

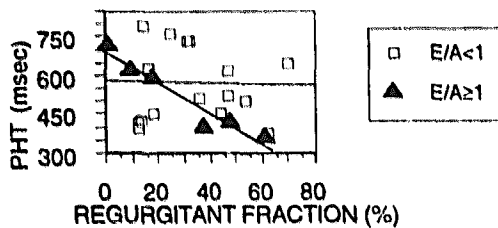
1094-19 Influence of Left Ventricular Relaxation on Pressure Half Time of Aortic Regurgitation Flow Velocity Spectrum

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Background: The severity of aortic regurgitation (AR) can be estimated using pressure half time (PHT) of the AR flow velocity, but the correlation between regurgitant fraction (RF) and PHT is weak. The purpose of this study was to test the hypothesis that the association between PHT and RF is substantially influenced by LV relaxation.

Methods: In 45 patients (age 57 ± 15) with pure AR, RF was calculated from the difference of LV and RV stroke volumes. Diastolic function was assessed using calculation of LV relaxation constant τ from the upstroke of digitized AR Doppler velocity curves, the E/A-ratio of the Doppler tissue velocity curves of the mitral annulus and the E/A-ratio of mitral inflow.

Results: There was a weak overall correlation between RF and PHT (regression equation: $PHT = 619 - 2.3 \times RF$; $r = 0.32$; $p = 0.10$). Correlation between E/A-ratio of mitral annulus velocity and E/A-ratio of mitral inflow was good ($r = 0.65$, $p < 0.0001$). In patients with a mitral annulus velocity E/A-ratio ≥ 1 a strong correlation between RF and PHT was found ($PHT = 687 - 6.0 \times RF$, $r = 0.96$, $p = 0.002$, see figure), whereas in patients with E/A-ratio < 1 (impaired LV relaxation), no correlation was found ($PHT = 582 - 0.3 \times RF$, $r = 0.04$, $p = 0.87$). Similarly, RF and PHT did not correlate in patients with $\tau \geq 70$ ms (impaired LV relaxation) ($PHT = 533 - 0.6 \times RF$, $r = 0.09$, $p = 0.81$) but did better in patients with $\tau < 70$ ($PHT = 641 - 3.3 \times RF$, $r = 0.40$, $p = 0.10$).



Conclusions: Only a normal LV relaxation allows a faster decay of PHT with increasing AR severity. In abnormal relaxation, there is a wide variety of prolonged LV filling resulting in variable slopes of the PHT-RF relation, and thus making PHT unsuitable for AR assessment.

1094-20 The Use of the Pressure Drop-Flow Slope as a Measure of Severity in Patients With Aortic Stenosis

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Pressure drop is dependent on flow in patients with aortic stenosis (AS) and there is some uncertainty about flow correction formulae. Pressure drop-flow slopes avoid the need for such formulae, but have not been previously validated in vivo. We therefore compared pressure drop-flow slopes and continuity equation effective orifice area (EOA) during dobutamine infusion in 46 patients with AS (29 male, mean age 65 (12) yrs) - 18 with mild AS (EOA 1.0-1.7 cm²), 12 with moderate AS (EOA 0.76-0.95 cm²) and 16 with severe AS (EOA 0.44-0.75 cm²).

Methods: Dobutamine was infused from 5 μ g/kg/min to 40 μ g/kg/min in 5 minute stages. Doppler estimation of mean pressure drop, EOA and flow, calculated from the stroke volume and ejection time were performed at each stage. There was a linear relationship between pressure-drop and flow in individual patients and the slope was calculated in each.

Results: There was a significant correlation between baseline EOA and slope ($R = 0.6$, $p < 0.0001$). EOA increased significantly with flow (mean resting EOA 0.92 cm² - peak EOA 1.2 cm², $p < 0.0001$). The individual slopes were steeper with increasing severity of AS - the mean slope in mild AS was 0.064 (95%CI 0.05-0.078), in moderate AS was 0.124 (0.88-0.16) and in severe AS was 0.183 (0.122-0.244), ($p = 0.0003$ by ANOVA).

Conclusion: Pressure drop is directly related to flow in individual patients with aortic stenosis. The slope of the relationship correlates well with effective orifice area. The pressure-drop flow slope is a promising new method of assessing haemodynamic severity in aortic stenosis.

1094-21 Doppler Echocardiography Can Predict the Magnitude of Pressure Recovery in Patients With Aortic Valve Disease

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Aortic valve pressure gradients (ΔP) calculated by Doppler echocardiography (E) can exceed those recorded by cardiac catheterization (C). One possible cause for this difference is the effect of pressure recovery (PR). To quantify the magnitude of PR, ΔP was simultaneously measured across the aortic valve by both E and C in 13 patients with aortic valve disease (valve area: 0.99 ± 0.65 cm²; mean ΔP : 62.5 ± 23.0 mm Hg). PR was measured as follows: ΔP was recorded using a double-sensor micromanometer catheter. The proximal sensor was initially positioned in left ventricle and was then withdrawn through the aortic valve into the ascending aorta. ΔP was recorded at the aortic valve (maximal ΔP) and at multiple sites above the valve until complete pressure recovery was noted. PR was predicted from Doppler echocardiography data by the formula:

$$PR = 8V^2(AVA/AOA)[1 - (AVA/AOA)] \quad (1)$$

where V is the aortic valve velocity and AVA and AOA are the aortic valve and aortic root areas respectively. Pressure recovery was observed in 12/13 patients. Peak PR averaged 10.9 ± 8.4 mm Hg (range: 0-27.9), and mean PR was 6.0 ± 4.3 mm Hg (range: 0-15.8). There was excellent agreement between predicted and measured values for peak PR (M = 2.4 ± 3.0 mm Hg) and mean PR (M = 0.5 ± 1.5 mm Hg) (both P = Ns). Mean PR by E significantly correlated with PR by C ($R = 0.56$, $P < 0.05$).

We Conclude: The magnitude of pressure recovery can be calculated by Doppler echocardiography. Correcting E-derived ΔP for PR can reduce the discrepancy between C and E pressure gradient measurements.

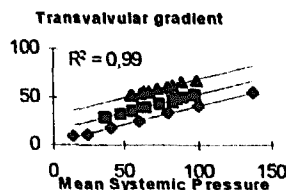
1094-22 Pressure-dependence of the Aortic Valve Gradient

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Background: Transvalvular gradient is described as flow-dependent; a pressure-dependence of gradient, irrespective of flow, has never been demonstrated.

Methods: We used the Sheffield Pulse Duplicator equipped with a X-Cell 21 porcine valve mounted in aortic position. We measured transaortic gradient at constant rate of 80 b/min, while flow was kept at 2, 5, and 8 l/min, and systemic pressure was increased from 50 to 200 mmHg by setting peripheral resistances manually. Valve area was computed with Gorlin's formula.

Results: For each flow, transvalvular gradient increases linearly with pressure (fig 1: $\diamond =$ Flow 2 l/min, $\square =$ flow 5 l/min, $\triangle =$ 8 l/min), and computed area decreases ($y = 0.0042x + 0.975$; $R^2 = 0.98$). The slope of pressure-gradient relation is independent of flow. The intercept on y axis was 2.3 mmHg, 13.5 mmHg and 30.5 mmHg for flow = 2 l/min, 5 l/min and 8 l/min respectively.



Conclusion: Transaortic gradient depends not only on flow, but shows also a pressure-dependence, that should be taken into account when evaluating aortic stenosis, especially in hypertensive and hypotensive states.

1095 Noninvasive Assessment of Congenital Heart Disease

Tuesday, March 31, 1998, 9:00 a.m.-11:00 a.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 9:00 a.m.-10:00 a.m.

1095-154 Enhanced Size and Shape Analysis of Aortic Coarctation by Three-dimensional Imaging: An In-Vitro Study Using Volume Rendered Intravascular Echo

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Background: Accurate delineation of the size and shape of luminal narrowing