1143-28 Cardiovascular Response to Supplemental Oxygen in Patients With Coronary Artery Disease

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Background: There is recent evidence that supplemental oxygen (O_2) has deleterious hemodynamic effects (increase in systemic resistance (SVR) and wedge pressure) when given to normoxic patients with congestive heart failure. To address this issue in normoxic patients with coronary disease (CAD), a group of patients commonly given O_2, we evaluated the hemodynamic effects of O_2 in 11 patients with stable ischemic symptoms.

Methods: Patients with ≥70% lesions in the LAD or circumflex, EF >40%, and O_2 saturation >0.95 on room air were eligible. Left and right heart hemodynamics as well as myocardial blood flow (via coronary sinus thermodilution) were measured during sequential inhalation for 20 minutes of room air, 40%, and 100% O_2 by mask (n = 8), or room air by mask for three 20 minute periods (n = 3, controls). For all measured parameters, there was no significant difference between study periods in the control group.

	Room air	*40% O_2*	*100% O_2*
MVO_2 (ml/min)	8.49 ± 5.16	9.83 ± 5.12	8.50 ± 5.94
SVR (dyne sec cm^{-5})	1656 ± 547	1678 ± 528	1736 ± 554
LVEDP (mm Hg)*	22 ± 7	22 ± 6	25 ± 6
Cardiac Output (l/min)*	5.24 ± 1.33	5.06 ± 1.23	4.95 ± 1.17
CVR*	1.67 ± 0.77	1.42 ± 0.68	1.84 ± 0.87

*p < 0.10 by ANOVA. Coronary vascular resistance (CVR) = Mean aortic pressure-Mean right atrial pressure/coronary blood flow. LVEDP = left ventricular end diastolic pressure. MVO_2 = myocardial O_2 demand.

Conclusion: Supplemental O_2, even at low doses, does not appear to offer hemodynamic benefits and may even be deleterious in normoxic patients with CAD. In addition, we observed a biphasic dose-related response of MVO_2 and CVR to O_2 supplementation.

1144 The Calculation of Valvular Hemodynamics

Tuesday, March 31, 1998, 3:00 p.m.–5:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 3:00 p.m.–4:00 p.m.

1144-18 Is Orifice Area Ratio a Determinant of Left Ventricular Work in Aortic Valve Stenosis? An in Vitro Study

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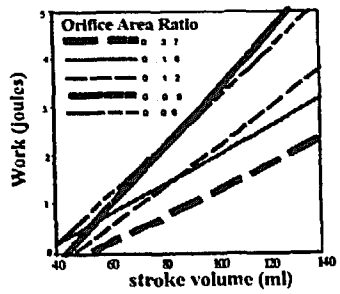
Background: The reason for clinical tendency towards hemodynamic decompensation at a critical value of aortic stenosis remains unclear. The purpose of this study was to determine if there was a fundamental fluid mechanical explanation for observed clinical complications.

Methods: Stenotic porcine bioprostheses with varying valve areas were studied in an in vitro model of human circulation (aortic annular area: 8.0 cm^2) under pulsatile flow conditions (flowrate: 2–8 l/min) at 60 bpm. Varying degrees of stenosis (3.0–0.5 cm^2, orifice area ratio: 0.37–0.09) were produced by suturing the porcine leaflets. The left ventricular (LV) work was calculated by applying conservation of energy principles to measured pressures and flowrates in the model.

Results: LV work increased significantly as orifice area changed from 3.0 to 1.0 to 0.5 cm^2 for all stroke volumes (SV) studied. There were statistically no differences in work for the 1.0 and 1.27 cm^2 (moderate stenosis) valves, and similarly between the 0.75 and 0.5 cm^2 (severe stenosis) valves. Work increased 55% as the valve area decreased from 3.0 to 1.0 cm^2, and by 70% from 1.0 to 0.75 cm^2 (SV: 80 ml), indicating exponential behavior in LV workload.



Conclusions: (i) LV work increases dramatically as the degree of aortic stenosis changes from mild to moderate to severe. (ii) Workload is better determined by orifice area ratio rather than valve area (iii) As the orifice area ratio reached the fluid mechanically established critical value (0.10) the LV work increased dramatically and could lead to hemodynamic decompensation.

1144-19 Development of a Correction Factor for Quantifying Stenotic Valve Area at Low Output States Using the Doppler Continuity Equation: In Vitro Validation

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Background: We have shown that the Doppler continuity equation (DCE) severely underestimates actual *vena contracta* area due to a breakdown of the flat velocity profile assumption at low (<65 cc/sec) flows. Modifying the continuity equation to include the true mean spatial cross-sectional velocity at the *vena contracta* should correct this underestimation.

Methods: We imaged the *vena contracta* region using digital color Doppler (DCD) and laser flow visualization (LFV) on an *in vitro* model of valvular stenosis. DCD imaging was used to extract cross-sectional velocities at the *vena contracta*, immediately downstream (2 mm) of the orifice. Steady (40 cc/sec–150 cc/sec) and pulsatile (30–80 cc/beat; 60–90 bpm) flows were directed through orifices of various shapes (circular, slit, Y-shaped) and sizes (0.8 cm^2–1.8 cm^2). DCD cross-sectional velocities were averaged across the *vena contracta* to obtain a mean spatial velocity, which was then used in DCE to obtain effective flow areas. For pulsatile flows, mean spatial velocities were integrated over the ejection cycle to produce a corrected velocity time integral.

Results: Use of peak velocities in DCE produced significant underestimation (mean = 43.1% ± 12.5%) of actual *vena contracta* areas at low flows (<2.4 L/min), but minimal underestimation at higher flows (mean = 7.4% ± 4.1%). The use of mean spatial velocities corrected the low flow underestimation, producing DCE areas that agreed well with *vena contracta* areas for steady ($y = 0.94x + 28.1$; $R = 0.95$; SEE = 8.4 cc/sec; mean error = 4.9% ± 2.0%) and pulsatile ($y = 0.89x + 7.2$; $R = 0.95$; SEE = 6.2 cc/beat; mean error = 9.1% ± 3.1%) flow states.

Conclusions: The use of a spatial mean velocity in the continuity equation corrects for Doppler underestimation of actual *vena contracta* areas at low flow states. This digital technique can be integrated into current ultrasound machines for on-line correction of Doppler areas in the clinical setting.

1144-20 The Proximal Isovelocity Surface Area Technique: When Is Flow Rate Not Equal to Surface Area Times Velocity?

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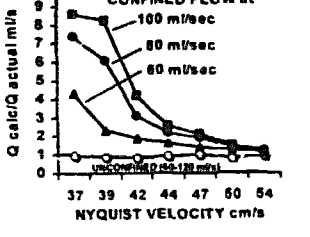
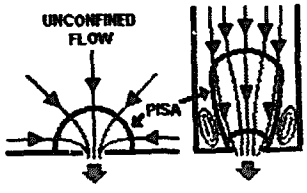
Recent techniques calculate regurgitant flow rate (Q) by conservation: Q entering a control volume proximal to the orifice must equal Q leaving via the orifice. *Flow is assumed to be perpendicular to the Doppler proximal isovelocity surface area (PISA)*, so that Q = surface area × the velocity (v) component perpendicular to the surface = area × v. This holds for unconfined flow approaching from all sides; confinement channels flow parallel to chamber walls, so that v is perpendicular only to the leading edge of the PISA, and





TUESDAY POSTER