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Chapter 1 : Cardiovascular Hemodynamics for the Clinician (, Paperback) | eBay

McLaughlin, D. P. and Stouffer, G. A. () Aortic Stenosis, in Cardiovascular Hemodynamics for the Clinician (ed G. A. Stouffer), Blackwell Publishing Ltd, Oxford.

Artikel bewerten Cardiovascular Hemodynamics for the Clinician, 2nd Edition, provides a useful, succinct and understandable guide to the practical application of hemodynamics in clinical medicine for all trainees and clinicians in the field. Cardiovascular Hemodynamics for the Clinician, 2nd Edition, provides a useful, succinct and understandable guide to the practical application of hemodynamics in clinical medicine for all trainees and clinicians in the field. He served as Director of the Cardiac Catheterization Laboratories and Director of Interventional Cardiology for 14 years before assuming his current role. He has published three textbooks and more than articles including several dealing with the hemodynamics of heart disease. List of contributors, viii Part I: Basics of hemodynamics 1 Introduction to basic hemodynamic principles, 3 James E. Faber and George A. Stouffer 4 Arterial pressure, 56 George A. Stouffer 5 The atrial waveform, 69 David P. McLaughlin and George A. Stouffer 6 Cardiac output, 82 Frederick M. Costello and George A. Stouffer 7 Detection, localization, and quantification of intracardiac shunts, 91 Frederick M. Valvular heart disease 8 Aortic stenosis, David P. Stouffer 9 Hemodynamics of transcatheter and surgical aortic valve replacement, John P. Vavalle, Michael Yeung, Thomas G. Caranasos and Cassandra J. Ramm 10 Mitral stenosis, Robert V. Kelly, Chadwick Huggins and George A. Stouffer 11 Aortic regurgitation, George A. Stouffer 12 Mitral regurgitation, Robert V. Cohen and George A. Stouffer 13 The tricuspid valve, David A. Tate and George A. Cardiomyopathies 15 Hypertrophic cardiomyopathy, Jayadeep S. Varanasi and George A. Stouffer 16 Heart failure, Geoffrey T. Jao, Steven Filby and Patricia P. Chang 17 Restrictive cardiomyopathy, David P. Pericardial disease 18 Constrictive pericarditis, David P. Stouffer 19 Cardiac tamponade, Siva B. Mohan and George A. Stouffer 20 Effusive constrictive pericarditis, Eric M. Smith and George A. Hemodynamic support 21 Hemodynamics of intra aortic balloon counterpulsation, Richard A. Santa J Cruz and George A. Stouffer 22 Hemodynamics of left ventricular assist device implantation, Brett C. Sheridan and Jason N. Coronary hemodynamics 23 Coronary hemodynamics, David P. Wu and George A. Stouffer 24 Fractional flow reserve, Paul M. Miscellaneous 25 Right ventricular myocardial infarction, Robert V. Stouffer 26 Pulmonary hypertension, Lisa J. Selzman, Lukas Jantac and George A. Stouffer 28 Systematic evaluation of hemodynamic tracings, George A. Stouffer Index, Verlagsort.

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As aortic valve disease progresses and the valve orifice narrows, resistance to blood flow increases. In order to maintain stroke volume, the velocity of blood exiting the LV increases and a pressure gradient develops from the LV to the aorta Figure 8. This adaptation allows the LV to generate the necessary pressure to maintain cardiac output, but can lead to abnormalities in diastolic LV function, coronary perfusion, and eventually LV systolic dysfunction. Progressive increase in the pressure gradient across the aortic valve and cardiac maladaptation explain the stages of hemodynamic findings that patients go through as AS progresses. In mild AS, intracardiac pressures and cardiac output will appear normal. As the valve becomes more stenotic, the patient may have normal hemodynamic findings at rest, but may be unable to increase cardiac output during exercise. Progressive narrowing of the valve leads to decreased stroke volume and cardiac output, even at rest. In a minority of patients LV systolic failure also occurs, which may lead to further elevation in intracardiac pressures. It is important to remember that the pressure gradient across the aortic valve increases exponentially not linearly with decreasing aortic valve area. Thus, in patients with severe AS, small changes in aortic valve area can lead to large changes in hemodynamics Figure 8. Adaptive mechanisms of the LV and circulation allow most patients to remain asymptomatic until advanced narrowing of the aortic valve orifice occurs. The prognosis of patients with asymptomatic severe AS is good, with a sudden death rate of less than 0. The onset of symptoms, however, heralds a marked change in the natural history of AS, with poor outcomes unless the patient undergoes aortic valve replacement. The classic clinical presentation of severe AS is typically an insidious onset of any of the triad of effort angina, exertional syncope, or dyspnea [1]. The average survival in untreated patients with severe AS is classically thought to be 2, 3, or 5 years after the onset of heart failure, syncope, or chest pain, respectively. Importantly, many patients report being asymptomatic, although careful questioning reveals that they have gradually decreased their level of activity and would, in fact, be symptomatic at their previous level of exertion. Extensive questioning, often involving a family member, is imperative. Physical exam The physical examination of patients with AS can often be helpful in predicting which patients have hemodynamically severe disease. A low volume, late onset arterial pulse, referred to as *parvus et tardus*, is often present in severe AS. This finding can be appreciated on palpation of the carotid upstroke and radial artery. It may not be manifest in the elderly owing to a noncompliant vasculature. Other physical findings include a systolic murmur that is *crescendo-decrescendo* in intensity. The duration will vary with the severity of disease, but the murmur always begins after S1 and ends prior to S2. The murmur is generally heard best in the right second intercostal space and can radiate to the carotid arteries. A diminished aortic component of the second heart sound is not uncommon and the second heart sound can also split paradoxically in severe AS delayed aortic component with narrowing of the splitting with inspiration. Flow through a stenotic AV is well approximated by flow through a convergent orifice e . The point of maximum velocity is termed the vena contracta VC of the jet and the area of the flow jet at the VC is known as the effective orifice area EOA. Doppler evaluation enables the noninvasive measurement of blood flow velocity with estimation of aortic valve gradient and valve area. The LV aortic pressure gradient is estimated by measuring blood flow velocity across the aortic valve and then using the Bernoulli equation to determine pressure gradient. Note the calcified aortic valve and left ventricular hypertrophy. A few additional caveats: Mean transaortic pressure gradient is the average difference in pressure between the LV and aorta during systole. Peak velocity and mean gradient provide independent information regarding AS severity, with the relationship depending on the shape of the velocity curve. The mean gradient is calculated by averaging the instantaneous gradients over the ejection period followed by calculation of pressure from velocity using a simplification of the Bernoulli equation: Maximum velocity and mean pressure gradients across a stenotic

aortic valve are both flow dependent. In contrast, aortic valve area is independent of the conditions in which it is measured at least in theory. When using echocardiography, aortic valve area is calculated based on the continuity equation, which was derived based on the assumption that the stroke volume flowing through the LVOT and the stenotic aortic valve are the same Figure 8. There are several important caveats to keep in mind: Despite these limitations, continuity equation valve area calculations have been well validated [2,3]. A simplified continuity equation can be used, although there tends to be more variability when using maximum velocities rather than velocity time integral VTI. The simplified equation assumes that the shape of the velocity curve in the outflow tract and aorta are similar, so that the ratio of LVOT VTI to aortic valve VTI is identical to the ratio of the LVOT maximum velocity to aortic valve maximum velocity:

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Chapter 3 : 8: Aortic stenosis | Thoracic Key

David P. McLaughlin and George A. Stouffer The basic function of the aortic valve is to separate the aorta from the left ventricle cavity during diastole. At peak left ventricular (LV) ejection the aortic valve opens briskly to an average area of 4 cm².

Descrizione Cardiovascular Hemodynamics for the Clinician, 2nd Edition, provides a useful, succinct and understandable guide to the practical application of hemodynamics in clinical medicine for all trainees and clinicians in the field. Basics of hemodynamics 1 Introduction to basic hemodynamic principles, 3 James E. Faber and George A. Stouffer 4 Arterial pressure, 56 George A. Stouffer 5 The atrial waveform, 69 David P. McLaughlin and George A. Stouffer 6 Cardiac output, 82 Frederick M. Costello and George A. Stouffer 7 Detection, localization, and quantification of intracardiac shunts, 91 Frederick M. Valvular heart disease 8 Aortic stenosis, David P. Stouffer 9 Hemodynamics of transcatheter and surgical aortic valve replacement, John P. Vavalle, Michael Yeung, Thomas G. Caranasos and Cassandra J. Ramm 10 Mitral stenosis, Robert V. Kelly, Chadwick Huggins and George A. Stouffer 11 Aortic regurgitation, George A. Stouffer 12 Mitral regurgitation, Robert V. Cohen and George A. Stouffer 13 The tricuspid valve, David A. Tate and George A. Cardiomyopathies 15 Hypertrophic cardiomyopathy, Jayadeep S. Varanasi and George A. Stouffer 16 Heart failure, Geoffrey T. Jao, Steven Filby and Patricia P. Chang 17 Restrictive cardiomyopathy, David P. Pericardial disease 18 Constrictive pericarditis, David P. Stouffer 19 Cardiac tamponade, Siva B. Mohan and George A. Stouffer 20 Effusive constrictive pericarditis, Eric M. Smith and George A. Hemodynamic support 21 Hemodynamics of intra aortic balloon counterpulsation, Richard A. Santa Cruz and George A. Stouffer 22 Hemodynamics of left ventricular assist device implantation, Brett C. Sheridan and Jason N. Coronary hemodynamics 23 Coronary hemodynamics, David P. Wu and George A. Stouffer 24 Fractional flow reserve, Paul M. Miscellaneous 25 Right ventricular myocardial infarction, Robert V. Stouffer 26 Pulmonary hypertension, Lisa J. Selzman, Lukas Jantac and George A. Stouffer 28 Systematic evaluation of hemodynamic tracings, George A. Stouffer Index, Catalogo.

Chapter 4 : April | | Thoracic Key

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David P. McLaughlin MD, Samuel calendrierdelascience.com MD, George A. Stouffer MD (University of North Carolina). 22 Hemodynamics of intra-aortic balloon counterpulsation. Richard A. Santa-Cruz MD (Interventional Cardiology).

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Chapter 8 : Table of contents for Cardiovascular hemodynamics for the clinician

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George A. Stouffer, MD, FACC, FAHA is Chief of Cardiology and the Henry A. Foscue Distinguished Professor of Medicine at the University of North calendrierdelascience.com served as Director of the Cardiac Catheterization Laboratories and Director of Interventional Cardiology for 14 years before assuming his current role.