Right Ventricle: *The other ventricle*

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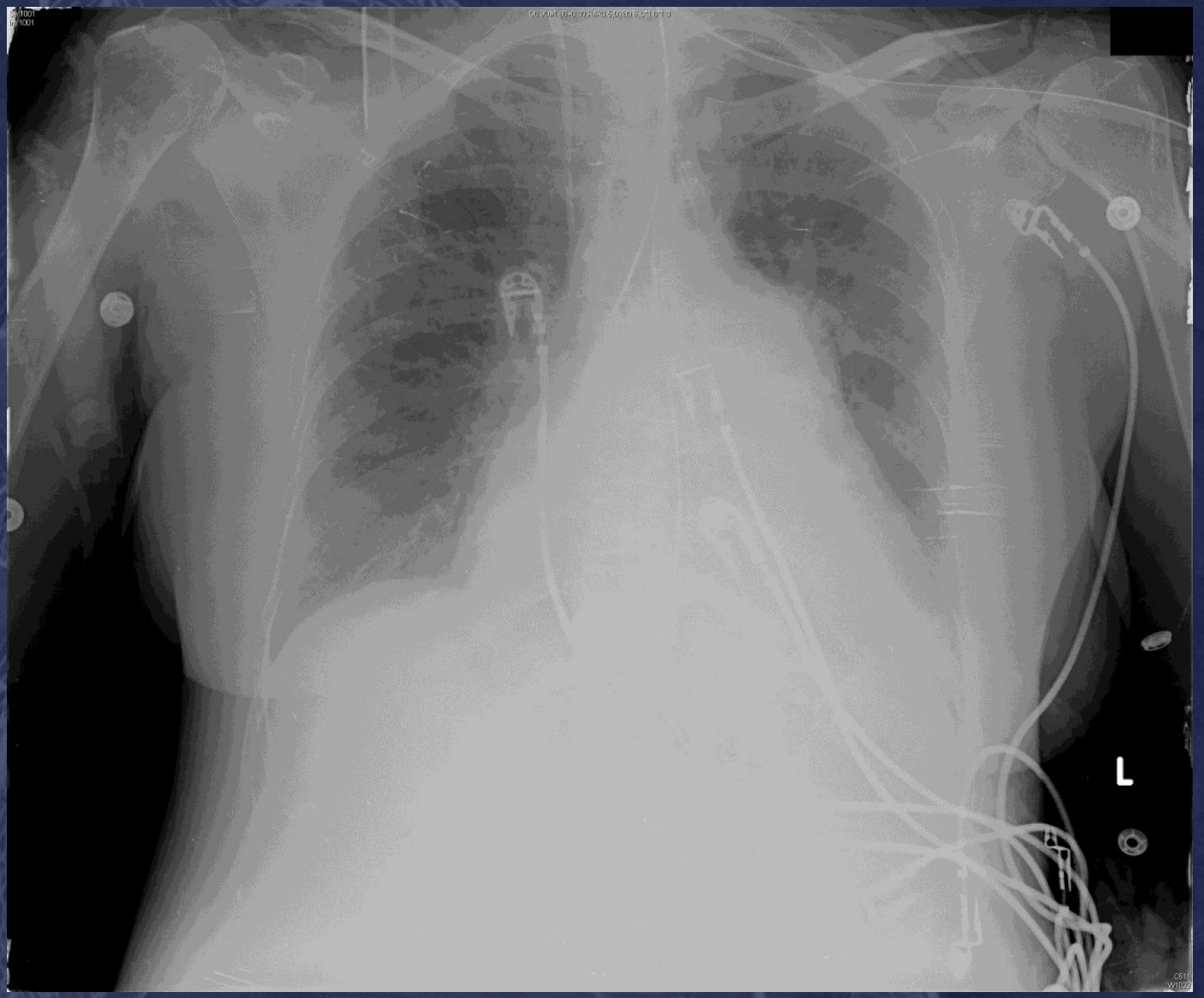
I have no financial relationship to disclose
I will discuss off label or investigational use

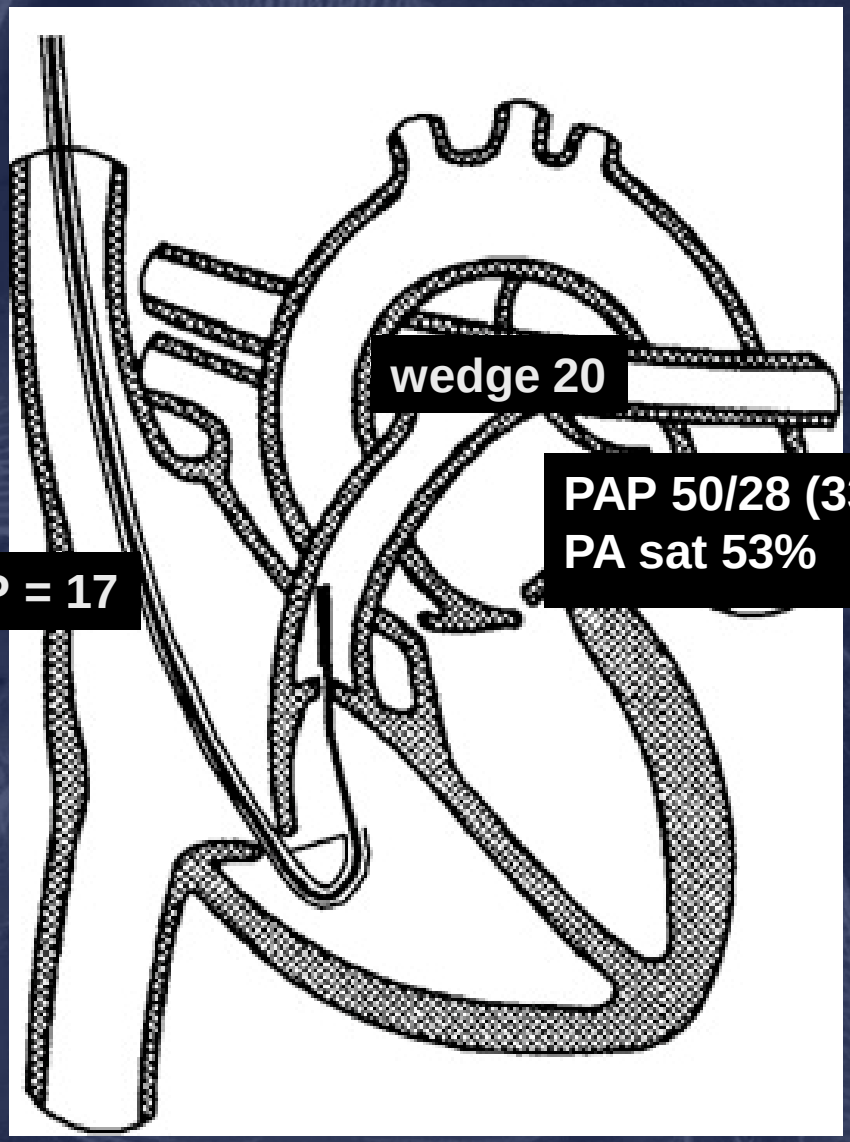
Case presentation

- 68 yo female for AVR, MVR
- AKI on CKD III (Cr 1.9), HTN, asthma, NYHA class III-IV, h/o GI bleed, severe spinal stenosis

  - ECHO: Severe AS. Normal left ventricle size. Moderate hypertrophy. Ejection fraction=55-60%. Moderate diastolic dysfunction and elevated filling pressure.
  - Pulmonary hypertension. Increased right ventricle size with mild dysfunction. Mild tricuspid regurgitation.


L

Right heart catheterization Data



Fick CI 0.95 L/min
TPG = 13
DPG = 8

Gradients

- What does TPG stand for?
  - mean PAP – LAP (estimated by PAOP)
- What does DPG stand for?
  - PAD – LAP (estimated by PAOP)

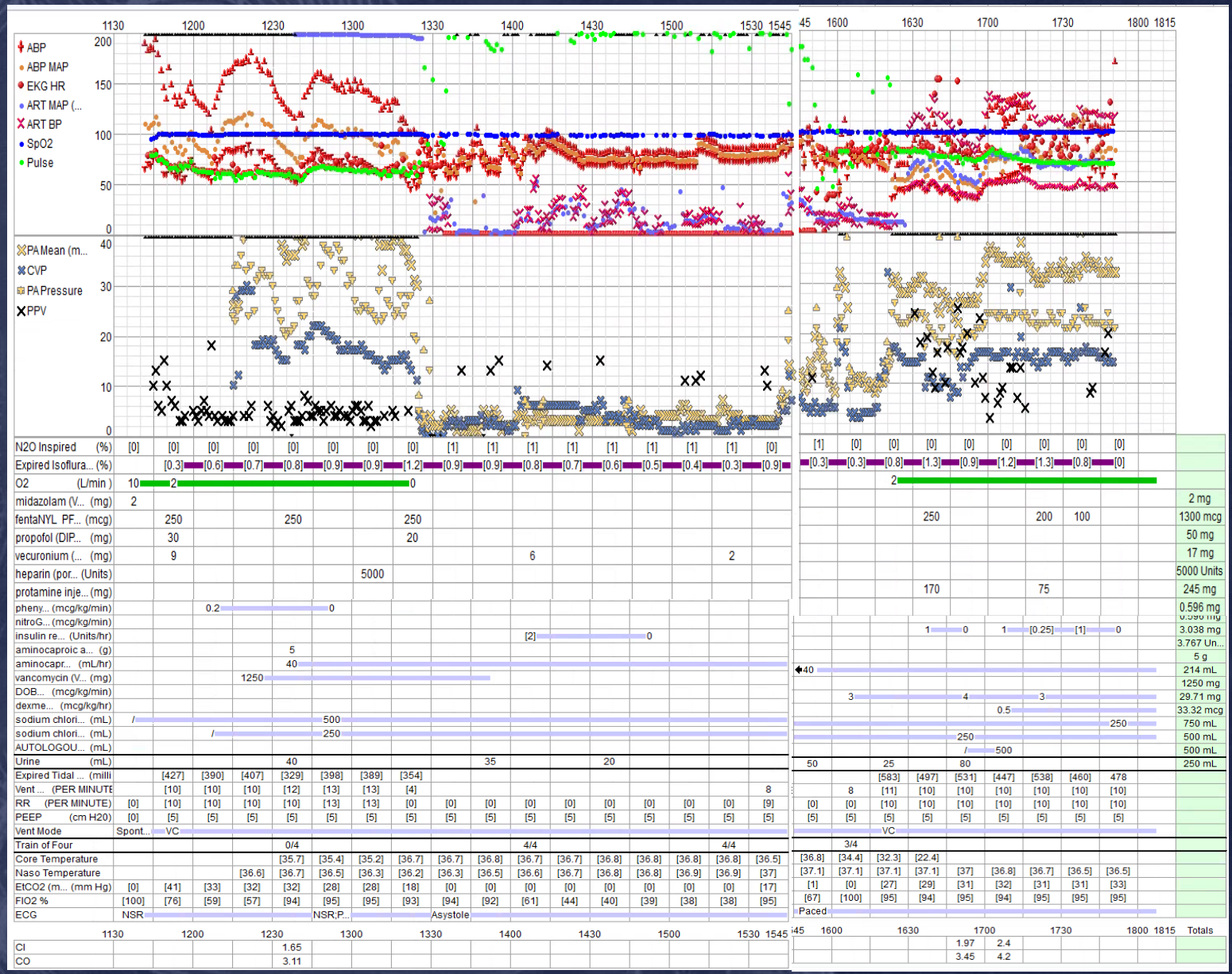
- TPG of >12 mmHg is “out of proportion” PHTN; not just LV failure
- *TPG > 15 relative contraindication to OHT*
- *DPG >7; not shown to predict post-OHT outcome*

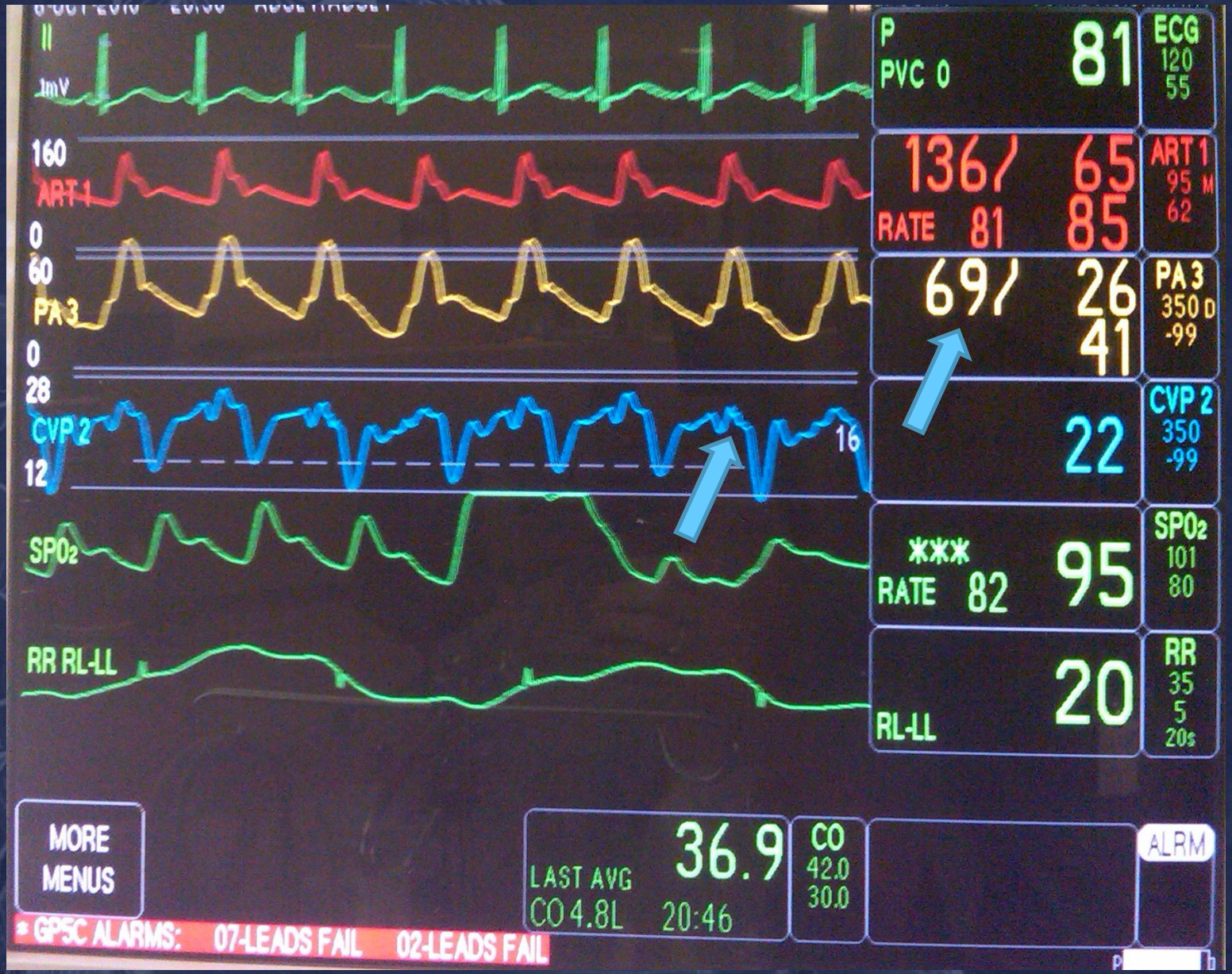
Mehra M. J Heart Lung Transplant. 2006;25:1024-42
Tedford R. J Heart Lung Trasnplant. 2014;33:289-97

OR record

Post CPB TEE: LV EF 60%, mildly reduced RV, mild TR. Valves ok.

DBA 4
mcg/k/min



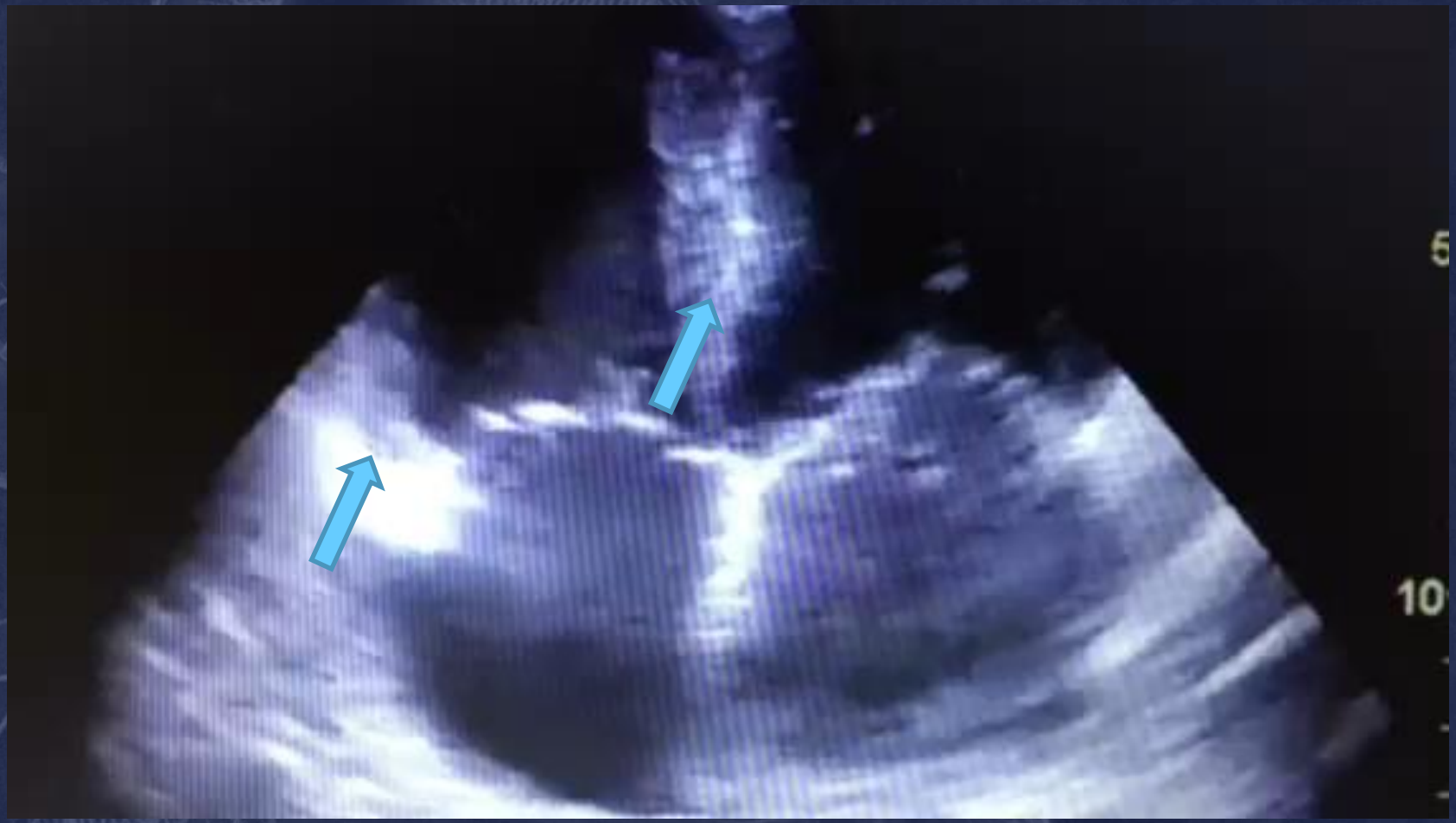

II
1mV
160
ART 1
0
60
PA 3
0
28
CVP 2
12
16
SPO2
RR RL-LL
P
PVC 0
81
ECG
120
55
136/ 65
RATE 81 85
ART 1
95 M
62
69/ 26
41
PA 3
350 D
-99
22
CVP 2
350
-99
***
RATE 82
95
SPO2
101
80
20
RL-LL
RR
35
5
20s
MORE
MENUS
LAST AVG
36.9
CO 4.8L 20:46
CO
42.0
30.0
ALRM
* GPSC ALARMS: 07-LEADS FAIL 02-LEADS FAIL

POD 2

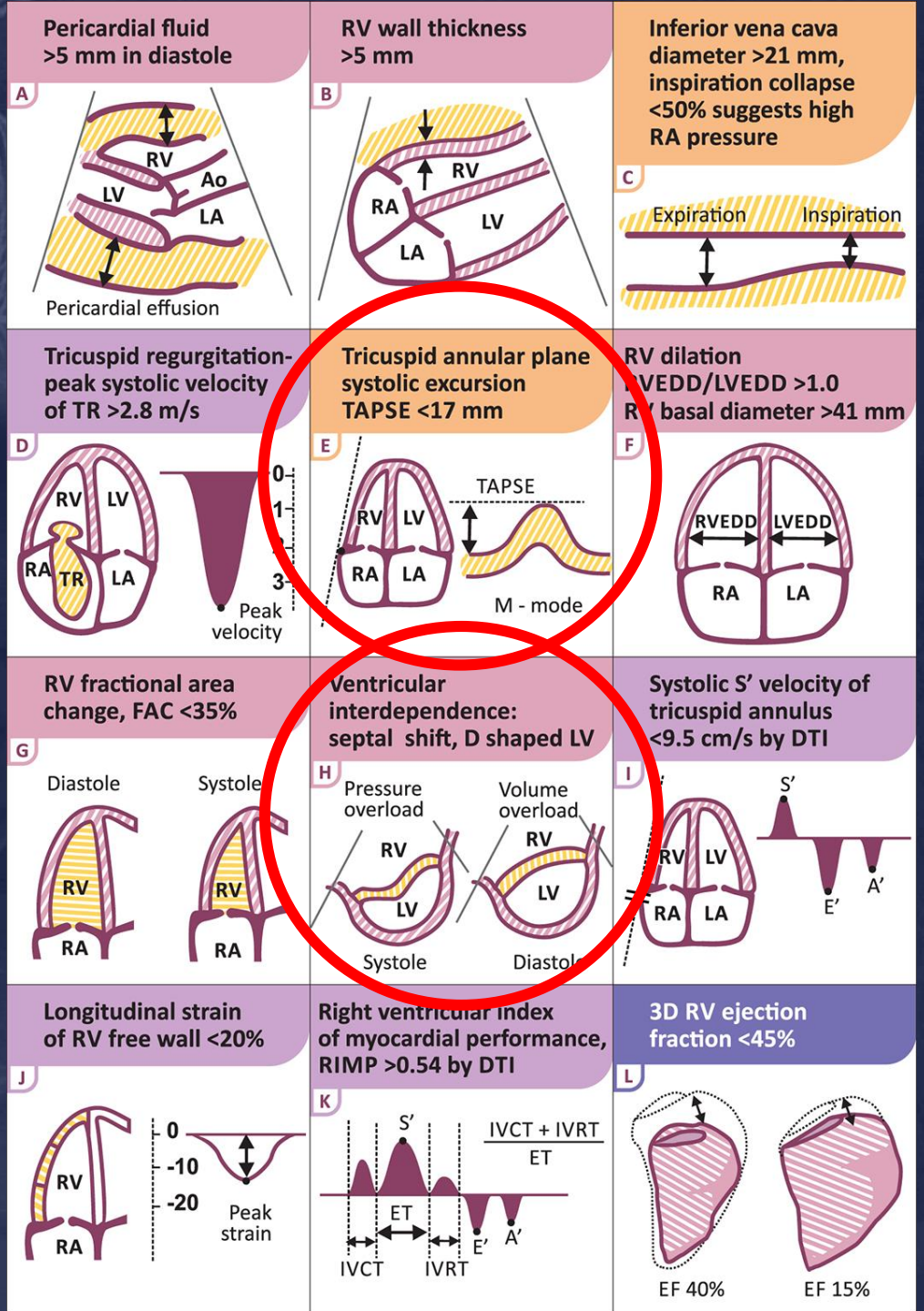
1 hr:	21-22	22-23	23-00	00-01	01-02	02-03	03-04	04-05	05-06	06-07	07-08	08-09	09-10	10-11	11-12	12-13	13-14	14-15	15-16	16-17	
Vitals																					
Weight										81.5 kg (...											Weight
Temp	36.8 (98.2)	36.8 (98.2)	36.8 (98.2)	36.7 (98...+	36.8 (98.2)	36.6 (97.9)	36.4 (97...+	36.4 (97...+		36.3 (97...+		36.3 (97.3)	36.4 (97.5)	36.6 (97.9)	36.8 (98.2)	36.8 (98.2)	36.9 (98...+	36.9 (98.4)	36.8 (98.2)	36.8 (98.2)	Temp
Temperature Source	Blood	Blood	Blood	Blood+	Blood	Blood	Blood+	Blood+		Blood+		Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Tempe...
Pulse	86+	86	84	89+	86	81	83+	83+		82+		81+	82+	83	81	81	65+	69	67	67	Pulse
Monitored Rhythm	SR;BBB	SR;BBB	SR;BBB	SR;BBB+	SR;BBB	SR;BBB	SR;BBB+	SR;BBB+		SR;BBB+		SR;BBB	SR;BBB	SR;BBB	SR;BBB	SR;BBB	SR;BBB	SR;BBB	SR;BBB	SR;BBB	Monito...
Ectopic Frequency	Rare	Rare	Rare	Rare+	Rare	Rare	Rare+	Rare+		Rare+		Rare		Rare	Rare					Rare	Ectopi...
PVC / Minute	0	0	0	0+	0	1	0+	0+		0+		0	0	0	3	1	14+	0	0	0	PVC /...
Respirations	31+	19	13	25+	33	12	21+	21!		23+		21+	22+	18	19	21	18+	26	26	30	Respir...
ABP	115/59	133/50	138/51	148/54+	142/51	135/51	145/53+	131/41+		126/49+		130/55	137/58	137/56	131/60	139/56	62/29!	122/63	107/52	122/43	ABP
O2 Percent	70+	70	70	60+	60	60	60+	60+		60+		50+	50			40	100	80		80	O2 Pe...
O2 Delivery																				Other (Co...	O2 Del...
O2 Liter Flow	Comment			Comment				Comment				--+	--+	Comment	Comment	Comment	--			--+	O2 Lit...
SpO2	100+	99	99	94+	97	99	99+	99+		99+		99+	97+	97	96+	[illegible]	99+		99	94	SpO2
Hemodynamics																					
CO				4.67				4.89											2.64		CO
CI				2.67				2.79											1.51		CI
SV (Calculated)				54.3				53											39.4		SV (C...
SVI (Calculated)				31				30											22.5		SVI (C...
CVP	17	14	13	13!	16	14	13!	8!		15!		17	20	19	25	22	18!	25	25	18	CVP
SVR (Calculated)				1199				997											1242		SVR (...
SVRI (Calculated)				2097				1746											2173		SVRI (...
PA Pressure	56/22	49/17	51/20	53/21!	58/22	51/19	55/19!	50/14!		59/21!		59/23	62/23	62/24	64/30	58/25	25/14!	37/23	38/21		PA Pr...
PA Mean (mm Hg)	33	28	29	30!	32	29	30!	25!		32!		33	36	35	41	35	18!	27	27		PA Me...
PAOP				20				19											21		PAOP
PVR (Calculated)				171				180											182		PVR (...
PVRI (Calculated)				300				315											318		PVRI (...
LVSWI (Calculated)				26.6				23											13.8		LVSWI...
RVSWI (Calculated)				7.17				7											0.61		RVSW...
LCW (Calculated)				4															1.6		LCW (...
LCWI (Calculated)				2.3															0.9		LCWI (...
RCW (Calculated)				1.08															0.07		RCW (...
RCWI (Calculated)				0.62															0.04		RCWI...
LVSW (Calculated)				46.5															24.1		LVSW...
RVSW (Calculated)				12.55															1.07		RVSW...
Intake																					
P.O.												120		225							P.O.
I.V.	38.9	38.9	38.9	38.9	38.9	38.9	38.9	36.6	32.9	32.9	32.9	32.9	32.9	32.9	26.2	12.9	12.9	12.9	32.3	32.4	I.V.
Total In	38.9	38.9	38.9	38.9	38.9	38.9	38.9	36.6	32.9	32.9	32.9	152.9	32.9	257.9	26.2	12.9	12.9	12.9	32.3	32.4	**Total In**
Output																					
Urine	15	17	10	25	7	10	10	15	10	10	10	9	5	5	15	10	7	20	5	0	Urine
Drains	10	0	0	20	10	0	0	10	10	120	0	0			125						Drains
Total Out	25	17	10	45	17	10	10	25	20	130	10	9	5	5	140	10	7	20	5	0	**Total...**
I/O Net	13.9	21.9	28.9	6.4	21.9	28.9	28.9	11.6	12.9	97.1	22.9	143.9	27.9	252.9	113.8	2.9	5.9	7.1	27.3	32.4	I/O Net

Epinephrine drip added

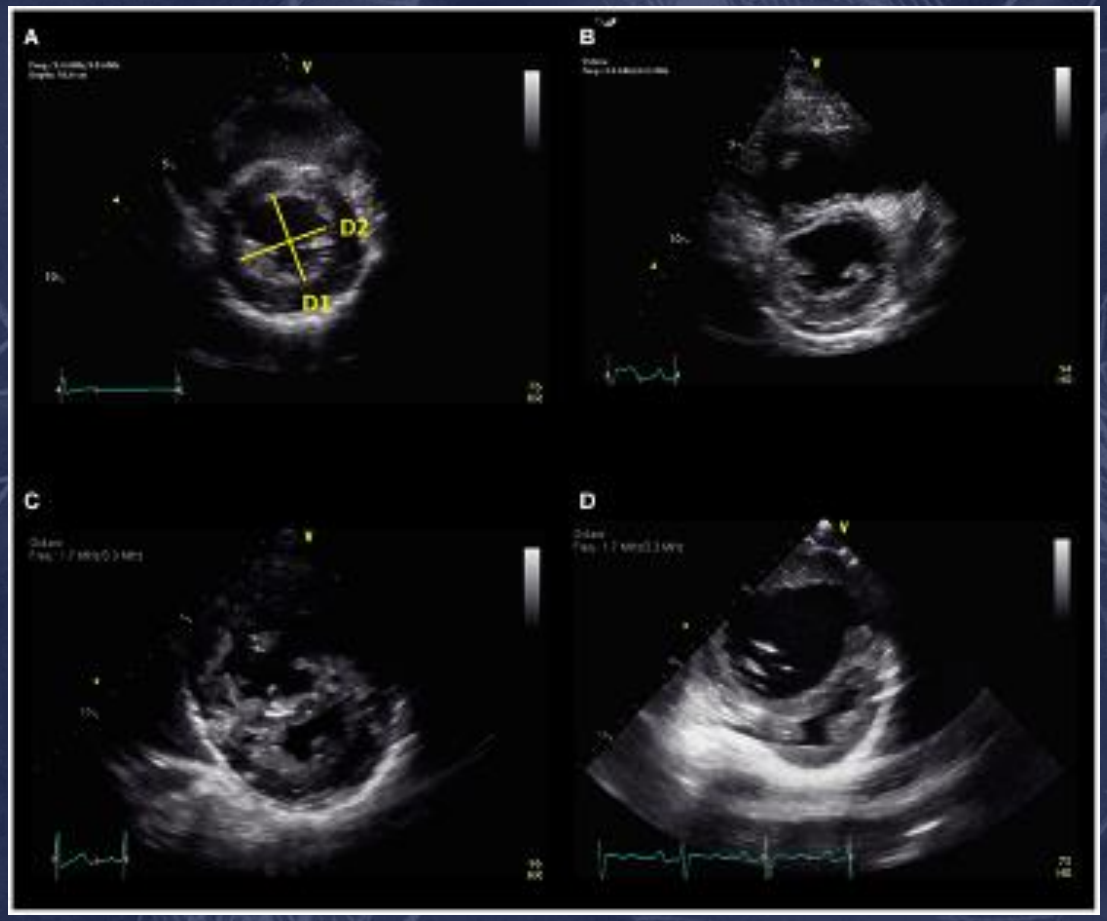
TTE obtained



RV function assessments by TTE



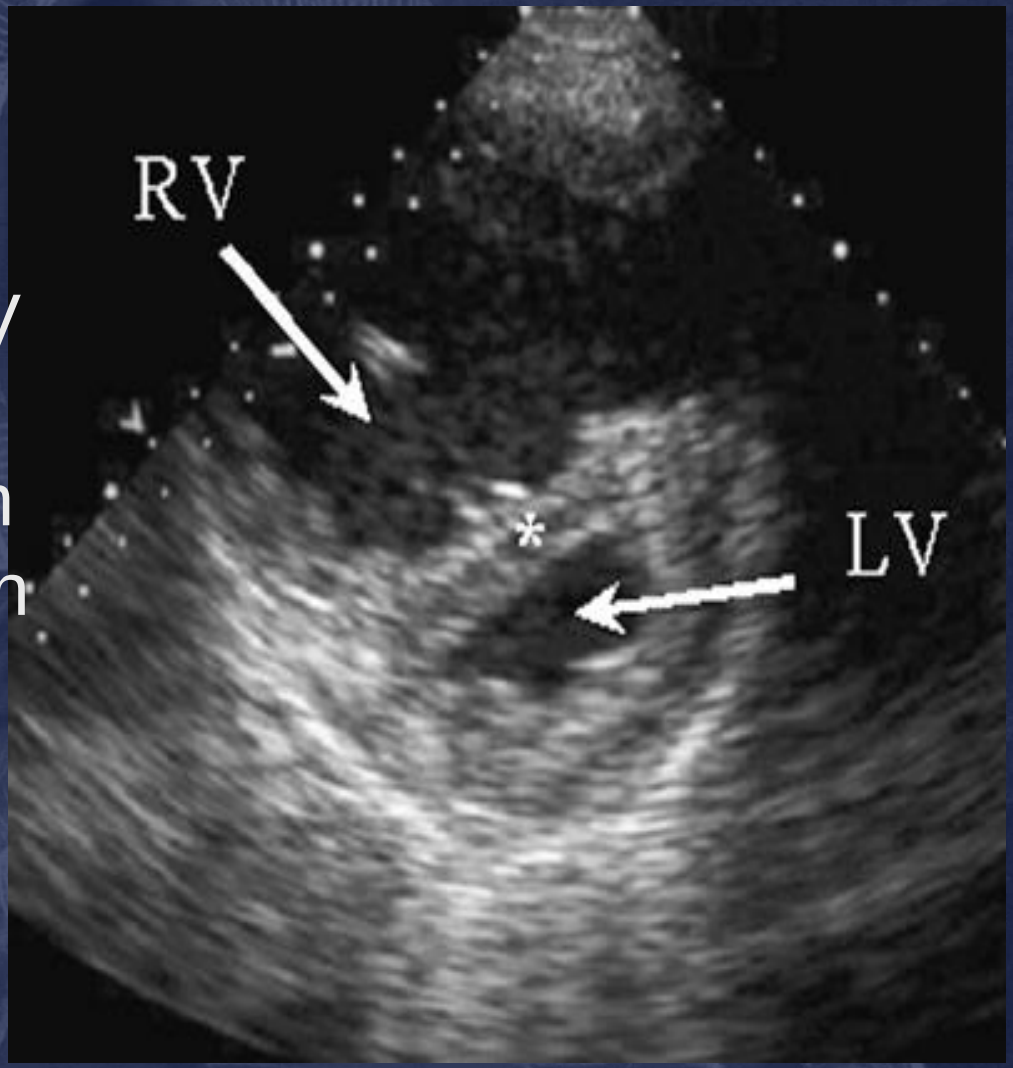
RV function assessments



- Septal shape
  - eccentricity index
  - D2/D1 > 1
    - In diastole = RV volume overload
    - In systole = RV volume and pressure overload

Ventricular Interdependence

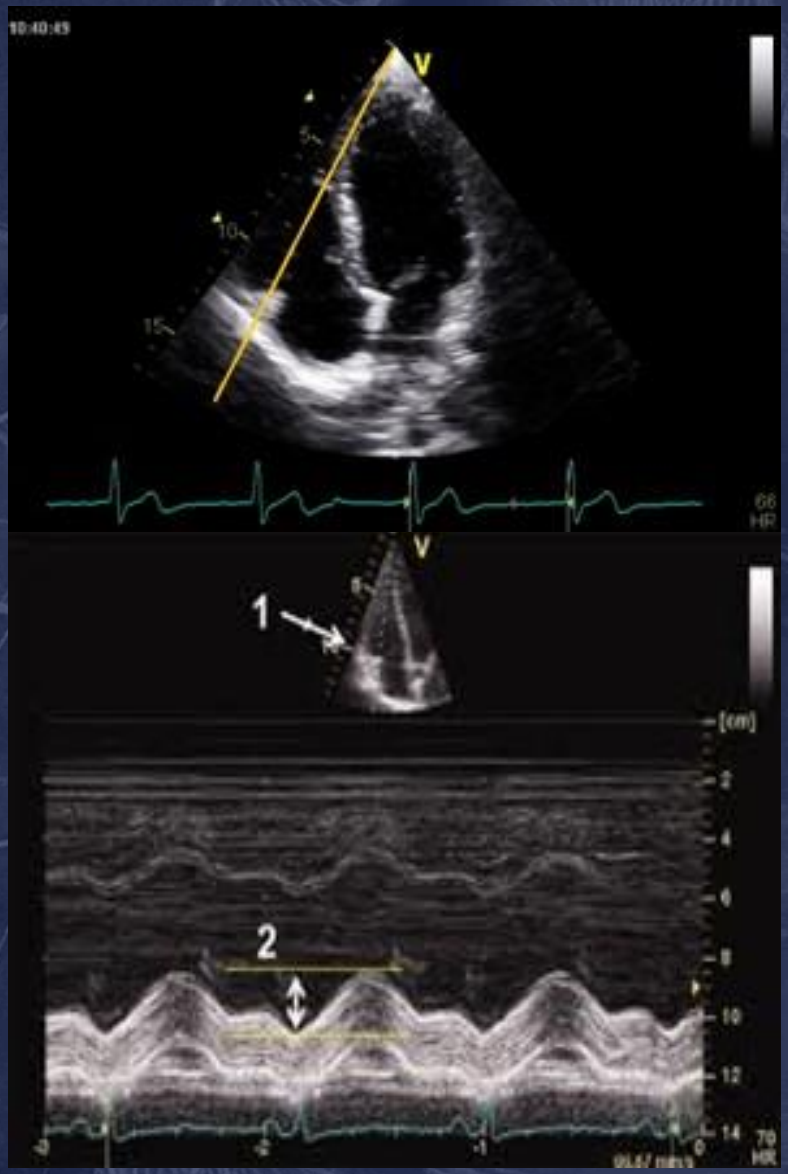
20-40% of RV contraction is provided from LV contraction via septum



LV filling is impaired by a left shift of the inter-ventricular septum

Wiedemann HP, et al. Heart-Lung Interactions in Health and Disease. 1989:920-926

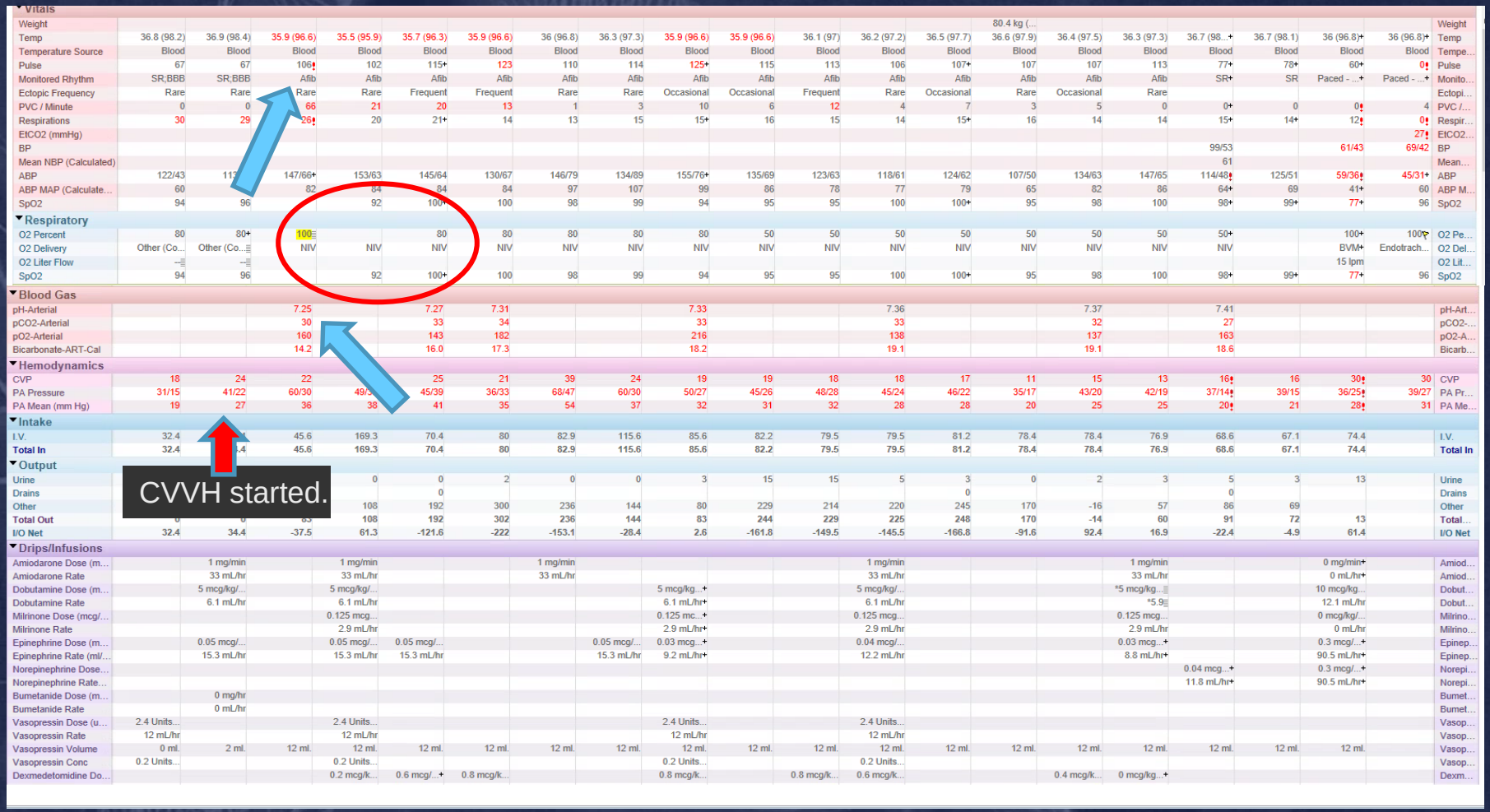
RV function assessments



- TAPSE (tricuspid annular plane systolic excursion)
  - Normal >20mm
  - Angle and load dependent
  - <8.5mm = RV EF 25%

J Am Soc Echocardiogr. 2010;23:685-713
Haddad S. JASE 2006;19:902

POD 2

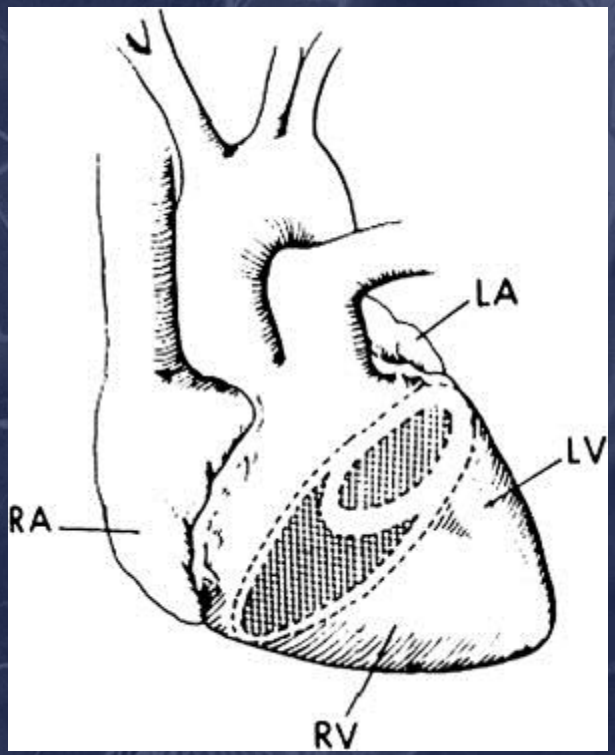


Vitals																				
Weight													80.4 kg (...							Weight
Temp	36.8 (98.2)	36.9 (98.4)	35.9 (96.6)	35.5 (95.9)	35.7 (96.3)	35.9 (96.6)	36 (96.8)	36.3 (97.3)	35.9 (96.6)	35.9 (96.6)	36.1 (97)	36.2 (97.2)	36.5 (97.7)	36.6 (97.9)	36.4 (97.5)	36.3 (97.3)	36.7 (98...+	36.7 (98.1)	36 (96.8)+	36 (96.8)+	Temp
Temperature Source	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Blood	Tempe...
Pulse	67	67	106↑	102	115+	123	110	114	125+	115	113	106	107+	107	107	113	77+	78+	60+	0↓	Pulse
Monitored Rhythm	SR;BBB	SR;BBB	Afib	Afib	Afib	Afib	Afib	Afib	Afib	Afib	Afib	Afib	Afib	Afib	Afib	Afib	SR+	SR	Paced - ...+	Paced - ...+	Monito...
Ectopic Frequency	Rare	Rare	Rare	Rare	Frequent	Frequent	Rare	Rare	Occasional	Occasional	Frequent	Rare	Occasional	Rare	Occasional	Rare					Ectopi...
PVC / Minute	0	0	66	21	20	13	1	3	10	6	12	4	7	3	5	0	0+	0	0↓	4	PVC /...
Respirations	30	29	26↓	20	21+	14	13	15	15+	16	15	14	15+	16	14	14	15+	14+	12↓	0↓	Respir...
EtCO2 (mmHg)																				27↓	EtCO2...
BP																	99/53		61/43	69/42	BP
Mean NBP (Calculated)																	61				Mean...
ABP	122/43	117	147/66+	153/63	145/64	130/67	146/79	134/89	155/76+	135/69	123/63	118/61	124/62	107/50	134/63	147/65	114/48↓	125/51	59/36↓	45/31+	ABP
ABP MAP (Calculate...	60		82	84	84	84	97	107	99	86	78	77	79	65	82	86	64+	69	41+	60	ABP M...
SpO2	94	96		92	100+	100	98	99	94	95	95	100	100+	95	98	100	98+	99+	77+	96	SpO2
Respiratory																					
O2 Percent	80	80+	100		80	80	80	80	80	50	50	50	50	50	50	50	50+		100+	100	O2 Pe...
O2 Delivery	Other (Co...	Other (Co...	NIV	NIV	NIV	NIV	NIV	NIV	NIV	NIV	NIV	NIV	NIV	NIV	NIV	NIV	NIV		BVM+	Endotrach...	O2 Del...
O2 Liter Flow	---	---																	15 lpm		O2 Lit...
SpO2	94	96		92	100+	100	98	99	94	95	95	100	100+	95	98	100	98+	99+	77+	96	SpO2
Blood Gas																					
pH-Arterial			7.25		7.27	7.31			7.33			7.36			7.37		7.41				pH-Art...
pCO2-Arterial			30		33	34			33			33			32		27				pCO2-...
pO2-Arterial			160		143	182			216			138			137		163				pO2-A...
Bicarbonate-ART-Cal			14.2		16.0	17.3			18.2			19.1			19.1		18.6				Bicarb...
Hemodynamics																					
CVP	18	24	22		25	21	39	24	19	19	18	18	17	11	15	13	16↓	16	30↓	30	CVP
PA Pressure	31/15	41/22	60/30	49/3	45/39	36/33	68/47	60/30	50/27	45/26	48/28	45/24	46/22	35/17	43/20	42/19	37/14↓	39/15	36/25↓	39/27	PA Pr...
PA Mean (mm Hg)	19	27	36	38	41	35	54	37	32	31	32	28	28	20	25	25	20↓	21	28↓	31	PA Me...
Intake																					
I.V.	32.4	34.4	45.6	169.3	70.4	80	82.9	115.6	85.6	82.2	79.5	79.5	81.2	78.4	78.4	76.9	68.6	67.1	74.4		I.V.
Total In	**32.4**	**34.4**	**45.6**	**169.3**	**70.4**	**80**	**82.9**	**115.6**	**85.6**	**82.2**	**79.5**	**79.5**	**81.2**	**78.4**	**78.4**	**76.9**	**68.6**	**67.1**	**74.4**		**Total In**
Output																					
Urine				0	0	2	0	0	3	15	15	5	3	0	2	3	5	3	13		Urine
Drains					0								0				0				Drains
Other				108	192	300	236	144	80	229	214	220	245	170	-16	57	86	69			Other
Total Out				**108**	**192**	**302**	**236**	**144**	**83**	**244**	**229**	**225**	**248**	**170**	**-14**	**60**	**91**	**72**	**13**		**Total...**
I/O Net	**32.4**	**34.4**	**-37.5**	**61.3**	**-121.6**	**-222**	**-153.1**	**-28.4**	**2.6**	**-161.8**	**-149.5**	**-145.5**	**-166.8**	**-91.6**	**92.4**	**16.9**	**-22.4**	**-4.9**	**61.4**		**I/O Net**
Drips/Infusions																					
Amiodarone Dose (m...		1 mg/min		1 mg/min			1 mg/min					1 mg/min				1 mg/min			0 mg/min+		Amiod...
Amiodarone Rate		33 mL/hr		33 mL/hr			33 mL/hr					33 mL/hr				33 mL/hr			0 mL/hr+		Amiod...
Dobutamine Dose (m...		5 mcg/kg/...		5 mcg/kg/...					5 mcg/kg...+			5 mcg/kg/...				*5 mcg/kg...			10 mcg/kg...		Dobut...
Dobutamine Rate		6.1 mL/hr		6.1 mL/hr					6.1 mL/hr+			6.1 mL/hr				*5.9			12.1 mL/hr		Dobut...
Milrinone Dose (mcg/...				0.125 mcg...					0.125 mc...+			0.125 mcg...				0.125 mcg...			0 mcg/kg/...		Milrino...
Milrinone Rate				2.9 mL/hr					2.9 mL/hr+			2.9 mL/hr				2.9 mL/hr			0 mL/hr		Milrino...
Epinephrine Dose (m...		0.05 mcg/...		0.05 mcg/...	0.05 mcg/...			0.05 mcg/...	0.03 mcg...+			0.04 mcg/...				0.03 mcg...+			0.3 mcg/...+		Epinep...
Epinephrine Rate (ml/...		15.3 mL/hr		15.3 mL/hr	15.3 mL/hr			15.3 mL/hr	9.2 mL/hr+			12.2 mL/hr				8.8 mL/hr+			90.5 mL/hr+		Epinep...
Norepinephrine Dose...																	0.04 mcg...+		0.3 mcg/...+		Norepi...
Norepinephrine Rate...																	11.8 mL/hr+		90.5 mL/hr+		Norepi...
Bumetanide Dose (m...		0 mg/hr																			Bumet...
Bumetanide Rate		0 mL/hr																			Bumet...
Vasopressin Dose (u...	2.4 Units...			2.4 Units...					2.4 Units...			2.4 Units...									Vasop...
Vasopressin Rate	12 mL/hr			12 mL/hr					12 mL/hr			12 mL/hr									Vasop...
Vasopressin Volume	0 ml	2 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml	12 ml		Vasop...
Vasopressin Conc	0.2 Units...			0.2 Units...					0.2 Units...			0.2 Units...									Vasop...
Dexmedetomidine Do...				0.2 mcg/k...	0.6 mcg/...+	0.8 mcg/k...			0.8 mcg/k...		0.8 mcg/k...	0.6 mcg/k...			0.4 mcg/k...	0 mcg/kg...+					Dexm...

CVVH started.

Discussions with daughters to not repeat ACLS.
Patient expired due to complications of RV failure.

History of the Oft' *"Misunderstood"* Right Ventricle



1940's:

- First PAP and PVR measurements
- Belief that RV simply a conduit
- Contraction not necessary
- Shrinks in thickness after birth
- Single organ responsibility

Chan C, et al. Clinics in Chest Med 2008;29:661-76

American Heart Journal

VOL. 26 SEPTEMBER, 1943 No. 3

Original Communications

THE ABSENCE OF CONSPICUOUS INCREMENTS OF VENOUS PRESSURE AFTER SEVERE DAMAGE TO THE RIGHT VENTRICLE OF THE DOG, WITH A DISCUSSION OF THE RELATION BETWEEN CLINICAL CONGESTIVE FAILURE AND HEART DISEASE

ISAAC STARR, M.D., WILLIAM A. JEFFERS, M.D., AND RICHARD H. MEADE, JR., M.D.
PHILADELPHIA, PA.

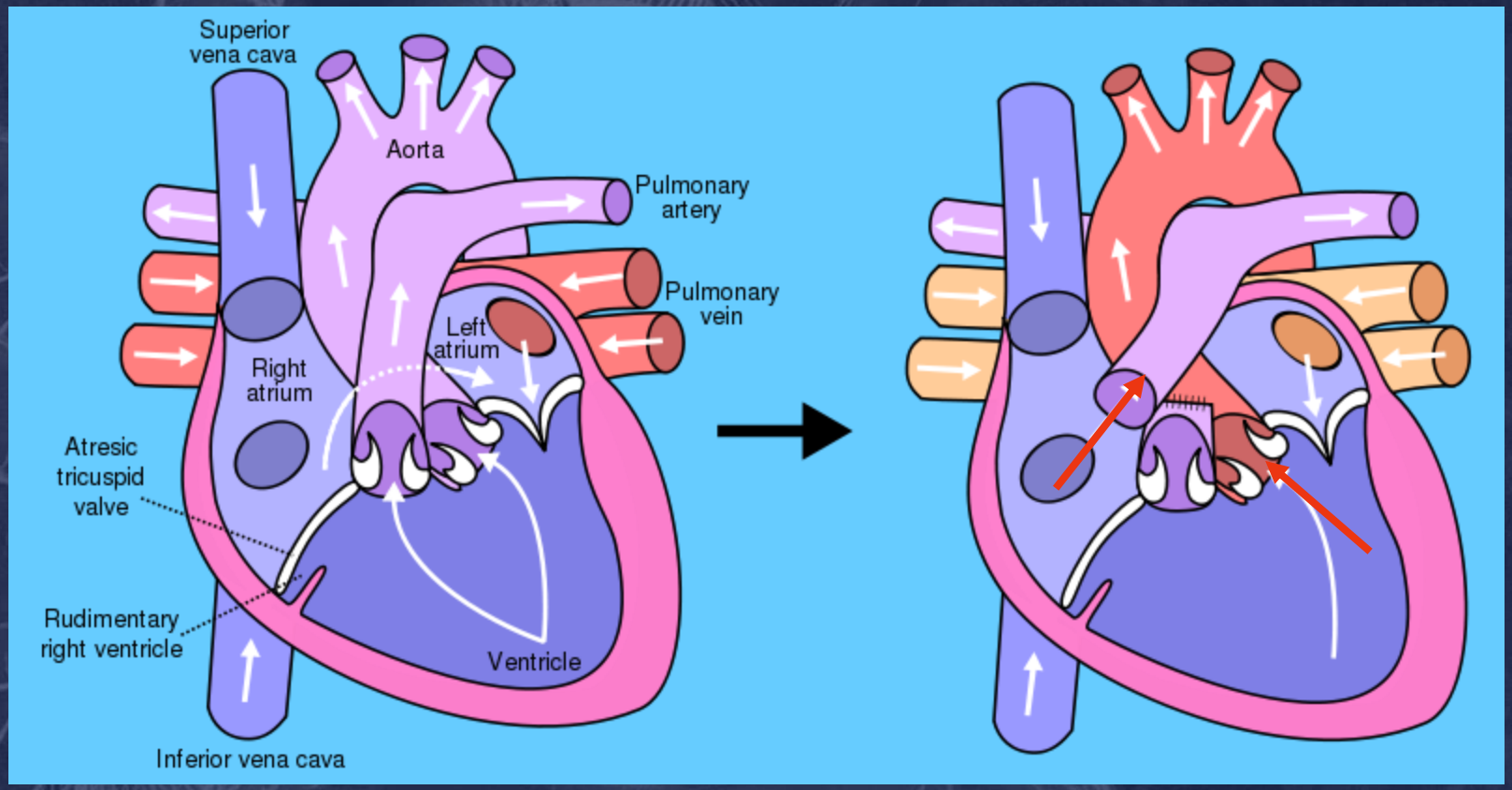
PERIPHERAL venous congestion is often interpreted by clinicians as indicating disproportionate failure of the right ventricle.[1, 2] Doubt of the validity of this interpretation[3, 4] stimulated us to attempt direct experiments on dogs. The controversy between those who believe that one side of the heart can fail while the other remains relatively competent and those who can conceive only of failure of the whole heart has been recently reviewed by Luisada.[5] In the experimental attack on this problem, interest has centered in the production of pulmonary edema by damaging the left side of the heart.[5] Therefore, although the right side of the heart has been damaged by ligation of the right coronary artery[6] or the injection of silver nitrate into the right ventricular wall,[7, 8, 9] these experiments were designed as controls, and the facts which chiefly interested us were not recorded.

Therefore, in acute experiments, we damaged the exposed right ventricular wall with a cautery, and, in chronic experiments, ligated the vessels supplying this wall, closed the incision, and studied the animals until death or recovery ensued. Only minimal changes of venous pressure followed the most extensive damage to the right side of the heart that we knew how to inflict. With the results of these experiments before us, we have reconsidered the dynamics of clinical congestive failure and discussed its relationship to weakness of the heart.

- Obliterated RV free wall with electrocautery
- Minimal change in CVP & CO
- But…pericardium open…

Starr I, et al. Am Heart J 1943;26:292-301

1971: Fontan procedure



Special Report

Right Ventricular Function and Failure

Report of a National Heart, Lung, and Blood Institute Working Group on Cellular and Molecular Mechanisms of Right Heart Failure

Norbert F. Voelkel, MD; Robert A. Quaife, MD; Leslie A. Leinwand, PhD; Robyn J. Barst, MD; Michael D. McGoon, MD; Daniel R. Meldrum, MD; Jocelyn Dupuis, MD, PhD; Carlin S. Long, MD; Lewis J. Rubin, MD; Frank W. Smart, MD; Yuichiro J. Suzuki, PhD; Mark Gladwin, MD; Elizabeth M. Denholm, PhD; Dorothy B. Gail, PhD

Knowledge about the role of the right ventricle in health and disease historically has lagged behind that of the left ventricle. Less muscular, restricted in its role to pumping blood through a single organ, and less frequently or obviously involved than the left ventricle in diseases of epidemic proportions such as myocardial ischemia, cardiomyopathy, or valvulopathy, the right ventricle has generally been considered a mere bystander, a victim of pathological processes affecting the cardiovascular system. Consequently, comparatively little attention has been devoted to how right ventricular pulmonary vascular diseases (cor pulmonale). Other diseases affect the right ventricle in different ways, including global, left ventricular– , or right ventricular–specific cardiomyopathy; right ventricular ischemia or infarction; pulmonary or tricuspid valvular heart disease; and left-to-right shunts.

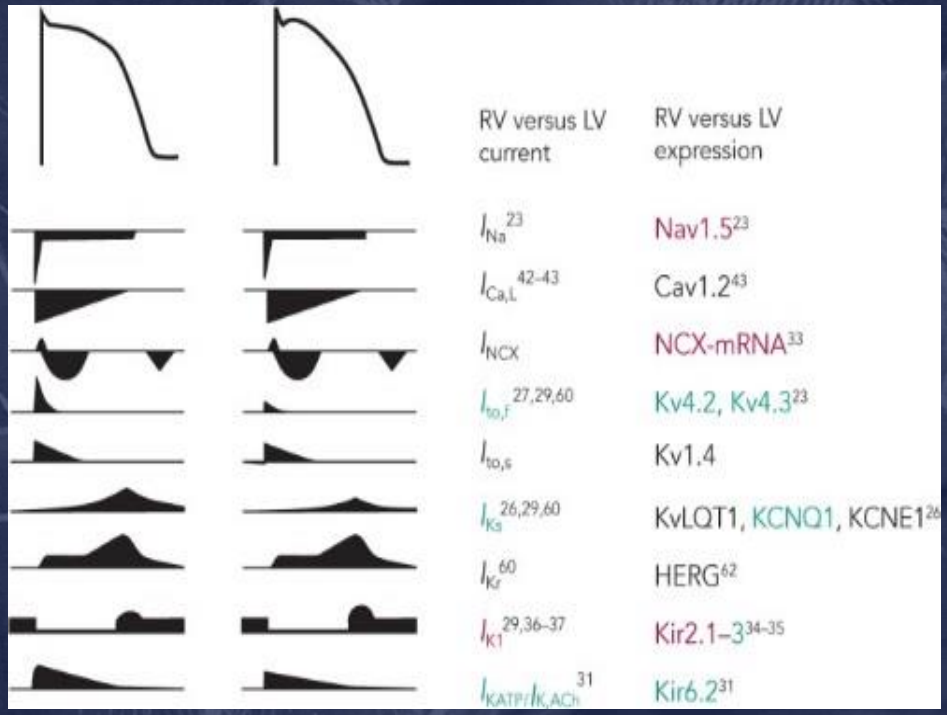
The Normal Right Ventricle

The right ventricle pumps the same stroke volume as the left ventricle but with ≈25% of the stroke work because of the low resistance of the pulmonary vasculature. Therefore, by

Voelkel NF, et al. Circulation 2006;114:1883-91

	RV	LV
Embryologic origin	Cardiac precursor cells in the anterior heart field	Heart tube
Transcription factors expressed during differentiation	dHAND	eHAND
Adult thickness	1/3 of LV	0.6-1cm
Shape	Triangular/conical	spherical
Number of myocyte layers	2	3
Direction of contraction	longitudinal	torsion, shortening, thickening
Moderator band present?	Yes (heavy trabeculations)	no
Afterload (dyne/s/cm^{-5})	70 (20-130)	1100 (700-1600)

RV and LV Electrophysiology



Green = upregulated in RV
Red = downregulated in RV
Black = similar

- known differences in Ca^{2+} handling at baseline and during pathophysiological conditions
- effects remodeling of each ventricle causing subsequent impact on cardiac arrhythmogenesis

Molina C. Arrhythm Electrophysiol Rev. 2016; 5:14-19

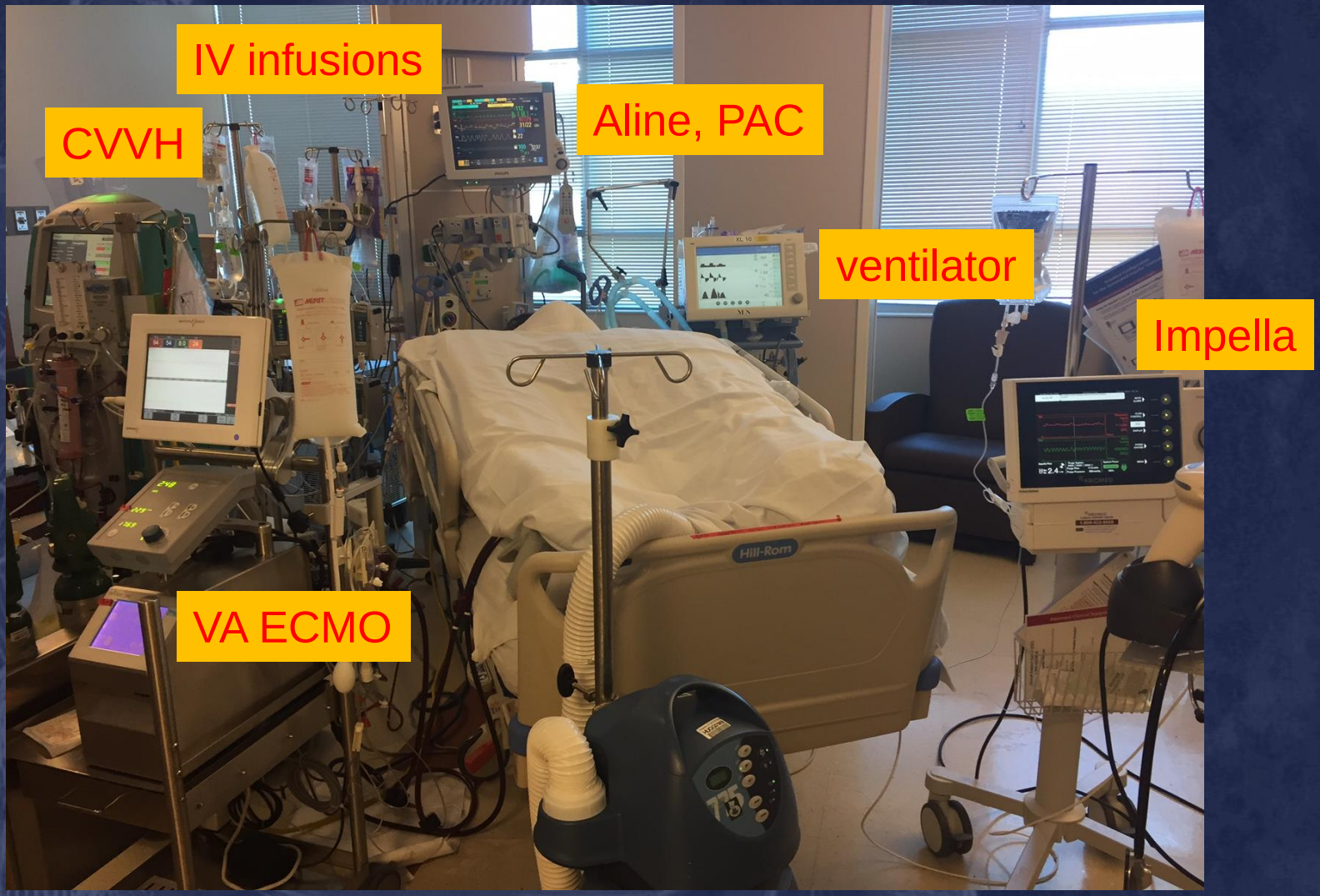
Shampoo is better,

I go on first and clean the hair!

Conditioner is better,

I leave the hair silky and smooth!

Causes of RV failure



Decreased Contractility

RV Infarct
Cardiac tamponade
Cardiomyopathies
Poor RV protection
Sepsis
Post-transplantation

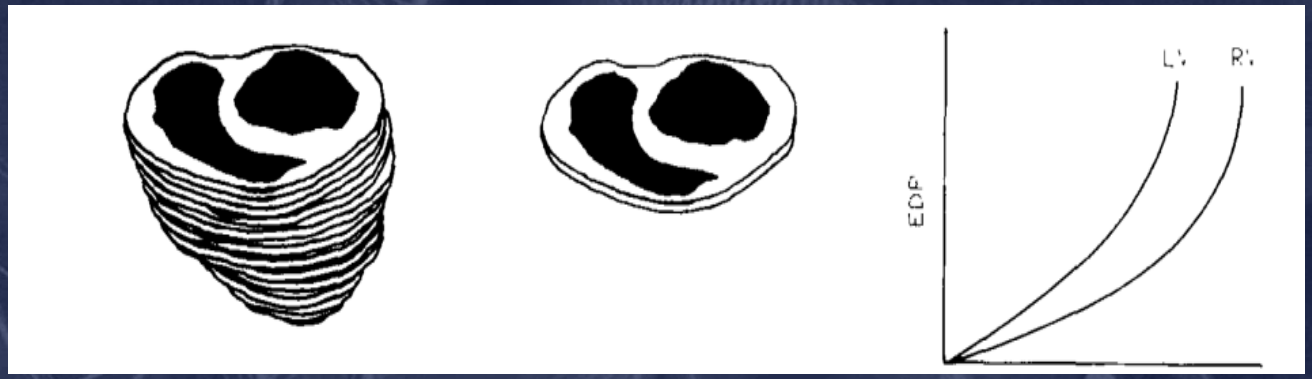
Tricuspid regurgitation
Pulmonary regurgitation
ASD
LVAD
Iatrogenic
Carcinoid

Left-sided failure/valve disease
Pulmonary Embolism
Obstruction of RVOT
Positive pressure ventilation
Pulmonary Hypertension
ARDS
Post-transplantation

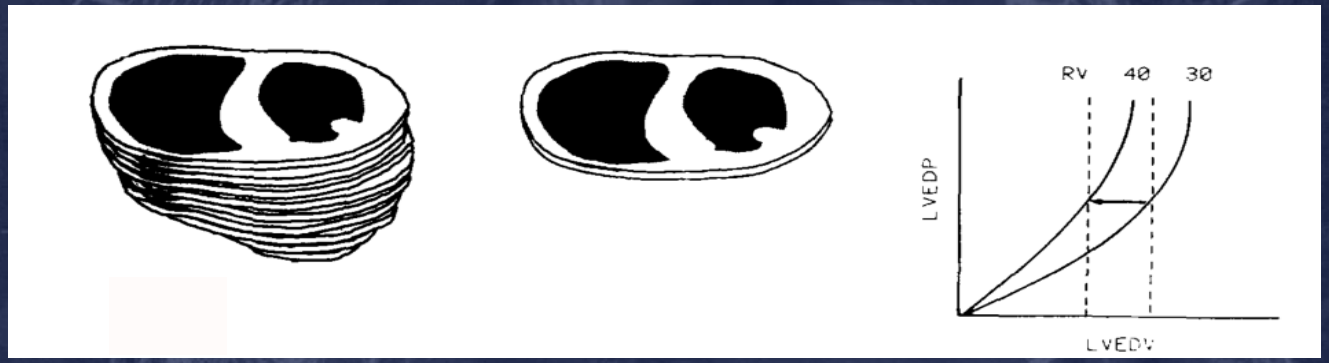
Volume Overload

Pressure Overload

Pressure Overload



Normal: RV is twice as distensible as LV



Failure: RV curve starts to look like LV curve

Weber KT, et al. Am J Cardiol 1981, 47:686-95

Which patient is in RV failure?

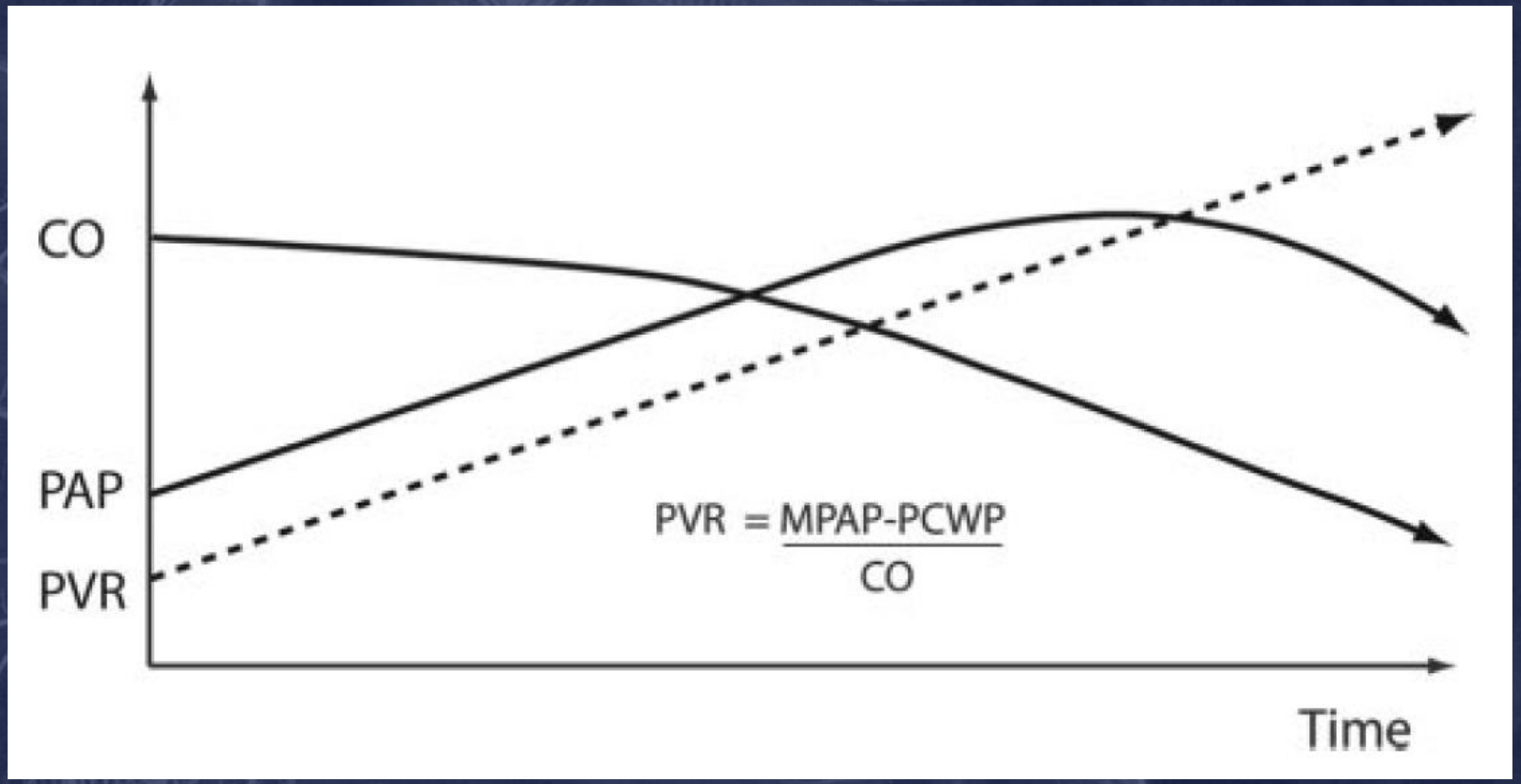
A. PAP = 25/15

B. PAP = 52/30

C. PAP = 80/38

D. I need more data.

RV response to afterload:



Haddad F, et al. Circulation 2008; 117:1717-31

How do you define the risk attributed to RV dysfunction associated with cardiac surgery?

Exact definitions of risk factors in Europe (EuroSCORE) and America (STS)

Risk factor	EuroSCORE definition	STS definition match
Age	Per 5 years or part thereof over 60 years	Per 5 years or part thereof over 60 years
Sex	Female	Female
Chronic pulmonary disease	Long-term use of bronchodilators or steroids for lung disease	Patient required pharmacologic therapy for the treatment of chronic pulmonary compromise, or patient has a FEV1 <75% of predicted value
Extracardiac arteriopathy	Any one or more of the following: claudication, carotic occlusion or >50% stenosis, previous or planned intervention on the abdominal aorta, limb arteries or carotids	Patient has peripheral vascular disease as indicated by claudication either with exertion or rest; amputation for arterial insufficiency; aorto-iliac occlusive disease reconstruction; peripheral vascular bypass surgery, angioplasty or stent; documented AAA, AAA repair, or stent; positive non-invasive testing documented – or – Patient has cerebrovascular disease, documented by any one of the following: Unresponsive coma >24 h; CVA (symptoms >72 h after onset); RIND (recovery within 72 h); TIA (recovery within 24 h); or non-invasive carotid test with >75% occlusion
Neurological dysfunction disease	Severely affecting ambulation or day-to-day functioning	A central neurologic deficit persisting more than 24 h
Previous cardiac surgery	Requiring opening of the pericardium	Prior cardiac surgical operation(s) with or without the use of cardiopulmonary
Serum creatinine	> 200 mmol/l preoperatively	> 20 mmol/l preoperatively
Active endocarditis	Patient still under antibiotic treatment for endocarditis at the time of surgery	Patien rrently under antibiotic treatment for endocarditis at the time of surg
Critical preoperative state	Any one of more of the following: ventricular tachycardia or fibrillation or aborted sudden death, preoperative cardiac massage, preoperative ventilation before arrival in the anaesthetic room, preoperative inotropic support, intra-aortic balloon counterpulsation or preoperative acute renal failure (anuria or oliguria <10 ml/h)	A ne or more of the following: sustained ventricular tachycardia or ventricular fibrillation requiring cardioversion and/or IV amiodarone, preoperative inotropic support, preoperative intra-aortic balloon pump, or patient required cardiopulmonary resuscitation within 1 h before the start of the perative procedure
Unstable angina	Rest angina requiring iv nitrates until arrival in the anaesthetic room	Preoperative use of iv nitrates
LV dysfunction	Moderate or LVEF 30–50%; Poor or LVEF <30%	LVEF 30–50%; LVEF <30%
Recent myocardial infarction	<90 days	<21 days
Pulmonary hypertension	Systolic PA pressure >60 mmHg	Systolic PA pressure >30 mmHg
Emergency	Carried out on referral before the beginning of the next working day	Procedure status is emergent or salvage. *Emergent*: The patient's clinical status includes any of the following. a. Ischaemic dysfunction (any of the following): (1) ongoing ischaemia including rest angina despite maximal medical therapy (medical and/or IABP); (2) acute evolving myocardial infarction within 24 h before surgery; or (3) pulmonary oedema requiring intubation. b. Mechanical dysfunction (either of the following): (1) shock with circulatory support; or (2) shock without circulatory support. *Salvage: The patient is undergoing CPR en route to the OR or prior to anaesthesia induction*
Other than isolated CABG	Major cardiac procedure other than or in addition to CABG	Any valve procedure in addition to or separate from CABG
Surgery on thoracic aorta	For disorder of ascending, arch or descending aorta	Aortic aneurysm/dissection repair
Post-infarct septal rupture		Ventricular septal defect

Bernstein AD, et al. Ann Thorac Surg 2000;69:823-8
Nashef SA, et al. Eur J Cardiothorac Surg 2002;22:101-

Precardiopulmonary Bypass Right Ventricular Function Is Associated with Poor Outcome After Coronary Artery Bypass Grafting in Patients with Severe Left Ventricular Systolic Dysfunction

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Patients with severe left ventricular systolic dysfunction (LVSD) undergoing coronary artery bypass grafting (CABG) have an increased risk for morbidity and mortality. The purpose of this study was to assess the association of pre-CABG right ventricular (RV) function with outcome for patients with severe LVSD. We performed a retrospective evaluation of 41 patients with severe LVSD (left ventricular ejection fraction [LVEF] ≤25%) scheduled for nonemergent CABG. Data were obtained from review of medical records, transesophageal echocardiography tapes, and phone interview. The pre- and post-cardiopulmonary bypass (CPB) LVEF and the RV fractional area of contraction (RVFAC) were calculated by using intraoperative transesophageal echocardiography. Group 1 patients had an RVFAC ≤35% ($n = 7$), whereas Group 2 patients had RVFAC >35% ($n = 34$). The durations of mechanical ventilation and of intensive care unit and hospital stays are presented as the median. Pre-CABG LVEF was similar between Groups 1 and 2 (15.8% ± 3.3% versus 17.8% ± 3.9%). Compared with Group 2, Group 1 patients required greater duration of mechanical ventilation (12 days versus 1 day; $P < 0.01$), longer intensive care unit (14 versus 2 days; $P < 0.01$) and hospital (14 versus 7 days; $P = 0.02$) stays, had a more frequent incidence and severity of LV diastolic dysfunction, and had a smaller change in LVEF immediately after CPB (4.1% ± 8.3% versus 12.5% ± 9.2%; $P = 0.03$). All Group 1 patients died of cardiac causes within 2 yr of surgery; five died during the same hospital admission. Three Group 2 patients died: one of colon cancer at 18 mo after CABG and two of cardiac causes 24 and 48 mo after surgery. A fourth patient was awaiting cardiac transplantation 4 yr after surgery. The remaining Group 2 patients were New York Heart Association Classification I or II. For patients with severe LVSD undergoing CABG, pre-CPB RV dysfunction was associated with poor outcome. Patients with RVFAC >35% had a relatively uneventful perioperative course and good long-term survival, whereas patients with RVFAC ≤35% had a poor early and late outcome. Assessment of RV function is useful to further assess the risk of CABG.

(Anesth Analg 2002;95:1507–18)

- Preop RV dysfunction associated with:
  - greater duration mechanical ventilation
  - increased hospital LOS
  - more frequent and severe LV dysfunction

Maslow Ad, et al. Anesth Analg 2002;95:1507-18

Right Ventricular Myocardial Performance Index Predicts Perioperative Mortality or Circulatory Failure in High-Risk Valvular Surgery

François Haddad, MD, André Y. Denault, MD, Pierre Couture, MD, Raymond Cartier, MD, Michel Pellerin, MD, Sylvie Levesque, MSc, Jean Lambert, PhD, and Jean-Claude Tardif, MD, *Montreal, Quebec, Canada*

Background: **The prognostic value of right ventricular myocardial performance index (RVMPI) and right ventricular fractional area change (RVFAC) in mitral or aortic valve surgery has not been well described. The main objective of this study is to assess the prognostic value of RVMPI and RVFAC in predicting postoperative mortality or circulatory failure.**

Methods: **RVMPI and RVFAC were prospectively measured after induction of anesthesia using transesophageal echocardiography in 50 consecutive patients undergoing corrective mitral or aortic valve surgery. Univariate and multivariate analyses were performed for the primary clinical end point of in-hospital mortality or circulatory failure.**

Results: **In the study population, the mean age was 67 ± 9 years. The primary end point occurred in 17 patients (34%); three patients died, and 14 patients presented signs of circulatory failure. Multivariate regression analysis identified RVMPI and RVFAC as variables of prognostic significance.**

Conclusion: **Preoperative RVMPI and RVFAC could have an incremental value in predicting postoperative mortality and morbidity in valvular heart surgery. Future studies are needed to validate these results in a larger population.** (J Am Soc Echocardiogr 2007;20:1065-1072.)

- Preop RV dysfunction is significantly correlated to in-hospital mortality and/or circulatory failure
- Better predictor than PAP

Haddad F, et al. J Am Soc Echocardiogr 2007;20:1065-72

Prognostic Value of Biventricular Function in Hypotensive Patients After Cardiac Surgery as Assessed by Transesophageal Echocardiography

Constant L.A. Reichert, MD, Cees A. Visser, MD, Renee B.A. van den Brink, MD, Jacques J. Koolen, MD, Harry B. van Wezel, MD, Adriaan C. Moulijn, MD, Arend J. Dunning, MD

In patients after cardiac surgery, hypotension, defined as a mean arterial pressure less than 65 mmHg despite adequate filling pressures and positive inotropic medication, poses a problem. In addition, it is often difficult to determine whether these patients have suffered irreversible myocardial injury or if they are likely to recover. In this study, left and right ventricular function, as assessed by transesophageal echocardiography (TEE), was related to mortality both (1) quantitatively, using fractional area change (FAC), and (2) qualitatively, using a segmental wall motion analysis, which assigned a score to myocardial wall segments, in order to determine whether this technique can be used to predict survival. Mortality rate was very high in patients with biventricular and especially right ventricular failure (FAC < 35%). Left and right ventricular wall motion abnormality indices were significantly better in survivors compared to nonsurvivors, but no distinct cut-off value could be determined. A wall motion index derived from only 6 segments at the mid-papillary muscle level was found to be as reliable as one based on 16 segments of the entire left ventricle. Thus, TEE provided information about the degree of left and right ventricular dysfunction by using a single cross-section at the papillary muscle level. It identified patients at high risk of death, ie, those with compromised right and biventricular function.

KEY WORDS: transesophageal echocardiography, hypotension, ventricular function, cardiac surgery, ejection fraction

- LV failure alone: mortality 20-40%
- Biventricular failure: mortality 86%

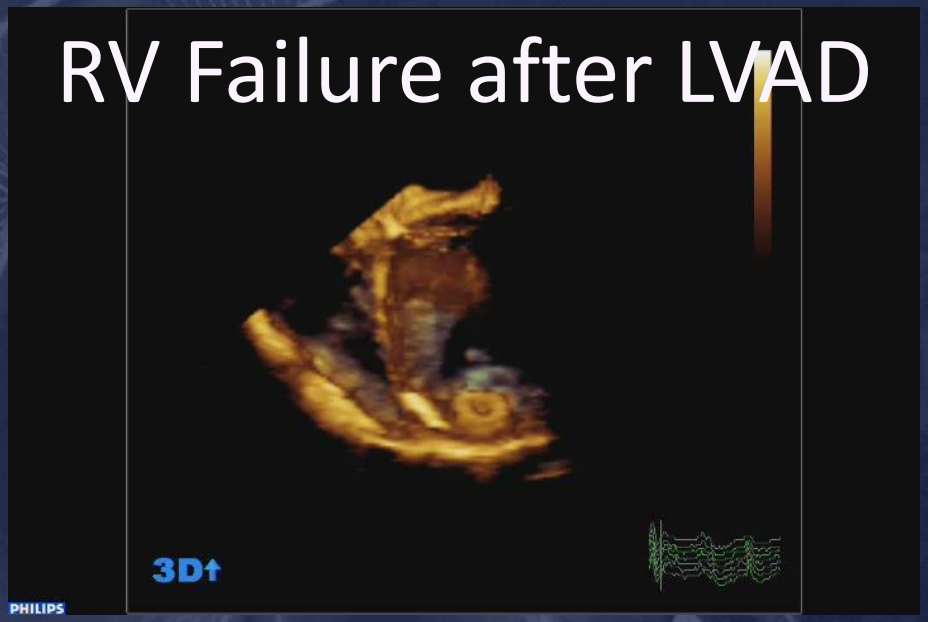
Reichert CL, et al. J Cardiothorac Vasc Anesth 1992;6:429-32

Why is RV failure worse after CPB?

- Inflammatory mediators
  - Lack of equilibrium between

    NO, prostacyclins, thromboxane A2, endothelin
- Ischemic/reperfusion injury
- Protamine administration
- Pulmonary microembolism (clot or air)
- RV ischemia
- Acidosis, hypothermia, fluid shifts
- Dysrhythmias

RV Failure after LVAD



Unloading of left heart

Ventricular septal shift

Lower septal contribution to RV contraction

RV geometry and contractility altered

More RV volume

Predictors of Severe Right Ventricular Failure After Implantable Left Ventricular Assist Device Insertion: Analysis of 245 Patients

Yoshie Ochiai, MD; Patrick M. McCarthy, MD; Nicholas G. Smedira, MD; Michael K. Banbury, MD; Jose L. Navia, MD; Jingyuan Feng, MS; Amy P. Hsu, MS; Michael L. Yeager, RN; Tiffany Buda, RN; Katherine J. Hoercher, RN; Michael W. Howard, MD; Masami Takagaki, MD, PhD; Kazuyoshi Doi, MD; Kiyotaka Fukamachi, MD, PhD

Background—Insertion of an implantable left ventricular assist device (LVAD) complicated by early right ventricular (RV) failure has a poor prognosis and is largely unpredictable. Prediction of RV failure after LVAD placement would lead to more precise patient selection and optimal device selection.

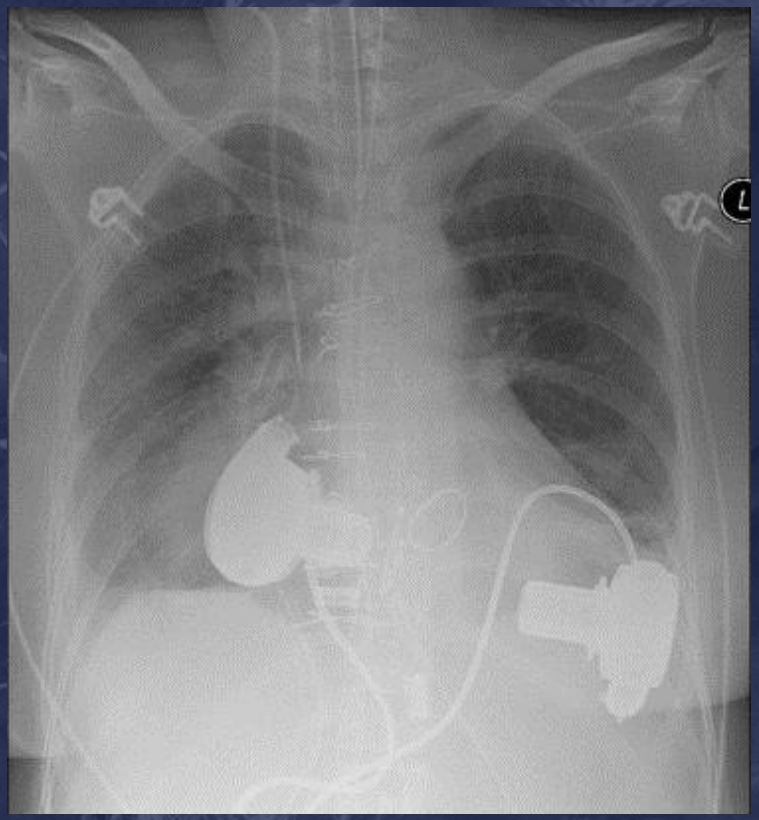
Methods and Results—We reviewed data from 245 patients (mean age, 54±11 years; 85% male) with 189 HeartMate (77%) and 56 Novacor (23%) LVADs. Ischemic cardiomyopathy predominated (65%), and 29% had dilated cardiomyopathy. Overall, RV assist device (RVAD) support was required after LVAD insertion for 23 patients (9%). We compared clinical and hemodynamic parameters before LVAD insertion between RVAD (n=23) and No-RVAD patients (n=222) to determine preoperative risk factors for severe RV failure. By univariate analysis, female gender, small body surface area, nonischemic etiology, preoperative mechanical ventilation, circulatory support before LVAD insertion, low mean and diastolic pulmonary artery pressures (PAPs), low RV stroke work (RVSW), and low RVSW index (RVSWI) were significantly associated with RVAD use. Elevated PAP and pulmonary vascular resistance were not risk factors. Risk factors by multivariable logistic regression were preoperative circulatory support (odds ratio [OR], 5.3), female gender (OR, 4.5), and nonischemic etiology (OR, 3.3).

Conclusions—The need for circulatory support, female gender, and nonischemic etiology were the most significant predictors for RVAD use after LVAD insertion. Regarding hemodynamics, low PAP and low RVSWI, reflecting low RV contractility, were important parameters. This information may lead to better patient selection for isolated LVAD implantation. **(*Circulation*. 2002;106[suppl I]:I-198-I-202.)**

- RV failure requiring RVAD after LVAD occurs in 10-30%
- Associated with high mortality

Ochiai Y, et al. Circulation 2002;106:I198-I202

Risk Factors for RVAD after LVAD



Heartware BIVAD

Factors	Odds Ratio	95% CI	*P*-value
Preoperative circulatory support	5.3	2.0–14.0	0.001
Female gender	4.5	1.7–12.4	0.003
Nonischemic etiology	3.3	1.3–8.4	0.015

TABLE 2. Preoperative Hemodynamic Variables

Variables	RVAD (n=23)	No-RVAD (n=222)	*P*-value univariate
Cardiac output (L/min)	3.4±0.8 (n=17)	3.8±1.2 (n=189)	0.205*
Cardiac index (L/min/m^2)	1.8±0.4 (n=17)	1.9±0.6 (n=189)	0.621*
LAP (mm Hg)	23±5 (n=9)	24±8 (n=125)	0.713†
RAP (mm Hg)	20±7 (n=17)	19±7 (n=188)	0.354*
Systolic PAP (mm Hg)	47±11 (n=17)	53±13 (n=194)	0.118*
Mean PAP (mm Hg)	33±7 (n=17)	37±9 (n=194)	0.041†
Diastolic PAP (mm Hg)	25±7 (n=17)	29±8 (n=194)	0.030*
Heart rate (bpm)	104±18 (n=16)	98±20 (n=178)	0.186*
PVR (dynes · sec · cm^{-5})	271±194 (n=8)	317±200 (n=117)	0.379*
PVRI (dynes · sec · cm^{-5}/m^2)	467±325 (n=8)	614±379 (n=117)	0.230*
RVSW (mm Hg · mL)	543±392 (n=14)	780±437 (n=166)	0.037*
RVSWI (mm Hg · mL/m^2)	285±196 (n=14)	400±221 (n=166)	0.046*

Ochiai Y, et al. Circulation 2002;106:I198-I202

REGISTRY REPORT

Registry of the International Society for Heart and Lung Transplantation: Tenth Official Pediatric Lung and Heart/Lung Transplantation Report—2007

Paul Aurora, MRCP, PhD, Mark M. Boucek, MD, Jason Christie, MD, MS, Fabienne Dobbels, PhD, Leah B. Edwards, PhD, Berkeley M. Keck, MPH, Axel O. Rahmel, MD, David O. Taylor, MD, Elbert P. Trulock, MD, and Marshall I. Hertz, MD

This tenth official pediatric report of the International Society for Heart and Lung Transplantation (ISHLT) covers the international pediatric lung and heart-lung transplantation experience from 1982 to 2006. As of last year's report, pediatric lung and heart-lung transplant data are now reported separately from pediatric heart transplant data and adult lung transplant data. For the first time this year, Registry data are analyzed by geographic region in addition to the usual aggregate analyses. All figures and tables included in this report and additional supplementary slides are available from the ISHLT website (www.ishlt.org/registries). J Heart Lung Transplant 2007;26:1223-8.

Acute RV failure accounts for
up to 20% of deaths following OHT

J Heart Lung Transplant 2007;26:1233-8

Risk Factors for RV Failure after OHT

1. Most common: Donor heart not adapted to recipient PH (preexisting or acquired)

2. Marginal organ preservation/long ischemic time/reperfusion injury

3. Mechanical obstruction of PA anastomosis

4. Significant donor-recipient size mismatch

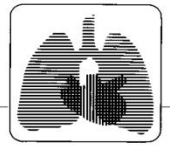
5. Acute allograft rejection

J Heart Lung Transplant 2007;26:1233-8

How can we manage RV failure?

1. Optimize RV preload
2. Increase RV contractility
3. Decrease RV afterload
4. Optimize blood pressure
5. Treat dysrhythmias
6. Mechanical support

1. Optimize preload



critical care review

Predicting Fluid Responsiveness in ICU Patients*

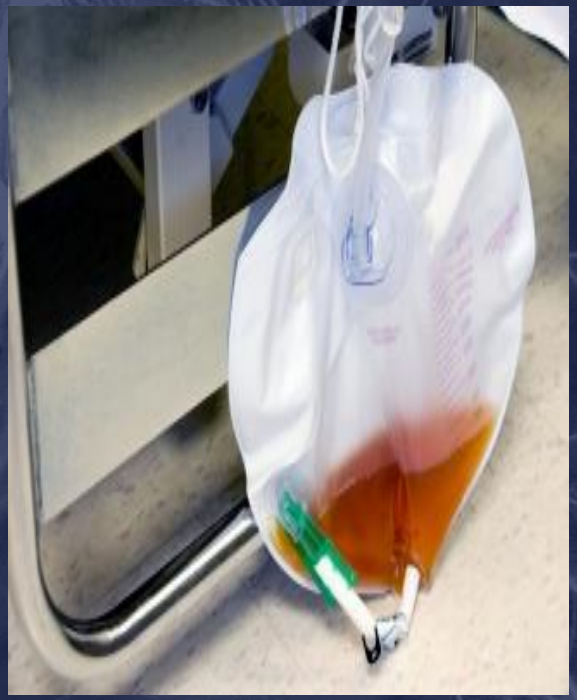
A Critical Analysis of the Evidence

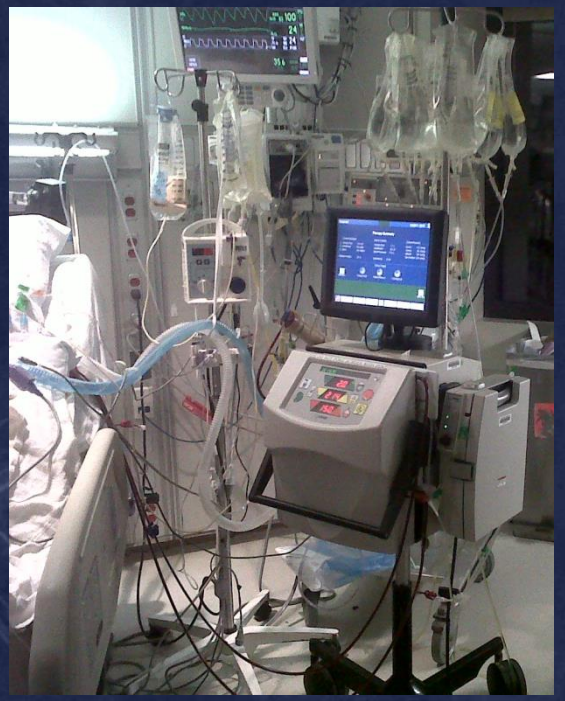
Frédéric Michard, MD, PhD; and Jean-Louis Teboul, MD, PhD

Study objective: **To identify and critically review the published peer-reviewed, English-language studies investigating predictive factors of fluid responsiveness in ICU patients.**
Design: **Studies were collected by doing a search in MEDLINE (from 1966) and scanning the reference lists of the articles. Studies were selected according to the following criteria: volume expansion performed in critically ill patients, patients classified in two groups (responders and nonresponders) according to the effects of volume expansion on stroke volume or on cardiac output, and comparison of responder and nonresponder patients' characteristics before volume expansion.**
Results: **Twelve studies were analyzed in which the parameters tested were as follows: (1) static indicators of cardiac preload (right atrial pressure [RAP], pulmonary artery occlusion pressure [PAOP], right ventricular end-diastolic volume [RVEDV], and left ventricular end-diastolic area [LVEDA]); and (2) dynamic parameters (inspiratory decrease in RAP [ΔRAP], expiratory decrease in arterial systolic pressure [Δdown], respiratory changes in pulse pressure [ΔPP], and respiratory changes in aortic blood velocity [ΔVpeak]). Before fluid infusion, RAP, PAOP, RVEDV, and LVEDA were not significantly lower in responders than in nonresponders in three of five studies, in seven of nine studies, in four of six studies, and in one of three studies, respectively. When a significant difference was found, no threshold value could discriminate responders and nonresponders. Before fluid infusion, ΔRAP, Δdown, ΔPP, and ΔVpeak were significantly higher in responders, and a threshold value predicted fluid responsiveness with high positive (77 to 95%) and negative (81 to 100%) predictive values.**
Conclusion: **Dynamic parameters should be used preferentially to static parameters to predict fluid responsiveness in ICU patients.** ***(CHEST 2002; 121:2000–2008)***

Michard F, et al. Chest 2002;121:2000-8

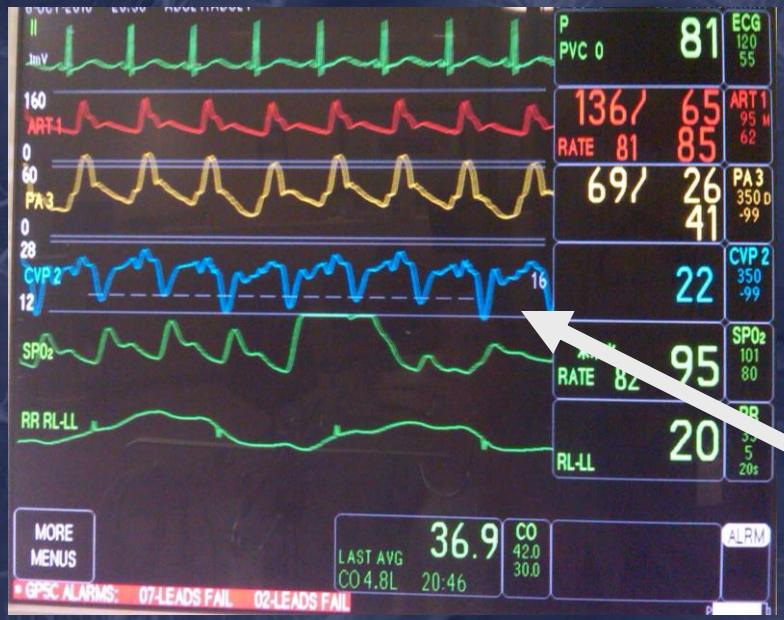
1. Optimize preload





- Problem = RV can accommodate large volume
- ….but less ability to pump it out

CVP?


P
PVC 0
81
ECG
120
55
160
ART 1
0
136/ 65
RATE 81
85
ART 1
95
62
60
PA 3
0
69/ 26
41
PA 3
350
-99
28
CVP 2
12
22
CVP 2
350
-99
SPO2
RATE 82
95
SPO2
101
80
RR RL-LL
RL-LL
20
5
20s
MORE
MENUS
LAST AVG
CO 4.8L
20:46
36.9
CO
42.0
30.0
ALARM
* GPSC ALARMS:
07-LEADS FAIL
02-LEADS FAIL

2. Increase Contractility

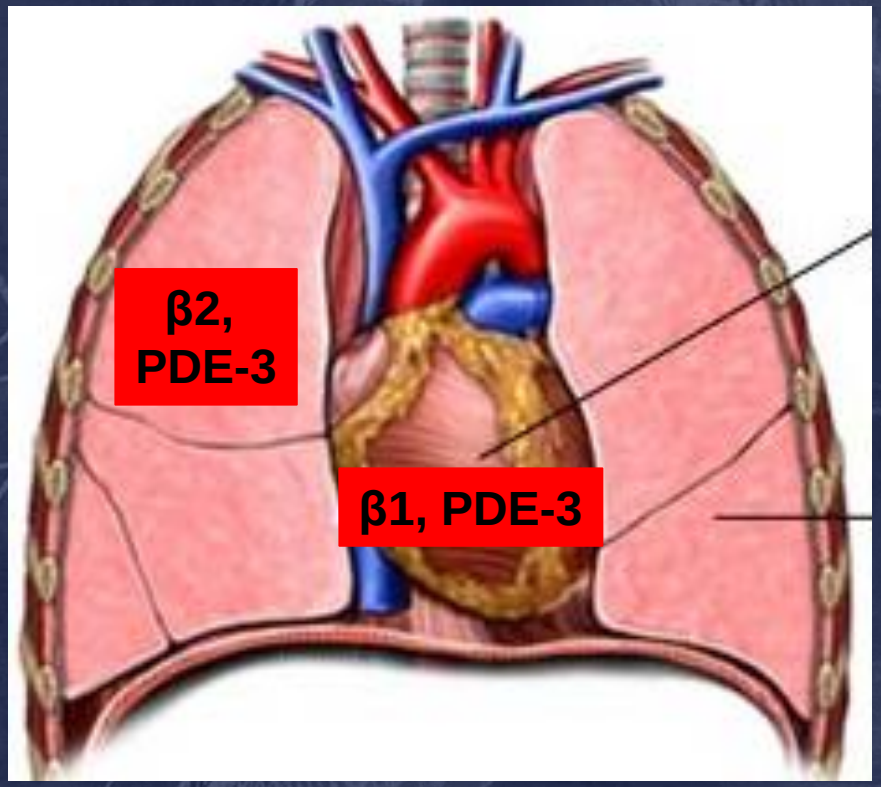
cAMP
β1, 2

cAMP
PDE-3 inhibition

cAMP + Inositol phospholipid calcium

Calcium sensitizer + ATP-dep K^+ channel

2. Increase Contractility:
Dobutamine and Milrinone



- Inodilation and lusitropy
- ↓ PAP, PVR, SVR, PCWP
- ↑ CO passively decreases PVR:

Ohm's Law:
V = IR
PAP = PBF x PVR

3. Reduce RV afterload:

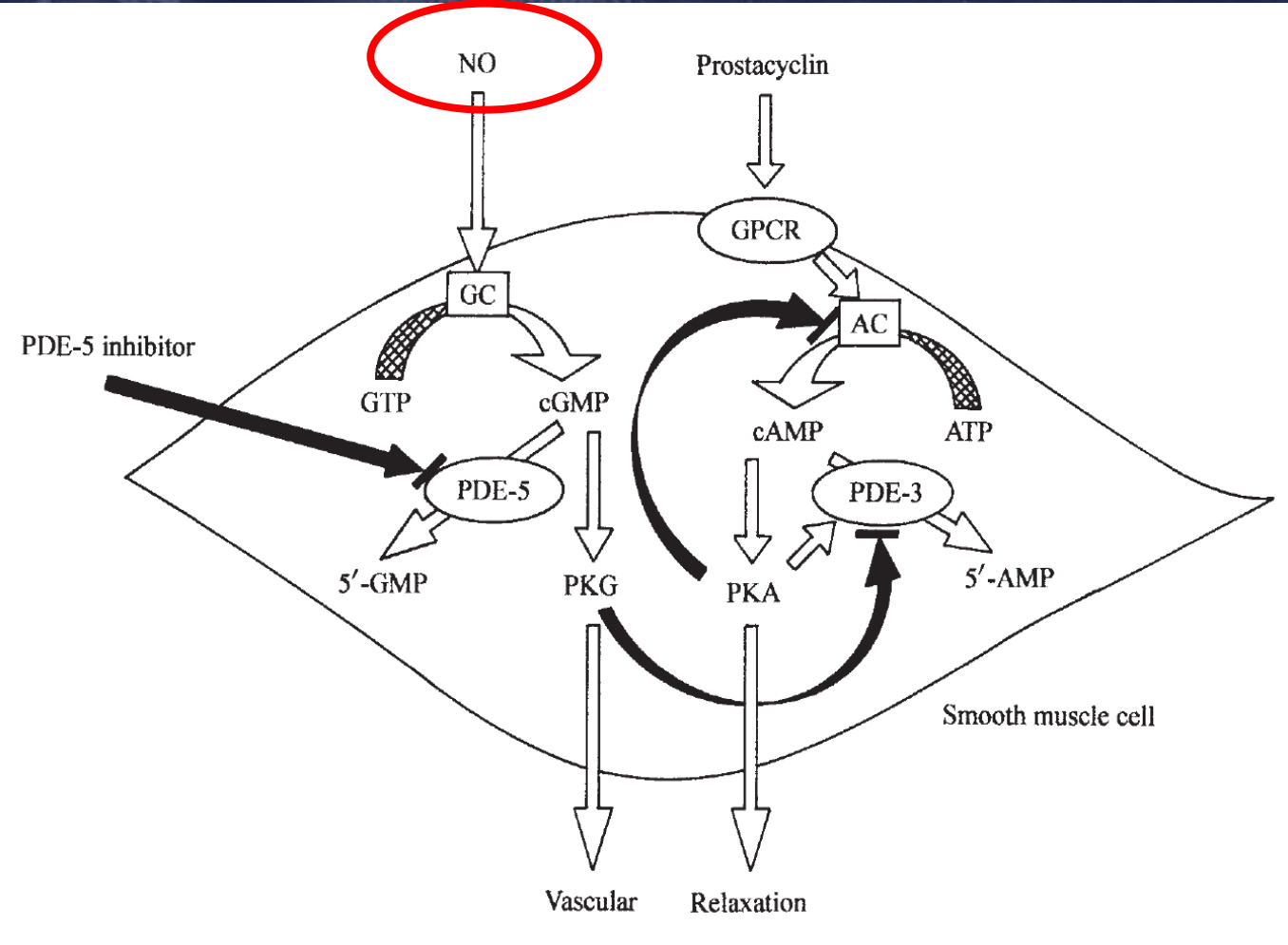
Pulmonary vasodilators

cAMP
Prostacyclin analogs
Phosphodiesterase-3 Inhibitors: Milrinone
Dobutamine (β2)

cGMP
Inhaled Nitric Oxide
Phosphodiesterase-5 Inhibitors: Sildenafil
Synthetic BNP: Nesiritide

Effects of cyclic AMP and cyclic GMP
Anti-remodelling
Vasodilatory
Anti-inflammatory
Anti-apoptotic
Inotropic

3. Reduce RV afterload:
Inhaled Nitric Oxide



Watanabe H, Internal Med 2004 Oct;43(10):891-3

3. Reduce RV afterload:
Inhaled Nitric Oxide

- Potent pulmonary vasodilator
  - cGMP $t_{1/2}$ is 1 minute; NO action ends when withdrawn
  - Affinity of Hb for NO is 3,000 times > O_2
- Anti-platelet activity
- Protects K+ channel function
- Improves V/Q matching
  - Effects limited to ventilated areas
    - attenuates HPV
    - improves oxygenation without increasing intrapulmonary shunt

Randomized, Double-Blind Trial of Inhaled Nitric Oxide in LVAD Recipients With Pulmonary Hypertension

Michael Argenziano, MD, Asim F. Choudhri, BS, Nader Moazami, MD, Eric A. Rose, MD, Craig R. Smith, MD, Howard R. Levin, MD, Arthur J. Smerling, MD, and Mehmet C. Oz, MD

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Background. Pulmonary vascular resistance is often elevated in patients with congestive heart failure, and in those undergoing left ventricular assist device (LVAD) insertion, it may precipitate right ventricular failure and hemodynamic collapse. Because the effectiveness of inotropic and vasodilatory agents is limited by systemic effects, right ventricular assist devices are often required. Inhaled nitric oxide (NO) is an effective, specific pulmonary vasodilator that has been used successfully in the management of pulmonary hypertension.

Methods. Eleven of 23 patients undergoing LVAD insertion met criteria for elevated pulmonary vascular resistance on weaning from cardiopulmonary bypass (mean pulmonary artery pressure >25 mm Hg and LVAD flow rate <2.5 $L \cdot min^{-1} \cdot m^{-2}$) and were randomized to receive either inhaled NO at 20 ppm (n = 6) or nitrogen (n = 5). Patients not manifesting a clinical response after 15 minutes were given the alternative agent.

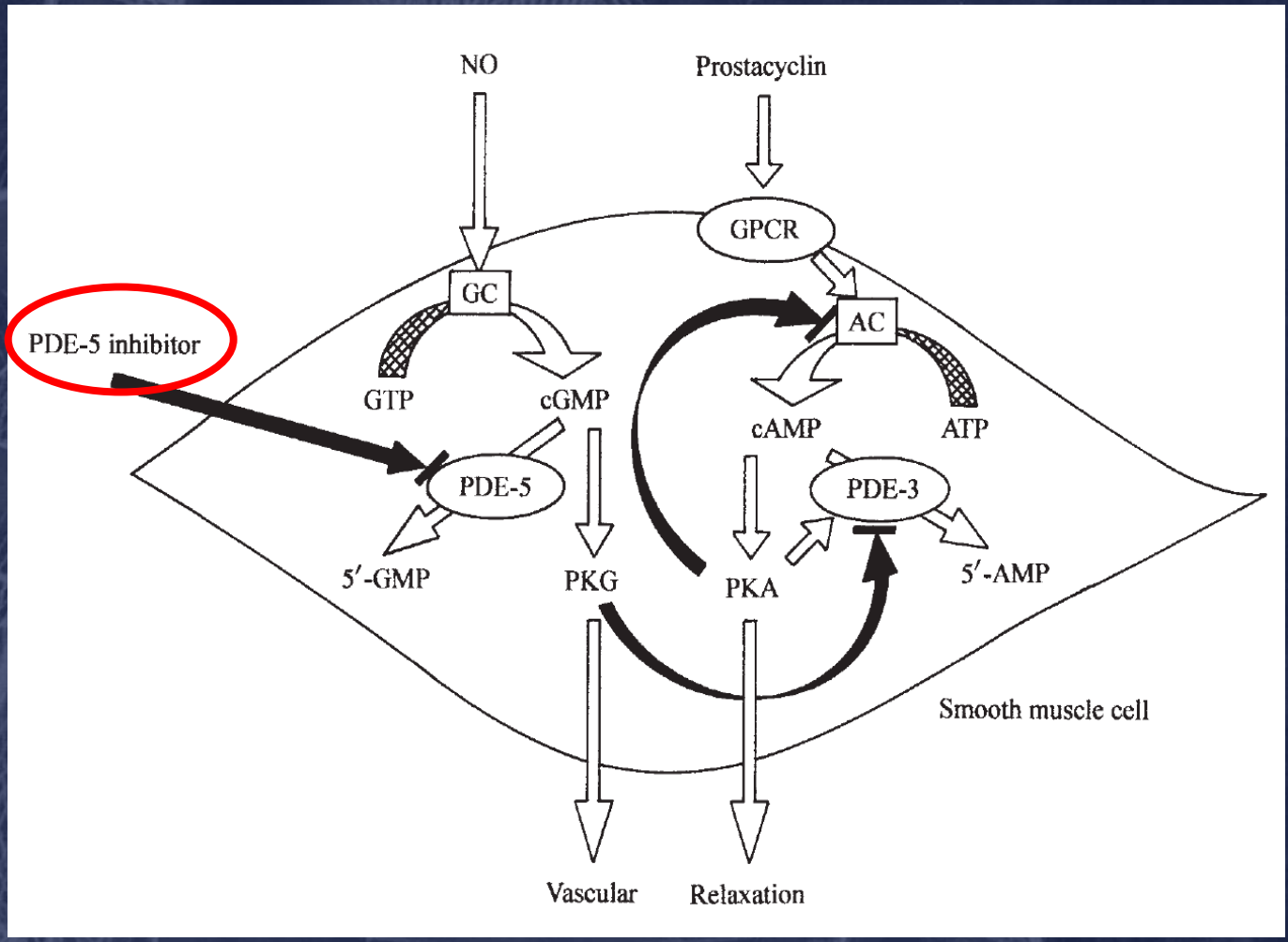
Results. Hemodynamics for the group at randomization were as follows: mean arterial pressure, 72 ± 6 mm Hg; mean pulmonary artery pressure, 32 ± 4 mm Hg; and LVAD flow, 2.0 ± 0.3 $L \cdot min^{-1} \cdot m^{-2}$. Patients receiving inhaled NO exhibited significant reductions in mean pulmonary artery pressure and increases in LVAD flow, whereas none of the patients receiving nitrogen showed hemodynamic improvement. Further, when the nitrogen group was subsequently given inhaled NO, significant hemodynamic improvements ensued. There were no significant changes in mean arterial pressure in either group.

Conclusions. Inhaled NO induces significant reductions in mean pulmonary artery pressure and increases in LVAD flow in LVAD recipients with elevated pulmonary vascular resistance. We conclude that inhaled NO is a useful intraoperative adjunct in patients undergoing LVAD insertion in whom pulmonary hypertension limits device filling and output.

(Ann Thorac Surg 1998;65:340–5)

Argenziano M. Ann Thorac Surg. 1998;65:340-5

3. Reduce RV afterload:
PDE 5 Inhibitors



Watanabe H, Internal Med 2004 Oct;43(10):891-3

PDE-5 Inhibitors

- Selective decrease in pulmonary-to-systemic vascular resistance and increases CO
- No direct anti-platelet effects
- Heightened SNS activity
  - PDE6 present in brain causes blue vision

Effect of Sildenafil on Pulmonary Artery Pressure, Systemic Pressure, and Nitric Oxide Utilization in Patients With Left Ventricular Assist Devices

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Background. Pulmonary artery hypertension can complicate the early postoperative care of patients with left ventricular assist devices (LVADs). Inhaled nitric oxide (INO) is frequently used to manipulate pulmonary resistance after LVADs have been placed. We evaluated the effect of oral sildenafil therapy on pulmonary artery pressure, systemic pressure, and nitric oxide utilization.

Methods. After Institutional Review Board approval, the records of 10 consecutive adult patients with LVADs and pulmonary hypertension who received sildenafil were reviewed. Demographics, surgical history, INO use, inotrope requirements, and hemodynamic response to oral sildenafil at multiple intervals were collected. Hemodynamic data were analyzed with a two-way analysis of variance of repeated measures with correction for multiple comparisons.

Results. There were 8 men and 2 women with 6 Heartmate XVE LVADs and 4 Thoratec LVADs (both, Thoratec, Pleasanton, California). When weaning was attempted, 8 patients who received INO demonstrated rebound pulmonary hypertension or increased right heart dysfunction. All patients were on inotropic therapy with dobutamine and milrinone. Sildenafil produced a significant reduction in pulmonary artery systolic pressure within 90 minutes of oral administration ($p = 0.042$). Significant changes in systolic blood pressure, mean arterial pressure, systemic vascular resistance, and heart rate were not observed. All 8 patients receiving INO were weaned within 12 hours without recurrent pulmonary hypertension. All 10 patients were weaned from inotropic support within 72 hours. No patient suffered right-side heart failure requiring intervention.

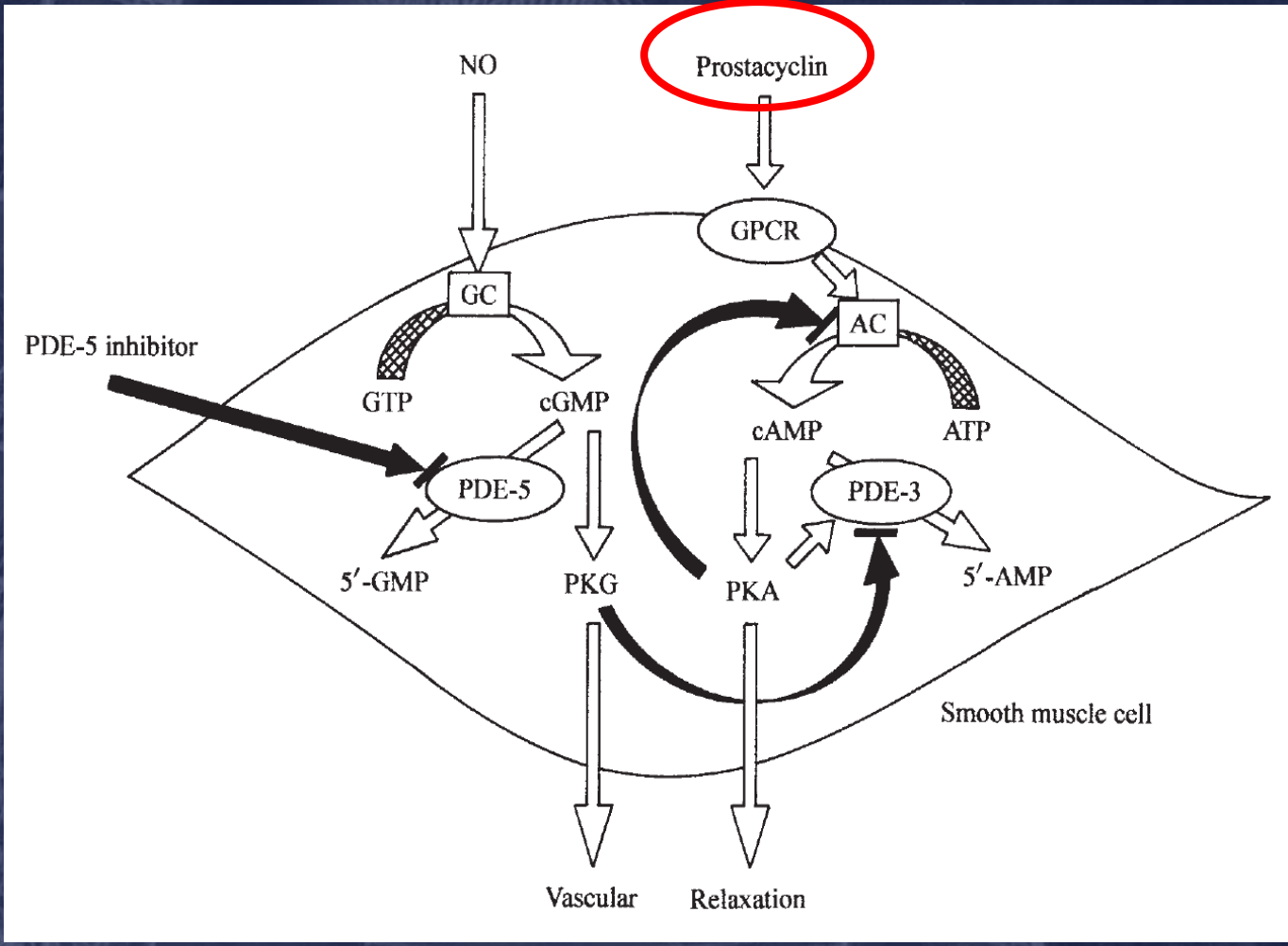
Conclusions. Oral sildenafil represents a useful adjunctive therapy for patients with LVADs. In our series, it provided additional reduction of pulmonary artery pressure, and facilitated weaning from INO and inotropes without deleterious hemodynamic consequences.

(Ann Thorac Surg 2007;83:68–71)

- Sildenafil may provide additional reduction in PAP
  Facilitates weaning if iNO and inotropes

Klodell C. Ann Thorac Surg. 2007. 83;68-71

3. Reduce RV afterload:
Prostacyclin (PGI_2) analogs



Watanabe H, Internal Med 2004 Oct;43(10):891-3

3. Reduce RV afterload:

Prostacyclin (PGI_2) analogs

- Naturally occurring metabolite of arachidonic acid
- Pulmonary vasodilation, anti-proliferative, anti-platelet
  - Iloprost, Ventavis (IV or inhaled)
  - epoprostenol, Flolan (IV or inhaled)
  - trepostinil, Remodulin, Orenitram, Tyvaso (IV, oral, inhaled)

Inhaled iloprost controls pulmonary hypertension after cardiopulmonary bypass

[L'inhalation d'iloprost permet de contrôler l'hypertension pulmonaire après la circulation extracorporelle]

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Purpose: Severe pulmonary hypertension (PH) is a major cause of right ventricular (RV) dysfunction. Various *iv* vasodilator modalities have been used with limited results because of lack of pulmonary selectivity. The aim of the present controlled study was to evaluate the efficacy of inhaled iloprost, a synthetic prostacyclin analogue, in patients with elevated pulmonary vascular resistance (PVR) immediately after separation from cardiopulmonary bypass (CPB).

Methods: Twelve patients with persistent PH after discontinuation of CPB were included in the study. In all patients standard hemodynamic monitoring was used. Inhaled iloprost was administered via nebulized aerosol at a cumulative dose of 0.2 $\mu g \cdot kg^{-1}$ for a total duration of 20 min. Complete sets of hemodynamic measurements were performed before inhalation (baseline), during and after cessation of the inhalation period. Echocardiographic monitoring of RV function was also used.

Results: Inhaled iloprost induced a reduction in the transpulmonary gradient at the end of the inhalation period in comparison to baseline (9.33 ± 3.83 mmHg *vs* 17.09 ± 6.41 mmHg, $P < 0.05$). The mean pulmonary artery pressure to systemic artery pressure ratio decreased over this period (0.28 ± 0.08 *vs* 0.45 ± 0.17, $P < 0.05$). A statistically significant decrease of the PVR to systemic vascular resistance ratio was also observed (0.15 ± 0.05 *vs* 0.21 ± 0.05, $P < 0.05$). Improved indices of RV function were observed in echocardiographic monitoring.

Conclusion: Inhaled iloprost appears to be a selective pulmonary vasodilator and may be effective in the initial treatment of PH and the improvement of RV performance in the perioperative setting.

Objectif : *L'hypertension pulmonaire sévère (HP) est une cause majeure de dysfonction du ventricule droit (VD). Diverses modalités vasodilatatrices iv ont été utilisées et ont donné des résultats limités, étant donné le manque de sélectivité pulmonaire. Notre but était d'évaluer l'efficacité de l'inhalation d'iloprost, un analogue de la prostacycline synthétique, chez des patients qui présentent une résistance vasculaire pulmonaire (RVP) élevée immédiatement après le sevrage de la circulation extracorporelle (CEC).*

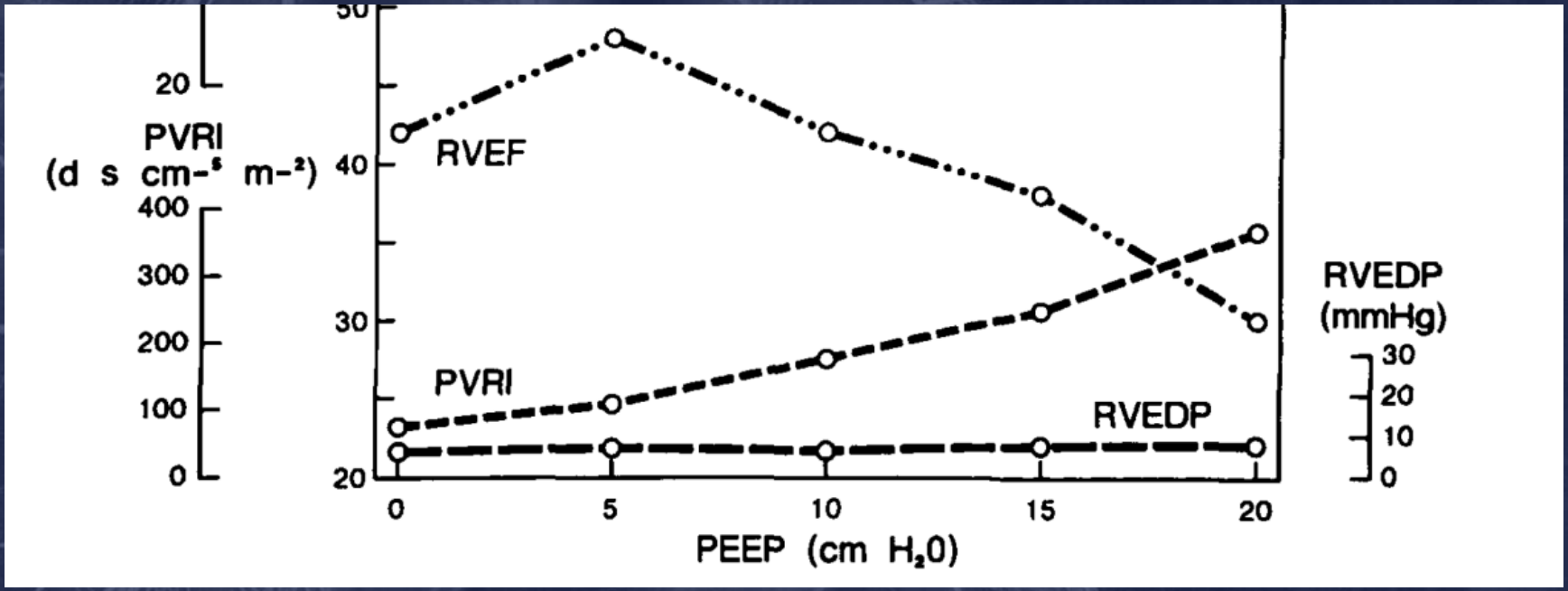
Méthode : *Douze patients présentant une HP persistante après l'interruption de la CEC ont été inclus dans l'étude. Une surveillance standard des paramètres hémodynamiques a été utilisée pour tous les patients. L'administration d'iloprost a été faite à l'aide d'un nébuliseur selon une dose de 0,2 $\mu g \cdot kg^{-1}$ pendant au plus 20 min. Des ensembles complets de mesures hémodynamiques ont été réalisés avant l'inhalation (mesures de base), pendant et après la période d'inhalation. La surveillance échocardiographique de la fonction du VD a aussi été utilisée.*

Résultats : *L'iloprost inhalé a provoqué une réduction du gradient transpulmonaire à la fin de l'inhalation, en comparaison avec les mesures de base (9,33 ± 3,83 mmHg vs 17,09 ± 6,41 mmHg, $P < 0,05$). Le ratio de la pression artérielle pulmonaire moyenne sur la pression artérielle systémique a diminué pendant cette période (0,28 ± 0,08 vs 0,45 ± 0,17, $P < 0,05$). Une baisse statistiquement significative du ratio de la RVP sur la résistance vasculaire générale a été aussi observée (0,15 ± 0,05 vs 0,21 ± 0,05, $P < 0,05$). Des indices supérieurs de la fonction du VD ont été observés par la surveillance échocardiographique.*

- Reduces TPG, mPAP, PVR
- Improved indices of RV function by echocardiography

Theodoraki K. Can J Anesth. 2002;49:963-7

3. Reduce RV afterload:
Effect of Intrathoracic Pressure and PEEP

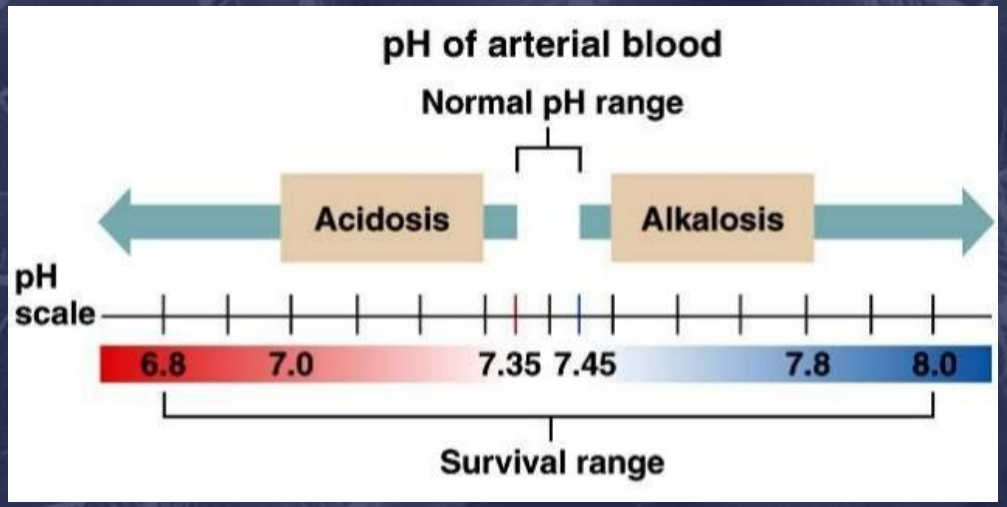


Increased PAP and RV pericardial pressure

Biondi J, et al. Anesth Analg 1988;76:144-51

3. Reduce RV afterload:

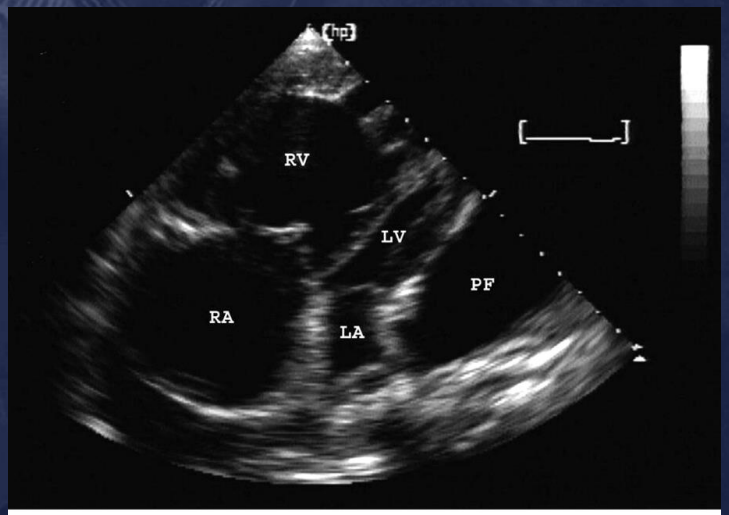
Acidosis and hypoxia



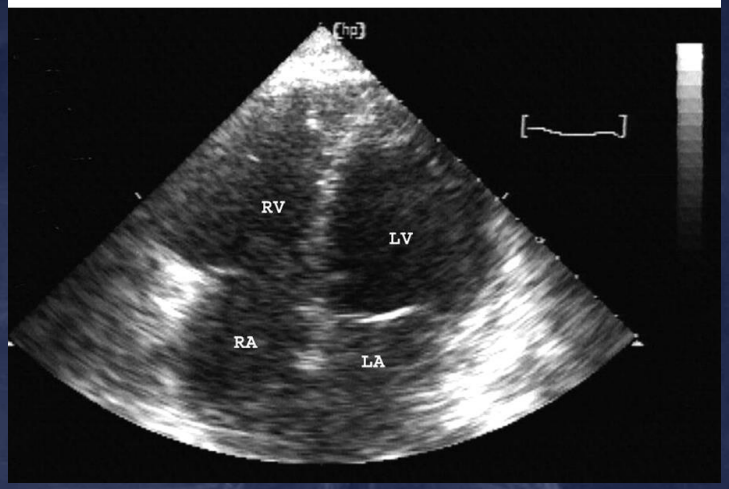
3. Reduce RV afterload:

Pulmonary disease treated with lung transplant

Pre-operative

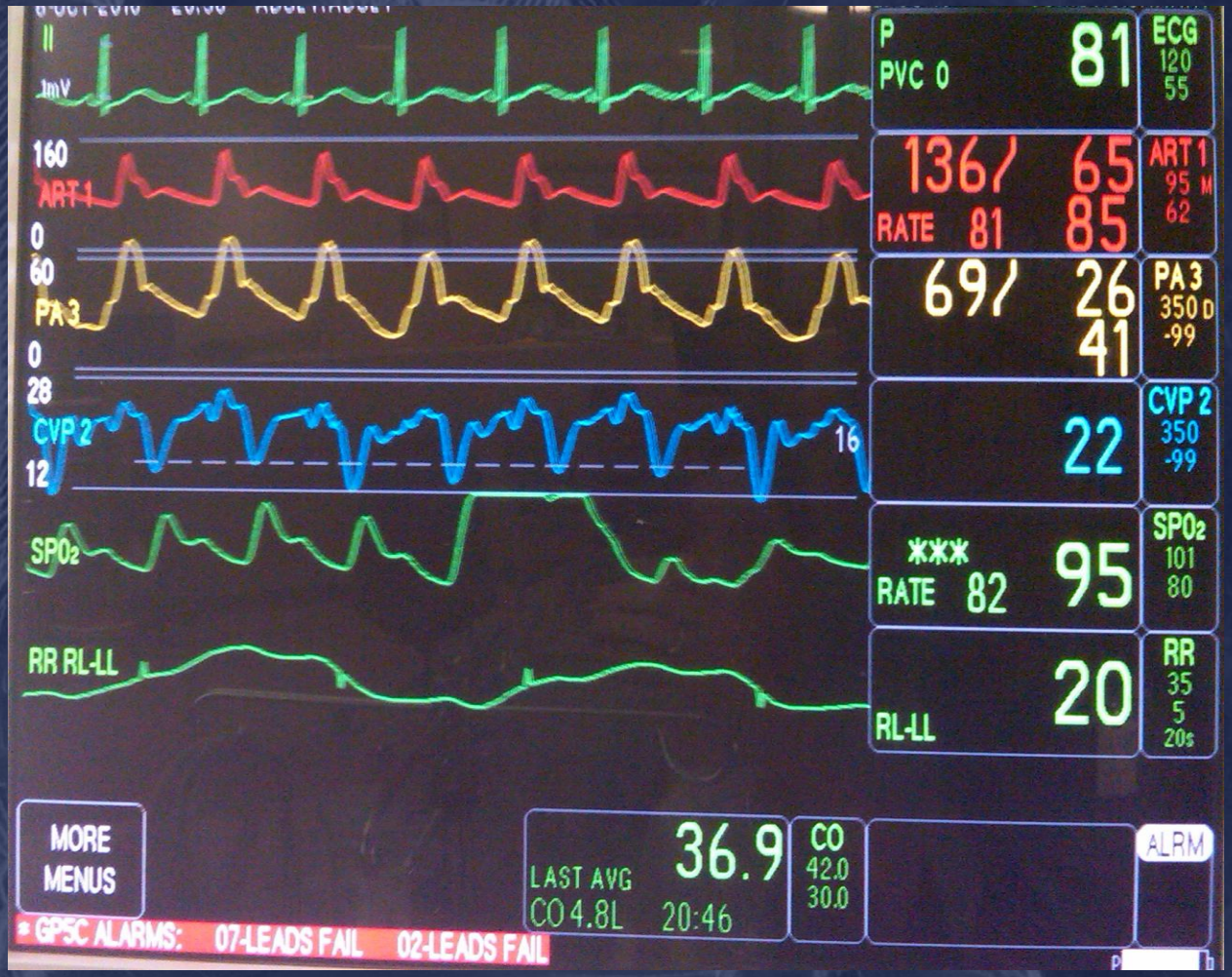


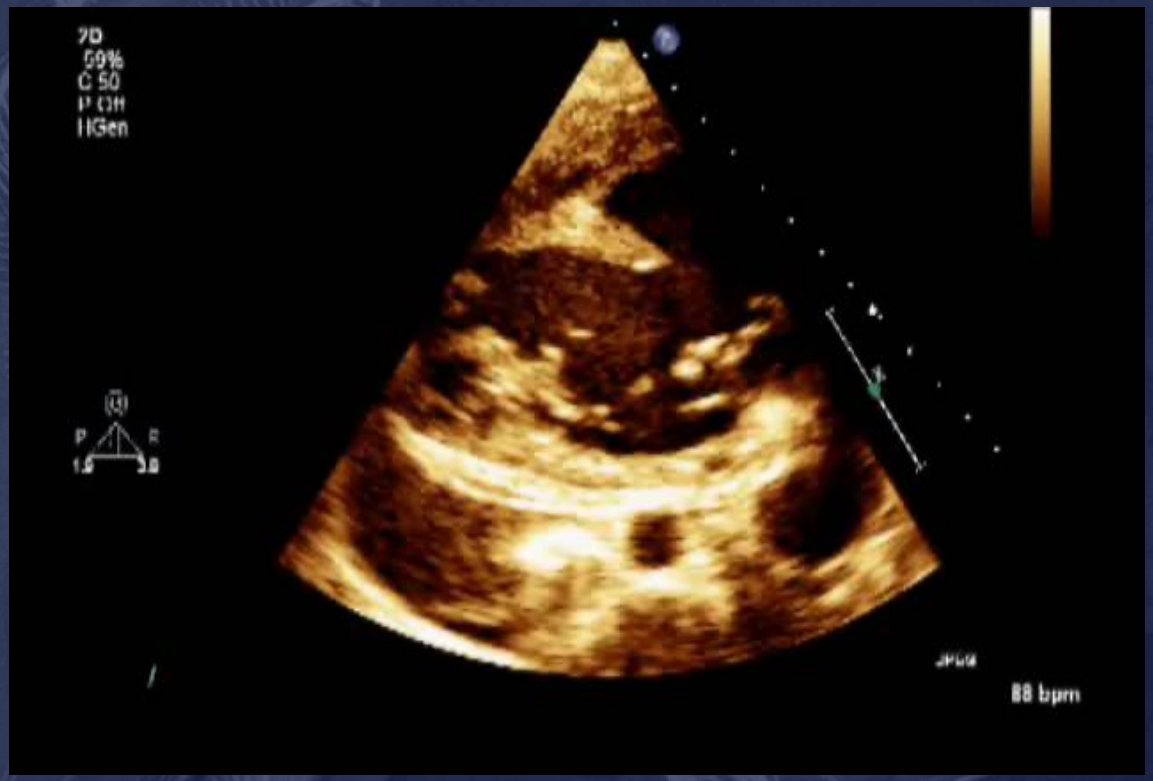
3 months post-lung transplant



Kasimir MT, et al., Eur J Cardiothorac Surg 2004;26:776-781

4. Optimize blood pressure





Hypertrophied RV

↓

Increased wall tension

↓ ↓

Increased O_2 demand

Reduced CPP

4. Optimize Blood Pressure:
Vasopressin vs Norepinephrine

Comparative hemodynamic effects of vasopressin and norepinephrine after milrinone-induced hypotension in off-pump coronary artery bypass surgical patients

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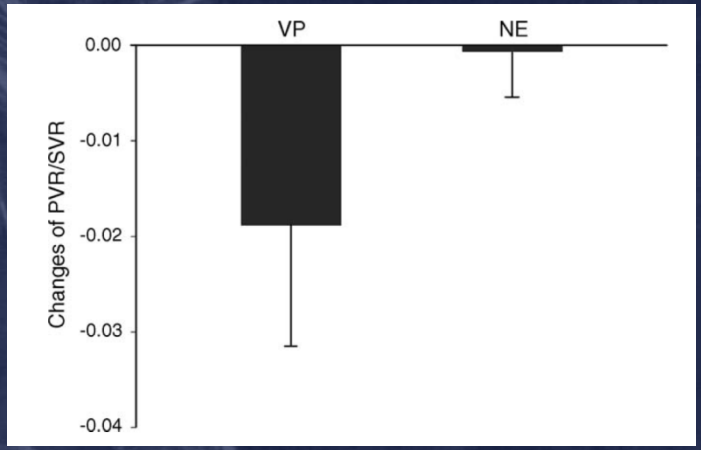
- Vasopressin decreases PVR/SVR ratio; i.e. less effect on PVR
- *Vasopressin can double SVR and only reduce CI by15%



Jeon Y, et al. Eur J Cardiohorac Surg 2006;29:952-6

Effect of Arginine Vasopressin on the Canine Epicardial Coronary Artery. Experiments on V_1-Receptor-Mediated Production of Nitric Oxide

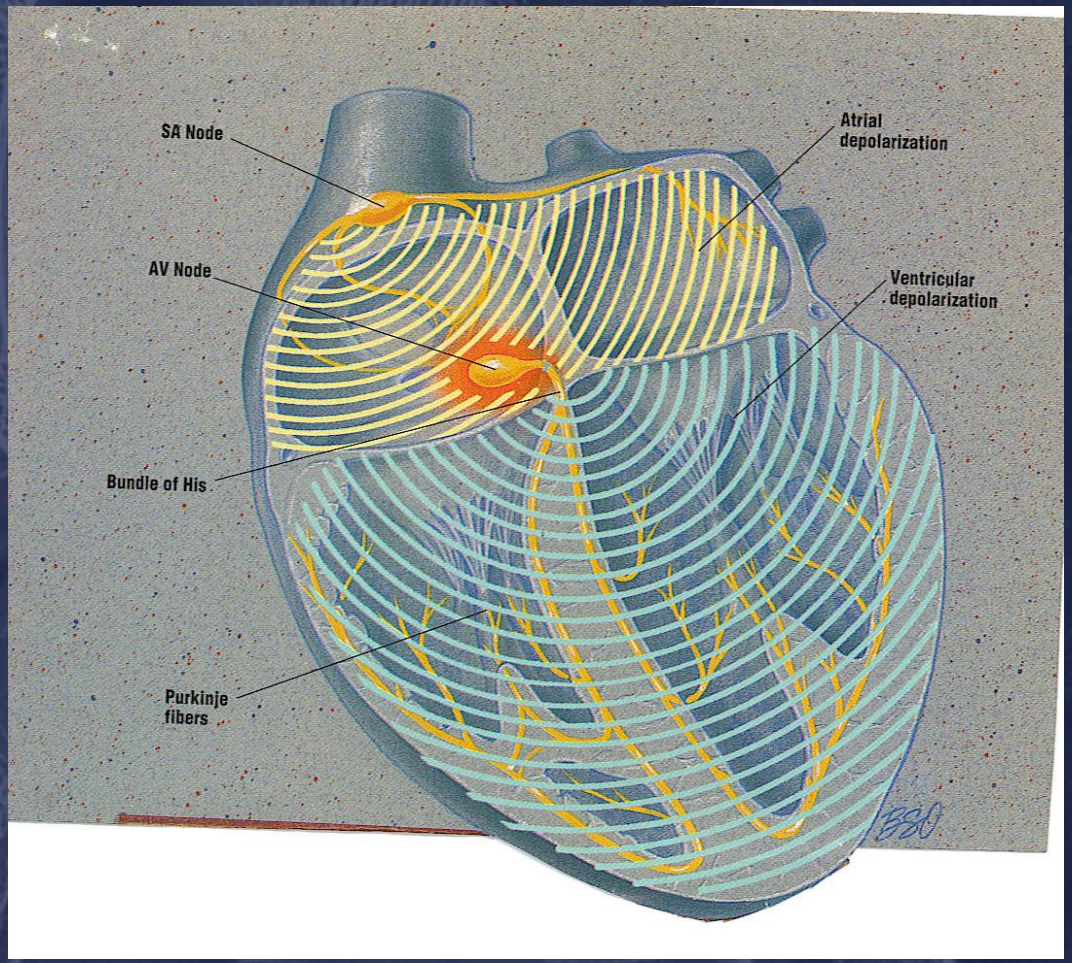
Paulo Roberto B. Evora, Paul J. Pearson, Alfredo J. Rodrigues, Fernanda Viaro, Hartzell V. Schaff

Rochester, MN, USA – Ribeirão Preto, SP - Brazil

- At low plasma concentrations, mediates vasodilation in coronary, cerebral, and pulmonary arterial circulations
  - likely mediated via oxytocin receptor and local NO production
- PVR does not increase until very high levels

Evora PR, et al. Chest 1993:103:1241-5

5. Treat dysrhythmias



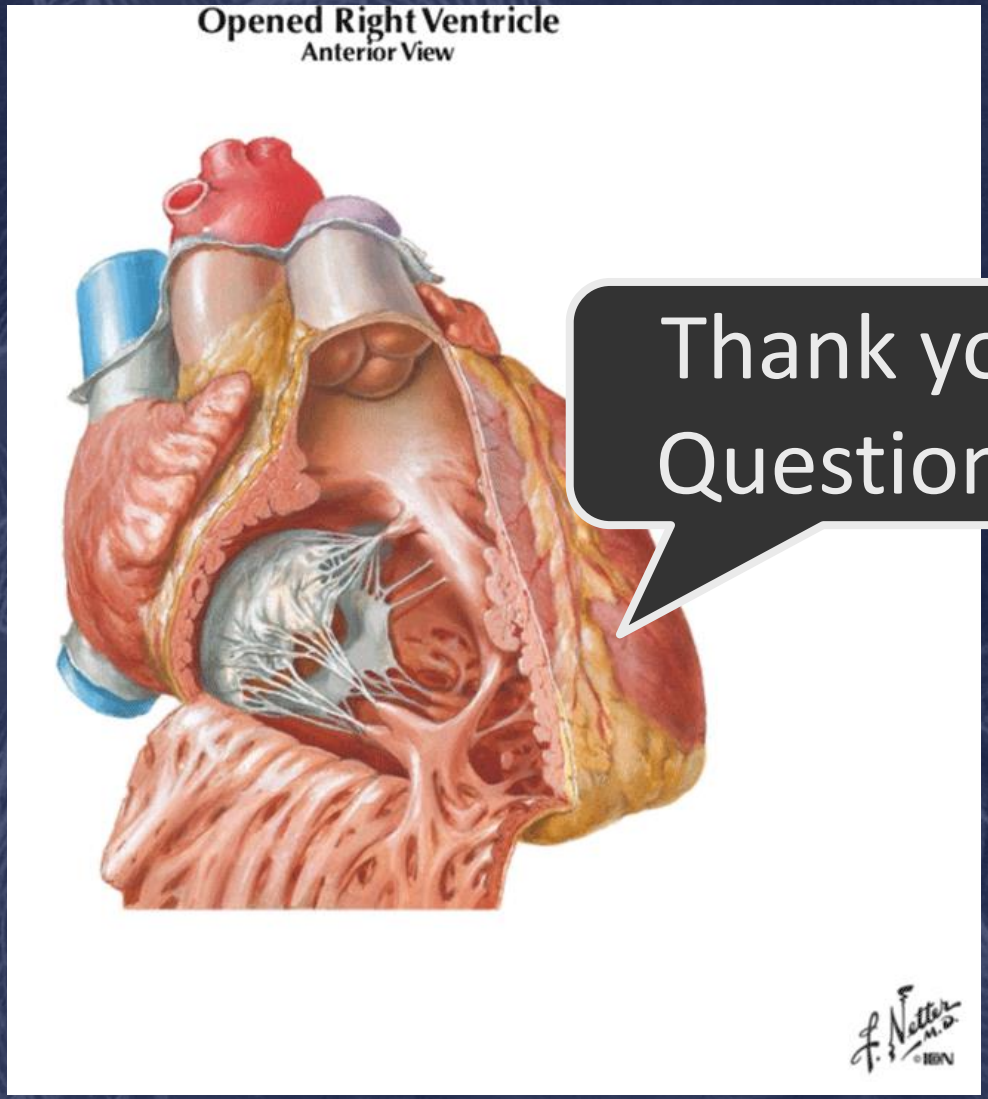
Arrhythmias	Selected Forms of Acquired RV Disease
Supraventricular tachycardia	
Atrial flutter	Pulmonary hypertension
	Tricuspid valve disease
	Advanced lung disease
Atrial fibrillation	RV myocardial infarction
	Pulmonary embilism
	Pulmonary hypertension
	Advanced lung disease
Multifocal atrial tachycardia	Advanced lung disease
Accessory pathways	. . .
Twin AV nodes	. . .
Ventricular tachycardia	ARVD
	Pulmonary hypertension
	RV myocardial infarction
Bradycardia	
Sinus node dysfunction	RV myocardial infarction
	Infiltrative disease
AV block	RV myocardial infarction
	Infiltrative disease
	Myocarditis

- Loss of RA contraction exacerbates difficulties with RV filling
- Atrial or AV pacing (but not ventricular) reverses hypotension and shock in RV failure complicated by AV dissociation

Haddad F, et al. Circulation. 2008;117:1717-31

6. Mechanical Devices

- IABP
  - augments RCA perfusion
  - may preserve ventricular interdependence
- RVAD
- ECMO
- Atrial septostomy


Opened Right Ventricle
Anterior View
Thank you.
Questions?
F. Netter M.D.