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Cardiovascular Hemodynamics

An Introductory Guide

Second Edition

💥 Humana Press

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(SV2 > SV1). Vasodilators do not affect the LV contractility. Black = baseline hemodynamic profile; Fig. 7.6 The hemodynamic effect of inotropes. The net effect is a leftward shift in the end-systolic pressure-volume relation (LV end-systolic elastance ($E_{es1} \rightarrow E_{es2}$)) indicating augmented contractility, which results in improved cardiac performance as indicated by the increased stroke volume (SV2 > SV1). Black = baseline hemodynamic profile; Fig. 7.7 Hemodynamics of catecholamine vasopressor agents. The actions of catecholaminergic vasopressors result in improved contractile function denoted by the slightly leftward shift in the end-systolic pressure-volume relation $(E_{es1} \rightarrow E_{es2})$. However, the dominant effect from these agents is peripheral vasoconstriction leading to an increase in afterload ($E_{a2} > E_{a1}$). Depending on the inotropic effect that these agents confer in comparison to the increased afterload, stroke volume might be expected to increase although to a lesser extent as compared to pure inotropic agents, remain unchanged, or in the setting of an overwhelming increase in afterload, actually decrease (SV1 \approx SV2). These agents are therefore best suited in cases of vasodilatory shock where their effect in maintaining adequate tissue perfusion pressure is required. Black = baseline hemodynamic profile; red = vasopressor effect; pink = arterial elastance (afterload) at baseline; Fig. 7.8 Hemodynamics of pure vasoconstrictors. These agents act primarily to increase the systemic vascular resistance resulting in an upward shift of the pressure-volume relationship. The increase in afterload does not affect intrinsic contractile function but does result in an increase in the end-systolic blood pressure, increased LV end-diastolic pressure, and an increase in the LV afterload $(E_{a1} \rightarrow E_{a2})$. The combined effects result in elevated myocardial oxygen demand and a reduced stroke volume (SV2 < SV1). Therefore, while systemic blood pressure is improved, it comes at the expense of the cardiac output. Black = baseline hemodynamic profile; red = vasoconstrictor effect; pink = arterial elastance (afterload) at baseline; blue = arterial elastance (afterload) Fig. 8.1 Angiotensin receptor-neprilysin inhibitors have the potential to modulate two counter-regulatory neurohormonal systems in HF: the renin-angiotensin-aldosterone system and natriuretic peptide system. ANG angiotensin, AT1 angiotensin type 1, HF heart failure, NP natriuretic

peptide, RAAS renin-angiotensin-aldosterone system. (Indian Heart Journal Volume 70, Supplement 1, July 2018, Ivabradine's primary mechanism of action on cardiac tissue Fig. 8.2 is on the sinoatrial (SA) node, which occupies a predominantly subepicardial position at the junction of the superior vena cava (SVC) and the right atrium (RA). (a) Heart with position of the Sinoatrial (SA) node. (b) In the sinoatrial node, ivabradine blocks the intracellular aspect of the hyperpolarization-activated cyclic nucleotide-gated (HCN) transmembrane channel, which is responsible for the transport of sodium (Na⁺) and potassium (K⁺) ions across the cell membrane, in the open state. This results in the inhibition of the inward funny current (I_f) , which is specifically activated at hyperpolarized membrane potentials. (c) By selectively inhibiting I_f , there is a reduction in the slope of diastolic depolarization of the pacemaker action potential (shaded region) and an increase in the duration of diastole, without altering other phases of the action potential. This results in heart rate reduction. Ao aorta, IVC inferior vena cava, PA pulmonary artery, RV right ventricle. (PMID:28958335)110 Fig. 8.3 Approval timeline of ivabradine across Europe and the United States. The indications for the use of ivabradine have evolved over time and differ based on region. Since it was first approved for use in angina by the European Medicines Agency (EMA) in 2005, the findings of several randomized controlled trials have resulted in expanded indications to include select heart failure patients and only recent approval by the US Food and Drug Administration (FDA) for this indication. BEAUTIFUL Morbidity-Mortality Evaluation of the Ir-Inhibitor Ivabradine in Patients With Coronary Disease and Left Ventricular Dysfunction, CAD coronary artery disease, CV cardiovascular, HFrEF heart failure with reduced ejection fraction, LVEF left ventricular ejection fraction, MI myocardial infarction, NYHA New York Heart Association, NSR normal sinus rhythm, SHIFT Systolic Heart Failure Treatment with the I-Inhibitor Ivabradine Trial, SIGNIFY Study Assessing the Morbidity-Mortality Benefits of the Ir-Inhibitor Ivabradine in Patients With Coronary Artery Disease. (PMID:28958335)112 Kaplan-Meier cumulative event curves for different end Fig. 8.4 points in SHIFT. Primary composite outcome (Panel A); cardiovascular mortality or heart failure hospitalization and its two components cardiovascular mortality (Panel B); heart failure hospitalizations (Panel C) and heart failure deaths (Panel D) in the ivabradine and the placebo arms.

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| | To him rig. In cardiac tamponade, the pulsus paradoxus |
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| | |

presence of cardiac tamponade, the reciprocal changes seen in the normal heart are exaggerated when the pericardial sac is filled with fluid, thus limiting distensibility of the entire heart. This results in a more dramatic reduction in filling of the left ventricle during inspiration, exacerbating the normal inspiratory decrease in stroke volume and blood Fig. 10.5 Relationship of murmurs to LV, aortic, and LA pressure waveforms and hemodynamics in various valve diseases. (a) Aortic stenosis (AS). The gradient between the left ventricle and aorta in AS is greatest in mid-systole and is relatively small early and late in systole. The murmur therefore has a diamond shape, i.e., starts soft and builds to a peak in mid-systole and then becomes quiet in late systole immediately before S2. In young AS patients, there may be an early ejection sound. In senile AS, A2 becomes diminished or absent. An S4 is common due to LV noncompliance. (b) Aortic regurgitation (AR). The carly diastolic murmur (EDM) begins with S2 and has a decrescendo contour. The duration of the murmur continues for a variable time in diastole, depending on severity and acuity of AR. Notice that aortic diastolic pressure is low. (c) *Mitral regurgitation*. There is a pansystolic murmur with a flat profile due to the regurgitant flow into the left atrium. Note that the left atrial pressure rises during systole, a "v" wave. An early diastolic S3 or flow rumble may occur. (d) Mitral stenosis (MS). A loud S1 is heard since the mitral gradient is high and the leaflets are not very thickened or calcified. An important indicator of the severity of MS is the time interval between the S2 and opening snap (OS), which is the time it takes for LV pressure to fall from late systolic aortic pressure to early diastolic left atrial pressure. The mitral valve snaps open due to the increased gradient, causing an opening snap (OS). The diastolic murmur correlates with the gradient across the mitral valve, which is largest in early diastole, and increases again in late diastole in this patient in sinus The Doppler principle and Bernoulli equation. Bottom Fig. 11.1 right: The echo transducer sends ultrasound waves at a given frequency (f_0) to the heart, and the sound waves are reflected back to the transducer at a different frequency $(f_{\rm r})$. The difference between (f_0) and (f_r) is the Doppler shift. As shown in the equation, the Doppler shift is directly proportional to the transmitted frequency (f_0) , the cosine of the angle of incidence θ (angle between the ultrasound wave and vector of the red blood cell), and the velocity of the red blood cells, however, is inversely proportional to the

speed of ultrasound in the medium (c). Rearrangement of the equation allows one to determine the velocity of the red blood cells. Top right: The Bernoulli equation enables one to determine the pressure gradient across a stenosis, in this case, a stenotic aortic valve. Flow accelerates just before and at the level of the stenosis. The velocity proximal to the stenosis is V_1 , and the velocity distal to the stenosis is V_2 . Based on certain assumptions (see text), the Bernoulli equation can be simplified to $P1 - P2 = \Delta P = 4(V_2)^2$. In this case, the peak gradient is 64 mmHg based on the peak velocity across the aortic valve (V_2) of 4 m/s. Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2011.....156 Fig. 11.2 Various forms of Doppler in echocardiography. (a) Pulse wave (PW) Doppler of the mitral inflow with the sample volume placed at the leaflet tips. In PW, the same transducer crystal sends and receives waves to determine the Doppler shift at a particular sample volume, marked by the white arrow. Because PW obtains information about a particular location, it is said to have "range specificity or range resolution," but it is prone to aliasing. Note that in diastole there is early filling (E wave) and Late Filling (A wave). Diastasis is known as the period between the E and the A wave. The E velocity is 68 cm/s. (b) Continuous wave (CW) Doppler across the aortic valve. In CW, one crystal sends sound waves continuously and another crystal receives the sound waves. Because the CW profile represents all the velocities along the path of interrogation (represented by the *dotted line*), the peak velocity cannot be localized based on the CW signal alone. This phenomenon is known as "range ambiguity." The y axis is velocity and the x axis is time, and therefore the area under the curve is the velocity time integral (VTI), or the aortic valve VTI, in units of distance (cm). In this example, the peak velocity is 1.3 m/s and the Aortic Valve VTI is 22 cm. (c) Tissue Doppler of the mitral annulus characterizes annular velocities, with the corresponding annular e'and a' waves. These waves correspond temporally with the *E* and *A* waves of the mitral inflow. Because E = 68 cm/s and e' = 13 cm/s, the ratio E/e' is roughly 5, suggesting normal PCWP pressures. (d) Color Doppler in which the color pixels represent the mean velocity vector at a Fig. 11.3 Right-sided pressures. (a) M-mode through the IVC from the subcostal view. Note that the IVC size is <2.1 cm and collapses greater than 50%, suggesting normal right atrial pressure (0-5 mmHg). (b) Pulse wave (PW) Doppler of the hepatic vein showing normal hepatic vein flow. Note that

there are two antegrade waves (S and D) and one retrograde wave (a reversal). The representative portions on the JVP waveform are shown (S corresponds to the x descent, and Dcorresponds to the y descent). The onset of the S wave corresponds to the onset of the QRS (isovolumic contraction), although the peak occurs in mid to late systole. In this example, the velocity of the S wave is larger than the D wave, indicating normal right atrial pressures. (c) A plethoric IVC greater than 2.1 cm in width which does not collapse, suggesting a right atrial pressure between 10 and 20 mmHg. (d) Systolic flow reversal in the hepatic veins in severe tricuspid regurgitation. Notice that the S wave is above the baseline, indicating flow reversal. This corresponds to the blunted x descent and tall y wave in the Fig. 11.4 Pulmonary pressures and signs of pulmonary hypertension. (a) The right ventricular systolic pressure can be estimated from the peak tricuspid regurgitation velocity obtained in the right ventricular inflow view (see Question 1). (b) The continuous wave (CW) Doppler profile of the pulmonary regurgitation jet. The early peak velocity can be used to determine the mean pulmonary artery (PA) pressure by the following formula: Mean PA Pressure = $4v_{\text{EarlyDPR}}^2$. In this case, early pulmonary regurgitation (PR) jet velocity is 3.9 m/s and the end-diastolic PR velocity is 1.9 m/s. Therefore, the mean PA pressure is roughly 39 mmHg. Also, the pulmonary artery end-diastolic pressure (PAEDP) can be determined from the end-diastolic velocity and estimated right atrial (RA) pressure: PAEDP = RA + $4v_{\text{EDPR}}^2$ (see Question 2). Note that in pulmonary hypertension, there is absence of the typical end-diastolic dip in the pulmonary regurgitation CW profile that normally corresponds to atrial systole. (c) The sample volume is in the RVOT, just below the pulmonic valve. In pulmonary hypertension, there is a steep slope in early systole (acceleration phase becomes shorter, upper left corner) and there can be a mid-systolic dip in the RVOT profile (yellow stars), due to high afterload. A simplified formula to calculate the mean pulmonary artery pressure (MPAP) is MPAP = 80 - 0.5 (acceleration time (ms)). Acceleration time is roughly 90 ms, yielding a MPAP of 35 mmHg. (d) Note the D-shaped septum during systole, suggestive of RV Fig. 11.5 Stroke volume and aortic valve area (AVA) calculation using the continuity equation. (a) Based on the continuity equation, the flow through the left ventricular outflow tract (LVOT), or the volume of the blue cylinder, must

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the *red cylinder*. The stroke volume (represented by the blue cylinder) is estimated by multiplying the LVOT area by the LVOT VTI. The LVOT area is obtained using the equation Area = πr^2 = (Diameter)²*0.785, with the diameter measured in the parasternal long axis view. Because the LVOT diameter in this case is 1.9 cm, the LVOT area is 2.84 cm². From the apical 5 chamber or apical long axis view, the LVOT VTI is obtained, which in this case is 28.1 cm (bottom right and (b)). Therefore, the stroke volume = $28.1 \text{ cm} \times 2.84 \text{ cm}^2 = 79.8 \text{ cm}^3$. The product of the stroke volume and the heart rate (SV*HR) can give an estimate of cardiac output. The volume of the *red cylinder* is the product of the AVA and the AV VTI (c, d). Because the volume of the *blue cylinder* (LVOT Area*LVOT VTI) must equal the volume of the red cylinder (AVA*AV VTI) to satisfy the continuity equation, it follows that AVA = [LVOTVTI * (LVOTdia meter)² * 0.785]/[AVVTI] = Strokevolume/AVVTI (see Question 3). (b) Pulse wave Doppler Sample volume is placed just below the aortic valve in the 5 chamber view. and the LVOT VTI is traced. (c) Continuous wave Doppler measures the highest velocity along its path to estimate the peak and mean gradient across the aortic valve. The peak and mean gradients are 95/55 mmHg from the 5 chamber view, which is an underestimation of peak flow in this particular patient. Multiple views are necessary to obtain the highest, most representative jet velocity, as seen in (d). Right sternal border view obtains peak and mean gradients of 119/74 mmHg, higher than the peak gradient of 95 mmHg from the apical 5 cham-Fig. 11.6 Shunt calculation in a patient with a secundum atrial septal defect (ASD). (a) Color flow Doppler demonstrates left to right flow across the ASD in this subcostal view. (b) Pulse wave Doppler at the level of the ASD confirms that there is left to right continuous flow. During peak systole, based on the velocity of 1.2 m/s, the pressure gradient between the right atrium (RA) and the left atrium (LA) is $4v^2 = 4(1.2)^2 = 5.8$ mmHg. The RA pressure was estimated at 10 mmHg, so the LA pressure during systole is estimated at 15.8 mmHg (LA = RA + $4v^2$). (c) Measurement of systemic flow (Q_s) based on the LVOT area and LVOT VTI (pulse wave Doppler from the apical 5 chamber view, right upper corner). (d) Measurement of pulmonary flow (Q_n) based on the RVOT area and the RVOT VTI (pulse wave

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