

VENOUS

Peripheral Venous Anatomy

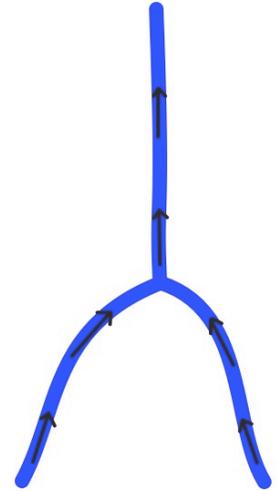
Think direction of flow!!

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

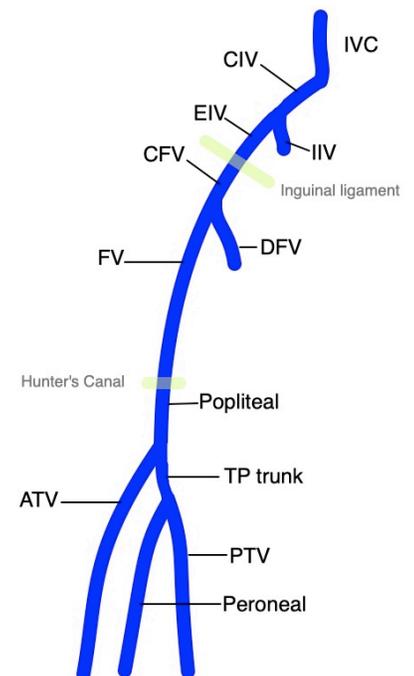
PTV and PER drain into tibioperoneal trunk. TP trunk and ATVs join to form the POP v.

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.



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Superficial veins

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

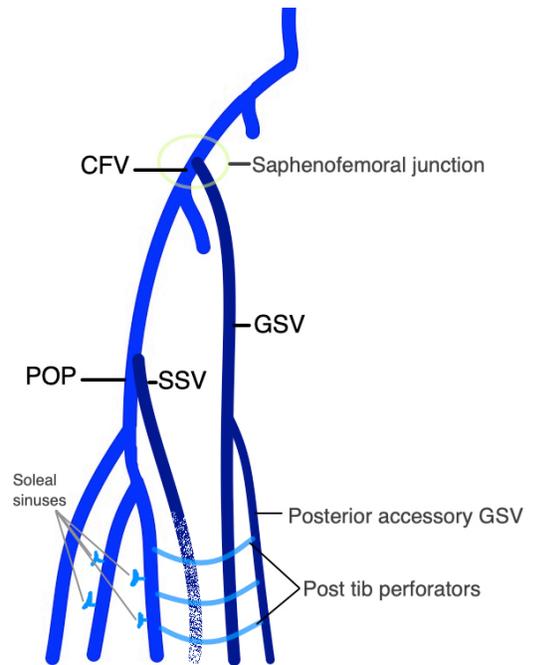
Greater saphenous vein = longest vein in body. Medial leg. Drains into the CFV at the saphenofemoral junction.

Small saphenous vein = posterior calf. Drains into POP.

Perforating 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.

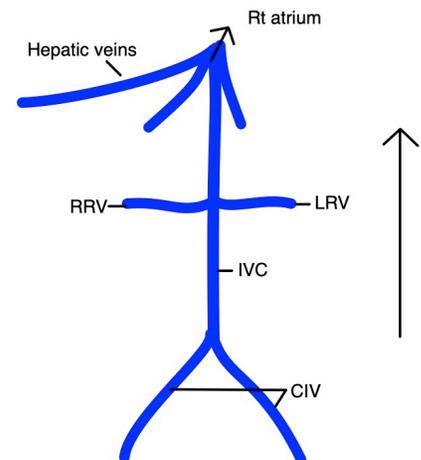
Venous sinuses = Potential reservoirs or channels that allow blood to accumulate in the calf and during calf contraction > ejected into peroneals and PTVs. Important role in calf muscle pump.



Abdominal venous system

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.



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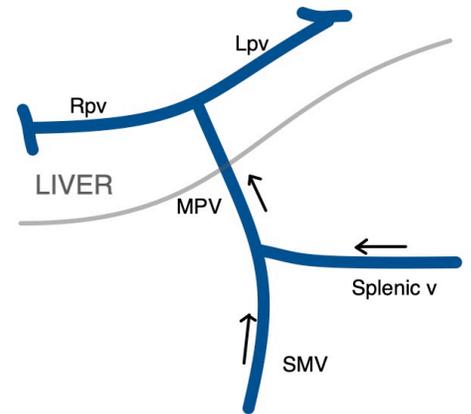
Portal venous system

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



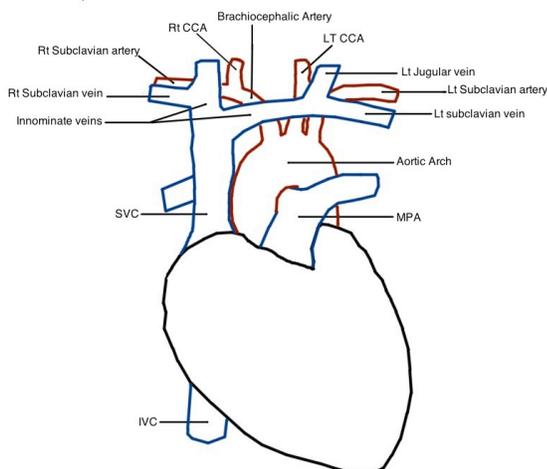
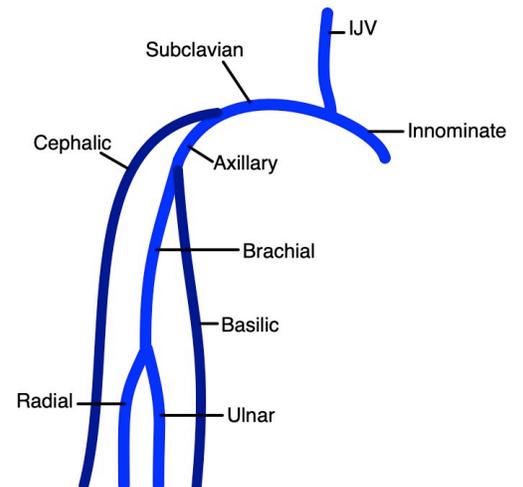
Upper Extremities

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.



Venous return to heart

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

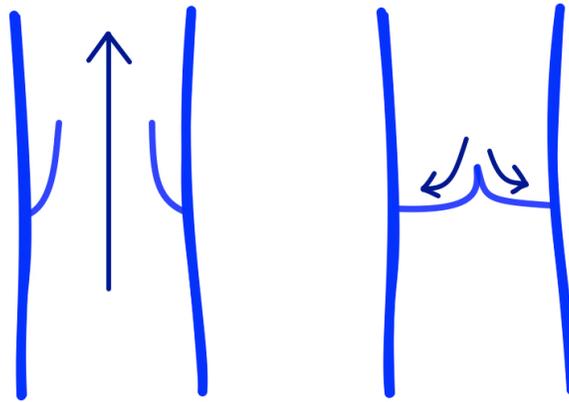
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Venous structure

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

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Venous Hemodynamics

Venous compliance

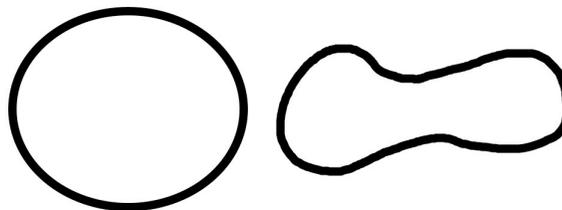
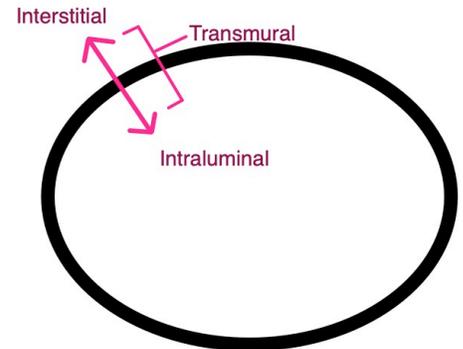
Ability to handle large changes in volume without greatly affecting the pressure.

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



Inc volume = Inc transmural pressure
Circular shape

Dec volume = Dec pressure
Elliptical shape

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

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

Respiration "Phasic flow"

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

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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 maneuver = 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

>60% blood ejected from calf
Increased venous return to heart
Decreases venous pooling
Decreases venous volume
Decreases venous pressure

Ineffective Calf Pump

Incompetent valves
Increased venous pooling
Increases venous volume
Increases venous pressure
Venous hypertension

Venous hypertension

Any abnormal increase in venous pressure due to high venous volume

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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)

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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 iliofemoral 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-Trenaunay (hypoplastic or absent deep veins)

Post-thrombotic / post-phlebotic syndrome

Pt had previous DVT. Damaged valves lead to chronic insufficiency

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

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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

Edema

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

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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

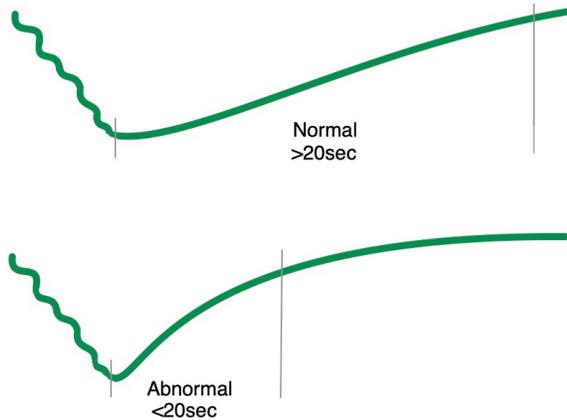
Incompetent valves = Fills up quickly <20sec

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

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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



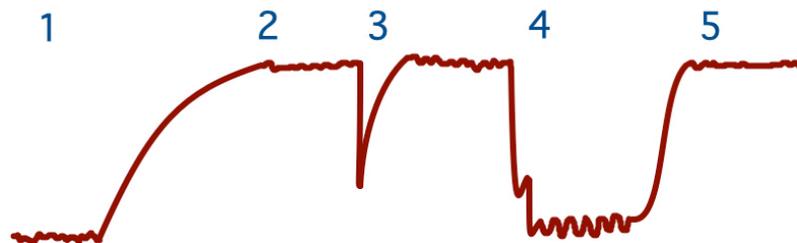
Venous PPG reading	
>20 sec VRT	Normal
<20 sec VRT	Insufficiency
Tourniquet normal AK	GSV insuff
Tourniquet normal BK	SSV insuff
Never normalizes	Deep insuff

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)

Vascular Registry Review

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

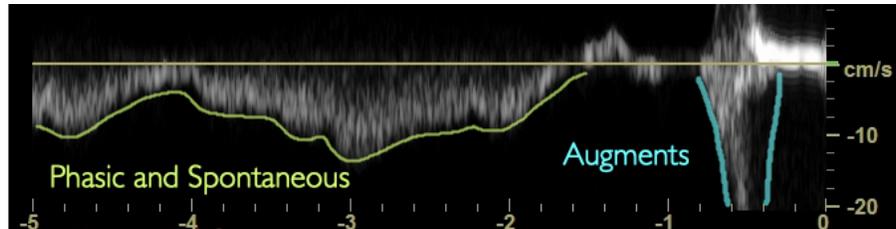
LE externally rotated with knee slightly bent

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

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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

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Venous Duplex

Capabilities and limitations

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

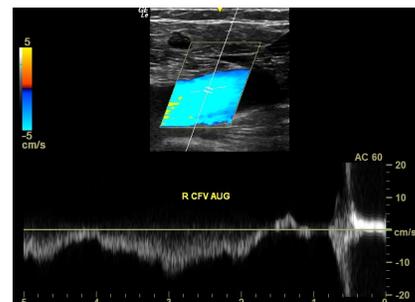
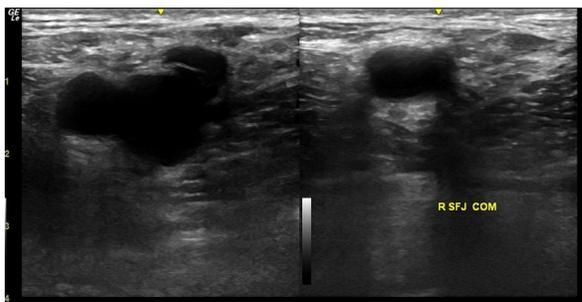
Technique

- 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

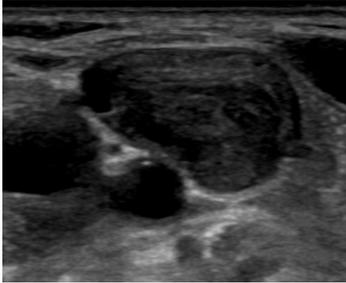
- ▶ Normal veins fully compress = Coaptation. No thrombus
- ▶ Fully fill with color
- ▶ Phasic, spontaneous and augmentation
- ▶ No reflux when standing or during valsalva/prox comp



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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

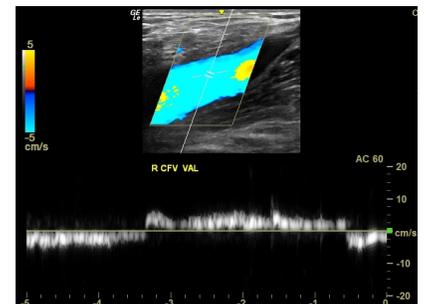
Smaller vessel size, thicker walls.
Hyperechoic striations, linear bands scattered within vessel.
Flow recanalizes throughout vessel, patchy color flow. And partially compressible vessel. Collateral veins may also be seen

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

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- Additional findings

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

Edema - fluid found infiltrating tissues

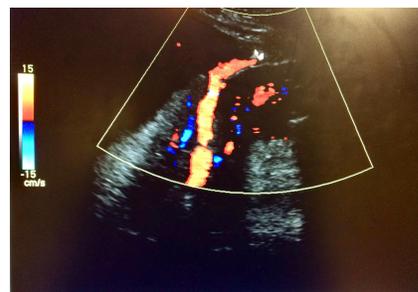
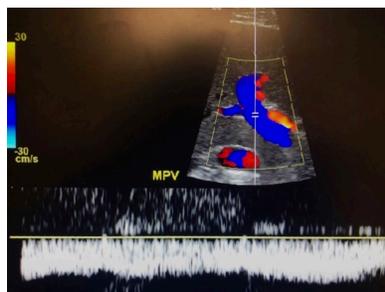
Joint effusion - fluid at joint, anterior to bone

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

Abdominal venous duplex

- 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

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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

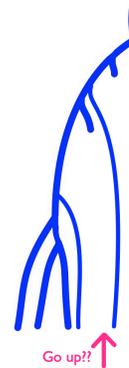
Same idea as angio. Dye is injected into vein and x-rays show the flow of dye material

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

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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.

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Varicose veins/ Insufficiency

Deep venous insufficiency cannot be corrected. Only symptoms minimized and reduced progression of disease. 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

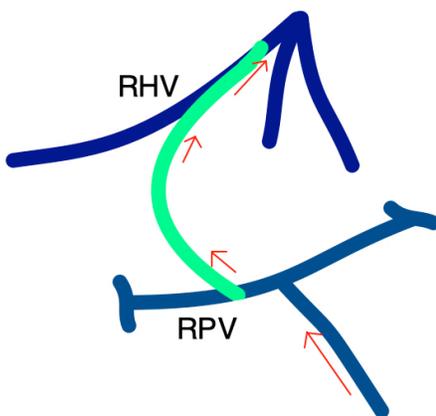
Sclerotherapy: Injections for the removal/closure of spider veins

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)

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Quality Assurance

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

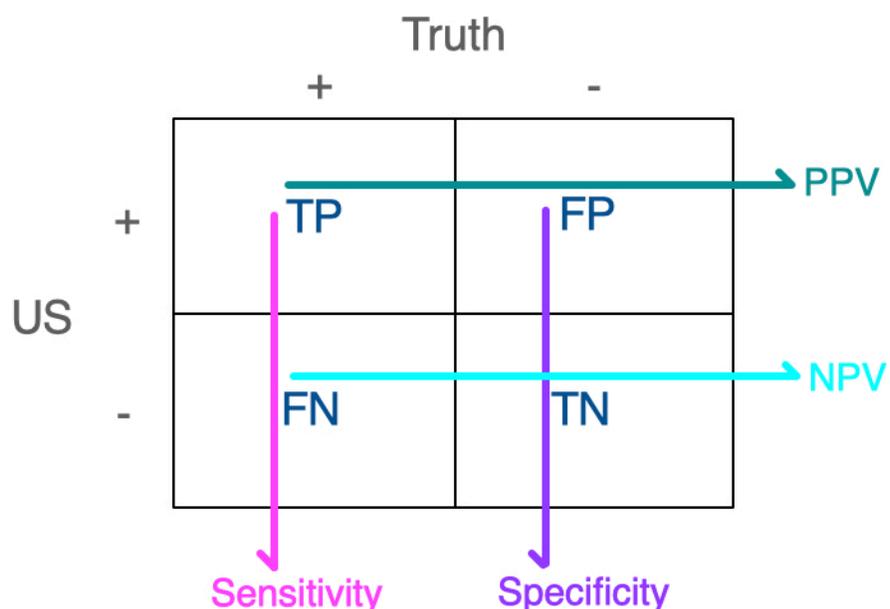
True positive- our test found disease and there really was disease

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



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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 by both methods

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

Vascular Registry Review

Physics Review

** basic physics coverage as applied to Vascular

Transducer choice and principles of frequency

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

- Scan type
- Body habitus

Increase frequency = better axial resolution / poorer penetration

To scan deeper > decrease frequency

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

Vascular Registry Review

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 echoes deep to real reflector
Examples: micro calcifications, 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

Vascular Registry Review

The DOPPLER EFFECT

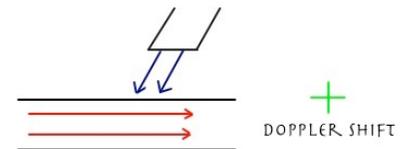
Object in motion will produce echoes that come back at a slightly different frequency than what was sent out.

Moving TOWARDS the sound source = HIGHER received frequency

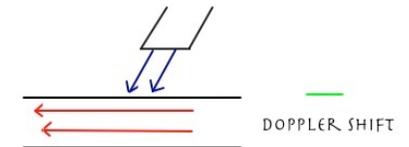
Moving AWAY from sound source = LOWER received Frequency

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

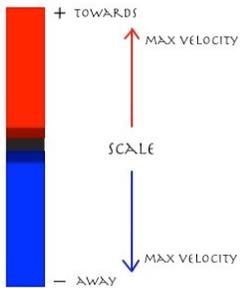
3 things that affect the shift:

- 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 = Greatest shift

Vascular Registry Review

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.

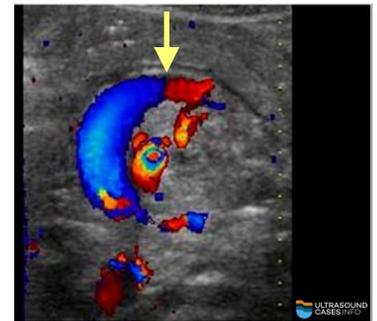
PRF (displayed doppler shift freq) and the range it's able to detect (maximum velocity)

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.



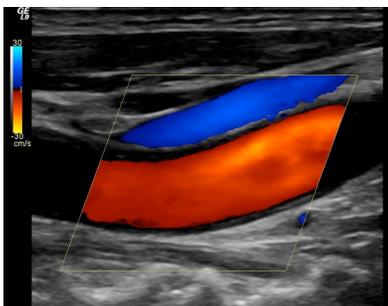
How to correctly use Color Doppler

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.



Determining flow direction

First, notice the scale. The red is negative because it's on the bottom.

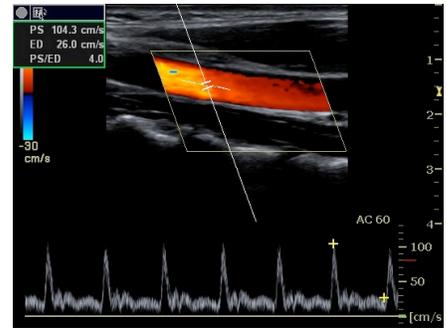
Negative means away or downhill. Box is steered to left, so flow is going away from us (downhill) to the LEFT. The negative color is always going towards the side the box is steered. The positive color is the opposite.

Vascular Registry Review

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.*

Vascular Registry Review

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

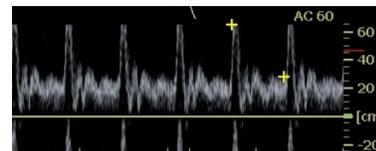
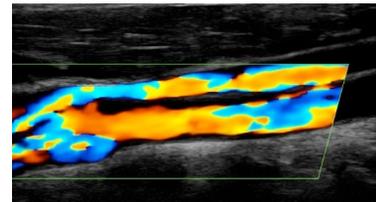
Fine tuning

- 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

