Assessment of Cardiac Hemodynamics by Echocardiography

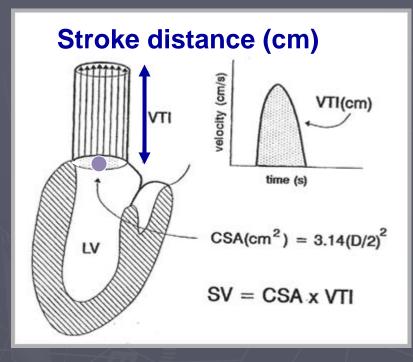
## M. Vaturi MD

Department of Cardiology Rabin Medical Center, Beilinson Hospital Sackler Faculty of Medicine Tel Aviv University Caesarea, 2010

Flow Rate, Volume Not constant (pulsatile flow) Flow rate = CSA x Flow velocity  $(cm^3/s)$   $(cm^2)$  (cm/s) $CSA = \pi \times r^2 = \pi \times (D/2)^2$ 

Volume = CSA x  $\int$  Velocity (time) VTI(cm<sup>3</sup>) (cm<sup>2</sup>) (cm)

# Stroke Volume - LVOT



 Assumptions:
 Circular orifice
 Constant orifice size
 Flat velocity profile
 Laminar flow
 Velocity & orifice measurements
 At the same level

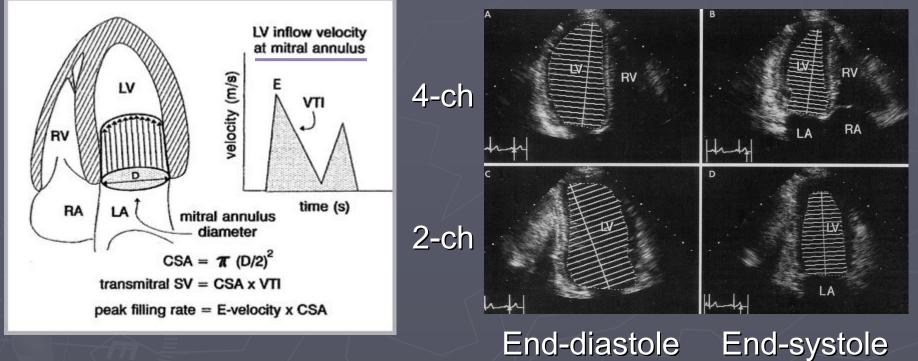
## Assessment of forward stroke volume

## ►MR → valid measurements of LV <u>forward</u> (systemic) SV

## ►AR → LV SV ≠ forward (systemic) SV

# SV – Other Techniques

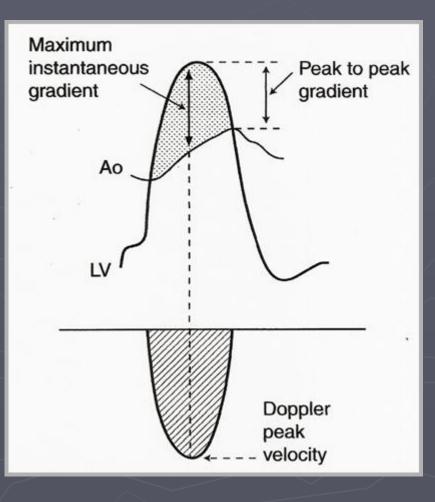
LV volumes



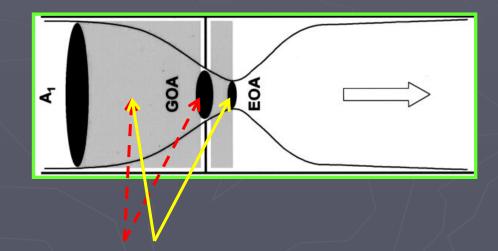
MR / AR → LV output ≠ forward (systemic) output
 LV SV = forward (systemic) SV + regurgitant volume (MR/AR)

# Aortic Stenosis

# Transvalvular Pressure Gradients



# Doppler does not measure AV pressure gradient directly



### $\Delta P$ Doppler = PLV-Pvena contracta

## $\Delta P$ – Aortic Stenosis

► ~ Constant relationship between peak & mean ΔP: AVMG ≈ 2/3 AVPG

Severe AS (AVA <1.0 cm<sup>2</sup>)

- Vmax > 4.0 m/s (peak ΔP >60 mmHg, mean >40)
- Assumption: normal transaortic flow (SV)

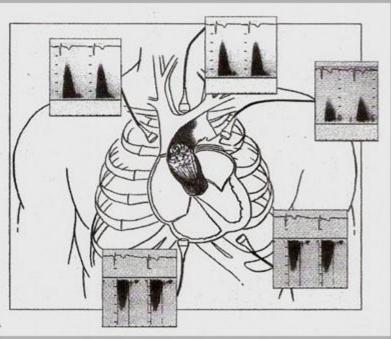
Incorrect assumption

- LV / RV dysfunction / MR / TR ++  $\rightarrow \Delta P \downarrow$ 
  - Low-gradient AS

► Hyperdynamic LV / AR ++  $\rightarrow \Delta P \uparrow$ 

# $\Delta P AS - Pitfalls$

## Inadequate Doppler alignment

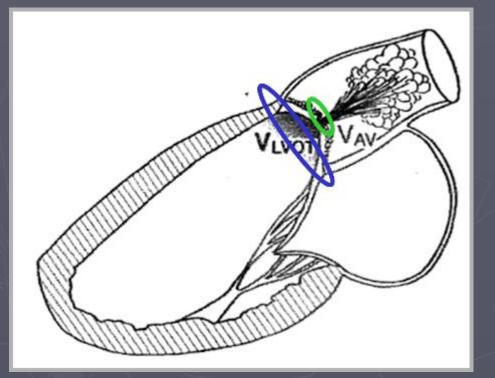


Serial stenoses
 Subaortic / mid-LV & aortic

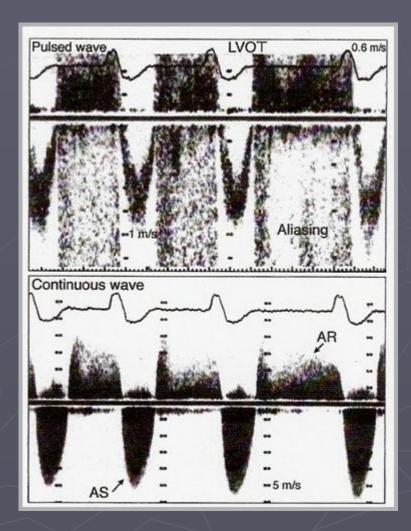
## When might pressure gradient calculation by the Bernoulli equation fall?

- Underestimation of poststenotic flow velocity v<sub>2</sub> (suboptimal angle to flow, calcified valve)
- Tunnel shaped muscular subaortic stenosis (viscous friction not negligible - underestimation)
- Prestenotic flow velocity v<sub>1</sub> not negligible (severe AR, high CO) or close to v<sub>2</sub> (prostheses) overestimation
- Pressure recovery (tapered outlet geometry, e.g. bileaflet prostheses) - overestimation

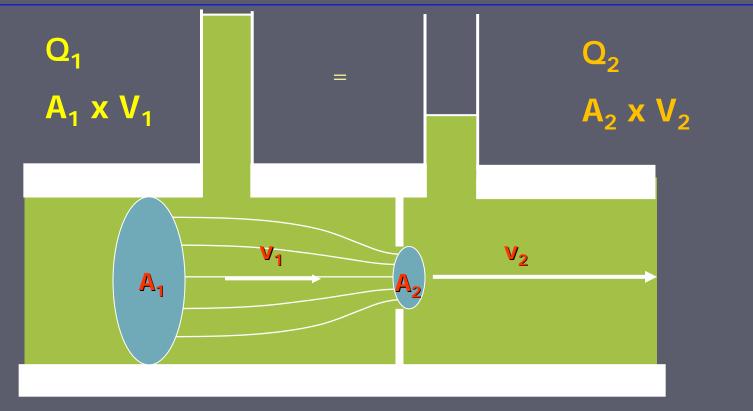
# **AVA – Continuity Equation**



Conservation of mass



# What causes the pressure gradient across a stenotic valve?

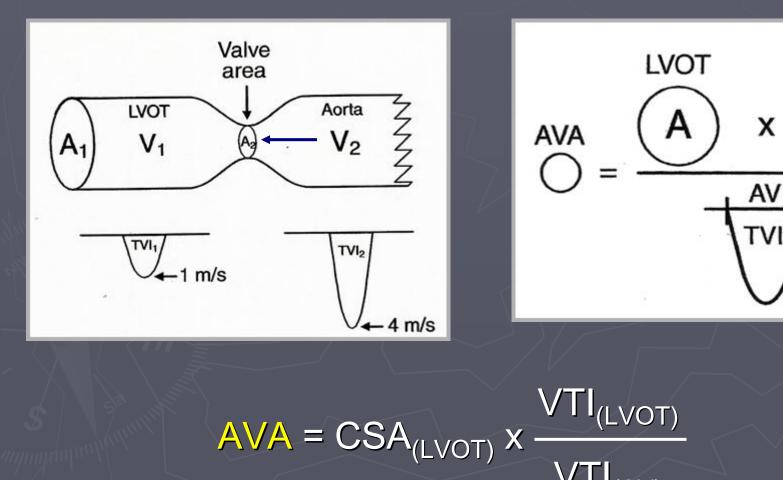


Fluids are incompressible. By continuity (conservation of mass) flow rate Q must therefore be the same through any cross section A of the flow path: Q1 = Q2. Since  $Q = A \times v$ , it follows that if A decreases, v must increase.

# **AVA – Continuity Equation**

LVOT

τvi



Continuity Equation – Pitfalls Inadequate PW sample location in LVOT Too close (to AoV)  $\rightarrow$  AVA  $\uparrow$ Too far (within LV)  $\rightarrow$  AVA  $\downarrow$  $AVA = CSA_{(LVOT)} \times \frac{V\Pi_{(LVOT)}}{V}$ VTI<sub>(AS)</sub> Usually underestimated Inadequate alignment  $(\rightarrow AVA \downarrow)$ with max flow velocity Mistake<sup>2</sup>



European Heart Journal (2008) **29**, 2526–2535 doi:10.1093/eurheartj/ehn022

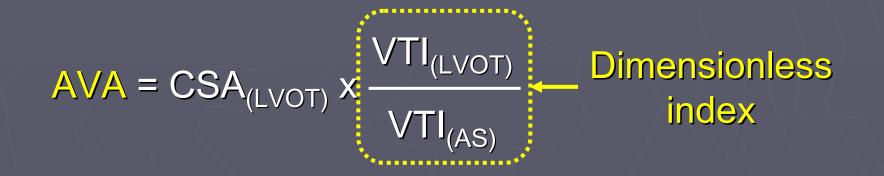
#### Assessing aortic valve area in aortic stenosis by continuity equation: a novel approach using real-time three-dimensional echocardiography

Kian Keong Poh<sup>1,2</sup>, Robert A. Levine<sup>1</sup>, Jorge Solis<sup>1</sup>, Liang Shen<sup>3</sup>, Mary Flaherty<sup>1</sup>, Yue-Jian Kang<sup>1</sup>, J. Luis Guerrero, and Judy Hung<sup>1\*</sup>



- AVA derived from 2D continuity equation correlates only modestly with that derived from 3D color Doppler
- Significant discrepancies between both methods are predicted by presence of <u>upper septal</u> <u>hypertrophy</u>, representing distorted LVOT geometry.
- RT3DE measurement of LVOT SV agrees better with the gold standard of aortic flow probe measurement in an animal model of varying LVOT geometry than 2DE.
- A better agreement of AVA derived from RT3DE color Doppler and AVA planimetry guided by RT3DE

# **Dimensionless Index**



 Index
 AS severity

 > > 1/2.5
 Mild

 1/2.5-1/4
 Moderate

 < 1/4</td>
 Severe

 (< 1/5</td>
 Critical)

Shortcut

Calculate with peak velocities

VTI ratio ≈ Peak V ratio

Journal of the American College of Cardiology © 2003 by the American College of Cardiology Foundation Published by Elsevier Science Inc. Vol. 41, No. 3, 2003 ISSN 0735-1097/03/\$30.00 doi:10.1016/S0735-1097(02)02764-X

#### **Cardiac Ultrasound**

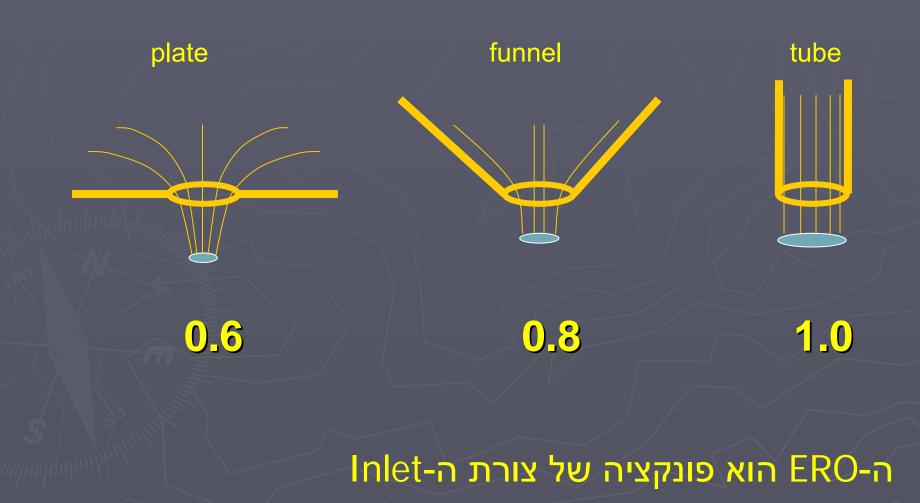
#### Discrepancies Between Catheter and Doppler Estimates of Valve Effective Orifice Area Can Be Predicted From the Pressure Recovery Phenomenon

Practical Implications With Regard to Quantification of Aortic Stenosis Severity

Damien Garcia, ENG,\* Jean G. Dumesnil, MD, FACC,† Louis-Gilles Durand, ENG, PHD,\* Lyes Kadem, ENG,† Philippe Pibarot, DVM, PHD, FACC\*†

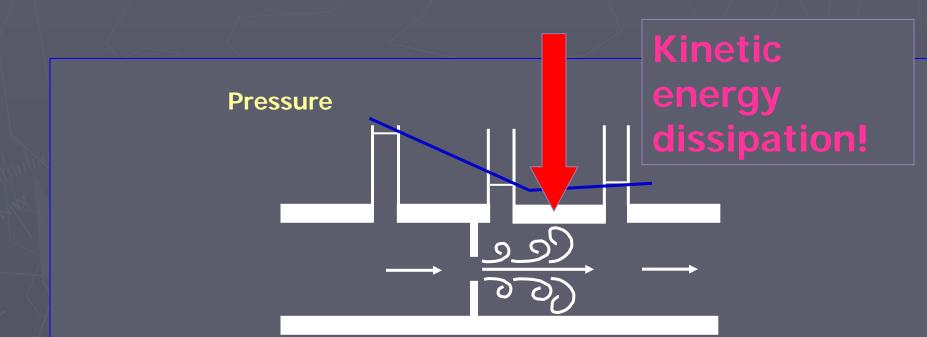
Sainte-Foy and Montreal, Quebec, Canada

### **Coefficient of contraction**



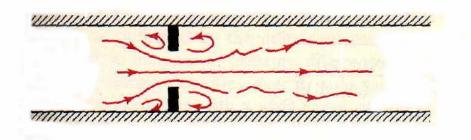
# Pressure Recovery

# Pressure gradient across a stenotic valve - energy dissipation



Most of the kinetic energy is dissipated in turbulence and is therefore not reconverted into static pressure

# Three Types of Pipe Flowrate Meters (obstruction type flowmeters)



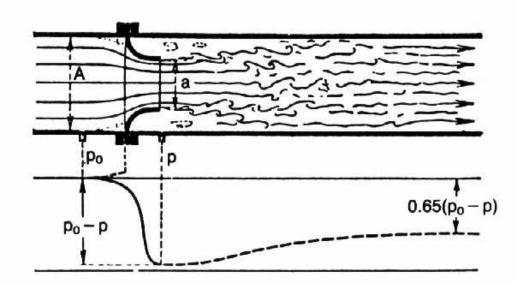
mmmmmm

## **Orifice Meter**

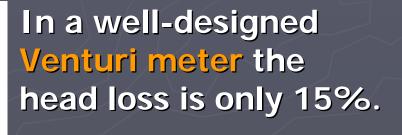
### **Nozzle Meter**

## **Venturi Meter**

Munson, Young, Okiishi: Fundamentals of Fluid Mechanics, New York 1990

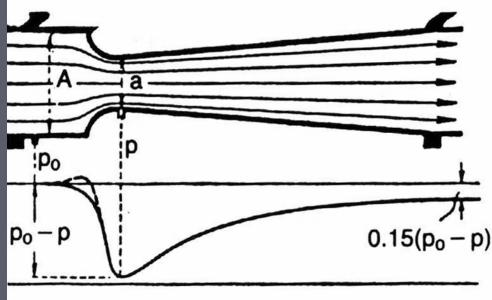


In a nozzle meter (e.g. valve prosthesis) there is a head loss of 65%.

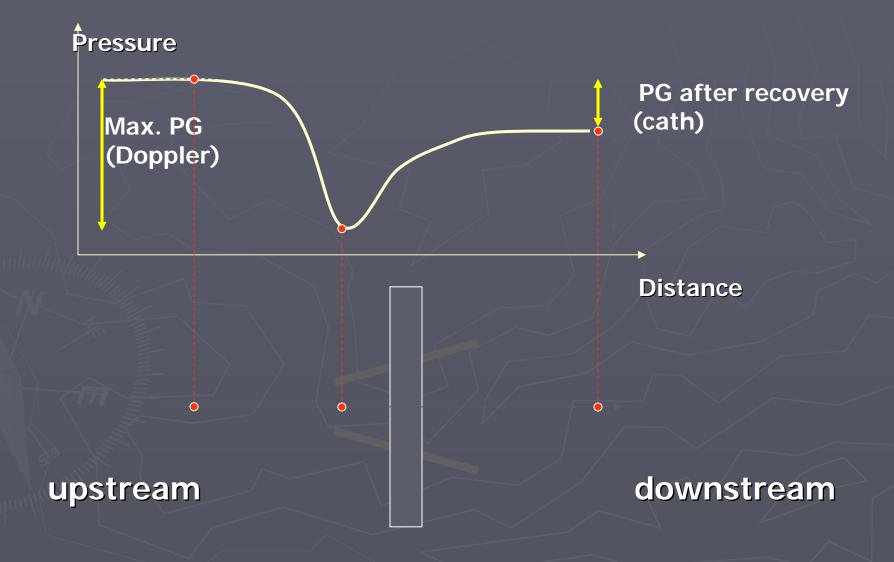


85% of the pressure drop is recovered !

*Prandtl L, Tietjens O: Applied Hydro- and Aeromechanics, New York, Dover 1957* 



## Pressure recovery in bileaflet prosthesis (SJM type)



The lowest pressure (the greatest pressure drop) after the stenotic valve is at the vena contracta. From there forward the pressure rises (E kinetic drops, E potential rises. This is the pressure recovery.

If aorta is small, P recovers more steeply (than in wider aorta). Hence, in small aorta (big pressure recovery) the delta P (P LV-P asc Ao) is smaller (in pull back) compared to delta P in Doppler => greater discrepancy in measurement of pressure gradient.

$$ELCo = \frac{EOA_{Dop} \times A_A}{A_A - EOA_{Dop}} = \frac{Q}{50\sqrt{EL}}$$

## EL index = ELCo/BSA

#### EOA cath or EL index $\leq 0.55 - 0.6 \text{ cm}^2/\text{m}^2$ is indicative of severe AS

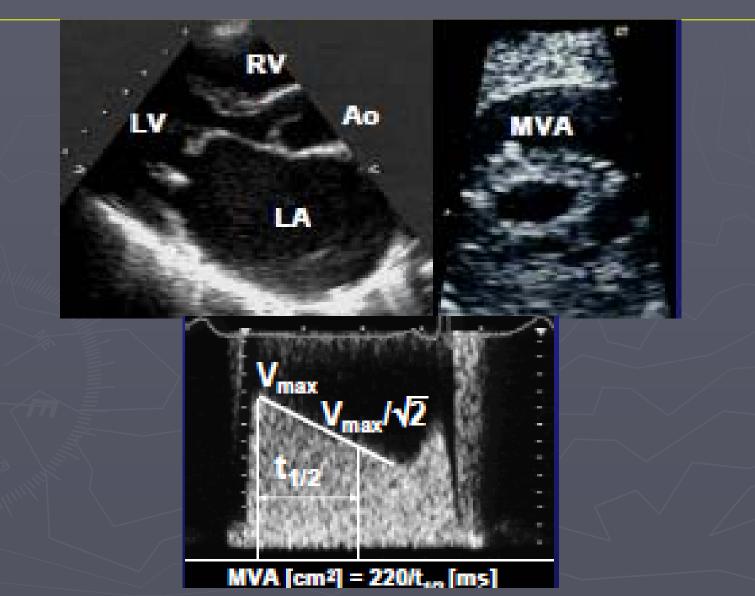
	Doppler-Derived EOA (cm <sup>2</sup> )		
Catheter-Derived EOA (cm <sup>2</sup> )†	Aortic Diameter = $2.0 \text{ cm}$ (A <sub>A</sub> = $3.14 \text{ cm}^2$ )	Aortic Diameter = $3.0 \text{ cm}$ (A <sub>A</sub> = $7.07 \text{ cm}^2$ )	Aortic Diameter = $4.0 \text{ cm}$ $(A_A = 12.0 \text{ cm}^2)$
1.50 (1.69)	1.02	1.24	1.34
1.00 (1.13)	0.76	0.88	0.93
0.75 (0.85)	0.61	0.68	0.71
0.50 (0.56)	0.43	0.47	0.48

Other factors (beside pressure recovery) can be associated with AVG – AVA dissociation (low gradients but severe valve area stenosis)

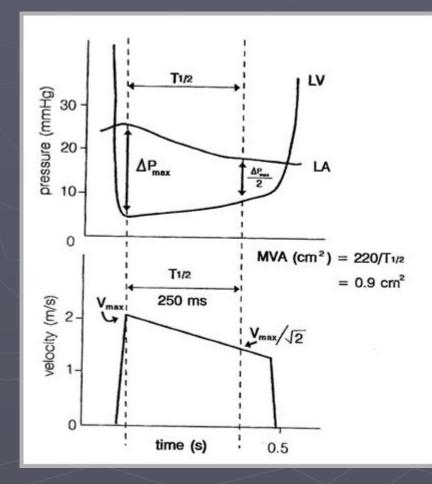
- Low cardiac output (LV dysfunction)
- Small stroke volume in LV with nEF
- Valvulo-arterial impedance (the impact of SBP)

# Mitral Stenosis



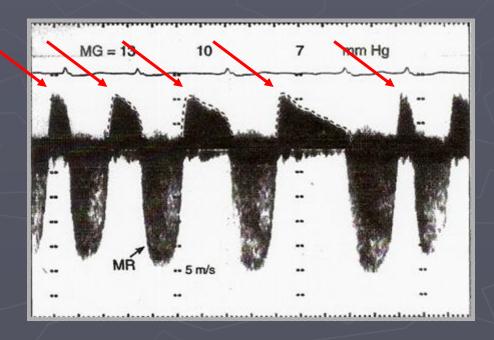


# **Transvalvular Pressure Gradients**

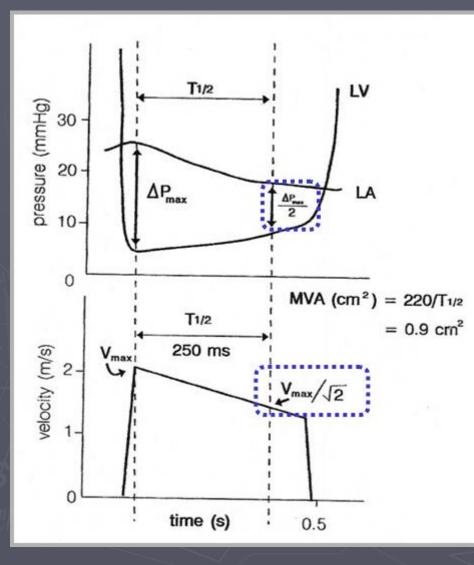


## $\Delta P$ – Mitral Stenosis

# Heart rate dependence + + + Non-constant (HR-dependent) relationship Peak ↔ mean ΔP



## MVA – Pressure Halftime



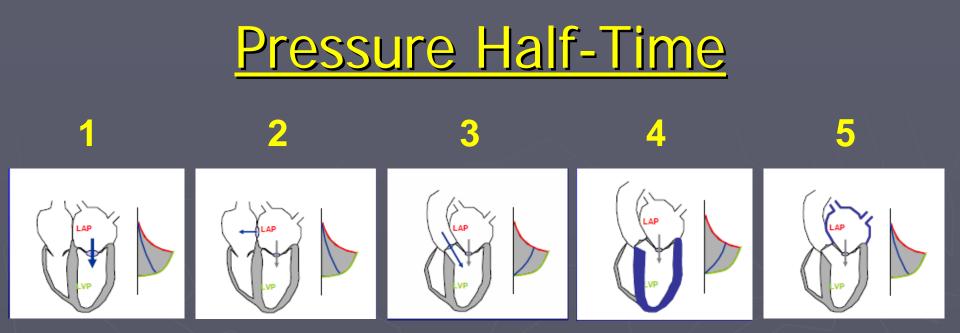
## $MVA = 220 / P t_{1/2}$

•  $P \propto V2$   $V \propto sqr(P)$ •  $P_{max} \rightarrow \frac{1}{2} P_{max}$  $V_{max} \rightarrow sqr(\frac{1}{2}) V_{max}$ 

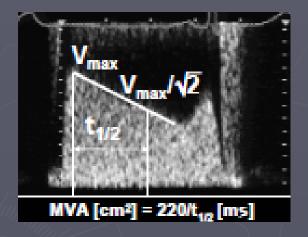
# MVA By P t<sub>1/2</sub> Rule of Thumb

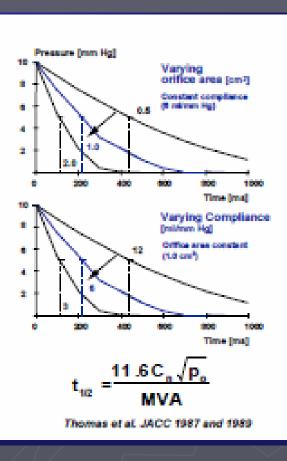
 $\triangleright$  P t<sub>1/2</sub> may be shorter than expected for specific MVA Presence of confounding LA / LV variables Overestimation of MVA (less severe MS) P t<sub>1/2</sub> "never" underestimates MVA\* • Calculated MVA  $\downarrow$  = severe MS - Calculated MVA > expected (morphology,  $\Delta P$ ) ► May still be severe MS !

\*Exception – Mild MS & LV relaxation  $\downarrow \downarrow$  (HHD)



- The larger the MVA, the more rapidly LAP drops, and diastolic LVP increases (equilibrium is reached quickly) - t1/2 will be shortened. However,
- 2. LAP may also drop rapidly if LA has a second outlet (ASD) t1/2 will be shortened.
- 3. Or LVP may rise rapidly if LV fills from a second source (AR) t1/2 will be shortened.
- 4. Or LVP may rise rapidly if LV is stiff (low ventricular compliance) t1/2 will be shortened.
- 5. Or LAP may drop rapidly if LA is stiff (low atrial compliance) t1/2 will be shortened.

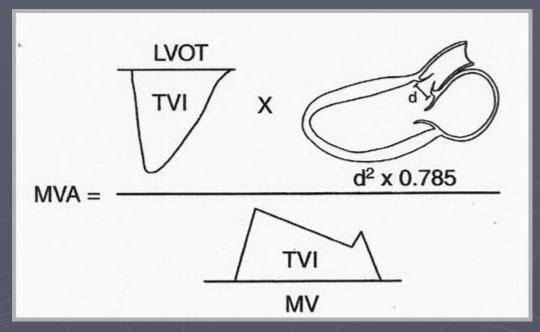




The decay of the pressure gradient across a stenotic mitral valve is not only determined by the size of the orifice but also by net AV compliance (Cn).

►T1/2 never underestimates MVA.  $\triangleright$  Therefore, if t1/2 is > 200-220 ms, MS is always severe.  $\triangleright$  However, if t1/2 is < 200 ms, look at the mean gradient, and pulmonary artery pressure, try mitral valve planimetry, and consider exercise echo

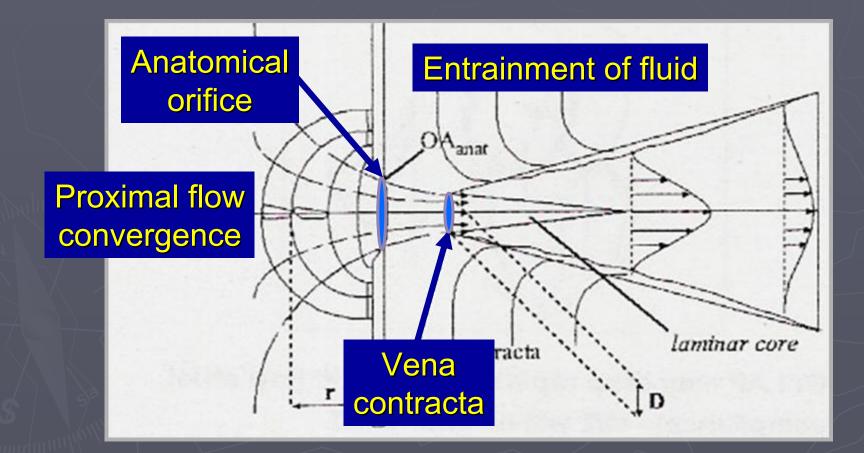
# MVA – Continuity Equation



Assumptions
 No MR (MR ≤ mild)
 No AR (AR ≤ mild)

# Mitral Regurgitation

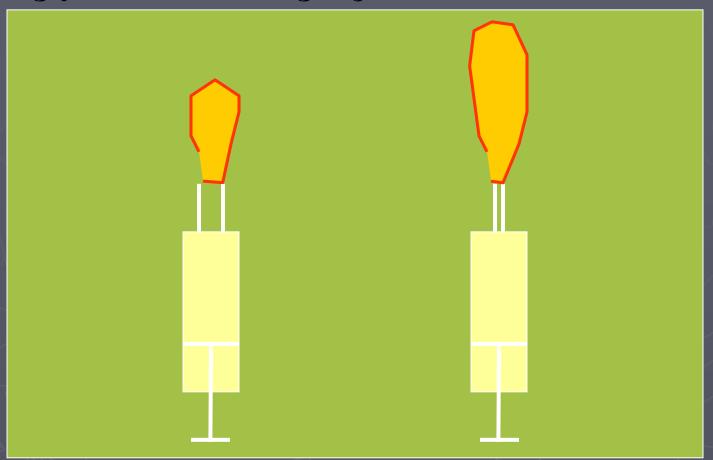
# Qualitative Assessment Color Flow Imaging



# Regurgitation jet depends on:

ERODriving pressure

If the same amount of fluid is injected with the same speed through a thinner needle (i.e. at a higher driving pressure) a larger jet results



This explains why regurgitant jets (of the same flow rate) are larger in patients with hypertension, aortic stenosis, HOCM

# <u>PISA – Formulas</u>

Flow @ PISA = flow @ regurgitant orifice Flow (t) = surface area x velocity (t)  $2\pi r^2 \times V_{aliasing} = ERO \times V_{regurg}$ 

Similar timing (mid-systole)

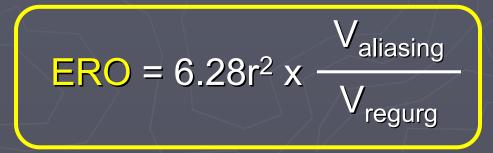
Hemispheric surface area

$$\frac{\text{ERO (cm^2)} = 6.28r^2 \text{ x} \frac{V_{\text{aliasing}}}{V_{\text{regurg}}}$$

Reg Vol (ml/beat) = ERO x VTI

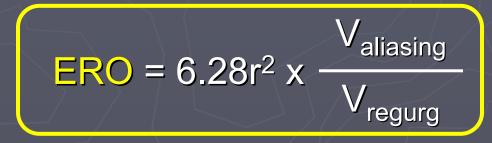
# <u>MR PISA – Shortcut I</u>

V<sub>aliasing</sub> → set @ 30 cm/s
 Assume V<sub>MR</sub> ~ 5 m/s (500 cm/s; 100 mmHg)
 If r ≥1 cm → ERO ≥ 0.4 cm<sup>2</sup> (severe MR)



# <u>MR PISA – Shortcut II</u>

•  $V_{aliasing} \rightarrow set @ 40 cm/s$ Assume  $V_{MR} \sim 5 m/s$  $\rightarrow ERO = r^2 / 2$ 



# PISA – Caveats

Multiple technical caveats Dynamic MR Timing of measurement e.g. – MVP, ischemic MR Multiple MR jets Angle correction Eccentric jets +++

# **MR** Severity

**RF (%) ERO** ( $cm^2$ ) RV (ml) ► Mild (I) < 0.2 < 30 < 30 ► Moderate (II) 30-45 0.2-0.3 30-40 ► Mod-severe (III) 0.3-0.4 45-60 40 - 50Severe (IV) >0.4 >50 >60

> Grades III & IV = surgical MR >0.3 >45

>40

# Aortic Regurgitation

# AR Severity (PISA / QD)

ERO (cm<sup>2</sup>) **RF** (%) RV (ml) ► Mild (I) < 30 < 0.1 < 30 ► Moderate (II) 30-45 0.1-0.2 30-40 ► Mod-severe (III) 0.2-0.3 45-60 40 - 50Severe (IV) > 0.3 > 60 >50

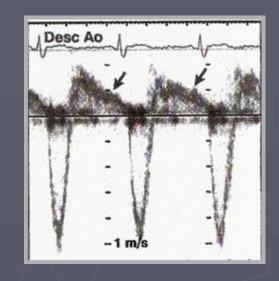
> 45

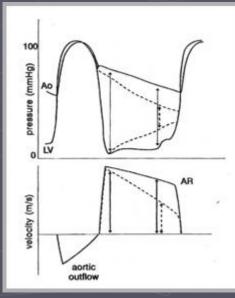
>40

Grades III & IV = surgical AR > 0.2

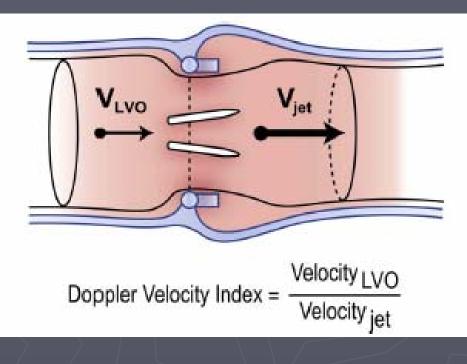
# Additional Doppler Findings - AR

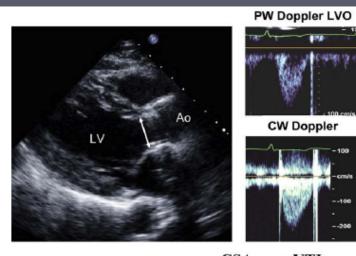
- Flow reversal descending aorta
- P t1/2 Multiple determinants
  - AR severity  $\uparrow \rightarrow P t1/2 \downarrow$
  - LV compliance ↓ → P t1/2 ↓
    SVR ↓ → P t1/2 ↓
- Do not confuse chronic with acute AR
- Cutoffs: >400 ms / 250-400 / <250 ms</li>





# Prosthetic Valves





VTI<sub>jet</sub>

#### Table 5 Doppler parameters of prosthetic aortic valve function in mechanical and stented biologic valves\*

Parameter	Parameter Normal Possible stenosis		Suggests significant stenosis	
Peak velocity (m/s) <sup>†</sup>	3	3-4	>4	
Mean gradient (mm Hg) <sup>†</sup>	<20	20-35	>35	
DVI	≥0.30	0.29-0.25	<0.25	
EOA (cm <sup>2</sup> )	>1.2	1.2-0.8	<0.8	
Contour of the jet velocity through the PrAV	Triangular, early peaking	Triangular to intermediate	Rounded, symmetrical contour	
AT (ms)	<80	80-100	>100	

PrAV, Prosthetic aortic valve.

\*In conditions of normal or near normal stroke volume (50-70 mL) through the aortic valve.

†These parameters are more affected by flow, including concomitant AR.

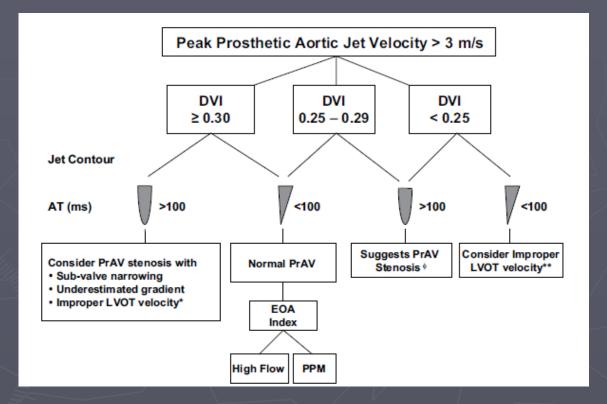
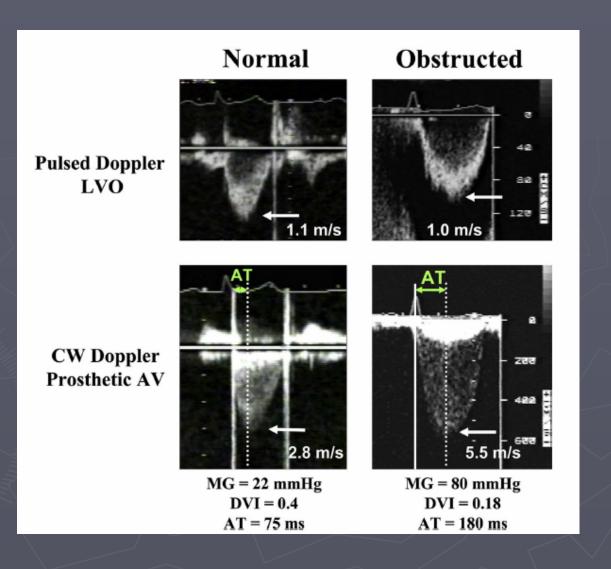


Table 9 Transthoracic echocardiographic findings suggestive of significant prosthetic MR in mechanical valves with normal pressure half-time

Finding	Sensitivity	Specificity	Comments
Peak mitral velocity ≥1.9 m/s*	90%	89%	Also consider high flow, PPM
$VTI_{PHMV}/VTI_{LVO} \ge 2.5^{*}$	89%	91%	Measurement errors increase in atrial fibrillation due to difficulty in matching cardiac cycles; also consider PPM
Mean gradient $\geq$ 5 mmHg <sup>*</sup>	90%	70%	At physiologic heart rates; also consider high flow, PPM
Maximal TR jet velocity > 3 m/s*	80%	71%	Consider residual postoperative pulmonary hypertension or other causes
LV stroke volume derived by 2D or 3D imaging is >30% higher than systemic stroke volume by Doppler	Moderate sensitivity	Specific	Validation lacking; significant MR is suspected when LV function is normal or hyperdynamic and VTI <sub>LVO</sub> is <16 cm
Systolic flow convergence seen in the left ventricle toward the prosthesis	Low sensitivity	Specific	Validation lacking; technically challenging to detect readily

PrMV, Prosthetic mitral valve. \*Data from Olmos et al.<sup>148</sup> When both peak velocity and VTI ratio are elevated with a normal pressure half-time, specificity is close to 100%.



# Sample Ouestions



76 yo woman; Hx of CHF; syst murmur Hyperdynamic LV contraction Small LV cavity Mild concentric LVH No sub-aortic obstruction Calcified aortic valve (difficult to image) valve) Peak velocities Aortic valve (CW): 4.8 m/s LVOT (PW): 1.9 m/s



### What is the Peak Transaortic PG?

Hint:  $4.8^2 = 23$ 

92 mmHg
 76 mmHg
 106 mmHg
 Can't tell

#### Answer #1

92 mmHg
 76 mmHg
 106 mmHg
 Can't tell

Correction for high proximal velocity (1.9 m/s) Bernoulli: **PG = 4 \* (V<sub>dist</sub><sup>2</sup> - V<sub>prox</sub><sup>2</sup>)** PG = 4 \* (23 - 4) = 76 mmHg



# 78 man; CHF; systolic murmur

- Severe LV systolic dysfunction (LVEF ~ 20%)
- Calcified aortic valve
- ► Aortic valve
  - Peak PG: 41 mmHg; mean PG: 28 mmHg
  - VTI: 72 cm; peak velocity: 3.2
- **LVOT** 
  - Diameter: 2.2 cm
  - VTI: 25 cm; peak velocity: 1.1 m/s



- 1. Mild
- 2. Moderate
- 3. Severe (low-gradient AS)
- 4. I need more time to calculate
- 5. Not enough data to answer the question



- Mild
   Moderate
   Severe (low-gradient AS)
   I need more time to calculate
  - 5. Not enough data to answer the question

### Answer #3

Low-gradient AS – suspected (LVEF ↓↓)
 But

Normal LV output
►LVOT VTI: 25 cm, LVOT diameter: 2.2 cm
→ SV: 95 ml (CO = 5.7 L/min)
■ VTI ratio (or peak vel ratio) ~ 1:3
►VTI ratio: 25 / 72
► Peak vel ratio: 1.1 / 3.2
■ AVA = 1.3 cm<sup>2</sup>



## 40 yo woman; MS; NYHA III

► MVA

Planimetry MVA: 1.3 cm2

P t<sub>1/2</sub> MVA: 1.3 cm2 (P t1/2: 170 ms)

►MR: mild

Mean PG: 12 mmHg (@ HR 70)
 SPAP: 50 mmHg

Discrepancy



### Possible causes of discrepancy

All of the possibilities **EXCEPT** 

- 1. Significant sub-valvular disease
- 2. Severe MR
- 3. Heart rate
- 4. LA compliance ↓
  5. LV compliance ↓



All of the possibilities **EXCEPT** 

1. Significant sub-valvular disease Planimetry overestimates physiologic MVA 2. More severe MR MV PG  $\uparrow \rightarrow$  severe combined MV disease 3. Heart rate ■ HR  $\uparrow \rightarrow$  MV PG / symptoms  $\uparrow$  for given MVA 4. LA compliance ↓
 5. LV compliance ↓  $\rightarrow$  MVA  $\uparrow$  (P t1/2  $\downarrow$ )



60 woman; carcinoid heart disease

Severe TR (severe leaflet malcoaptation)
 RV systolic dysfunction
 Peak TR PG: 20 mmHg
 Estimated RA pressure: 25 mmHg



PA systolic pressure ?

- 1. 45 mmHg
- 2. Can't calculate TR malcoaptation may cause overestimation of peak TR PG
- 3. Can't calculate associated pulmonary valve disease likely
- Can't calculate calculation invalid due to RV dysfunction



PA systolic pressure ?

- 1. 45 mmHg
- 2. Can't calculate TR malcoaptation may cause overestimation of peak TR PG
- Can't calculate associated pulmonary valve disease likely
   Can't calculate – calculation invalid due to RV dysfunction

# Good Luck with the test

...but just in case it doesn't go so well, the next slide is not an option !



# Thank You

Acknowledgment: Dr. Yoram Agmon