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Editors

Cardiovascular Hemodynamics

An Introductory Guide

Second Edition

 Humana Press

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To our families for their unwavering support.

Preface to the Second Edition

For any cardiovascular care provider, an understanding of physiology is absolutely essential for delivering patient care. Whether sitting in the echocardiography reading room, performing procedures in the catheterization laboratory, or rounding in the coronary care unit, such an understanding is equally vital. While the underpinnings of cardiovascular physiology from a systems perspective have changed little in recent time, there have been substantial advances in the ability to apply these concepts to newer technologies.

Our primary goal for this second edition of *Cardiovascular Hemodynamics: An Introductory Guide* is not only to expound on the fundamental education of cardiovascular physiology but also to focus additionally on the clinical application of these hemodynamic principles. In order to achieve this, we have updated all of the first edition chapters and have added a new section, “Effects of Selected Interventions on Cardiovascular Hemodynamics.”

Born out of the huge economic burden of heart failure is an increased pressure on the healthcare system to decrease the often-avoidable readmissions for those patients with heart failure. We believe that a solid knowledge of heart failure physiology will arm those charged with the care of these patients with the tools necessary to decrease these costly readmissions. In addition, we have added another chapter, “Objective Evaluation of Hemodynamics in the Outpatient,” that aims to familiarize the reader with the recent advancements (medical and technological) that have proven helpful with regard to optimizing patient care.

Of course, an emphasis on the basic tenets of cardiovascular physiology remains our central focus, and we use numerous figures, hemodynamic tracings, tables, board style review questions, and hemodynamic “pearls” in order guide our readers. We believe this manual will be of immense value and interest to every student and practitioner of cardiovascular medicine who wishes to fully learn the hemodynamic foundation of cardiovascular medicine.

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We would like to acknowledge the authors, who, in many cases, were also mentors.

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Fig. 11.1 The Doppler principle and Bernoulli equation. *Bottom 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_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 $P_1 - P_2 = \Delta P = 4(V_2)^2$. 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 particular location 157

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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 *D* corresponds 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 *v* wave in the JVP waveform. 160

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- resistance, VTI velocity time integral, LVOT left ventricular outflow tract, D diameter, LA left atrial pressure, SBP systolic blood pressure, V_{MR} peak mitral regurgitation velocity, PCWP pulmonary capillary wedge pressure, LVEDP left ventricular end-diastolic pressure, DBP diastolic blood pressure, V_{EDAI} end-diastolic aortic insufficiency velocity, SVR systemic vascular resistance, Q_p pulmonary flow, Q_s systemic flow. (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2011) 177
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