OXFORD

Revalidation FOR ANAESTHETISTS
RCA Revalidation matrix
Matrix reference
1A03, 2A04, 3G00

BJA Education, 16 (2): 46–52 (2016)

doi: 10.1093/bjaceaccp/mkv015
Advance Access Publication Date: 10 June 2015

Quantitative Doppler echocardiography

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Key points

- Doppler and 2D imaging provide objective haemodynamic data to quantify ventricular function and grade valve lesion severities.
- Parallel alignment of Doppler ultrasound beam to the direction of blood flow is of paramount importance to avoid errors.
- Velocity–time integration of Doppler traces through valves, using area under the curve analysis, provides the basis for the majority of calculations.
- Pulse-wave Doppler measures slower velocities at a specified depth, whereas continuous-wave Doppler measures any velocity shift but without knowing where on the scan line they come from.
- The simplified Bernoulli equation can convert Doppler velocity to pressure, using $P=4V^2$.

Echocardiography provides information regarding volumes [e.g. stroke volume (SV), ejection fraction], flows [e.g. cardiac output (CO), shunt fractions], and pressures (e.g. gradients across valves). As the only directly measurable variables are length (using 2D imaging) and velocity (using spectral Doppler), quantitative haemodynamic data is acquired using extrapolation of these two modalities. Area can also be measured directly using planimetry in most systems. This review demonstrates how the physical principles behind Doppler echocardiography can be used to calculate volumes, flows and pressures. Systolic and diastolic ventricular function, filling status, and valve pathology can all be quantified, providing depth and accuracy to decision-making processes.

Principles of Doppler

$$f=\frac{2v f_0 \cos\theta}{c}$$

where f is Doppler shift frequency, v velocity of red cell target, f_0 frequency of transmitted ultrasound beam, θ angle between the ultrasound beam and vector of the red blood cell flow and c is velocity of ultrasound in blood.

The Doppler effect is the change in frequency of a reflected sound wave for an observer moving relative to its source. In blood flow, the frequency shifts relate to red blood cell velocities. If the source moves towards the observer, the observed frequency increases (and thus, wavelength decreases). If the source is moving away from the observer, the opposite occurs. The observer and source must be parallel to each other. As the angle between them becomes more perpendicular, the Doppler shift falls, until at 90° there is no shift. An angle >20° produces unacceptable error in Doppler velocity measurements. In general, all measurements should be done by using data from a minimum of three cycles (more with arrhythmias) and from more than one view. In 2D imaging, the ultrasound beam should be perpendicular and passing through the centre of the structure of interest to reduce measurement errors. The measurements are taken from inner-to-inner edge.[1]

Continuous-wave Doppler

Two separate crystals are used, one to transmit and one to receive ultrasound signals. Every single velocity along the line of interrogation is recorded, thus the trace appears filled in (see echo inset in Fig. 2). Continuous-wave (CW) Doppler can measure very high velocities, but it is unable to pinpoint where on the scan line they come from. For measurement, the outer edge of the velocity envelope is used.

Pulse-wave Doppler

A single crystal is used, both to transmit and receive ultrasound signals. By knowing the velocity of ultrasound in body tissue (~1540 m s^{-1}), the crystal is able to wait a defined period of time for the reflected signal to return, and thus interrogate a specified area. The trace appears hollow and for measurement the

outer edge of the brightest portion of the velocity tracing is used. A drawback to pulse-wave (PW) Doppler is that the further away the sample volume, the longer the round trip the signal has to take resulting in a lower pulse repetition frequency. If the blood flow being measured is fast, then the blood cells will have moved a long way between pulses and the direction of flow will not be able to be determined. This phenomenon is called *aliasing*. A visual example of aliasing is vehicle wheels appearing to rotate the opposite way when viewed on television as a result of the frame rate at which they are filmed. The maximum Doppler frequency that can be measured unambiguously is half the pulse repetition frequency (as sound waves have to be measured at least twice per wavelength to measure wavelength accurately). This maximum velocity is known as the *Nyquist limit*, above which aliasing occurs. In colour flow Doppler (a form of pulse-wave Doppler) this means the colour may change from red to blue (or vice versa), despite the blood flow continuing in the same direction, giving the false impression of turbulence. With velocity tracings, any velocities above the Nyquist limit will be displayed on the opposite side of the baseline. Aliasing can be reduced in a number of ways including shifting the baseline of the velocity tracing, decreasing the frequency, increasing the angle of incidence and by sampling at two points. For colour Doppler it can be minimized by adjusting the colour Doppler velocity scale and decreasing the width and depth of the sample volume.

Colour Doppler

A form of PW Doppler whereby single crystals both emit and receive signals but instead of focusing on a single point, multiple sample volumes are evaluated along each individual sampling line. The velocities detected are colour coded such that blue signifies velocities away from the transducer and red towards it. (Blue Away, Red Towards—'BART'.)

Tissue Doppler

Myocardial wall motion velocities can be interrogated as well as the blood velocities listed above, where the cursor is aligned over the ventricular wall rather than within a cavity. This provides additional information on the extent and timing of diastolic wall motion.

Calculations rely on several vital factors and also make several assumptions.[2]

Vital factors:

1. The Doppler beam must be parallel or within 15–20° of the direction of blood flow. An angle >20° produces an unacceptable error in Doppler velocity shift. At 20° the error is 6%, at 60° it is 50%.
2. Areas must be measured accurately. Errors in measuring cylinder diameters will be exponentially compounded in calculations. This is particularly relevant with calculating ejection fraction and SV.
3. No two heart beats are identical; therefore, several separate measurements should be averaged. This is time-consuming.

Assumptions:

1. *Blood flow is laminar with a flat velocity profile*. This may be true through the left ventricular outflow tract (LVOT) but cannot be said for a diseased valve or in a great vessel, where flow is more parabolic and turbulent in shape.
2. *For each equation, variables are measured simultaneously*. This is rarely possible as different modalities are used for areas and velocities.
3. *Cross-sectional areas (CSA) are circular* (e.g. oesophageal Doppler calculations). This is rarely, if ever, completely accurate.
4. *Area sizes are fixed*. Changes in CSA will occur during any flow period.

These are only 'ideals' but not necessarily achievable or practical in many clinical situations.

Pressure gradients

Pressure gradients quantify severity of stenotic lesions and can estimate unknown pressures from known pressures. Pressure and flow are integrally related but neither can be measured directly with echocardiography. Pressure (*P*) can be estimated from velocity (*V*) using the simplified Bernoulli equation: $P=4V^2$.

Total energy in a closed system is a constant (Newton's law of conservation of energy). When blood flows through a stenotic valve, kinetic energy increases and potential energy decreases proportionately to maintain constant total energy. Velocity increases through an orifice and pressure decreases. This explains why post-stenotic dilatation of the aorta is seen in severe aortic stenosis. The modified Bernoulli equation states:

$$\Delta P = 4(V_2{}^2 - V_1{}^2)$$

where V_1 is velocity pre-orifice and V_2 is velocity post-orifice. $V_1{}^2$ is significantly less than $V_2{}^2$ in most physiological conditions and can be ignored, thus: $\Delta P=4V^2$. In aortic stenosis, peak pressure gradient is 4×(peak velocity)^2 through the valve. If V_1 (LVOT) velocity is abnormally high, such as in obstructive cardiomyopathy (>1 m s^{-1}), the full equation should be used. If V_1 is <1 m s^{-1} (most common in clinical practice), then the simplified version is used.

Stenotic valve gradients

Pressure gradients across stenotic valves correlate with invasive, cardiac catheterisation data, particularly with the aortic valve (AV). Cardiac catheterisation measures the arithmetic difference between peak LV pressure and peak aortic pressure, which is non-simultaneous. Doppler measures velocities simultaneously and thus the pressure gradient may be slightly greater. True gradients are generally overestimated and this is exaggerated (pressure recovery)[3] if there is a small aortic root, prosthetic valves or aortic coarctation, where the pressure can increase post narrowing. The pressure recovery phenomenon can be responsible for gradients that are substantially higher, and valve areas that are lower, than those measured invasively. In practice, kinetic energy gained proximal to the stenotic orifice is converted to thermal energy or recovered as pressure energy (known as pressure recovery) distal to stenosis. Doppler underestimates the pressure gradient rather than overestimating it because of sampling errors from poor beam alignment.

Right Ventricular systolic pressure

Right Ventricular systolic pressure (RVSP) is commonly estimated using peak tricuspid regurgitation (TR) velocity (in m s^{-1}) and central venous pressure (CVP). RVSP correlates with pulmonary artery systolic pressure in the absence of RV outflow obstruction or pulmonary stenosis.

$$\mathrm{RVSP} = 4 \times \mathrm{TR}_{(\text{jet velocity})}{}^2 + \mathrm{CVP}$$

By using the same principle, an instantaneous pressure gradient across a ventricular septal defect (VSD_{PG}) can be measured and

RV systolic pressure can be calculated from the known LV systolic pressure.[4]

$$RVSP \approx LVSP - VSD_{PG}$$

Pulmonary artery (PA) pressures

In diastole, right atrial and ventricular pressures are equal if the tricuspid valve is normal. If pulmonary regurgitation (PR) is present, Continuous-wave Doppler can be applied to the jet to estimate PA diastolic pressure.

$$PA_{\text{end-diastolic pressure}} = 4 \times PR_{(\text{jet velocity})}{}^2 + CVP$$

Similarly, PA systolic pressure is estimated by using the velocity jet of a patent ductus arteriosus if present.

Left heart pressures

Left atrial pressure (LAP) and left ventricular end-diastolic pressure (LVEDP) can be estimated from mitral regurgitant (MR) and aortic regurgitant (AR) jets (in m s^{-1}), respectively:

$$LAP = LVSP - 4 \times MR_{(\text{jet velocity})}{}^2$$

$$LVEDP = \text{aortic diastolic pressure} - 4 \times AR_{(\text{end-diastolic jet velocity})}{}^2$$

Left heart pressures are included here for completeness but are more theoretical and not used in clinical practice.

Left ventricular diastolic assessment

Symptomatic heart failure with preserved ejection fraction suggests diastolic dysfunction, which is most readily identified using mitral valve inflow Doppler interrogation. PW Doppler at the mitral valve annulus produces two periods of forward blood flow from LA to LV during diastole, the first (*E* wave) corresponding to passive early filling of the ventricle and the second (*A* wave) corresponding to late diastolic atrial contraction. The ratio between the two changes as diastolic dysfunction worsens. In addition, the *E* wave velocity can be coupled with the mitral annulus velocities (measured with tissue Doppler, *E′*) to create an *E*/*E′* ratio, which is one of the more reliable ways of differentiating the aetiology of diastolic dysfunction. Thus, $E/E' < 8$ indicates normal filling pressures, whilst $E/E' > 12$ indicates elevated filling pressures. Diastolic function and dysfunction are covered in detail in a previous *CEACCP* article.[5]

Stroke volume and cardiac output

During one cardiac cycle the entire CO and SV pass through the LVOT and AV, making them ideal areas to interrogate, although other locations within the heart and great vessels can be used. Oesophageal Doppler uses the same principles for calculating CO from the descending aorta.

SV and CO can be derived as follows, using the AV as an example:

$$SV = \text{area} \times \text{length} = CSA_{AV} \times SD$$

where CSA_{AV} is cross-sectional area of AV and SD is stroke distance.

CSA_{AV} is easily measured using 2D imaging. A short-axis view of the AV is achieved, then the area calculated by tracing around the opened leaflets during mid-systole. The machine calculates the area bound by the line traced. This is known as planimetry. Descending aorta and the LVOT are assumed to be cylindrical and the area is calculated using πr^2, where r is the radius of the cylinder. Oesophageal Doppler acquires this area using nomograms from patient height and weight data correlated to aortic size from historical CT scan slices from multiple patients. Of importance, error in basic measurement can be exponentially compounded in further analysis. πr^2 is often seen written as $0.785 \times d^2$, as diameter (d) is usually measured, rather than radius.

$$\pi r^2 = \pi \times \left(\frac{d}{2}\right)^2 = 3.14 \times \frac{d^2}{2^2}.$$

In mid-systole, the normal open AV resembles an equilateral triangle. Thus, CSA can also be calculated using the formula:

$$CSA_{AV} = 0.433 \times S^2$$

where S is the length of one side (leaflet base).

Stroke distance cannot be measured directly. It is a theoretical concept equivalent to the distance that blood travels with each heartbeat. Doppler measures velocity (distance/time). The area delineated by a velocity/time curve gives the distance travelled for a given time. So, tracing the area under the curve of the Doppler flow velocity profile [known as the velocity–time integral (VTI)] yields SD. Thus

$$SV = CSA_{AV} \times VTI_{AV}$$

This integration of velocity and time by calculating areas under curves is a fundamental way in which 2D imaging and Doppler can achieve volume and CO measurements.

In practice, SV calculation is more commonly done using LVOT interrogation rather than AV, which may be diseased. It is paramount to place the PW Doppler sample volume at the same point as where the diameter of the LVOT is measured to ensure accuracy.

Whilst CO can be calculated from the SV above, Doppler can also be used to measure it directly, as follows.

$$\begin{aligned} \text{Flow (Q)} &= \text{volume/time} \\ &= (\text{area} \times \text{length})/\text{time} \\ &= \text{area} \times (\text{length/time}) \\ &= CSA_{AV} \times \text{velocity}_{AV} \end{aligned}$$

When CW Doppler is aligned through the AV, the predominant velocities reflect aortic ejection. By measuring the maximum velocity (V_{max}) during mid-systole, the equation for CO becomes as follows:

$$CO = CSA_{AV} \times V_{max_{AV}}$$

Calculation of pulmonary-to-systemic flow ratio (Q_P/Q_S)

It is possible to quantify the magnitude of shunt that exists from abnormal cardiac foramina such as atrial septal defect or ventricular septal defect or from a patent ductus arteriosus.

Q_P/Q_S normally equals 1 as the CO from left and right ventricles is equal. In the presence of a pathological shunt, Q_P increases. The ratio is important as $Q_P/Q_S>1.5$ may indicate the need for clinical intervention.

Q_S is calculated from the CO formula above, from measurements either at the LVOT or AV. Similarly, Q_P can be calculated using CO calculations from the RVOT or main pulmonary artery. It is important to make several measurements as errors in calculating both outputs may make the resultant ratio even more inaccurate (e.g. overestimating one whilst underestimating the other).

The continuity equation

The continuity equation expands upon the concepts of SV and CO calculation above and has many applications. It is the most widely used method of calculating AV area in aortic stenosis, where calcium deposition makes planimetry inaccurate. It is simply conservation of mass, in that flow or volume through the LVOT in systole must equal that through the AV in the same cardiac cycle.

Thus, using flow

$$CSA_{LVOT} \times V_{max-LVOT} = CSA_{AV} \times V_{max-AV}$$

CSA_{LVOT} can be measured directly with 2D echo. The diameter is measured in cm, then πr^2 applied, assuming it to be cylindrical. $V_{max\text{-}LVOT}$ is measured using PW Doppler aligned with aortic outflow and the cursor in the LVOT. $V_{max\text{-}AV}$ must be measured with CW Doppler as the velocity exceeds the Nyquist limit. AV area can then be calculated (Fig. 1).

Alternatively, using SV:

$$CSA_{LVOT} \times VTI_{LVOT} = CSA_{AV} \times VTI_{AV}$$

where VTI_{LVOT} is the area under the curve using PW Doppler at the LVOT. VTI_{AV} is the same, using CW Doppler aligned with aortic outflow.

It may be possible to use CW Doppler alone to calculate AV area. In correct alignment with aortic outflow a 'double envelope' may be seen, where the two predominant Doppler velocities of LVOT and aortic outflow are seen. This has the advantage of knowing that the velocities through LVOT and AV were from the same heartbeat, although they may not be as accurate. Because LVOT and aortic ejection occur in the same phase of the cardiac cycle, the continuity equation is accurate in the presence of aortic regurgitation. It can also be used to calculate other valve areas. For example, flow through the mitral valve in diastole must equal flow through the AV in systole, but the method must factor in any aortic and mitral regurgitation. This becomes a more time-consuming and complicated calculation, with the possibilities of inaccuracies from multiple measurements.

Calculation of regurgitant volumes

Regurgitation is the flow of blood backwards through an incompetent valve. Quantification of regurgitant volume and its fraction of SV provides objective information on severity and guides the clinician on the need for intervention. Regurgitant volume and fraction are validated for grading severity of mitral and aortic regurgitation, but are seldom used for tricuspid and pulmonary valve pathology.

Volumetric method:

$$\text{Systemic SV} = \text{Total SV} - \text{regurgitant volume (RV)},$$

where the total SV is forward flow through the regurgitant valve.

Thus, for mitral regurgitation:

$$SV_{LVOT} = SV_{MVI} - RV_{MV}$$

where MVI is mitral valve inflow and SV_{LVOT} is the systemic (true) SV.

This demonstrates conservation of mass as the passage of blood has only two options; forward through the LVOT or backwards through the incompetent mitral valve.

In practice, whilst possible, this method is seldom used because of the challenges involved in accurately measuring mitral valve area, which is more complex in shape than the AV and changes in shape during atrial systole. The presence of aortic regurgitation is a compounding problem as it is in the same phase as mitral inflow and thus erroneously increases total SV.

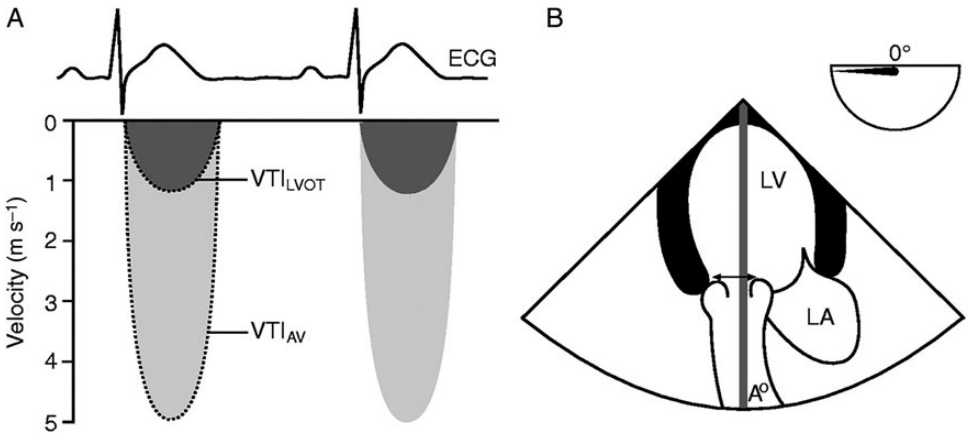

Fig 1 CSA_{LVOT} is measured from the diameter of LVOT, given as an arrow in the 2D echo image in (B). A 'double envelope' signal is obtained from the CW Doppler of LVOT and AV. The thick darker envelope represents low velocity flow through LVOT and the lighter envelope represents flow through stenotic AV in (A). The software calculates peak and mean gradients for each signal and the velocity–time integral (VTI) for AV VTI_{AV} and LVOT VTI_{LVOT} signals. SV is calculated by the equation $CSA_{LVOT} \times VTI_{LVOT}$. AV area is calculated from the equation $CSA_{LVOT} \times VTI_{LVOT}/VTI_{AV}$. (Reproduced after permission from Morgan-Hughes.)[6]

For aortic regurgitation, the equation is as follows:

$$SV_{MVI} = SV_{LVOT} - RV_{AV}$$

where SV_{MVI} is the systemic (true) SV after the removal of the aortic incompetent component from the LVOT ejection. Again, this process is seldom used as it is time-consuming and has the same error potential as that with the mitral valve. Similarly, mitral regurgitation will underestimate the aortic regurgitant volume and invalidate the calculation.

The regurgitant fraction can be calculated by dividing the regurgitant volume by total SV and is a gauge of severity. Quantification of all valve lesions is given in Table 1.

Proximal isovelocity surface area measurement

Proximal isovelocity surface area (PISA) is the term used to explain the phenomenon that occurs as blood flows through an orifice, whereby all equidistant points towards the orifice exhibit the same velocity. The velocity increases as blood nears the orifice. This creates the illusion of multiple concentric hemispheres of flow convergence, with each hemisphere having the same velocity, the smallest of which is the fastest. PISA can be used in all valves, but its commonest application is in mitral valve pathology. PISA hemispheres are seen using colour flow Doppler analysis.

Regarding the mitral valve, PISA hemispheres occur on the ventricular side of the valve during systole for mitral regurgitation, and on the atrial side of the valve during diastole for mitral stenosis. Thus, for mixed mitral valve disease, PISA hemispheres can be seen on both sides of the valve throughout the cardiac cycle.

PISA size can be artificially changed to suit the interrogation simply by altering the Nyquist limit, with no loss of accuracy. Reducing the Nyqyist limit increases the size of the PISA.

For mitral regurgitation, the flow rate through any proximal hemisphere equals regurgitant flow rate through the mitral valve during systole.

Using the same equation from above, flow=CSA×velocity and the law of conservation of mass (as in the Continuity Equation), the following equations must be true:

$$\text{PISA flow rate} = \text{Regurgitant flow rate}$$
$$CSA_{PISA} \times velocity_{PISA} = EROA_{MV} \times velocity_{MR}$$

where EROA is the effective regurgitant orifice area and MR is mitral regurgitant jet peak velocity.

CSA_{PISA} is assumed to be hemispherical, thus the area is $2\pi r^2$ (surface area of a sphere is $4\pi r^2$). The radius is the length of the PISA from the valve orifice and the velocity is simply the Nyquist limit set by the investigator. If the PISA angle (θ) is <180°, then the CSA should be multiplied by θ/180°.

Once the $EROA_{MV}$ is known (in itself a marker of MR severity), the regurgitant volume can be calculated in the same way that SV was calculated in the previous section.

$$\text{Regurgitant volume (MR)} = EROA_{MV} \times VTI_{MR}$$

CW Doppler aligned through the MR jet is used both for peak velocity to calculate EROA and the area under the curve (VTI) to calculate regurgitant volume.

The above process is used regularly in formal echocardiography but is time-consuming in the operating theatre. An abbreviated method of calculating $EROA_{MR}$ uses the following equation:

$$EROA_{MR} = \frac{r^2}{2}$$

where r is the PISA radius (Fig. 2).

This can only be done if the Nyquist limit is set to 40 cm s^{-1}, the PISA is a complete hemisphere, r is the radius of the first PISA seen and the MR peak velocity is 5 m s^{-1}. Whilst much simpler, errors are often small and acceptable. PISA radius alone at 40 cm s^{-1} can be used as a marker of MR severity.

PISA can also be used to calculate mitral valve area (MVA) in mitral stenosis:

Thus

$$PISA_{MV-inflow} \times velocity_{PISA} = MVA \times VTI_{MV-inflow}$$

Pressure half time (PHT)

PHT is a simple Doppler method for assessing MVA and the severity of aortic regurgitation, and uses the concept of pressure deceleration.

Mitral stenosis

The pressure gradient between LA and LV increases in mitral stenosis, with longer time required for blood to fill the LV through the stenotic valve. The early diastolic, transmitral Doppler velocity (E-wave) deceleration is subsequently prolonged. PHT

Table 1 Quantification of valve lesions

	Mild	Moderate	Severe
Aortic regurgitation			
Jet width/LVOT width (%)	<0.25	<0.25–0.64	>0.64
Vena contracta width (cm)	<0.3	0.3–0.6	>0.6
Jet area/LVOT area (%)	<4	4–60	>60
$P^{1/2t}$ (ms)	>500	500–200	<200
Regurgitant volume (ml $beat^{-1}$)	<30	30–59	>60
Regurgitant fraction	<30	30–49	>50
EROA (cm^2)	<0.10	0.10–0.29	0.3
Aortic stenosis			
Jet velocity (m s^{-1})	<3	3–4	>4
Mean gradient (mm Hg)	<25	25–40	> 40
Valve area (cm^2)	1.5–2.0	1.0–1.5	<1.0
Mitral regurgitation			
Jet area/LA area (%)	<20	20–40	> 40
Vena contracta (mm)	<3.0	3–6.9	>7
PISA radius (mm)	<4	4–10	>10
Regurgitant volume (ml)	<30	30–59	>60
Regurgitant fraction (%)	<30	30–49	>50
EROA (cm^2)	<0.2	0.2–0.4	>0.40
Mitral stenosis			
Mean gradient (mm Hg)	<5	5–10	>10
PHT (ms)	71–139	140–219	>219
MVA (cm^2)	1.5–2.0	1.0–1.5	<1
Tricuspid regurgitation			
Jet area (cm^2)	<5	5–10	>10
PISA radius (mm)	<5	6–9	>9
Vena contracta (mm)	–	<7	>7
Pulmonary stenosis			
Peak velocity (m s^{-1})	<3	3–4	>4
Pressure gradient (mm Hg)	<36	36–64	>64
LV dysfunction			
dP/dt (mm Hg s^{-1})	1200	800–1200	<800

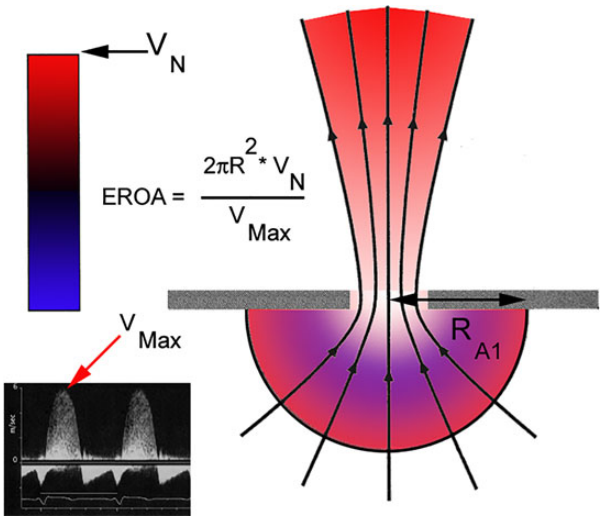

Fig 2 Proximal isovelocity surface area (PISA) principle applied to stenotic or regurgitant orifice area. EROA refers to effective regurgitant orifice area; $2\pi R^2$ surface area of hemispheric shell derived from the proximal flow convergence radius (R) in cm^2; V_N velocity at the radius of hemispheric shell (colour aliased velocity or Nyquist limit) (cm s^{-1}); V_{max} peak velocity across the stenotic orifice. (Figure belongs to A. Pybus. Reproduced with permission from ipad app; https://itunes.apple.com/gb/app/papworth-hospital-mcq-learning/id679626718?mt=8.)

measures the rate of decrease of the pressure gradient between LA and LV and is the time required for the peak pressure gradient to decline to 50% of its original value (in milliseconds). As stenosis increases in severity, the PHT increases. From before, pressure has to be extrapolated from Doppler velocities:

$$P_{half} = \frac{1}{2} \times P_{peak}$$

from Bernoulli $\Delta P = 4V^2$

$$4(V_{half})^2 = \frac{1}{2} \times 4(V_{peak})^2$$

Simplifying the above, we obtain

$$V_{half} = V_{peak}/\sqrt{2}$$

or more simply

$$V_{half} = V_{peak} \times 0.707$$

Thus PHT is the time taken for the initial peak mitral inflow velocity to decrease by 30% (Fig. 3).

PHT is quick to do and independent of CO, mitral regurgitation, and heart rate. It is thus very useful in mitral stenosis with coexistent mitral regurgitation. Conversely, in low CO status, severity of mitral stenosis can be underestimated by low transmitral pressure gradients.

From experimental models, a PHT of 220 ms equates to a MVA of 1.0 cm^2.[7]

Thus, MVA equals 220/PHT (shorter PHT = bigger valve area).[8]

Limitations of using PHT to calculate MV area

Any condition that alters the diastolic compliance of the LA or LV can affect flow velocity and pressure half time. It is not useful in the estimation of normal mitral valve area because it reflects only the compliance of the ventricle when there is no stenosis. It is inaccurate if the compliance of the ventricle is abnormal. Pressure half time method is not validated in calculating prosthetic mitral valve areas.[9] It is unreliable with severe aortic regurgitation because of rapid equilibration of LA and LV pressures and also because the mitral inflow and aortic back flow may be difficult to differentiate with Doppler. Sinus tachycardia, first-degree block and atrial flutter with fast atrial rates can affect deceleration slope by altering the mitral inflow E wave and artificially shorten the PHT and overestimate the MVA. Pressure half time method is also not accurate in the presence of an atrial septal defect.

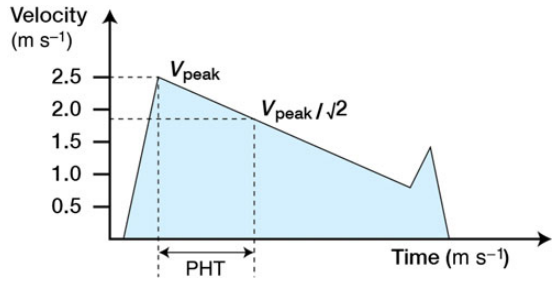

Fig 3 The method of measuring pressure half time from the Doppler velocity spectrum. PHT equals the time taken for the peak velocity (V_{peak}) to decrease to a value equitant to $V_{peak}/\sqrt{2}$. In this example, PHT equals the time taken for the velocity to decrease from 2.5 to 1.77 m s^{-1}. (From Anderson.[11] Reproduced with permission from MGA Graphics.)

Aortic regurgitation

In aortic regurgitation, CW Doppler velocity profile during diastole denotes the pressure decrease between aorta and left ventricle and the velocity at which the pressures equilibrate reflects severity of regurgitation. With mild disease, the early diastolic gradient is initially high and declines slowly over time. The gradual decrease in aortic diastolic pressure creates a small increase in left ventricular diastolic volume. In severe aortic regurgitation, the drop in pressure gradient during diastole occurs suddenly as a result of the rapid decrease in aortic diastolic pressure, with an associated increase in left ventricular end-diastolic volume (PHT < 300 ms). PHT is most useful in acute aortic regurgitation, which commonly manifests as a rapid increase in LVEDP and short PHT. PHT is not validated for estimating severity of aortic regurgitation in the presence of significant mitral disease.

Ventricular systolic function (dP/dt)

Many of the methods above calculate LV function during ejection and are thus influenced by preload and afterload. Using the mitral regurgitation jet (CW Doppler), dP/dt advantageously measures the rate of increase of ventricular pressure during the isovolumetric phase of contractility (units mm Hg s^{-1}). The MR jet is measured at 1 and 3 m s^{-1} (which corresponds to a pressure increase from 4 to 36 mm Hg, using the simplified Bernoulli equation) and the time interval between 1 and 3 m s^{-1} on the mitral regurgitation jet is measured to calculate dP/dt.[10]

$$\frac{dP}{dt} = \frac{32}{\Delta t}$$

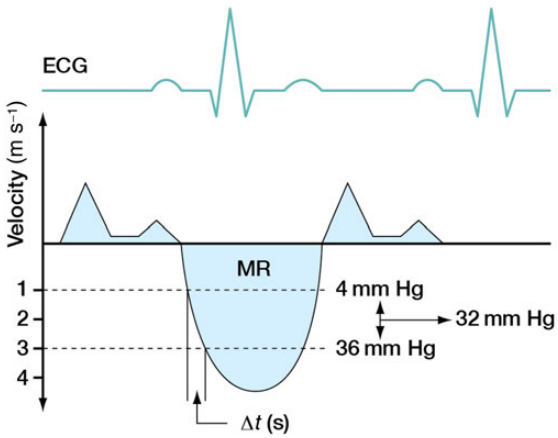

Fig 4 Measurement of the dP/dt from the mitral regurgitation Doppler signal. For explanation, please refer to text. (From Anderson.[11] Reproduced with permission from MGA Graphics.)

(i.e. time interval taken for the velocity to change from 1 to 3 m s^{-1}) (Fig. 4).

Normal values are 1000–1200 mm Hg s^{-1}.

It can also be used to assess right ventricular systolic function, usually between 0 and 2 m s^{-1}.

Conclusions

Echocardiography is not only used for evaluating cardiac anatomy but can also provide objective haemodynamic information about valve areas, pressure gradients, intra-cardiac volumes, ventricular systolic and diastolic function and cardiac output. These assessments guide clinicians and their advice to patients on treatment options and need for intervention, such as grading aortic stenosis and the need for valve replacement. However, they are measurements which require accuracy and expertise and have complex limitations in the presence of other cardiac pathologies and thus should be used in conjunction with other modalities in the final decision-making process.

Declaration of interest

None declared.

MCQs

The associated MCQs (to support CME/CPD activity) can be accessed at https://access.oxfordjournals.org by subscribers to *BJA Education*.

References

1. Quinones MA, Otto CM, Stoddard M, Waggoner A, Zoghbi WA. Recommendation for quantification of Doppler echocardiography: a report from the Doppler Quantification Task Force of the Nomenclature and Standards Committee of the American Society of Echocardiography. *J Am Soc Echocardiogr* 2002; **15**: 167–83
2. Savage RM, Aronson S. *Comprehensive Textbook of Intraoperative Transesophageal Echocardiography*, 2nd Edn. Lippincott Williams & Wilkins, 2004
3. Levine RA, Jimoh A, Cape EG, McMillan S, Yoganathan AP, Weyman AE. Pressure recovery distal to a stenosis: potential cause of gradient 'overestimation' by Doppler echocardiography. *J Am Coll Cardiol* 1989; **13**: 706–15
4. Silbert DR, Brunson SC, Schiff R, Diamant S. Determination of right ventricular pressure in the presence of a ventricular septal defect using continuous wave Doppler ultrasound. *J Am Coll Cardiol* 1986; **8**: 379–84
5. Kapila R, Mahajan R. Diastolic dysfunction. *Contin Educ Anaesth Crit Care Pain* 2009; **9**: 29–33
6. Brown J, Morgan-Hughes MJ. Aortic stenosis and non-cardiac surgery. *Contin Educ Anaesth Crit Care Pain* 2011; **5**: 1–4
7. Hatle L, Angelsen B, Tromsdal A. Non-invasive assessment of atrioventricular pressure half-time by Doppler ultrasound. *Circulation* 1979; **60**: 1096–104
8. Otto CM. *Textbook of Clinical Echocardiography*, 2nd Edn. Philadelphia: WB Saunders Company, 2000
9. Dumesnil JG, Honos GN, Lemieux M, Beauchemin J. Validation and applications of mitral prosthetic valvular areas calculated by Doppler echocardiography. *Am J Cardiol* 1990; **65**: 1443–8
10. Bargiggia GS, Bertucci C, Recusani F *et al*. A new method for estimating left ventricular dp/dt by continuous wave Doppler echocardiography. Validation studies at cardiac catheterisation. *Circulation* 1989; **80**: 1287–92
11. Anderson B. *Echocardiography: The Normal Examination and Echocardiographic Measurements*. 2nd Edn. Queensland, Australia: MGA Graphics, 2007; 223