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Position Of The Heart In The Chest

The heart lies in the chest on an angle, with the apex pointing to the left side of the body. Approximately two-thirds of the heart is on the left side of the chest and one-third is on the right. The normal heart is about the size of a clenched fist.

If you make a fist with your left hand, placing the knuckles on the sternum gives an indication of the position the heart lies within the chest.

Position Of The Valves

Two atrio-ventricular valves, tricuspid and mitral, sit between the atria and the ventricles. Two ventricular-arterial valves, pulmonary and aortic, sit between the ventricles and the great arteries. The aortic and pulmonary valves lie at different angles to each other and are opposed by approximately 60 degrees.
Direction Of Blood Flow In The Heart

The cardiovascular system is a closed circuit. The heart consists of two pumps. The right side of the heart directs blood to the lungs and the left side pumps blood to the body. Flow at any point must equal flow at any other point (in the absence of regurgitation or shunt).

Right Heart

Blood flows from the body through the IVC and SVC into the right atrium. The tricuspid valve (TV) opens and the deoxygenated blood flows into the right ventricle (RV). The tricuspid valve closes and pressure builds in the right ventricle (RV). This pressure forces the pulmonary valve (PV) open, pushing blood into the pulmonary artery (PA) and then to the lungs.

Left Heart

Oxygenated blood returns from the lungs via the pulmonary veins and enters into the left atrium. The mitral valve (MV) opens and the oxygenated blood flows into the left ventricle (LV). The mitral valve closes. Pressure builds up in the left ventricle forcing the aortic valve (AV) to open. With the aortic valve open, the blood is pushed forward into the aorta and to the body.

ANATOMY and PHYSIOLOGY

Systole

Contraction of the ventricles ejects blood through the aortic and pulmonary valves with high velocity.

Diastole

After the aortic valve and pulmonary valve close, the heart cycle enters diastole. The mitral and tricuspid valves open to allow blood flow into the ventricles. Three phases of diastole - Early filling, diastasis and atrial contraction.
Heart Sounds

‘Lub – dub’ is the classic sound heard with the stethoscope. ’Lub’ is end-diastolic valve closure (MV and TV). ’Dub’ is end-systolic valve closure (PV and AV).

These sounds are slightly different to those heard with Doppler. Doppler records the movement of blood flow, usually heard as soft, ‘whooshing’ sounds. ’Clicky’ sounds, which represent valve opening and closure, can also be heard and recorded.
Blood flow through the heart, valves and great vessels is either laminar or turbulent.

**Laminar Flow**

**Straight Vessels**

Normal flow is called laminar. All of the red blood cells travel at approximately the same velocity.

In the normal heart, flow from the left ventricle through the outflow tract, valves and great vessels is typically laminar.

A parabolic shape is formed (see diagram). As the blood passes along the arteries or veins, the walls of the vessel slow the flow down, due to friction. Hence, the blood at the centre moves at a faster rate.

In three dimensions this would look like a bullet. An example of a straight vessel is the ascending aorta.

Faster velocities occur as the blood flows from the ventricles through the normal aortic or pulmonary valve.

Imagine running water coming out of a hose or tap, this is laminar flow. Now think about what happens when you cover a small area of the opening, with your finger. The orifice is now smaller and the water runs at a faster rate. This increase in velocity of flow happens because the same amount of water is flowing from a large area to a smaller area.
FLOWdynamics

Curved Vessels

When the blood flows through the ascending aorta, aortic arch and descending aorta, changes can be seen in the laminar flow profile. As the blood travels along the ascending aorta the flow profile becomes asymmetric, with increased velocities along the inner wall due to the curvature in the vessel. As the blood travels down the descending aorta the flow profile shows increased velocities along the outer wall (see diagram).

Turbulent Flow

This typically occurs as a result of narrowed or stenotic valves. Regurgitant flow is also turbulent. During systolic contraction, the same stroke volume of blood must pass through the stenotic orifice. The obstruction to flow forces the blood to pass through the narrowed orifice at a higher velocity. As the flow of increased velocity passes the obstruction, resultant flow vorticies, or eddies occur. These eddies are made up of blood flowing in many different directions and velocities. With Doppler, the effect of a stenotic valve is seen as an increase in peak velocity, filling in of the profile and a high pitched sound.

Once again imagine running water coming out of the hose or tap. This time cover a lot more of the opening with your finger. The orifice is a lot smaller and the water now runs at a much faster rate and is spitting in all directions out of the hose. The sound has changed also. This increase in velocity of flow happens because the same amount of water is flowing from a larger area to a much smaller area. This result is the same as flow passing through a stenotic valve. The same volume of blood must pass the obstruction in the same amount of time, to maintain stroke volume.
**BLOODflow VOLUME**

The basic volume calculation is: \[ \text{Volume} = \text{Area} \times \text{Height} \]

The amount of blood travelling across the aortic or pulmonary valve in one beat can be measured as a volume.

**Cross Sectional Area**

The area of the valve or outflow tract can be used in the volume equation. If the diameter of the valve or outflow tract (OT) is known, the area can be calculated.

Diameter of the outflow tract (OTD) is 1.9 cm

\[
\text{OT area} = \pi r^2
\]

\[
\text{OT area} = 2.8 \text{ cm}^2
\]

**Stroke Distance - Velocity Time Integral**

When the volume equation is applied physiologically, the distance a column of blood travels in one beat is called the stroke distance. The stroke distance is measured by Doppler as the velocity time integral (vti).

vti is a method of calculating the distance blood has travelled, allowing for the velocity and time that the blood has been moving.

\[
\text{Stroke Volume} = \text{OT Area} \times \text{Stroke Distance (vti)}
\]

If the blood travels 20 cm with each beat, then

\[
\text{Stroke Volume} = \text{OT Area} \times \text{vti}
\]

\[
= 2.8 \text{ cm}^2 \times 20 \text{ cm}
\]

\[
= 56 \text{ cm}^3
\]

\[
\text{Stroke Volume} = 56 \text{ ml}
\]
OUTFLOW tract DIAMETER

The aortic or pulmonary outflow tract diameter is used to calculate area for stroke volume (SV) and cardiac output (CO).

**Aortic OTD**

The aortic OTD is measured from the aortic valve annulus. The aortic valve annulus is a fibrous structure made up of a ring of collagenous tissue surrounding and supporting the aortic orifice.

The aortic OTD is fairly rigid and therefore has little variability during systole. It also does not alter significantly in size after adulthood.

**Pulmonary OTD**

The pulmonary OTD is measured from the pulmonary valve annulus.

The pulmonary valve annulus is part of the fibrous skeleton of the heart. It is a subendocardial connective tissue thickening that has similar structure to the aortic valve annulus.
An anthropometric algorithm can be used to calculate flow volume.\textsuperscript{1} In adults and children, the OTD has been shown to correlate linearly with height.\textsuperscript{2} Usually the OTD is measured directly from a two-dimensional echocardiogram. A two-dimensional echocardiogram independent method for calculating flow volumes from Doppler spectral flow can also be used. This is a simpler method which is more accurate.

\[ SV = CSA \times vti \]

Right sided SV = Left sided SV

So:

\[ PV (CSA \times vti) = AV (CSA \times vti) \]

In children, the correlation is constant for both weight and height. As weight is a more commonly used measurement, it is used in the algorithm for neonates under 50cm in height.\textsuperscript{3}

In adults and children, the OTD has been shown to correlate linearly with height.
DOPPLER basics

Doppler Effect

The Doppler effect is a change in pitch or frequency associated with a moving sound source or observer. Pitch and frequency (f) increase as the source approaches and decreases as it moves away.

Medical ultrasound equipment uses Doppler to emit a constant frequency of sound waves that bounce off red blood cells.

The red blood cells are either travelling toward the transducer or away from the transducer.

When the sound waves and red blood cells meet, the waves that bounce back to the transducer are either at a higher or lower frequency than those originally emitted, depending on the direction of flow.

Oncoming blood cells change the sound waves to a higher frequency.

The blood cells moving away change the sound waves to a lower frequency.

Think about an approaching racing car. The pitch of the engine increases as the racing car approaches and decreases as it drives away.
DOPPLER basics

Doppler Display

The frequency change is displayed as a Doppler shift and the actual velocity of the moving red blood cells is calculated using the Doppler equation.

\[
F_d = \frac{2F_t \times V \times \cos \theta}{C}
\]

Fd is the Doppler frequency, Ft is transmitted frequency, V is velocity of red blood cells, \( \theta \) is the angle between the direction of the moving target and the ultrasound beam and C is velocity of sound in soft tissue and is a constant.

Doppler shift displayed as a velocity-time graph.

Continuous Wave Doppler

Continuous Wave Doppler (CW) uses ultrasound waves (frequency > 20,000 Hz) to provide information on the velocity and direction of blood flow.

CW is a form of Doppler that continuously transmits sound waves. At the same time, it receives the reflected sound waves with the Doppler shift information caused by a moving target. Moving targets are red blood cells in the heart and great arteries.

A unique feature of CW Doppler is that it continuously reflects from each moving red blood cell within its beam. This beam, when aimed at the transaortic flow, (suprasternal notch position) will reflect flow from the aorta, aortic valve, left ventricular outflow tract and the left ventricle.
**DOPPLER basics**

**Continuous Wave Doppler**

Continuous wave Doppler continuously sends and receives sound waves.

Blood travelling in line with the ultrasound beam, toward the transducer.

Blood travelling in line with the ultrasound beam, away from the transducer.

**Doppler Angle**

The aim is to direct the beam to reflect off the red blood cells ‘head on’ or ‘in line with the direction of blood flow’. When the Doppler beam is in line with the direction of blood flow, a maximal and optimal velocity is measured.

The ‘displayed’ velocity is affected by the angle of insonation to the blood flow, therefore it is important to minimise this angle.
**DOPPLER basics**

**Effects Of Air, Bone And Adipose Tissue**

If the CW Doppler beam encounters air, bone or thick layers of adipose tissue, the beam is altered. This reduces its ability to reach the red blood cells and be reflected back to the transducer.

![Well reflected profile](image)

**Air**

When the ultrasound beam encounters air, the beam is almost entirely reflected back to the transducer with practically no transmitted sound reaching the vessel. The resulting spectral display is noisy with no discernable shape or characteristic audible signal. If the lungs are in the path of the ultrasound beam, the air will prevent the signal from reaching blood flow across the pulmonary valve.

![No transmission due to reflection of the beam](image)

**Bone**

When the ultrasound beam encounters bone, no transmission of the beam occurs beyond this point. The CW ultrasound beam does not reach the red blood cells as the entire beam is reflected and absorbed by the bone.

The resulting spectral display has no discernable shape and no characteristic audible signal. The ribs and sternum prevent the ultrasound beam from reaching blood flow across the pulmonary valve.

![No transmission due to total absorption of the beam](image)
DOPPLER basics

Adipose Tissue

When the ultrasound beam encounters excessive fat the beam is attenuated. Attenuation results in a marked reduction in the strength of the ultrasound signal. The ultrasound beam still reaches the red blood cells although the signal is weakened. The spectral display has both characteristic shape and audible signal but of reduced intensity.

Reduced transmission due to attenuation of the beam.
DOPPLER flow PROFILES

The characteristic systolic flow profile is triangular shaped and provides information on:

**VELOCITY:** the speed the red blood cells are travelling at a given time
**DIRECTION OF FLOW:** toward or away from the transducer
**TIMING:** systolic ejection time and diastole
**INTENSITY:** the greater the intensity, the greater the number of red blood cells moving at that velocity

![Doppler flow profile showing velocity, direction, timing and intensity of blood flow.](image)

The flow profile is triangular because initially the ventricle pumps the blood with increasing velocity (onset to peak) and then reducing velocity (peak to baseline).

**Velocity**

During the cardiac cycle, the red blood cells move at different velocities. The ultrasound beam traverses the aorta, aortic valve and left ventricle. Slower velocities are recorded from the larger diameter areas (LV cavity) and higher velocities from the smaller areas (AV). The spectral display is filled in, reflecting the range of velocities recorded by the red blood cells.

**Direction Of Flow**

By convention, if the blood is flowing towards the transducer, the spectral profile is described as positive and displayed above the baseline.
From the suprasternal notch (SSN), flow through the aortic valve is seen as travelling toward the transducer.
From the SSN, the aortic profile is positive and is displayed above the baseline.

When the blood is flowing away from the transducer, the spectral profile is described as negative and displayed below the baseline.

From the left parasternal intercostal space, flow through the pulmonary valve is seen as travelling away from the transducer. The pulmonary profile, from the left intercostal space, is negative and is displayed below the baseline.

*The measure of life.*
Timing Of Flow

1. At the onset of systole, blood flow accelerates from the ventricles into the great arteries.
2. A vertical valve click is often seen, marking the valve opening.
3. The blood flow acceleration into the great artery stops and the peak velocity is reached.
4. The blood flow velocity reduces until the pressure in the great artery exceeds that of the ventricle and the valve snaps closed. A valve closure click may be seen.
5. The characteristic shape is a triangle. The area under the curve is calculated as the vti.
6. Following valve closure diastolic flow may be observed.

Diastolic Flow

Diastolic flows can also be observed with CW Doppler.
The characteristic diastolic flow profile is an ‘M’ shape, which reflects movement of the blood in the ventricle during the diastolic phases of early filling and atrial contraction.
The diastolic profile, seen on the spectral display, is a normal phenomenon.
The CW beam, when aimed down the ascending aorta, will continue through the aortic valve and into the left ventricle. The beam may pick up left ventricular flow as the blood travels, in a positive direction, toward the outflow tract (AV).
DOPPLER – SOUND and ANGLE

The Doppler equation shows that the angle relative to blood flow is important. When the beam is directed parallel to blood flow, or at 0 degrees, velocity calculations are most accurate.

In contrast when the beam is perpendicular to blood flow, or at an angle of 90 degrees, the measured velocity will be zero. The velocity error decreases as the angle to blood flow is aligned. Therefore the smaller the angle of the beam to actual blood flow or the closer the angle is to zero the more accurate and reliable is the measured velocity. As the angle decreases, both the velocity and signal strength of the Doppler flow profile increase. The audible signal also changes in pitch. To optimise the Doppler flow profile and peak velocity, align the Doppler beam with the blood flow.

Velocity \( \text{measured} = V_{\text{actual}} \times \cos \varnothing \)

The diagram shows the effect of varying angles on the ‘displayed’ velocity.

If the Doppler beam is misaligned by 20°, there is a velocity error of 6%.

Practically, for optimal beam alignment, an angle of <20° either side of the direction of blood flow is needed.

Vpk calculation changes with the cosine of the angle
DOPPLER monitoring POSITIONS

Aortic Access

The suprasternal notch and supraclavicular region are two positions for acquiring the aortic flow profile.

Suprasternal Notch

The transducer is positioned in the recessed, soft tissue area directly above the sternum. The transducer can be manoeuvred parallel to and around the trachea, aiming at the aortic valve.

Note: Often the first systolic flow to be detected is from the aorta as opposed to the aortic valve.

Supraclavicular

The transducer can be positioned above the left and right clavicle, lateral to the sternocleidomastoid muscle, again pointing toward the aortic valve.

Note: Often the first systolic flow to be detected is from the aorta as opposed to the aortic valve.
**DOPPLER monitoring POSITIONS**

**Pulmonary Access**

The transducer is positioned on the left side of the sternum, between the 2nd and 5th intercostal space, with the transducer cable running parallel to the ribs and angled towards the head.

When the best intercostal space has been established, optimisation of the pulmonary valve Doppler profile is pursued.
**AORTIC technique**

The aim is to optimise the Doppler profile and peak velocity by angling the transducer Doppler beam in line with the transvalvular blood flow.

A formalised process will quickly define the area of interest by way of eliminating large areas of the chest. This technique uses the 3 dimensional planes; sagittal (head / toe), coronal (side / side) and transverse, (front / back).

**A1**
Start at the suprasternal notch (SSN). Place the transducer face at the SSN and aim the beam to the back of the neck.

Angle the transducer face up, in an arc, toward the sternum, keeping the axis along the centre of the body from head to toe.

This defines an angle within the chest between the sternum and the back. i.e. ‘saggital plane’.

Maximize the Doppler profile.

**A2**
From that position and angle rotate the transducer toward the left (4 o’clock), back to the centre of the body and then toward the right (8 o’clock).

Maximise the Doppler profile along this coronal plane.

The intersection of these two planes defines a narrow window for finding the optimal Doppler profile.

**A3**
The general direction of the aortic valve flow, from the SSN, is now detected.

Use smaller, circular movements in this direction to optimise the Doppler profile.

Maintaining this position and angle, try some downward pressure in the transverse plane. Make small circular movements.
Aortic

1. Initially, acknowledge a low velocity, unfilled systolic profile, of short systolic duration. This is flow from the ascending aorta.

2. The systolic duration increases. Late systolic filling with increasing velocity superimposing over the ascending aortic profile. This indicates the beam is nearing the aortic valve.

3. Continue to angle the transducer in the direction of the increasing velocity. As the beam lines up with the direction of flow the two sides of the triangle will become more straight and have more definition.

4. The majority of the profile is formed. There is full systolic duration, filling in of the profile and straighter sides. The peak is not well defined and appears ‘wispy’.

5. Optimising the angle through the aortic valve via small circular movements, will maximise the velocity and sharpen the peak.

AV Doppler Profile Characteristics
- Triangular shape
- Triangle base - full systolic width
- Triangle sides - sides should emanate from the base and be straight and continuous, converging in a sharp, pointy peak
- Highest velocity
- Clear start and cessation of flow
- Diastolic components
- Filling in of profile
- Respiratory variation
- Change in audio feedback

More examples
**ACQUISITION protocol**

**Aortic**

Lay the patient flat or with the head of the bed slightly elevated.
The head and neck should be in a comfortable position. Remove the pillow if necessary.
Lift the chin slightly, to gain access to the suprasternal notch (SSN).

**Select transducer**

Apply gel to the flat face of the transducer.

**Locate the best window**

Suprasternal and Supraclavicular approaches should both be tried.

**Supraclavicular approach**

The supraclavicular fossa area is above the patient’s clavicle, lateral to the sternocleidomastoid muscle of the neck. Repeat the above transducer movements from both the left and right positions. Relax the neck muscle by turning the patient’s head to the side that the transducer is placed.
Positioning from the supraclavicular area provides another angle for aiming the beam at the aortic valve flow. Initially flow from the Ascending Aorta will be detected. Confirm this flow before aiming for the AV flow.
1. Find the Ascending Aortic flow.
It is most important to recognise the Ascending Aortic (Asc Ao) flow and distinguish it from the Valvular (AV) flow.

Begin by positioning the transducer, facing flat on the SSN, pointing towards the back of the neck.

Sometimes the Asc Ao flow can be seen even in this position. It is obvious that this flow cannot be at valve level and should not be mistaken for AV flow.

Ascending Aortic flow characteristics:
- Narrow, triangular & systolic
- Well defined sides with sharp peak
- A hollow or lightly filled interior
ACQUISITION protocol

2. Confirm the Ascending Aortic Flow.

What is its Peak Velocity? Ascending Aortic Flow will be lower than AV Peak Velocity
Note the Systolic Width Ascending Aortic Flow will be shorter than the AV Systolic width

Sometimes there appears to be 2 different flows, one thin triangle (Asc Ao) but with a full systolic width across the baseline.

Acknowledge the Time Gap between the Asc Ao flow and the diastolic flow.

(This will be wider than the time gap between the true end of systole and diastole.)

The full systolic width is there but the late systolic side of the triangle is not straight and continuous and falters along its length, giving the appearance of a bump or ledge.

The peak seen is still that of the Asc Ao not AV.

In healthy, young people the peak velocity can be over 1.0 m/s.

In older people and pathologic states, this can be as low as 0.5 m/s.
**ACQUISITION protocol**

3. **Aim for Aortic Valve Flow by Maximising the Systolic Time and Velocity.**
Maximise the peak velocity and systolic width by slowly raising the handle and arc the face of the transducer up toward the sternum, angling through the chest along the saggital or ‘head to toe’ plane.

Like a pendulum or when tuning a radio station, go beyond the point that gives the best signal and then swing back to it, maintaining the axis. Continue past the maximum velocity point and then back to it again. When maximised, hold this angle.

Now rotate the transducer face to the patient’s left side in the coronal or ‘side to side’ plane. If the velocity decreases, then you are moving away from the direction on the AV, if it increases, then you are moving toward the AV. Return to the centre position, then rotate the probe towards the right shoulder. Note whether it increases or decreases. Whichever direction improves the velocity then slowly aim back in that direction until the maximum velocity in this plane is achieved.

Now you have eliminated a large part of the entire chest in the 2 main planes. This technique has narrowed the field and allows you to focus in a small area. The Doppler profile should be significantly taking shape.

4. **Sharpen the peak**
Make small, circular movements focusing in this direction and narrowed area. Optimise the profile and maximise the peak velocity. Try some downward pressure in the transverse or Front to back plane. Confirm a full systolic width and a triangular shape with filling in of the profile.

Now fully define with very small movements to sharpen the peak.

The sides should emanate from the extremes of the full systolic width. They should be straight converging at a pointy peak.
Hold still, obtain a screen of profiles and freeze. Note the diastolic components of the Doppler profile. Once the profile is optimised, skin marking will make repeat imaging easier.
5. Confirm Aortic Valve Flow
Peak Velocity is greater than Ascending Aortic Flow.
Systolic Width is wider than that of the Ascending Aortic flow.

The time gap between end-systole and diastole should be shorter than that of the Ascending Aortic flow and diastole.

Confirm that the early and late sides of the systolic triangle emanate from the ‘Full width’ of systole.

Confirm the sides of the triangle are straight and continuous converging at a pointy peak.
Confirm a pointy peak.

6. Confirm FlowTracer

Incorrect HR or SV calculation will affect CO / CI calculations. Use TouchPoint if the FlowTracer tracking is inadequate.

Eliminate inadequate Doppler profiles.

Eliminate poor tracking by FlowTracer

Review contributors to the Stroke Volume calculation:

-Review peaks
-Review baseline

Start and end of systolic profile. Eliminate profiles where the diastolic flow has been traced. (If valve clicks are visible these will help to determine start and end of systole)

Review contributors to the HR calculation:

Confirm that the tracking has traced a complete beat, from start of systole to the start of the next systole. (If a profile is eliminated because the baseline was too broadly traced, then the previous heart rate may be overestimated)
Confirm with HR from monitor if available.

7. Add B/P
8. Save
9. Add Notes

ACQUISITION protocol

Ascending Aortic Flow
Aortic Valve Flow
Note: Scale has changed

The measure of life.
How can I be sure whether this is Ascending Aorta or Aortic Valve flow?

*Where are you angling?*

*Can the AV possibly be at this angle?*

*Are there signs of more signal evident in the Doppler profile?*
The appearance of soft shadows is a hint that there is more signal.

Are there shadows:
- Along the descending side of the triangle?
- Extending the base of the triangle?
- Above the peak of the triangle?

Does there appear to be a second small systolic profile before diastole?

Does there appear to be other signals ‘growing on’ or around the defined Ascending Aortic triangle?

*Look for a Time Gap before diastolic flow*

Is there a time gap between the end of systole and the start of diastolic flow?
How can I be sure whether this is Ascending Aorta or Aortic Valve flow?

If not, is the late systolic side of the triangle straight and continuous?

When the transducer is angled between ascending aortic and AV flow, the Doppler profile may have the appearance of a ‘ledge’, bump or ‘dicrotic notch’.

This is because it displays ‘full systolic’ width at the base but one or both sides of the triangle may not be straight and continuous.

If you look closely sometimes the outline of the Asc Ao flow is apparent.
AORTIC troubleshooting

Patient Habitus

The tall and thin patient’s heart is often more vertically oriented; with the apex angled to the midline. Angle the transducer more centrally.

The apex of a dilated heart or heart in a larger patient may be angled further toward the patient’s left.

Neonates

AV flow is better found from the Right Supraclavicular position and pointing up towards the sternum. The baby’s head needs to be tilted backward with support under the shoulders. It can also be found from the left subcostal position.

Ascending Aortic flow will be of a similar velocity to the Descending Aortic flow. Whereas the AV flow will be higher than both.

Confirm the obtained systolic flow by comparing with the Descending Aortic flow.

Position

In adults, if the aortic signal is difficult to acquire from the suprasternal position, changing position by tracking around the trachea and angling more toward the valve may help. The supraclavicular position could also be tried.

Technique

At a distance further from the source very small arcing or sweeping result in large movements of the ultrasound beam.
Imagine a wheel. Think of the distance between two spokes, first at the centre of the wheel and then at the rim.
Now imagine holding a transducer at the centre and sweeping from one spoke to the next. Notice that a small movement of the transducer face at the centre produces a greater distance travelled by the ultrasound beam at the rim.
AORTIC troubleshooting

Flow Profiles

**Ascending Aorta:**
A positive, narrow and unfilled triangular profile with low velocity is often first noted in the SSN approach. An unfilled profile indicates that most of the blood cells are travelling at a narrow range of velocities. This profile is characteristic of ascending aortic flow not aortic valve flow.

**Descending Aorta:**
A negative profile with no positive component indicates descending aortic flow. Aim the transducer towards the patient’s right hip and angle under the sternum to locate the aortic valve.

This can be used to confirm with suspicious forward flow. It will be a similar velocity to that of ascending aortic flow but much lower than the AV flow.

**Venous Flow:**
A continuous, low velocity and pulsatile profile with corresponding audio feedback, indicates venous flow. This venous flow can often be seen when using the supraclavicular approach.
PULMONARY technique

The aim is to optimise the Doppler profile and peak velocity by angling the transducer Doppler beam in line with the transvalvular blood flow.

The best window to obtain the pulmonary valve flow on an individual may vary from the 2nd left intercostal space to just below the ribs. We suggest a formalised process to quickly determine the best window by quickly eliminating a large part of the chest area.

Review of a chest x-ray or use of a stethoscope may also help.

P1 and P2

To determine the best interspace to use, hold the transducer with the face flat to the chest.

Glide along each left intercostal space, from the 2nd to the 5th, moving into the sternum and then laterally (keeping the transducer face flat to the chest).

Sample until the signal becomes loud and strong. Do not stop sampling here but continue to the next intercostal space to check if it is even better.

Return to the best window with the strongest signal and loudest sounds.
PULMONARY technique

P3 and P4
From the best intercostal space, angle the transducer at 9 (patient’s right side), 11 (patient’s right shoulder) and then 12 o’clock (head) and note at which angulation the systolic signal and peak velocity increases.

Perform these 3 angulations to rapidly determine which angle is best, will eliminate a large section of the chest in which to find the PV flow.

If 2 directions were equally good, then angle between them.

If one direction was best than optimise in that direction.

This should only take 15-30 secs to do.

P5
Make small lateral and circular movements with the aim to continue to maximise the peak velocity and optimise a sharp peak.

Use the transducer to pull back the skin in the opposite direction from the angle of the best profile and tilt up slightly.

To confirm the Doppler profile refer to ‘Optimal PV Doppler profile characteristics’.
The measure of life.

ACQUISITION sequence

**Pulmonary**

1. Place the transducer in the 2nd left intercostal space. Sample in each left intercostal space, aiming for the strongest and loudest signal indicating the best window. Often, the two diastolic components are initially detected.

2. Return to the intercostal space with the best window. With sagittal and transverse sweeps of the transducer interrogate the ‘face of the clock’ between 9 and 12. A fairly defined triangular systolic shape should be forming.

3. Make smaller movements angling the transducer up and down in line with this best direction in a pendulum like motion. The peak velocity is increasing and a triangular shape is becoming defined.

4. Maintain the best direction and angle and use small circular movements to improve the pulmonary Doppler profile.

5. Very subtle pulling of the skin away from the direction of flow while tilting up slightly to ‘sharpen the peak’ of the maximum velocity.

**PV Doppler Profile Characteristics**

- Triangular shape
- Triangle base - full systolic width
- Triangle sides - sides should emanate from the base and be straight and continuous, converging in a sharp, pointy peak

- Highest velocity
- Filling in of profile
- Clear start and cessation of flow
- Respiratory variation
- Diastolic components
- Change in audio feedback

More examples
**ACQUISITION protocol**

**Pulmonary**

Lay the patient flat or with the head of the bed slightly elevated. The muscles of the chest should be relaxed. The pulmonary valve is approximately positioned left of the sternum below the 2nd intercostal space.

Begin the examination at this position.

**Select transducer**

Apply gel to the flat face of the transducer.

All intercostal spaces along the left sternal edge should be sampled. The most common window will often be the 3rd or 4th intercostal space but occasionally it may be high at the 2nd intercostal space or as low as under the rib cage.

The window tends to be in a lower intercostal space on a longer torso. On a short torso the window tends to be in a higher intercostal space.

A quick process of elimination, to narrow down the possible area is recommended. The best window is the one that gives access to the strongest signal. The strongest signal may initially be from diastolic profiles. These will be dark and loud. The aim is to find the best interspace or window for acquiring the Doppler profile. Once the best window is found then transducer manipulation and targeting of the pulmonary valve flow can be optimised.
ACQUISITION protocol

1. Locate Best Window
Hold the transducer with the face flat to the chest.

Sample the 2\textsuperscript{nd} intercostal space sliding into the sternum and a little laterally keeping the transducer face flat to the chest.

Move to the 3\textsuperscript{rd} intercostal space, then 4\textsuperscript{th} etc and continue to sample until the signal becomes loud and strong. Do not stop sampling here, but continue to the next intercostal space to check if it is even better.

Return to the best window with the strongest signal and loudest sounds.

Confirm that you have the best window by sampling past the strongest signal i.e. the signal becomes less strong. This should only take 15-30 secs to do.
ACQUISITION protocol

2. Locate Best Direction
Slowly angle the transducer from flat, straight up toward 12 o’clock.
Beginning with a small angulation and then increasing it. A downward facing, triangular shape begins to form.

Sometimes, initially the diastolic flow may be more in line and display higher than the systolic flow. As the transducer lines up in the right direction with the PV flow, then the systolic flow will be higher.

Angle the transducer back to the flat position.
Now angle to 9 o’clock and note whether the signal increases or not.

Now angle the transducer back to the flat position again.

This time angle to 11 o’clock or the patient’s right shoulder and note whether the signal increases or not.

These 3 angulations will rapidly eliminate a large portion of the chest in which to find the direction of the PV flow.
If 2 directions were equally good, then angle between them.
If one direction was best than optimise in that direction.
This should only take 15-30 secs to do.

At this point a fairly defined triangular systolic shape should be forming.
ACQUISITION protocol

3. Maximise Systolic Width and Velocity
Angle the transducer up and down in line with this best direction, going beyond the highest velocity and back to it, using a pendulum like movement. Make small lateral and circular movements with the aim of optimising the profile and defining the peak.

If the Doppler profile is not well filled in or the systolic width appears short, then a change in angulation or position is needed. The valve clicks which are often seen on the Doppler pulmonary valve flow are excellent indicators of the full systolic width, highlighting the start and end of systole.

If one of the clicks is very strong and a lot of the filling is not seen, then again, some angulation or position change is needed.

4. Sharpen the Peak
If the peak is wispy or blunted, then continue angulation to optimise.
Use the transducer to pull the skin in the opposite direction from the angle of the best profile and tilt slightly up.

Occasionally, moving to the lower interspace and maintaining this last angle may also help. The profile should be of full systolic width with a defined, triangular shape with two straight sides meeting in a sharp peak.

The PV flow often has less edge definition which displays as a softer Doppler profile. Usually systolic valve clicks are seen with the PV flow.

5. Confirm FlowTracer
Eliminate inadequate Doppler profiles.
Eliminate poor tracking by FlowTracer
Use TouchPoint if the FlowTracer tracking is inadequate.

6. Add B/P
7. Save
8. Add Notes
PULMONARY troubleshooting

Patient Habitus

The tall and thin patient’s heart is more vertically oriented with the apex angled more to the midline. Reposition the transducer in a lower intercostal space and angle the transducer more steeply.

The apex of the heart in an obese patient may be angled further toward the left. A higher intercostal space may be a better position.

Neonates and small children

The transducer face is usually left fairly flat to the chest with little if any angulation.

Lungs and air

Ultrasound waves do not travel through air.

Depending on the position of the transducer, inspiration may intermittently reduce access to the pulmonary Doppler profile due to the air filled lung expanding into the path of the ultrasound beam.

During inspiration hold your position and attempt to optimise the profile during expiration only.

Gaining access to the pulmonary artery, therefore, may be difficult in the patient with severe cardiopulmonary disease or immediately post open heart surgery.

A lower intercostal space may be required, or repositioning the patient to shift air in the chest may also help.

Consider turning the patient to their left side with the body at a 15-30 degree left lateral tilt. If possible position the left arm toward the patient’s head; this will separate the ribs a little.
PULMONARY troubleshooting

Other Doppler Profiles

Tricuspid regurgitation is a systolic flow that is sometimes seen from the LSE position when acquiring the PV flow.

The TR Doppler profile is wider at the base and slightly rounded in its shape.

The flow begins immediately the TV closes, at end diastole and finishes when the TV opens, at the start of diastole.

The TR Vpk is usually >2m/s.

PROFILE optimisation CONFIRMATION

The TR Doppler flow may be picked up when aiming the transducer too far to the patient’s right side.

For PV flow aim the transducer more anteriorly or to the patient’s midline.

Tricuspid Regurgitation has a wider systolic width than the PV flow.
Potential sources of error

Potential errors of accuracy can occur from:
- Assumptions of normal anatomy
- Method of data collection
- Calculations used

Cardiac Output = Stroke Volume × HR

Stroke Volume = Flow Area × vti

Algorithm (OTD flow area) vti (stroke distance, flow length)

Potential errors can occur in the measurement of:
- HR - TouchPoint or FlowTracer
- vti - TouchPoint or FlowTracer

Potential errors can occur in the assumption of:
- OTD area - Height or weight based algorithm
  - Lack of presence of stenosis or narrowing of the targeted valve or anatomy
  - Lack of presence of significant regurgitation of the targeted valve

Potential errors can occur in the acquisition of:
- Incorrect or inadequate Doppler flow profiles
- Incorrect input of patient data

Questions to ask:

Have I acquired a correct and adequate Doppler profile?

Has FlowTracer correctly traced the profile?

Is the Heart rate correctly determined?
**VALVULAR** pathology

In the case of suspected or known ventriculo-arterial valvular pathology such as aortic stenosis, the calculation of CO will be overestimated. It will however, display the true Doppler flow profile and velocity, provided the peak velocity is acquired. In this case the peak velocity (Vpk) will be significantly higher than normal.

The two main causes of higher than normal velocities are obstruction to flow and increased volume.

**Obstruction To Flow**

- Subvalvular obstruction
- Valvular stenosis
- Supravalvular obstruction

Why is the velocity increased?

CW Doppler will reflect all velocities in the path of the beam. The narrowest area produces the highest velocities (Refer to Doppler Basics). In the case of a valvular stenosis the highest velocities will be from the stenotic valve. The stenotic diameter will be of a smaller value than the algorithm estimated OTD, resulting in an overestimation of the CO.

---

Once again imagine running water coming out of the hose or tap. This time cover most of the opening with your finger. The orifice is very small and the water now runs at a very fast rate and is spitting in all directions out of the hose. The sound has changed also. This increase in velocity happens because the same amount of water is flowing from a wide area to a much smaller area. This result is the same as flow passing through a stenotic valve. The same volume of blood must pass the obstruction in the same amount of time, to maintain stroke volume.
Grading Of Stenosis

The peak velocity helps to grade the severity of stenosis. Stenosis is graded from mild to severe depending on the obstruction to flow.

Valvular Aortic Stenosis - Peak Velocity (Vpk)
- Mild: < 3.5 m/sec
- Moderate: 3.5 - 4.0 m/sec
- Severe: > 4.0 m/sec

Valvular Pulmonic Stenosis - Peak Velocity (Vpk)
- Mild: < 3.5 m/sec
- Moderate: 3.5 - 4.3 m/sec
- Severe: > 4.3 m/sec

Monitoring Cardiac Output In The Presence Of Stenosis

Solution A:
Obtain the stenotic valve area that has been determined on echocardiography and from this, derive the diameter of the valve. Use this value to override the algorithm’s estimated OTD.

Example: Valve Diameter = approx. 1.13 \( \sqrt{x} \) where \( x \) is the stenotic valve area
Note: Acquiring the peak velocity from a smaller, tighter area requires a more precise angle than acquiring from a larger orifice. Therefore to use this method the true aortic stenotic profile must be acquired.

Solution B:
Compare left heart to right heart.
(L) CO = (R) CO
(L) vti x CSA x HR = (R) vti x CSA x HR
(L) CSA = (R) vti x CSA / (L) vti where CSA is the Cross Sectional Area of the valve.
(L) CSA = (R) SV / (L) vti

Solution C:
Use the alternative ventriculo-arterial valve position.
Example: If there is aortic stenosis (not severe), aim to acquire the pulmonary CO.

Prosthetic Valves:
Use solution (C).
The diameter of the prosthetic valve may not reflect the true diameter of the LVOT.
It is possible to use the measured SV or CI as a baseline for monitoring change. In this case the volumes measured may not be true.
**VALVULAR pathology**

**Increased Volume**

Aortic Regurgitation
Pulmonary Regurgitation

Why is the velocity increased?
In valvular regurgitation more blood is initially pumped through the valve than the volume which continues along the vessels, due to some flowing back into the ventricle. This increased volume also needs to travel faster through the same diameter than a normal stroke volume would. (Refer Doppler Basics)

---

Increased forward volume (120ml).
Regurgitant volume (50ml).

---

As the Doppler profile records the velocities during systole, it will record true increased velocity due to increased volume. This vti multiplied by the estimated normal valve area will overestimate the net stroke volume and net cardiac output.

Significant regurgitation will cause a marked increase in velocity.

**Monitoring Cardiac Output In The Presence Of Regurgitation**

Use the alternative ventriculo-arterial valve position.
Example: If there is significant aortic regurgitation, monitor the pulmonary CO.

The net volume is equal to the stroke volume of the other valves.
HAEMODYNAMICS

Circulation

The purpose of circulation is to provide oxygen (O₂) and nutrients to the cells. The heart is a pump, providing the push to supply available volume to the body.

The goal of perfusion is to supply adequate oxygen delivery to balance oxygen consumption.

Cardiac output is one of the most important haemodynamic measures as it regulates oxygen delivery:

\[
\text{Oxygen Delivery (DO₂)} = \text{Cardiac Output} \times \text{Oxygen Content (Hb X SaO₂)}
\]

Determinants Of Stroke Volume

**Preload**
Preload is the amount of blood in the left ventricle at the end of diastole, prior to contraction. It is related to the myocardial fibre stretch at the end of diastole (left ventricular end-diastolic volume or pressure).

**Contractility**
Contractility is the force and velocity of myocardial muscle fibre shortening independent of preload or afterload.

**Afterload**
Afterload is the pressure against which the ventricles must work and must overcome to force blood into the arteries. Afterload is related to vascular tone or resistance.

The tissues do not receive enough oxygen, (tissue hypoxia) because of a decreased supply or an increased demand. To prevent tissue hypoxia, preload and perfusion pressure need to be maintained as well as delivery of O₂ matched with O₂ consumption which involves maintaining cardiac output.

\[
\text{Cardiac Output} = \text{Stroke Volume} \times \text{Heart Rate}
\]

The measure of life.
HAEMODYNAMICS

Frank-Starling Curve

Frank and Starling described the relationship of myocyte stretch and contractility in the isolated heart. They observed that the more the myocyte was stretched, the greater the responding myocyte contraction, up to an optimal point. This is of clinical importance because it suggests that SV is dependent on preload or filling; the greater the filling, the greater the contraction and the greater the SV.

Conversely, beyond a certain point, increasing the stretch results in decreased contractility, suggesting each myocyte has an optimal stretch. This is demonstrated in a simple graph of left ventricular end-diastolic volume (LVEDV), analogous to preload and SV, known as the Frank-Starling curve.

With increased contractility for a given preload a higher stroke volume (SV) or cardiac output (CO) will be achieved.

With a low contractility at the same preload, a lower SV and CO will result.

Frank-Starling curve relates preload to stroke volume.

A) Increased contractility, higher SV
B) Decreased contractility, lower SV
HAEMODYNAMICS

Volume vs Pressure

The current clinical practice is to increase pressures such as central venous pressure (CVP) a surrogate for cardiac output (CO) arterial pressure to clinical targets. Optimising to a target pressure may not adequately optimise the volume status of every patient; one may be underfilled while another overfilled.

Restoring blood pressure does not guarantee an adequate volume status either due to the contribution of systemic vascular resistance (SVR).

Compliance

Restoring volume to a target value also does not ensure an adequate volume has been achieved, as each individual has a different capacity. There is no uniform response to adding fluid due to compliance of the heart muscle.

In myocardium which is compliant, additional volume will stretch the myocytes and greater SV will be achieved. In a less compliant myocardium, a heart muscle that has had its myocytes over stretched (hypertrophied as in CHF), does not have the same recoil with the additional volume. Therefore SV may not increase or be able to be maintained.

Think about a basketball and a balloon. When they are the same size and shape they will respond differently to volume and pressure. Due to its highly compliant nature, more air (volume) is needed by the balloon to reach the same pressure as the basketball. Adding extra volume will increase the pressure more rapidly in the basketball than in the balloon. When they contain the same volume, there is a higher pressure in the basketball.
**HAEMODYNAMICS**

**Fluid Challenge and Monitor the Effect**

Each individual has a characteristic Frank-Starling curve reflective of their underlying myocardial morphology and function. For this reason, individual optimisation may not be achieved using standard targets, but rather it would be better to make serial observations of each patient’s cardiac output to determine preload, afterload and contractility.

Optimising to an individual patient’s Frank-Starling curve is a better goal than optimising to a target number. Observing serial cardiac output measurements allows observation of changes in SV related to increases or decreases in fluid volume.

Dehydration or hypovolaemia from blood loss results in an underfilled heart, and understretched myocytes. Inotropes cannot increase contractility beyond a certain point in hypovolaemia; therefore the addition of fluid is a basic and fundamental treatment, increasing the myocyte stretch and SV.

![Graph showing serially monitored fluid challenge](image)

Serially monitored fluid challenge.
The addition of volume increases the individuals SV up to an optimal point thus replicating the Frank-Starling mechanism. Monitoring the fluid challenge confirms optimal stroke volume. Adding more fluid would not benefit the patient.
HAEMODYNAMICS

If changes in SV are not monitored, overfilling of the heart, heart failure and subsequent pulmonary oedema are possible. This overfilling is the point on the Frank-Starling curve beyond the apex. Additional fluid at this time does not result in an increased SV; it may in fact produce no change or decrease SV. Using diuretics to reduce the blood volume in fluid overload will return the SV to the apex of the curve, i.e. the point of optimal SV.

Most importantly, haemodynamic optimisation, whether associated with increasing or decreasing the blood volume, is associated with improved outcomes in a number of clinical conditions.⁵

Optimisation of preload to stroke volume.
Frank-Starling curve demonstrating point of SV optimisation and hypovolaemia and hypervolaemia.

REFERENCES


DISCLAIMER: The following information is drawn from unpublished studies by Smith, B. et al and has not been produced or authenticated by Uscom. The information is for general guidance only and must not be used for specific therapy management. Values stated have used aortic valve Doppler flow taken from the suprasternal notch position unless otherwise indicated.

Uscom recommends that the normal values and ranges for any particular demographic group should be established locally.

<table>
<thead>
<tr>
<th>Age</th>
<th>0 - 2</th>
<th>2 - 5</th>
<th>5 - 10</th>
<th>10 - 16</th>
<th>16 - 60</th>
<th>&gt;60</th>
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<tbody>
<tr>
<td>Vpk (m/s) AV</td>
<td>1.1-1.6</td>
<td>1.1-1.6</td>
<td>1.1-1.6</td>
<td>1.1-1.6</td>
<td>1.1-1.4</td>
<td>0.9-1.3</td>
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<tr>
<td>Vpk (m/s) PV</td>
<td>0.8-1.4</td>
<td>0.8-1.4</td>
<td>0.8-1.4</td>
<td>0.8-1.4</td>
<td>0.8-1.2</td>
<td>0.7-1.1</td>
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<td>MD (m/min) AV</td>
<td>18-30</td>
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<td>18-28</td>
<td>16-26</td>
<td>15-25</td>
<td>14-22</td>
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<tr>
<td>MD (m/min) PV</td>
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<td>14-22</td>
<td>12-20</td>
<td>11-22</td>
<td>10-20</td>
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<td>HR (bpm)</td>
<td>110-140</td>
<td>85-115</td>
<td>75-105</td>
<td>65-100</td>
<td>55-85</td>
<td>60-90</td>
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<tr>
<td>ET%</td>
<td>40-60</td>
<td>40-60</td>
<td>35-50</td>
<td>35-45</td>
<td>30-45</td>
<td>35-50</td>
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<tr>
<td>FTc (ms) AV</td>
<td>300-375</td>
<td>300-375</td>
<td>325-400</td>
<td>350-400</td>
<td>400-450</td>
<td>425-475</td>
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<tr>
<td>FTc (ms) PV</td>
<td>325-400</td>
<td>325-400</td>
<td>350-425</td>
<td>350-425</td>
<td>400-475</td>
<td>450-525</td>
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<td>SV (ml/kg)</td>
<td>1.5-2.25</td>
<td>1.5-2.4</td>
<td>1.25-2.2</td>
<td>1.25-1.75</td>
<td>1.1-1.75</td>
<td>0.9-1.4</td>
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<tr>
<td>SVI (ml/m²)</td>
<td>36-45</td>
<td>40-55</td>
<td>40-60</td>
<td>40-60</td>
<td>35-65</td>
<td>30-55</td>
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<tr>
<td>CO (l/min)</td>
<td>2.5-3.5</td>
<td>2.5-4</td>
<td>2.8-5</td>
<td>3.5-7.5</td>
<td>3.5-8</td>
<td>2.5-6</td>
</tr>
<tr>
<td>CI (l/min/m²)</td>
<td>4.2-5.5</td>
<td>3.5-5</td>
<td>3.5-5</td>
<td>3.2-4.8</td>
<td>2.8-4.2</td>
<td>2.4-3.6</td>
</tr>
<tr>
<td>SVR (d.s.cm⁻⁵)</td>
<td>1500-2000</td>
<td>1500-2000</td>
<td>1200-1800</td>
<td>900-1500</td>
<td>800-1600</td>
<td>1000-1800</td>
</tr>
<tr>
<td>SVRI (d.s.cm⁻⁵m²)</td>
<td>800-1200</td>
<td>1000-1600</td>
<td>1000-2000</td>
<td>1100-2300</td>
<td>1800-3200</td>
<td>2000-3400</td>
</tr>
<tr>
<td>DO₂ (ml/min)</td>
<td>500-700</td>
<td>500-800</td>
<td>560-1000</td>
<td>700-1500</td>
<td>700-1600</td>
<td>500-1200</td>
</tr>
<tr>
<td>DO₂I (ml/min/m²)</td>
<td>800-1100</td>
<td>700-1000</td>
<td>700-1000</td>
<td>650-950</td>
<td>550-850</td>
<td>480-720</td>
</tr>
</tbody>
</table>

¹ Extrapolated from other data. Neonate data limited.

² CI, DO₂ and DO₂I should be corrected for Hb level and SpO₂. Corrected CI = CI(measured) x 15,000 / Hb(g/L) x SpO₂. Corrected DO₂ = DO₂(measured) x 15,000 / Hb(g/L) x SpO₂. Corrected DO₂I = DO₂I(measured) x 15,000 / Hb(g/L) x SpO₂.
## HAEMODYNAMIC measurements

### Measurements

<table>
<thead>
<tr>
<th>Metric</th>
<th>Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CO - Cardiac Output</strong></td>
<td>( CO = SV \times HR )</td>
</tr>
<tr>
<td><strong>CI - Cardiac Index</strong></td>
<td>( CI = \frac{CO}{BSA} )</td>
</tr>
<tr>
<td><strong>SV - Stroke Volume</strong></td>
<td>( SV = vti \times CSA )</td>
</tr>
<tr>
<td><strong>SVI - Stroke Volume Index</strong></td>
<td>( SVI = \frac{SV}{BSA} )</td>
</tr>
<tr>
<td><strong>SVV - Stroke Volume Variation</strong></td>
<td>( SVV = \frac{(SV_{\text{max}} - SV_{\text{min}})}{(SV_{\text{max}} + SV_{\text{min}}) \times 100} )</td>
</tr>
<tr>
<td><strong>SVR - Systemic Vascular Resistance</strong></td>
<td>( SVR = 80 \times \frac{\text{MAP} - \text{CVP}}{CO} )</td>
</tr>
<tr>
<td><strong>SVRI - Systemic Vascular Resistance Index</strong></td>
<td>( SVRI = SVR \times BSA )</td>
</tr>
<tr>
<td><strong>DO2 - Oxygen Delivery</strong></td>
<td>( DO_2 = 1.34 \times Hb \times \frac{SpO_2}{100} \times CO )</td>
</tr>
<tr>
<td><strong>FT - Flow Time</strong></td>
<td>( FT = t_{1/2} )</td>
</tr>
<tr>
<td><strong>FTc - Flow Time Corrected</strong></td>
<td>( FTc = \frac{FT}{\sqrt{t_{1/2}}} )</td>
</tr>
<tr>
<td><strong>ET% - Ejection Time Percentage</strong></td>
<td>( ET% = \frac{ET}{CycleDuration} \times 100 )</td>
</tr>
<tr>
<td><strong>vti - Velocity Time Integral</strong></td>
<td>( vti = \int_{0}^{T} v(t) , dt )</td>
</tr>
<tr>
<td><strong>MD - Minute Distance</strong></td>
<td>( MD = vti \times HR )</td>
</tr>
<tr>
<td><strong>MAP - Mean Arterial Pressure</strong></td>
<td>( MAP \approx BP_{sys} + \frac{(BP_{dia} - BP_{sys})}{3} )</td>
</tr>
</tbody>
</table>
## Definitions

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>$DO_2$</td>
<td>Oxygen Delivery</td>
</tr>
<tr>
<td>$CO$</td>
<td>The volume of blood pumped by the heart in one minute</td>
</tr>
<tr>
<td>$CI$</td>
<td>Cardiac Output indexed with BSA</td>
</tr>
<tr>
<td>$SV$</td>
<td>The volume of blood pumped by the heart in one beat</td>
</tr>
<tr>
<td>$SVI$</td>
<td>Stroke Volume indexed with BSA</td>
</tr>
<tr>
<td>$SVV$</td>
<td>The variation of Stroke Volume over a group of a number of beats. (Calculated when in Group selection. Useful in ventilated patients)</td>
</tr>
<tr>
<td>$SVR$</td>
<td>Pressure against which the heart pumps</td>
</tr>
<tr>
<td>$SVRI$</td>
<td>Systemic Vascular Resistance indexed with BSA</td>
</tr>
<tr>
<td>$V_{pk}$</td>
<td>The highest velocity of blood flow through the valve</td>
</tr>
<tr>
<td>$HR$</td>
<td>Beats per minute. (Measured from systolic onset to systolic onset)</td>
</tr>
<tr>
<td>$FT$</td>
<td>The systolic flow time. (Systolic ejection)</td>
</tr>
<tr>
<td>$FT_c$</td>
<td>The systolic flow time corrected for heart rate</td>
</tr>
<tr>
<td>$ET%$</td>
<td>Percent of cycle duration occupied by systolic ejection</td>
</tr>
<tr>
<td>$v_{ti}$</td>
<td>The integral of the flow profile. (The distance the blood travels in one beat)</td>
</tr>
<tr>
<td>$MD$</td>
<td>The distance the blood travels in one minute, independent of valve area</td>
</tr>
<tr>
<td>$BSA$</td>
<td>Body surface area. (Used for indexing calculations)</td>
</tr>
<tr>
<td>$SpO_2$</td>
<td>Peripheral oxygen saturation</td>
</tr>
<tr>
<td>$P_{mn}$</td>
<td>Mean pressure gradient across the valve</td>
</tr>
</tbody>
</table>
Abbreviations

AV  Aortic valve
BPdia  Diastolic blood pressure
BPsys  Systolic blood pressure
BSA  Body surface area
CI  Cardiac index
CO  Cardiac output
CPO  Cardiac power
CVP  Central venous pressure
CW  Continuous wave Doppler
DO₂  Oxygen delivery
DO₂I  Oxygen delivery index
ET%  Ejection time percent
FT  Flow time
FTc  Flow time corrected
Hb  Haemoglobin
HR  Heart rate
LV  Left ventricle
LVEDP  Left ventricular end diastolic pressure
MAP  Mean arterial pressure
MD  Minute distance
MV  Mitral valve
OT  Outflow tract
OTD  Outflow tract diameter
PA  Pulmonary artery
Pmn  Mean pressure valve gradient.
PCWP  Pulmonary capillary wedge pressure
PV  Pulmonary valve
RV  Right ventricle
SpO₂  Peripheral oxygen saturation
SSN  Suprasternal notch
SV  Stroke volume
SVI  Stroke volume index
SVR  Systemic vascular resistance
SVRI  Systemic vascular resistance index
SVS  Stroke volume saturation
SVV  Stroke volume variation
SW  Stroke Work
TV  Tricuspid valve
Vpk  Velocity peak
vti  Velocity time integral

The measure of life.
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