TRANSESOPHAGEAL ECHOCARDIOGRAPHY AND HAEMODYNAMICS

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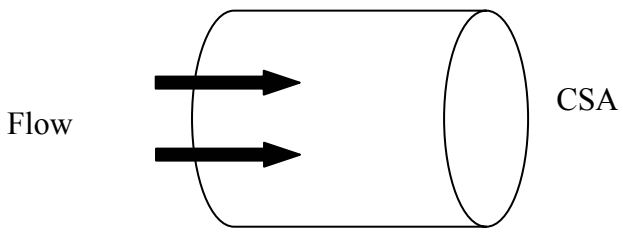
Basic equations [2, 12-15]

Three main equations are the basis for the following hemodynamic calculations:

(a) ***The flow equation*** states that the flow is the product of the cross section area (CSA) and average velocity of the blood cells passing through that CSA.



Flow = CSA x Velocity

Since volume is the product of flow and time, the above equation can be rewritten:

Volume = CSA x Velocity x Time

Velocity x Time is the velocity time integral (VTI) that is automatically given by all the common echo machines by tracing the profile of a PW or CW Doppler signal (figures 1 and 2). To assess a volume passing through an orifice at any beat, we therefore must apply the equation:

$$\textbf{Volume (cm}^3\textbf{) = CSA (cm}^2\textbf{) x VTI (cm)} \quad \textbf{(1)}$$

(b) ***The Bernoulli equation***, in its complete structure, determines the value of a pressure gradient for a flow passing through a restricted orifice. It can be semplified in the following equation:

$$\Delta P = \tfrac{1}{2} \rho (V_b^2 - V_a^2)$$

where ρ is the mass density of blood ($1.06 \times 10^3 kg/m^3$)

Vb is the velocity at point b (distal to the restriction)

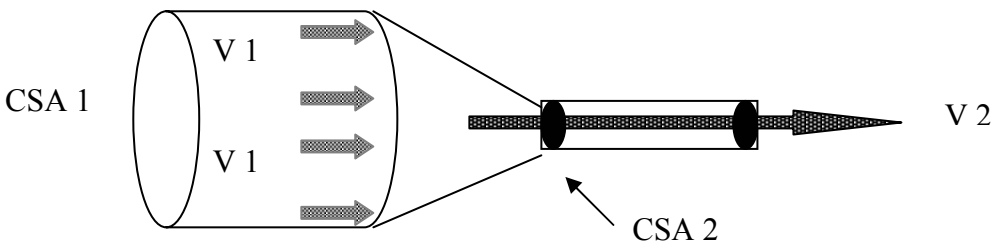
Va is the velocity at point a (proximal to the restriction)

Since Va is very low, it can be skipped, and therefore the final equation that is commonly applied is

$$\Delta P = 4 \times V^2 \qquad \textbf{(2)}$$

(c) ***The continuity equation*** is the Gorlin formula of echocardiography and is applied to calculate the area of a stenotic or regurgitant valve. Basically, it states the "conservation of flow" regardless of the CSA that is met by the flow itself:

Flow (Q) = CSA x Velocity



Q 1 = Q 2

Q 1 = CSA 1 x V 1 = CSA 2 x V 2 **(3)**

CSA 2 = Q 1/ V2

Stroke volume and cardiac output [2, 16-23]

To determine the stroke volume the equation (1) is applied. The most important technical factor to ensure a correct value of stroke volume will be to properly match the site of sample volume with the anatomic measure of CSA. It requires the assessment of the aortic flow VTI and of the CSA. It is assessed with the following steps:

1. Research of the best parallel orientation with the blood flow at the bidimensional (2D) echo examination. This is usually obtained using the transgastric views (long axis transgastric view at 90° with lateralization of the tip of the probe, or deep transgastric view at 0°).

2. PW Doppler signal tracing by placing the sampling volume at the left ventricle outflow tract (LVOT) level, about 5 mm proximal to the aortic valve; VTI determination by tracing the outer edge of the dense envelope of the spectral recording (figure 1).

3. CSA determination (figure 3): when using the PW Doppler for VTI determination, the LVOT area is to be considered. It can be assessed by the 2D examination, using the mid-oesophageal long-axis view at 120° , and by calculating the LVOT diameter. Trace the measurement line at the level of the aortic annulus during systole. From this diameter, and by assuming a circular section, the CSA is determined by the following equation:

$$\mathbf{CSA = D^2 \times 0.785} \qquad \mathbf{(4)}$$

4. Calculate the SV (CSA x VTI) and CO (SV x heart rate)

Whatever method is applied to assess the SV, the main question remains: is a TEE-based SV and cardiac output determination reliable? Of course, the theoretical calculations at the basis of all the equations applied are sound, but the main problem remains that both 2D and Doppler determinations are strongly operator-dependent. To this respect, the ability of the operator to limit the potential sources of error (non-parallel flow determination; incorrect border determination of

the Doppler signal; excessive approximation in CSA determination...) is of paramount importance, and limits the applicability of echo-derived SV measurements. Particular attention should be posed on the VTI measurement: it must be considered that under mechanical ventilation there is a considerable beat-to-beat variation of the stroke volume, that is reflected by different VTIs. In synus rhythm, at least three VTIs should be calculated and the mean should be used for SV assessment; in atrial fibrillation, at least 5-7 beats should be mediated. Nevertheless, TEE may be useful as a "trend monitor" of SV and cardiac output during cardiac and non-cardiac operations. Given that the CSA is relatively stable in the same patient, the VTI serial changes in time accurately reflect equivalent SV changes. When using a serial monitoring of VTI, the best way is to use the CW Doppler of the aortic flow, that appears to have less variability than the equivalent PW Doppler.

Pressures and gradients

The pressure gradients across a stenotic structure are assessed by the modified Bernoulli equation (2). A CW Doppler signal is commonly applied when exploring aortic stenotic lesions, while the mitral valve can be explored with a PW Doppler signal too.

All the commonly available echo machines automatically calculate the pressure gradients from the Doppler signal waveform. To obtain the mean pressure gradient it is needed a complete tracing of the waveform profile, while the peak gradient assessment only requires to settle the velocity peak. Transaortic pressure gradients calculations need a Doppler signal parallel to the transaortic flow; this is achievable in the transgastric views as shown in figure 2. Transmitral pressure gradients calculations can be obtained in the standard four chamber view (figure 5). Gradients across the pulmonary valve are more difficult to be obtained due to a difficult parallelism of the Doppler signal with transpulmonary flow. This can be obtained, in expert hands, by a 2D transgastric view or using an upper oesophageal 90° view.

Since the calculation of pressure gradients relies on the quadratic expression of velocity, little errors in determining this last parameter are amplified. It is therefore very important to find the best parallelism between Doppler signal and blood flow.
Once obtained this, the measure is reliable unless (1) the velocity proximal to the stenosis exceeds 1.5 m/s; (2) there are two stenotic lesions in the blood flow path (subaortic stenosis + aortic valve stenosis); and (3) the stenotic lesion is very long, tunnel-like.

Intracardiac pressures may be calculated with TOE providing that one or more cardiac valves are regurgitant. By measuring the peak velocity of the regurgitant flow, a pressure gradient can be established. The pressure gradient plus the pressure in the chamber receiving the regurgitant flow is the pressure inside the chamber driving the regurgitant flow:

Driving Pressure = Pressure Gradient + Pressure distal to the regurgitant flow

Many pressures can be calculated depending on the presence of regurgitant flows and septal defects. The most commonly measured pressures are:

(a) Systolic pulmonary artery pressure (sPAP) [24-29]

Requires a tricuspid valve regurgitation (figure 6). The peak velocity and the resulting pressure gradient are determined usually using a CW Doppler signal. The pressure distal to the regurgitant flow is the RAP. The equation is:

sPAP = Peak Pressure Gradient + RAP

This equation is true in absence of a pulmonary valve stenosis, assuming that the systolic right ventricular pressure is equal to the sPAP.

(b) Left atrial pressure (LAP) [30]

Requires a mitral valve regurgitation. The peak velocity and the resulting pressure gradient are determined usually using a CW Doppler signal: consider that the mitral regurgitation flow is often characterized by a very high peak velocity. The driving pressure is the systolic systemic arterial pressure (sSAP). The equation is:

LAP = sSAP – Peak Pressure Gradient

This equation is true in absence of an aortic valve stenosis, assuming that the systolic ventricular pressure is equal to the sSAP.The TOE assessment of the LAP is critical: the driving pressure is very high, and a litlle error in its determination leads to an unacceptable error in LAP measurement, moreover if we consider that this value has many clinically relevant meanings.

(c) Left Ventricle End Diastolic Pressure (LVEDP) [30].

Requires an aortic valve regurgitation. The end diastolic pressure gradient is assessed using a CW Doppler signal (figure 7). The driving pressure is the diastolic systemic arterial pressure (dSAP).The equation is:

LVEDP = dSAP – End Diastolic Pressure Gradient

Preload and fluid responsiveness assessment

The preload, expression of myocardial fiber stretch at the end of diastole, is represented by the LV volume and indirectly by the pressure required to fill the LV, and has a direct effect on cardiac performance. The achievement of an adequate pre-load to optimize cardiac performance remains a primary target in the immediate postoperative treatment of cardiac surgery patients. In clinical practice the measure of ventricular end-diastolic pressure can be assessed using left atrial pressure and pulmonary artery occlusion pressure (PAOP), but the relative invasiveness of the procedure and

the limitations of the interpretation have questioned their utility. Echocardiography and Doppler application allow in a non invasive manner to estimate a reliable LAP pressure and LV end-diastole pressure integrating volume and pressure information.

TOE allows for the bidimensional visualization on LV cavity helping to quantify with a strong correlation the LV volumes. The assessment of LV filling is routinely evaluated from transgastric short-axis view at papillary muscles because the volume variations are more evident in this plane than in long axis cut. The ***end-diastolic area*** (figure 10) at the transgastric short axis view has proved to be a more sensitive index of LV filling than PAOP during abdominal aortic aneurismectomy. An end-diastolic area $\leq$ 5 cm^2 body surface area is accepted as cut-off of a hypovolemic state accepted for hyperdynamic conditions where a hypercontactility is associated to normovolemia.

The use of ***mitral flow patterns*** as surrogate of left ventricular pressure has some limitations depending by the influence of loading conditions and ventricular compliance.

Factors that influences Left Ventricular compliance

- Myocardial ischemia
- Restrictive cardiomyopathy
- Right-to-left interventricular septal shift
- Aortic stenosis
- Cardiac tamponade
- Myocardial fibrosis
- Inotropic-drug use
- Hypertension

However LV end-diastolic pressure can be estimated looking at the deceleration time (DCT) of the early diastolic filling of mitral inflow using pulsed-wave Doppler. The sample volume (2 mm width) must be placed at the tips of mitral leaflets and the DCT must be measured on the early filing (E) wave, extrapolating the descending slope to the baseline. This method correlates with pulmonary capillary wedge pressure (PCWP) in those patients with an ejection fraction less than 35 %. The shorter is DCT the higher is the PCWP. A DCT$\geq$150 ms has a sensitivity of 93% and a specificity of 100 for predicting a PCWP $\leq$10 mmHg [54-75].

Pulmonary venous flow and especially the systolic-diastolic ratio strongly correlate with mean left atrial pressure, but this correlation depends on the ventricular function and cardiac output. Pulmonary venous flow is examined placing the sample volume at least 1 cm into the pulmonary vein. Colour flow can often help to locate the ostium of the pulmonary veins. The typical pulmonary vein flow is comprised of systolic (S), diastolic (D) and atrial reversal wave (rA) (figure 11). The S/D ratio and the velocity and duration of rA reflect ventricular compliance and ventricular filling pressure In the patients with preserved ventricular function the correlation is positive so that a high atrial pressure is represented by a high systolic wave. Conversely, whenever the contractility is depressed, a high atrial pressure is represented by a decreased systolic wave [76-83].

The developments of new modalities as **M-mode color** echocardiography and **tissue Doppler imaging** (**DTI**) have added other criteria of preload evaluation and their combination with classical transmitral flow indices allow to estimate atrial pressure.

The flow velocity from mitral inflow area towards apex (from mitral valve plane to 4 cm distally into the LV cavity) can be measured placing an M-mode cursor in the center of brightest colour inflow from the transoesophageal four chamber view .The information obtained by ***color M-mode*** is similar to the data obtained positioning simultaneously more samples volume at different levels from mitral annulus to the apex of the LV. When the mitral valve opens a first flow propagates from left atrium to left ventricle corresponding to the early filling (E wave in PW study of mitral valve) followed by a second flow depending on the atrial kick (A wave). Flow at mitral valve occurs earlier than at apical level. The time delay between mitral annulus and apex can be represented by the slope of the color wavefront. Adjusting the colour Doppler setting to produce color aliasing , the slope of the first color alias or of the color-non color interface (black-to-red transition zone) during the early filling, represents the propagation flow velocity (Vp) of the blood flowing towards the apex. Young health subjects typically have a Vp $\geq$ 55cm/sec.Older patients ,

those with left ventricular hypertrophy and/or advanced diastolic dysfunction have a lower Vp. The flow velocity propagation (Vp) has been shown to be correlated inversely with the time constant of LV isovolumic relaxation (τ) and to be relatively preload independent. The ratio E between inflow velocity and flow propagation velocity relates linearly to the mean left atrial pressure [84-95]

The **DTI** is a new technique that records the systolic and diastolic velocitie within myocardium and at the corners of mitral annulus positioning [96-100] . Doppler signal arising from tissue motion differs from blood motion by two main aspects: 1) tissue velocities are lower (20 cm/sec) than the red cells ones (20-100 cm/sec) 2) the amplitude of the signal arising from cardiac structures (myocardium, mitral and tricuspidal annulus) is significantly higher (approx. 100 times greater than blood cells). Conventional blood flow Doppler uses high pass filter to remove low velocities due to wall motion: by rearranging the filter and the amplification (both gains and filter must be set low) the Doppler signal reflected by the cardiac tissue can be displayed. The sample volume (5 mm) must be placed within the myocardium or at the mitral annulus (lateral or septal in four chambers section) and a spectral recording of velocities is reproduced.

The spectral longitudinal velocity of myocardium is represented by a systolic deflection (negative deflection) and two diastolic deflections (positive deflections), representing the early filling (E_m) and atrial flow (A_m). The early diastolic wave at mitral annulus show a reduction with age and has been demonstrated to be an index of LV relaxation that is relatively insensitive to left atrial pressure.

The ratio of transmitral E velocity to Em has been recently demonstrated to relate well with mean left atrial (or pulmonary capillary wedge) pressure in multiple clinical scenarios, such as depressed or normal systolic LV function, hypertrophic cardiomyopathy, sinus tachycardia, and atrial fibrillation.

The echo Doppler can be applied in the assessment of hemodynamic instability and hypotension caused by a reduction of biventricular preload (i.e. hemorrhage or pathologic fluid shifts) predicting

the hemodynamic response to fluid loading with the estimation of the left ventricular stroke volume variation on the basis of the specific interactions of the heart and the lungs under mechanical ventilation. It is known that under these conditions, a SV variation is induced by the effects of intrathoracic intermittent positive pressure on venous blood flow [101-106]. These variations are more evident in patients with a reduced pre-load ("fluid responders"). The SV variation may be appreciated with a CW Doppler study of the aortic flow, and can be expressed in terms of aortic blood flow velocity variation. To measure this effect, more than one respiratory cycle should be investigated: the velocity of recording should therefore be decreased in order to have many aortic flow profiles in the same screen (figure 12). The maximal and minimal velocities should be measured to aortic blood velocity variation: the difference between the two values divided by the mean value is the % variation. A value of about 12% has been demonstrated as a good cut-off for fluid responsiveness.

Doppler analysis of the transmitral flow has been used as an index of fluid responsiveness. The ratio between the E and A waves VTIs is an indicator of fluid responsiveness (figure 13): the lower it is, the more likely the patient will benefit from a fluid expansion, with a good cut-off settled at 1.26 [107].

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