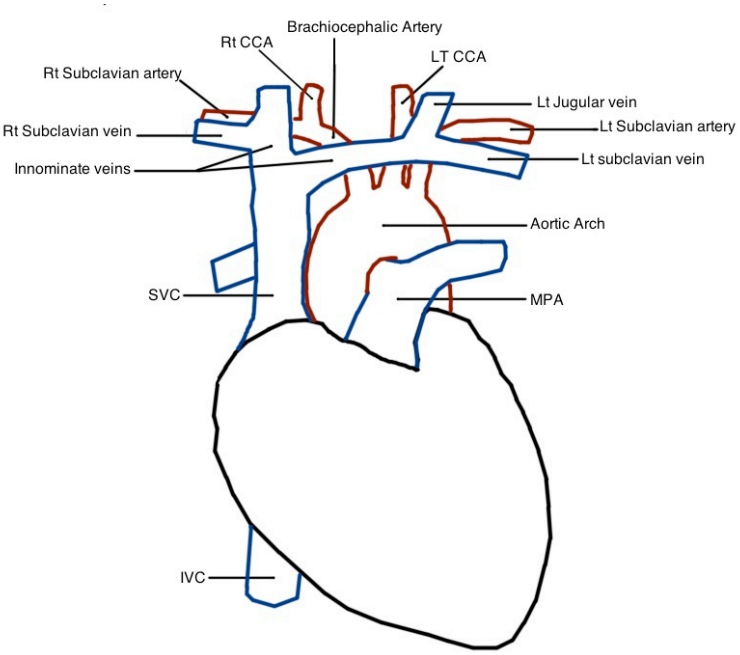
ARTERIAL

Peripheral Arterial Anatomy

Originates at aortic arch

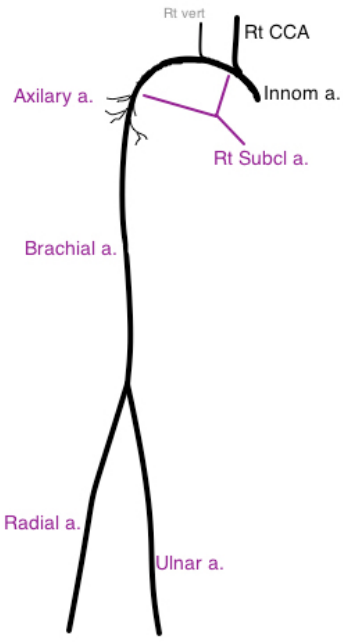
- 1st - Innominate/Brachiocephalic a,..bifurcates into Rt Subclavian and Rt CCA
- 2nd - Lt CCA
- 3rd - Lt Subclavian a



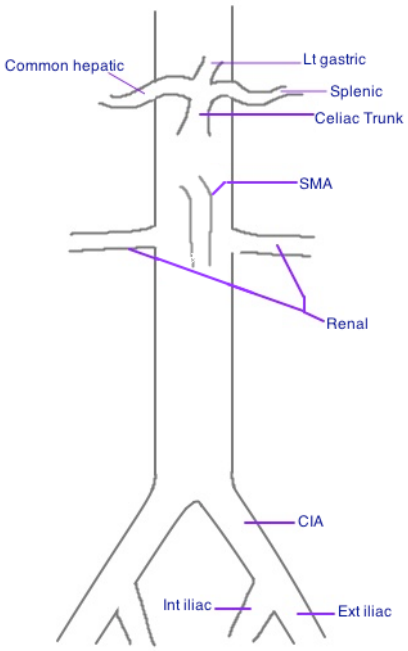


Upper Extremities

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

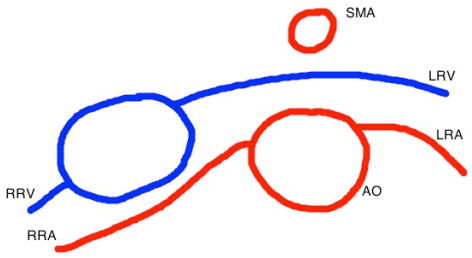


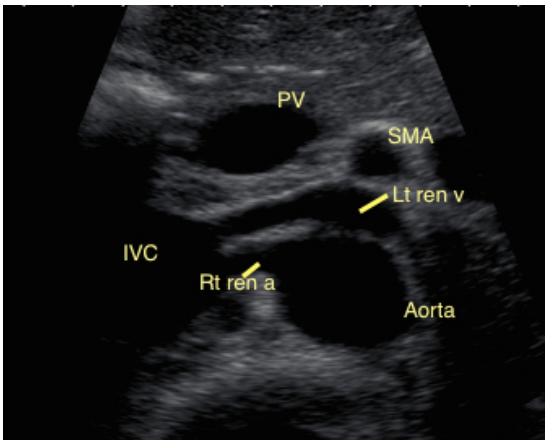
Abdominal Aorta

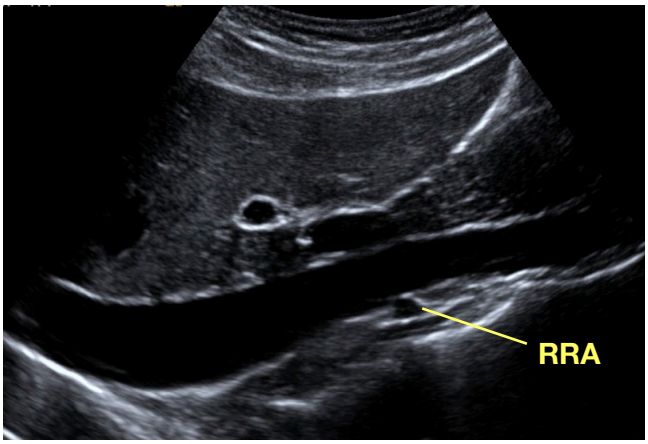


Originates at crus of the diaphragm.
Superior to Inferior branches from aorta
1st branch: *Celiac* trunk. Celiac gives rise to *common hepatic, lt gastric, splenic a.*
In transverse view (seagull sign) = only see common hep and splenic vis.
2nd: *Superior mesenteric a.* (*SMA*)
3rd: *Renal arteries* 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.

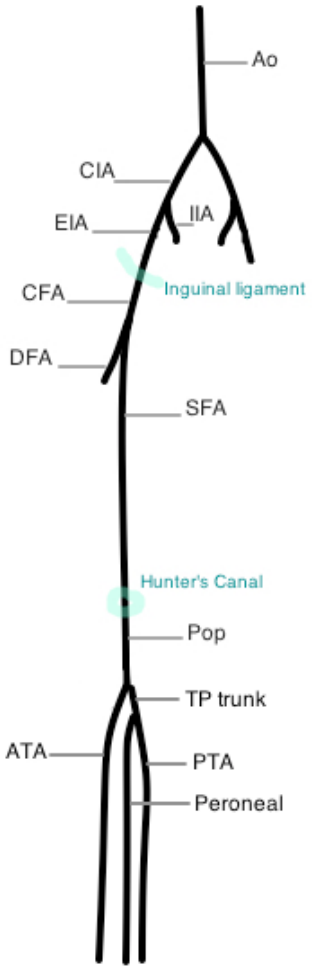


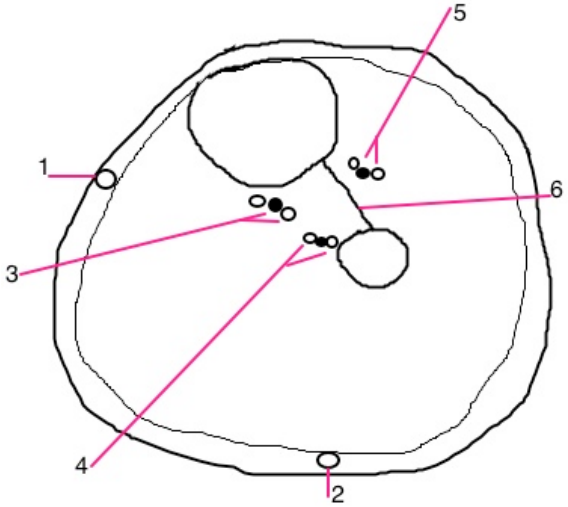




Lower Extremities

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.

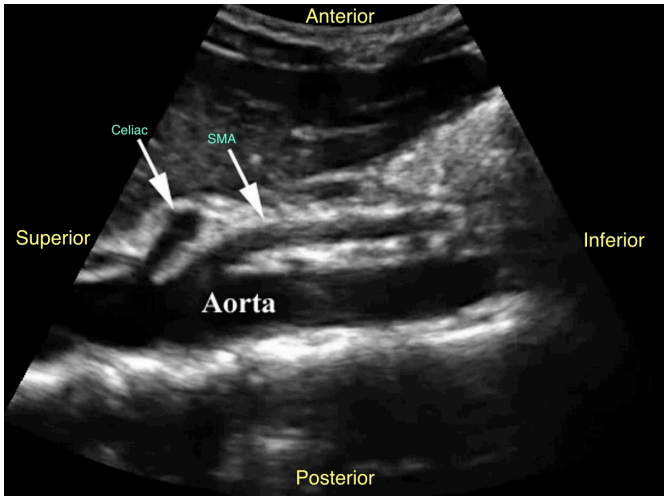
1. GSV
2. SSV
3. Post tibials
4. Peroneals
5. Ant tibials

✳ Relational anatomy

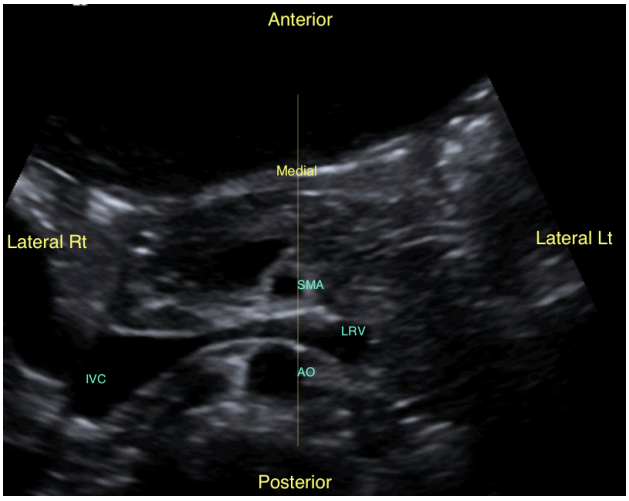
Anterior- Closer to the front of body AKA superficial
Posterior- Closer to back AKA deep
Superior - Towards the head AKA Cephalad
Inferior - Towards the feet AKA Caudal
Medial - Closer to middle
Lateral - Towards the side
Proximal - Closer to heart, flow comes from
Distal - Away from heart, flows goes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

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

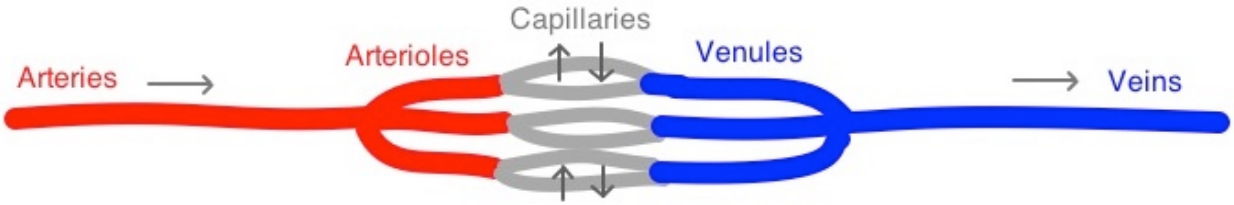
Sagittal



Transverse



Functional level of circulation



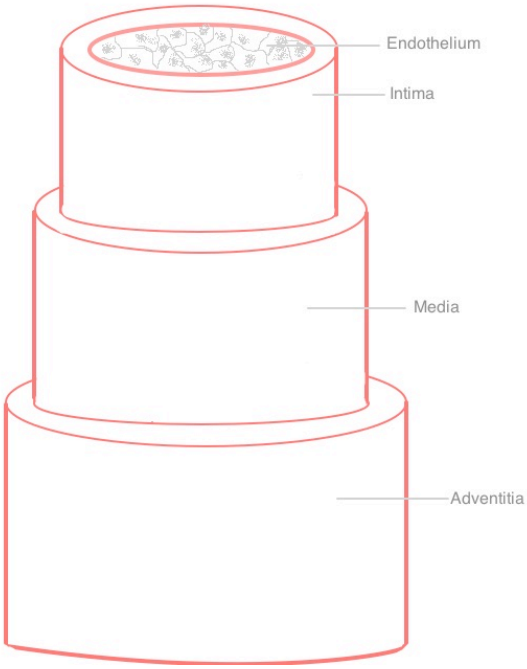
Arteries >> Arterioles >> Capillaries >> Venules >> Veins

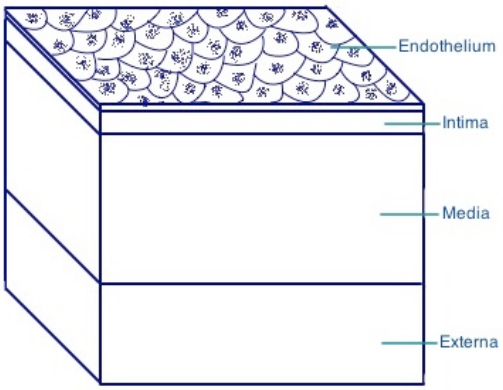
Arterioles : Smallest arteries. Vessels of resistance

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

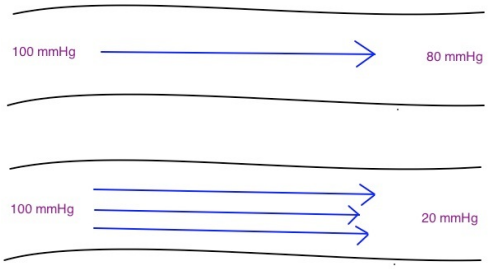
Blood flow depends on 2 main things
Pressure gradient and Resistance

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

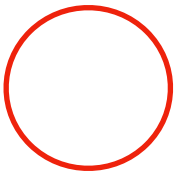
Decreasing diameter would increase resistance and decrease flow

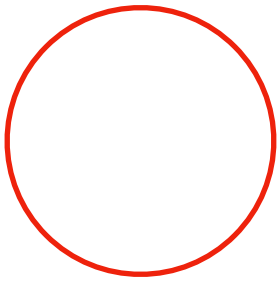
Flow volume directly proportional to diameter. Notice how radius is to the 4th power.
That means small changes in radius result in big changes to flow.
Decreased radius = increased resistance = decreased volume
Flow is inversely related to length and viscosity

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**

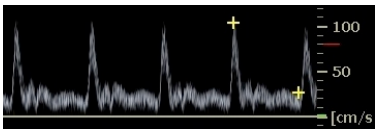
When there is a narrowing (stenosis), the velocities increase and the pressure drops

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

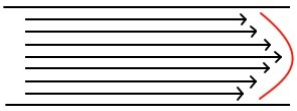
Laminar

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

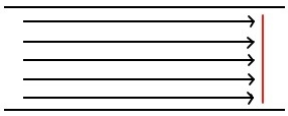


Spectral window indicates laminar flow. The PW is sampling one, neat layer which moves at its own speed. So the waveform displays a neat, organized flow pattern

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



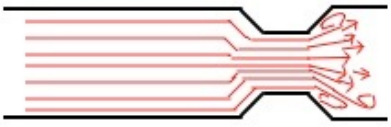
Turbulent

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



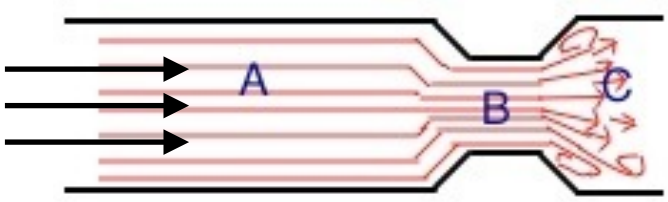
The 2 main factors are radius and velocity. Both DIRECTLY RELATED

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

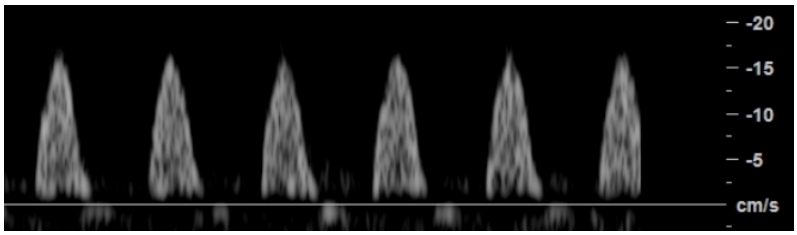
"Post-stenotic turbulence" = AFTER stenosis when vessel diameter is larger but velocity is increased. *Spectral broadening* indicates turbulence.

Stenosis profile

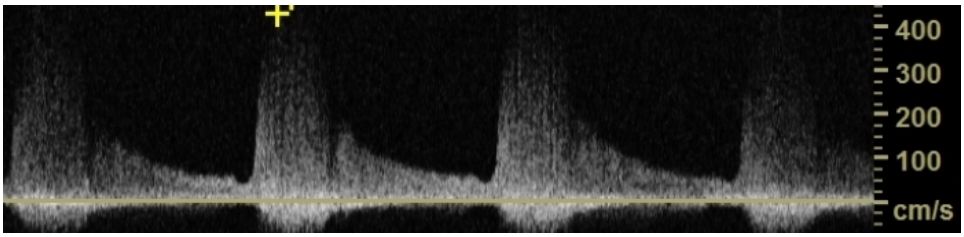
How stenosis affects blood flow. Hemodynamically significant means the stenosis has reached at least 50% diameter reduction and has altered flow patterns.
50% diameter = 75% area reduction



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



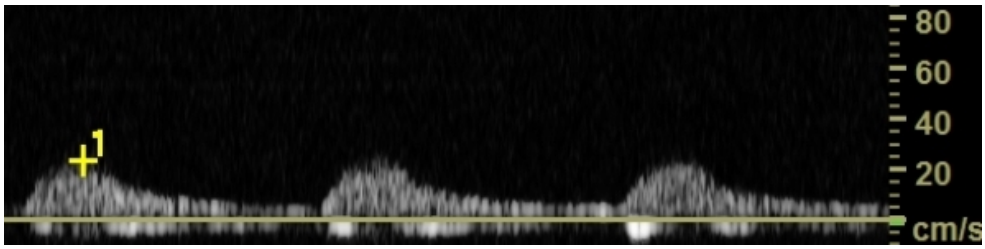
B. **At the stenosis.** Elevated PSV and EDV through the narrowed section. Velocity must increase when area decreases to maintain volume flow. Increase to velocity = decrease in pressure (Bernoulli). Highest velocity



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

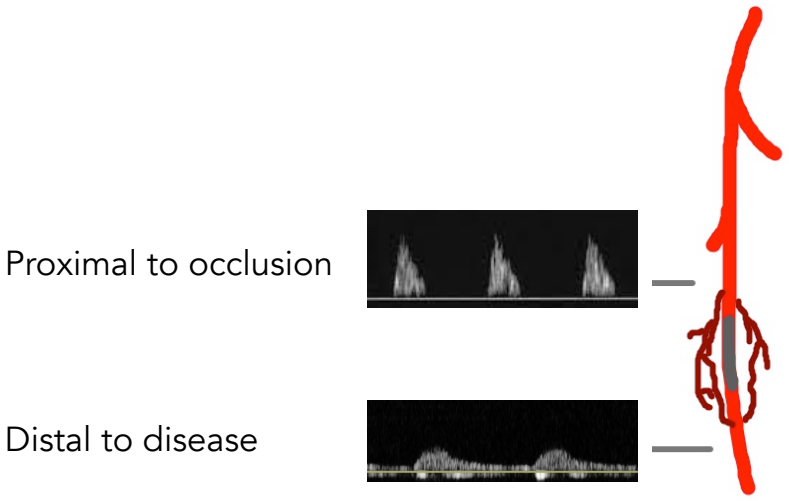


Proximal or Distal???

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??

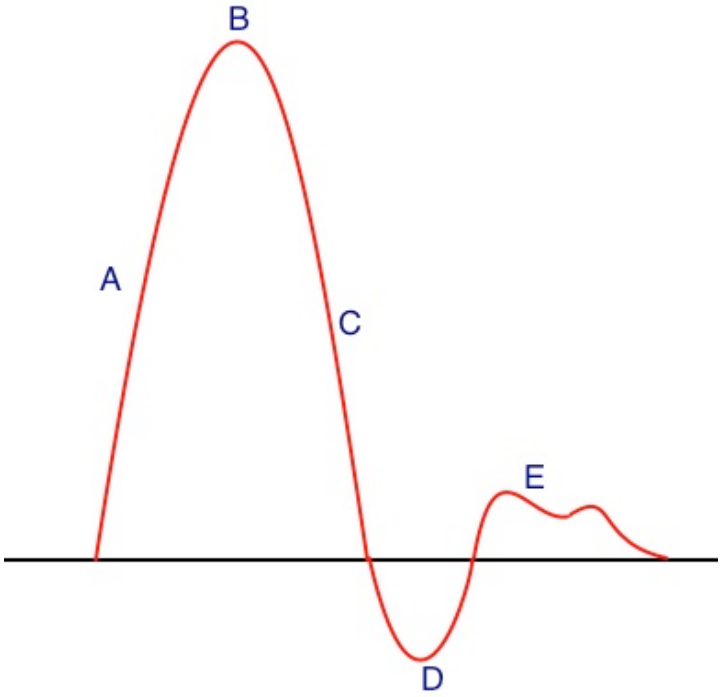
Proximal to stenosis = Distal obstruction

Distal to stenosis = Proximal obstruction



Cardiac effects on peripheral flow

Peripheral flow reflects the pulsatility of the cardiac cycle



A - Early systole

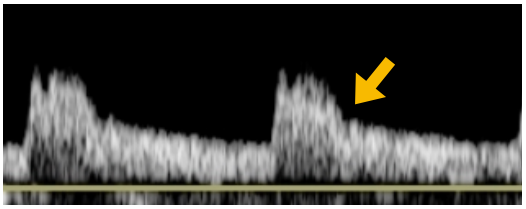
B - Peak systole - Maximum forward flow. Aortic walls distend = potential energy

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**

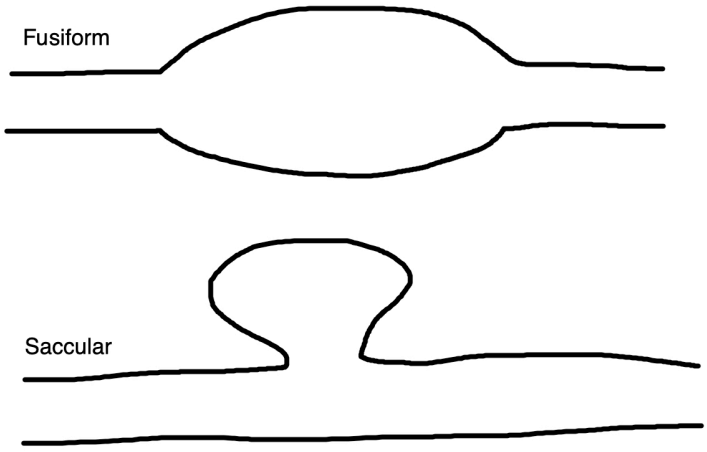
  Young men (<40yo) that are heavy smokers. Affects the smallest most distal vessels (digits).

  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



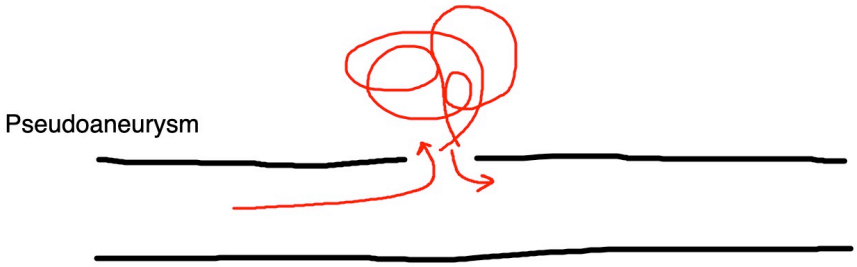
Most common location: Infrarenal aorta
Most common cause: Atherosclerosis
Most common type: Fusiform

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

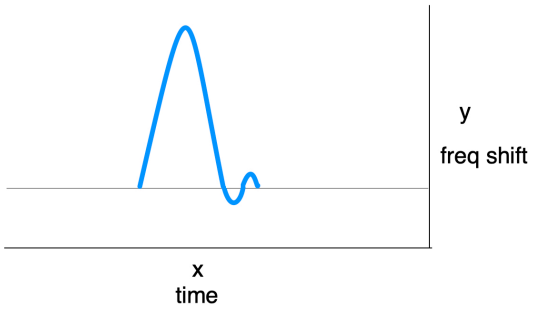
Presence/absence of arterial occlusive disease
Approximate level of disease, NOT exact location or severity
Tech dependent

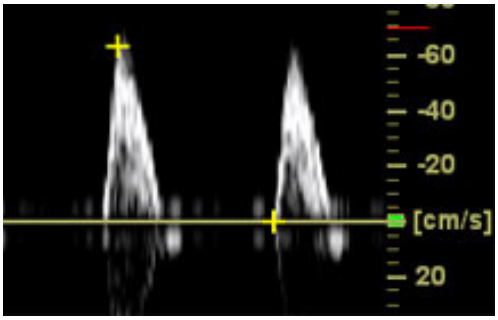
Physical principles

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

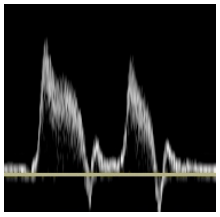
Supine - limbs same as heart
8-10 MHz CW probe
Probe angle 45-60 degrees to skin surface

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

Interpretation

Mainly qualitative

Normal: Triphasic or Biphasic



Abnormal: Monophasic or monophasic, continuous.

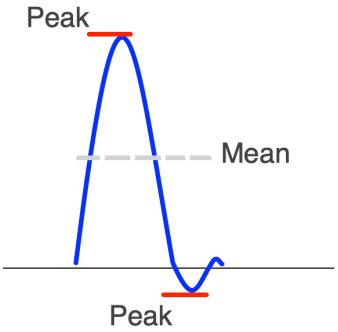
** Deterioration in signal contour from one level to the next =

Disease between those levels

Quantitative

Pulsatility index = measure resistance

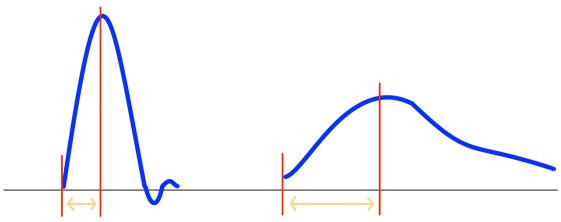
$$\frac{\text{Peak to peak freq}}{\text{Mean freq}}$$



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

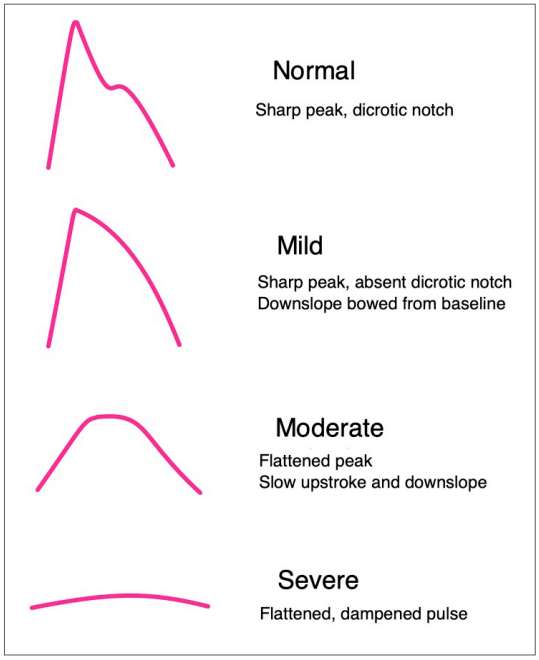
pneumo/true/air plethysmography = uses pneumatic cuffs (BP cuffs with special sensor) to detect changes in volume

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

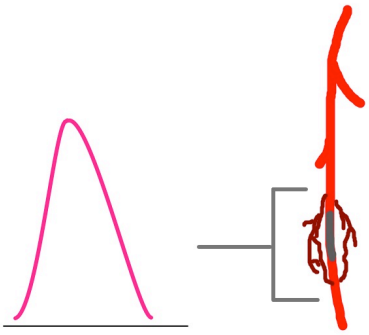
Qualitative only. Based on waveform contour!

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)

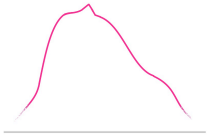
For Raynauds - *Cold stress:*
1st perform at rest
2nd soak in ice water for 3min or to tolerance
Obtain pressures and waveforms

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

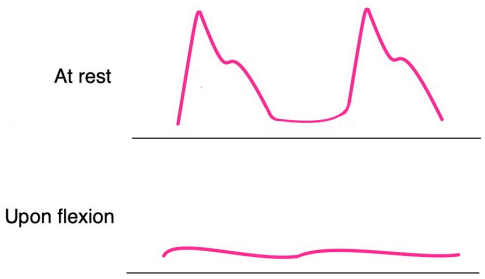
Normal >50mmHg
Poor <40mmHg (impaired wound healing)
Critical <30mmHg (non-healing)

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.

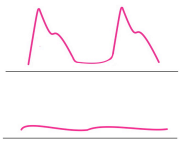
  Findings: *Normal at rest.* Only compressed when muscle is activated. *Waveforms will diminish or flatten upon flexions.*



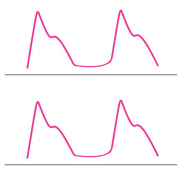
- Thoracic outlet syndrome
  Compression of neurovascular bundle by shoulder structures (ribs, ligaments, muscles). Pt complain pain, tingling, weakness in arm when in certain positions. *Most common cause is neurogenic (nerves).* Must also tested when replicating compression

  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

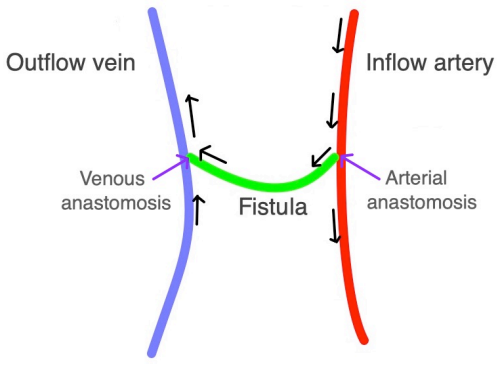
Duplex uses PW doppler (doppler physics covered in physics review)

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

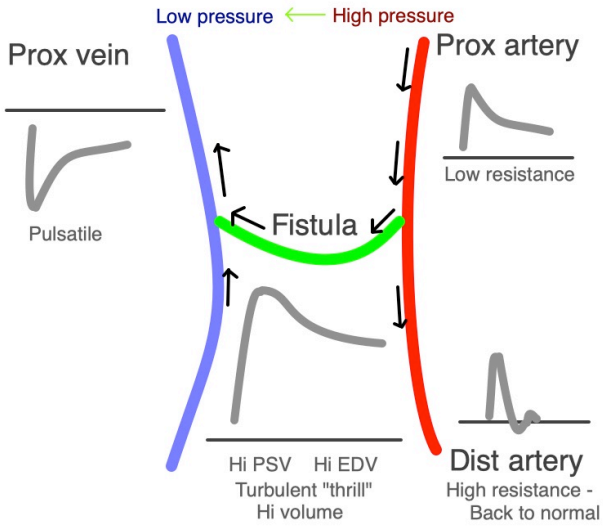


Interpretation

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

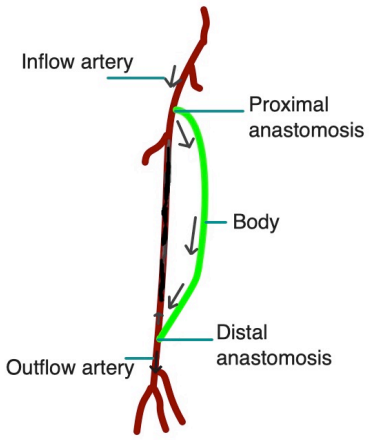
Technique

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
dis tal anastomosis
outflow artery



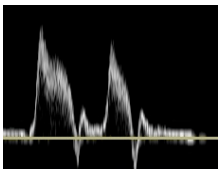
All successful bypasses must start above disease (patent inflow), have a patent conduit (graft), and end after any disease (patent outflow)

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

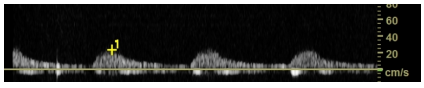
Clinical: hypertension
Renal artery stenosis > renal ischemia > renin > angiotensinogen > angiotensin

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'

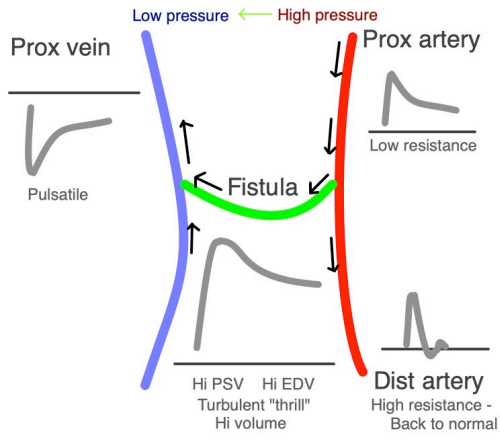
  Same hemodynamics as hemodialysis access graft = AVF!

  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

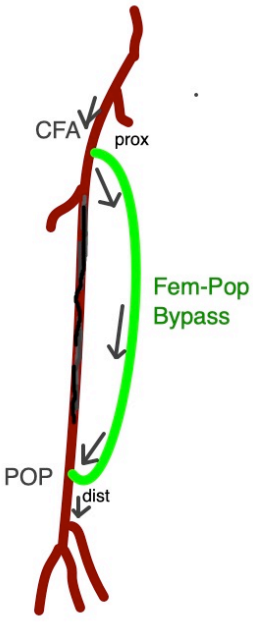
Exercise - promotes collateral formation

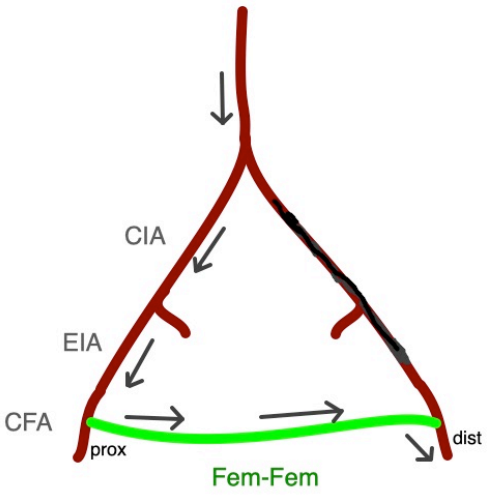
Aspirin : anti-platelet drug, reduces thrombotic activity

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
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