QUANTITATIVE ASSESSMENT OF AORTIC AND MITRAL STENOSIS BY MAGNETIC RESONANCE VELOCITY MAPPING

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The aim of the study was to compare magnetic resonance (MR) velocity mapping with Doppler echocardiography for the assessment of jet velocities through stenotic aortic and mitral valves.

We have studied 6 patients with aortic stenosis (3M,age 52-79) and 7 patients with mitral stenosis (7F,age 31-67, 2 in atrial fibrillation). We used a Picker 0.5T machine and a cine field even echo rephasing (FEER) sequence with echo times (TE) of 3,6,6 and 14ms. The stenotic jet was identified from the amplitude display by virtue of the signal loss associated with turbulence using the longer TE. Cine velocity mapping was performed using the shorter TE to measure jet velocities throughout the cardiac cycle. The imaging plane was oriented both parallel and perpendicular to the jet, and the maximum velocity in either plane was chosen. The results were compared with Doppler echocardiography. MR velocity maps clearly showed the shape and direction of the jet and peak velocity measurements agreed closely with those obtained by Doppler.

Mean (SD) peak velocity (m/s)

Magnetic Resonance	Doppler	Mean differences
2.6 (1.4)	2.8 (1.2)	0.1 (0.4)

Conclusion
The FEER sequence with short TE has allowed velocities in stenotic jets to be measured. The results agree closely with Doppler. Our previous studies have shown that MR has the advantage of greater accuracy,a wider dynamic range, and the lack of restriction to echocardiographic windows.

LIMITATIONS OF THE SIMPLIFIED FORMULA FOR CALCULATING VALVE AREA IN PATIENTS WITH MITRAL STENOSIS.

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In patients (pts) with mitral stenosis (MS), the mitral valve area is traditionally calculated with the Gorlin equation. A simplified formula:

$$\text{valve area } (cm^2) = \frac{\text{cardiac output } (l/min)}{[\text{pressure gradient } (mmHg)]^{\frac{1}{2}}}$$

has been proposed as a substitute for the Gorlin equation in determining valve area in pts with MS. This study was done to compare the results of this simplified formula with those of the Gorlin equation. In 96 pts (20 men, 76 women, aged 17-75 yrs) with MS, the mean transvalvular pressure gradient was determined by the simultaneous measurement of LV and LA pressures (n=31) or LV and PCW pressures (n=65), and cardiac output was determined by the Fick method. A substantial difference between the results of the simplified formula and those of the Gorlin equation (> 0.2 cm²) was noted in 43 pts (45%): in 33, the valve area determined by the simplified formula was greater than that determined by the Gorlin equation, and in the other 10 it was substantially less. A disparity between the results of the simplified formula and those of the Gorlin equation occurred with similar frequency in those with sinus rhythm (n=58) and those with atrial fibrillation (n=38). A difference of greater than 0.2 cm² was noted in 33 of 80 pts (41%) with a heart rate below 100 b/min but in 10 of 16 pts (63%) with a heart rate greater than 100 b/min. Thus, in patients with mitral stenosis, the simplified formula and the Gorlin equation for determining mitral valve area yield disparate results in almost half the patients. Such a disparity is particularly common in patients with tachycardia.

Phasic Characteristics of Transaortic Pressure Gradients in Valvular Aortic Stenosis

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Phasic characteristics and differences between obstructive and nonobstructive transaortic pressure gradients (ΔP's) may be helpful in analyzing LV systolic load dynamics in aortic stenosis (AS). To study such differences, we measured ΔP by solid-state multisensor catheter in 5 control (NL) subjects with normal LV function and 13 AS pts (aortic valve area: 0.7 ± 0.2 cm²) at rest (R) and exercise (E). Ensemble average (≥ 25 beats) data were analysed in time and frequency domains. Results follow:

	$\widehat{\Delta P}$ mmHg	$T_{\widehat{\Delta P}}$ ms	$T_{\Delta P}$ ms	TR%	VAR1%
NL-R	9 ± 2	32 ± 16	193 ± 69	17 ± 6	64 ± 23
AS-R	85 ± 28	126 ± 29	316 ± 40	39 ± 6	90 ± 2**
NL-E	12 ± 3	26 ± 16	161 ± 66	21 ± 20	75 ± 9
AS-E	89 ± 16	111 ± 22	298 ± 26	37 ± 5*	85 ± 2**

All $p < 0.001$ except *$p = 0.05$, **$p = 0.02$ (AS vs. NL);
$\frown$: peak value; $T_{\widehat{\Delta P}}$: time to $\widehat{\Delta P}$; $T_{\Delta P}$: time of positive ΔP; TR: ratio of $T_{\widehat{\Delta P}}/T_{\Delta P}$; VAR1: fraction of total variance of $\Delta P(t)$ curve accounted for by its 1st Fourier term.

Obstructive ΔP's peaked late and were symmetric and rounded, in contrast to nonobstructive ΔP's.

Conclusion:
Important phasic differences distinguish ejection ΔP waveforms in AS from NL. Symmetric, rounded ΔP's are equally as distinctive a hemodynamic sign of AS as the increased gradient magnitudes. This should be recognized in analyses of systolic LV load dynamics.

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Author Present: 10:00AM–11:00AM
Hall F, West Concourse
Acute Myocardial Infarction

DIFFERENT CORONARY ANGIOGRAPHIC FINDINGS IN UNHERALDED ACUTE MYOCARDIAL INFARCTION AND UNCOMPLICATED CHRONIC STABLE ANGINA

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To test whether angiographic findings and risk factor profile (RFP) differ in acute and chronic coronary artery disease (CAD), we identified 102 consecutive patients presenting with either: 1) Acute myocardial infarction (AMI) as first manifestation of CAD with a concomitant angiogram (55 pts) or 2) Stable angina (SA) for at least 2 years with a positive exercise test, no history of acute events, no ECG Q waves or akinesia on ventriculography, and angiography performed ≥2 years from initial symptoms (47 pts). Observers, blinded to all clinical data including ventriculography, evaluated these coronary angiograms for: 1) severity (number of vessel disease, stenoses ≥ 50%, occlusions); 2) extent (an index derived by assigning a score of 0-3 per segment depending on the extent of surface irregularity and dividing the sum by the number of visualized segments); 3) pattern (discrete = 0-3 loci of disease never affecting >50% of the length of any segment, or diffuse = anything exceeding this). The findings (mean±SD) were:

	Vessel Disease	Stenoses	Occlusions	Extent Index	Discrete Pattern
AMI	1.3±.8	2.1±1.8	0.6±0.6	0.6±0.5	54.6%
SA	2.1±.8*	3.9±1.8*	1.0±0.9**	1.2±0.5*	8.5%*

* p<0.001 ** p=0.02
Age at symptom onset, sex distribution, serum cholesterol, present and past smoking history combined, and positive family history were similar in the two groups. Hypertension was found in 64.4% of SA and 30.9% of AMI (p<0.01), a present smoking history in 60% of AMI and 31.1% of SA (p<0.01). There were 7 cases of diabetes in SA, one in AMI. Thus, SA is associated with markedly more severe and extensive atherosclerosis and rarely a discrete pattern and a different RFP, compared to AMI. This may reflect differing pathogenetic mechanisms in these two ischemic syndromes.