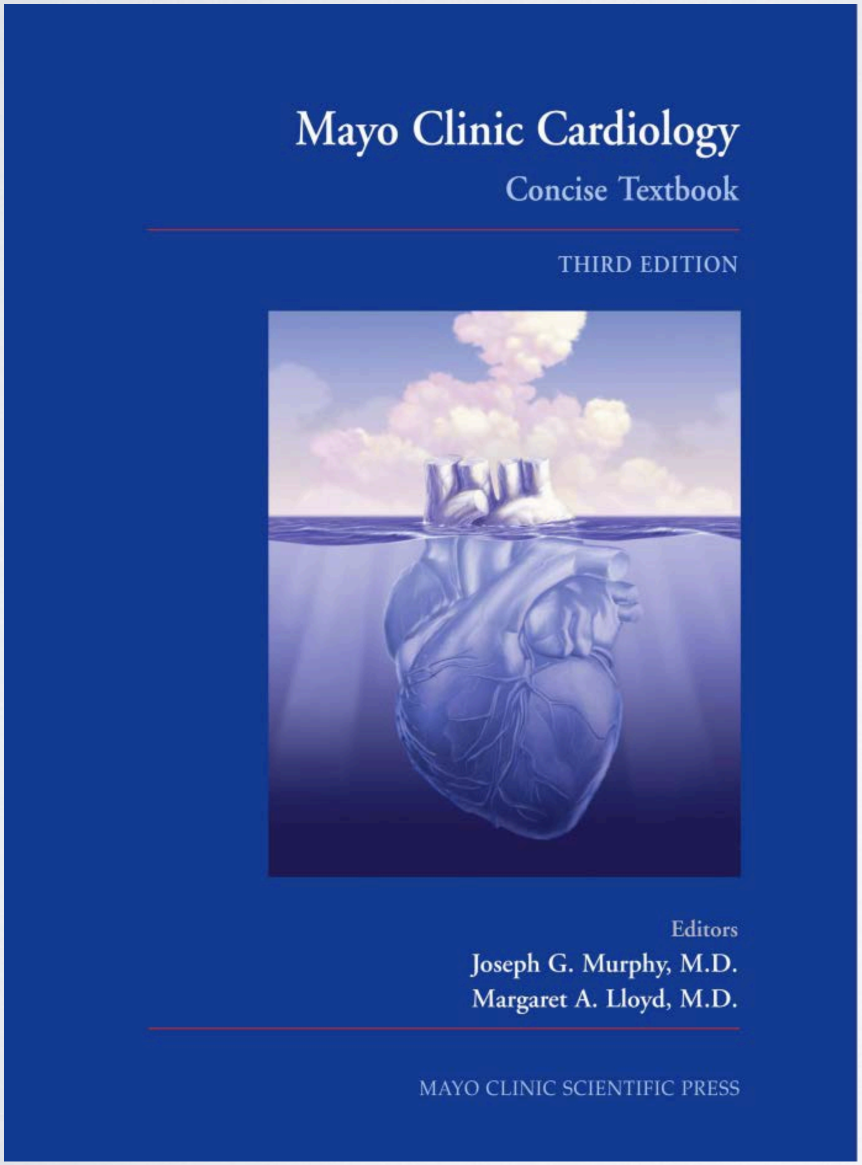
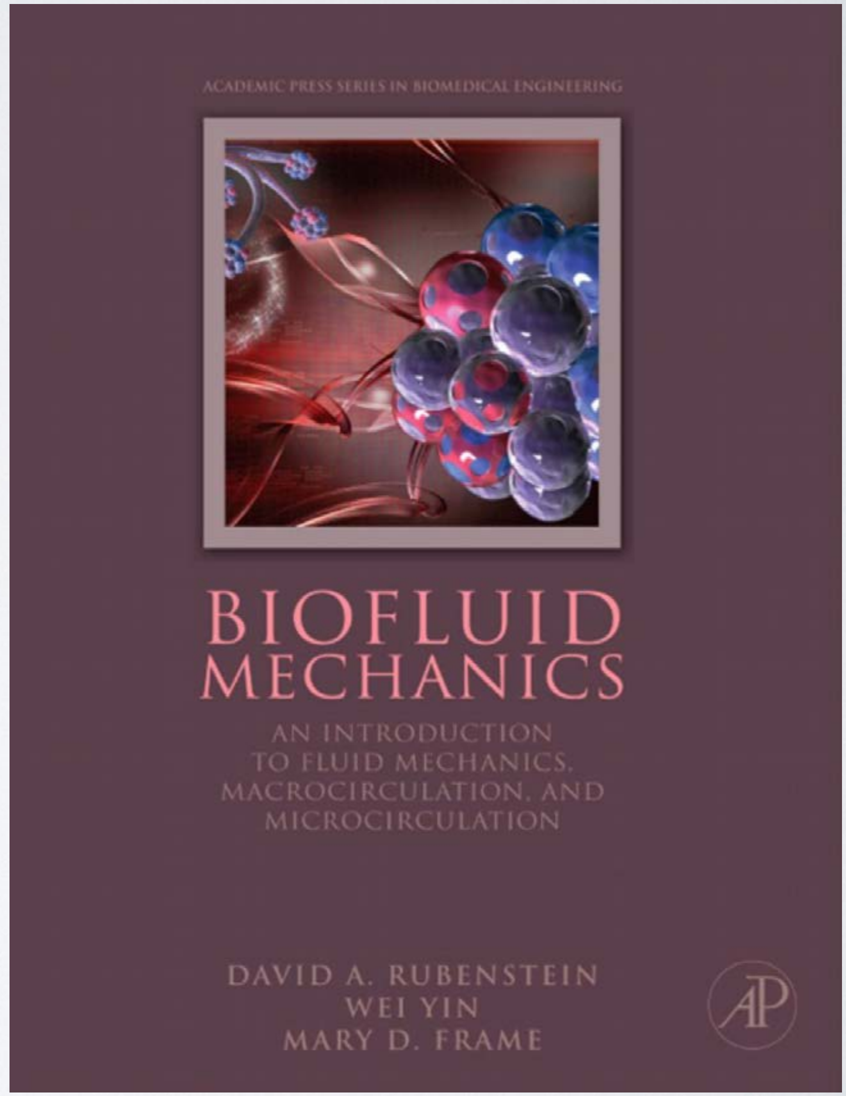
FLUID MECHANICS MOTIVATED BY PHYSIOLOGY AND CARDIOLOGY

11/21/17

REFERENCES FOR FIGURES





REFERENCES

Cardiovascular Biomechanics

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Cardiovascular Biomechanics,
Spring 2004 1

Physiological Fluid Mechanics

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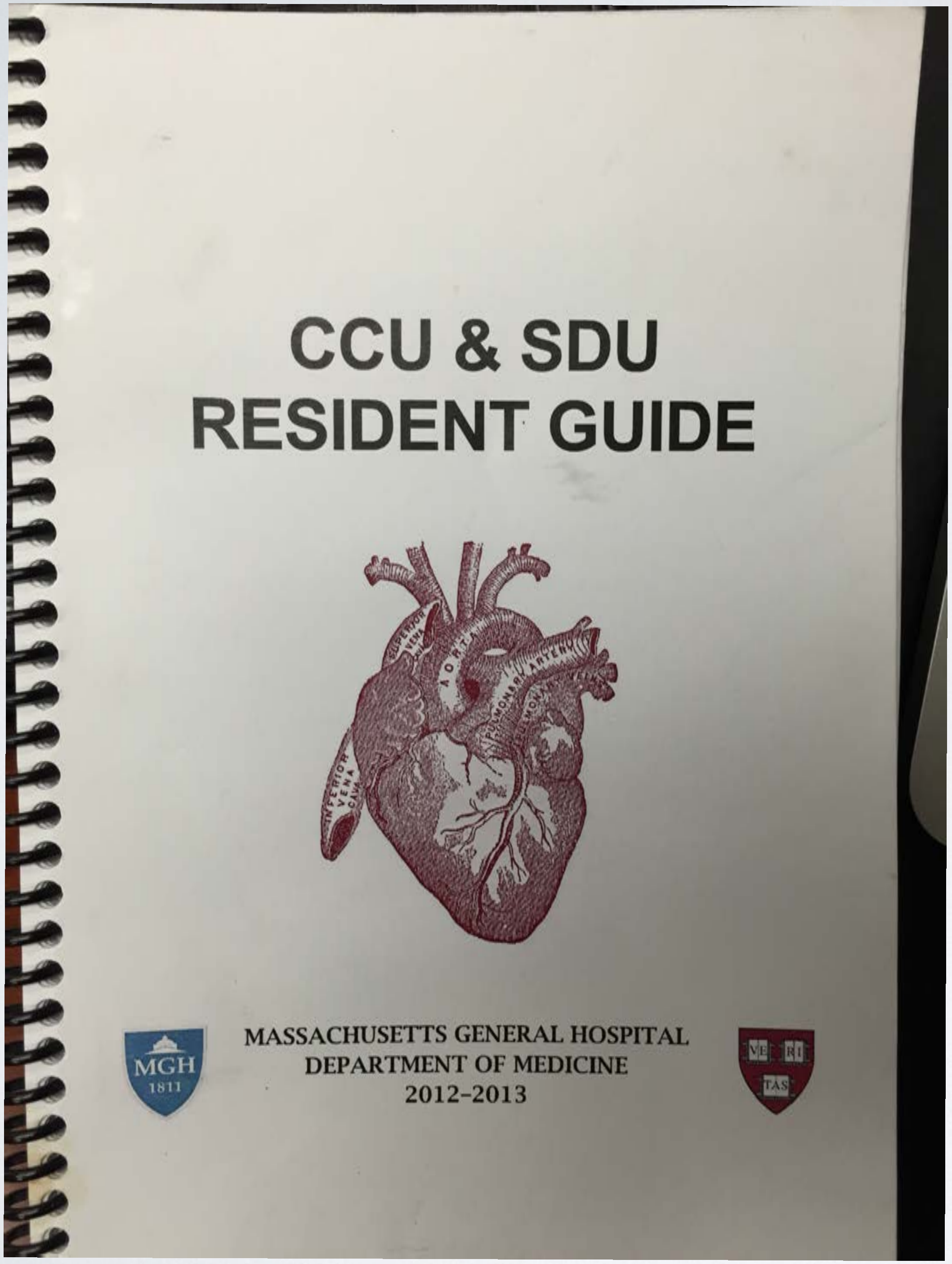
September 2009

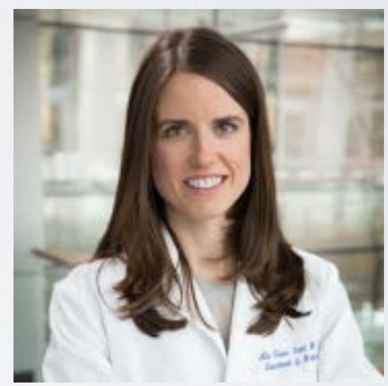
Jennifer Siggers (Imperial College London) Physiological Fluid Mechanics September 2009 1 / 166

WEB INFO

- Google searches:
  - coronary critical care monitoring
  - ventricular assist devices
  - angiography
  - computation of flow in heart valves
  - blood flow modeling



http://www.cocoonbiotech.com/team/



Thanks to:
Dr. Ailis Tweed-Kent
ChEg '07

(SOME) MOTIVATION

SPECIAL ARTICLE

EMC

Rev Bras Cir Cardiovasc 2010; 25(1): 1-10

Concepts of basic physics that every cardiovascular surgeon should know. Part 1-mechanics of fluids

Conceitos de física básica que todo cirurgião cardiovascular deve saber. Parte I - Mecânica dos fluidos

Marcos Aurélio Barboza de OLIVEIRA[1], Fernanda Tomé ALVES[2], Marcos Vinícius Pinto e SILVA[3], Ulisses Alexandre CROTI[4], Moacir Fernandes de GODOY[5], Domingo Marcolino BRAILE[6]

Do you have high blood pressure? You might, based on new guidelines

By Honor Whiteman | Published Tuesday 14 November 2017

Fact checked by Jasmin Collier

HEALTH

'Unbelievable': Heart Stents Fail to Ease Chest Pain

Leer en español

By GINA KOLATA NOV. 2, 2017

http://www.chp.edu/our-services/heart/cardiology/heart-failure-recovery-program/patient-stories?origin=sitelink+patient+stories&keyword=%252Bpediatric%2520%252Bventricular%2520%252Bassist%2520%252Bdevices&matchtype=b&gclid=EAIaIQobChMIxKynstnP1wIVDChpCh0YjgPqEAAYASAEEgKumPD_BwE

INTERSECTION OF ENGINEERING AND MEDICINE?

- Provides example situations to motivate <u>our</u> study of fundamentals.
- Engineering can provide quantitative understanding of flows that explain clinical observations.
- Engineering analysis can provide a foundation for advanced measurement/diagnosis technologies.
    - Determine what measurements could provide most insight
    - Better evaluate success of treatment
  - Help to design interventional and prosthetic devices
    - fabricate: by 3-D print from MRI/CAT scan images a complex internal structure to aid surgeon
- We hope that engineering can contribute in a broad sense to cost, efficiency and reliability of health care.

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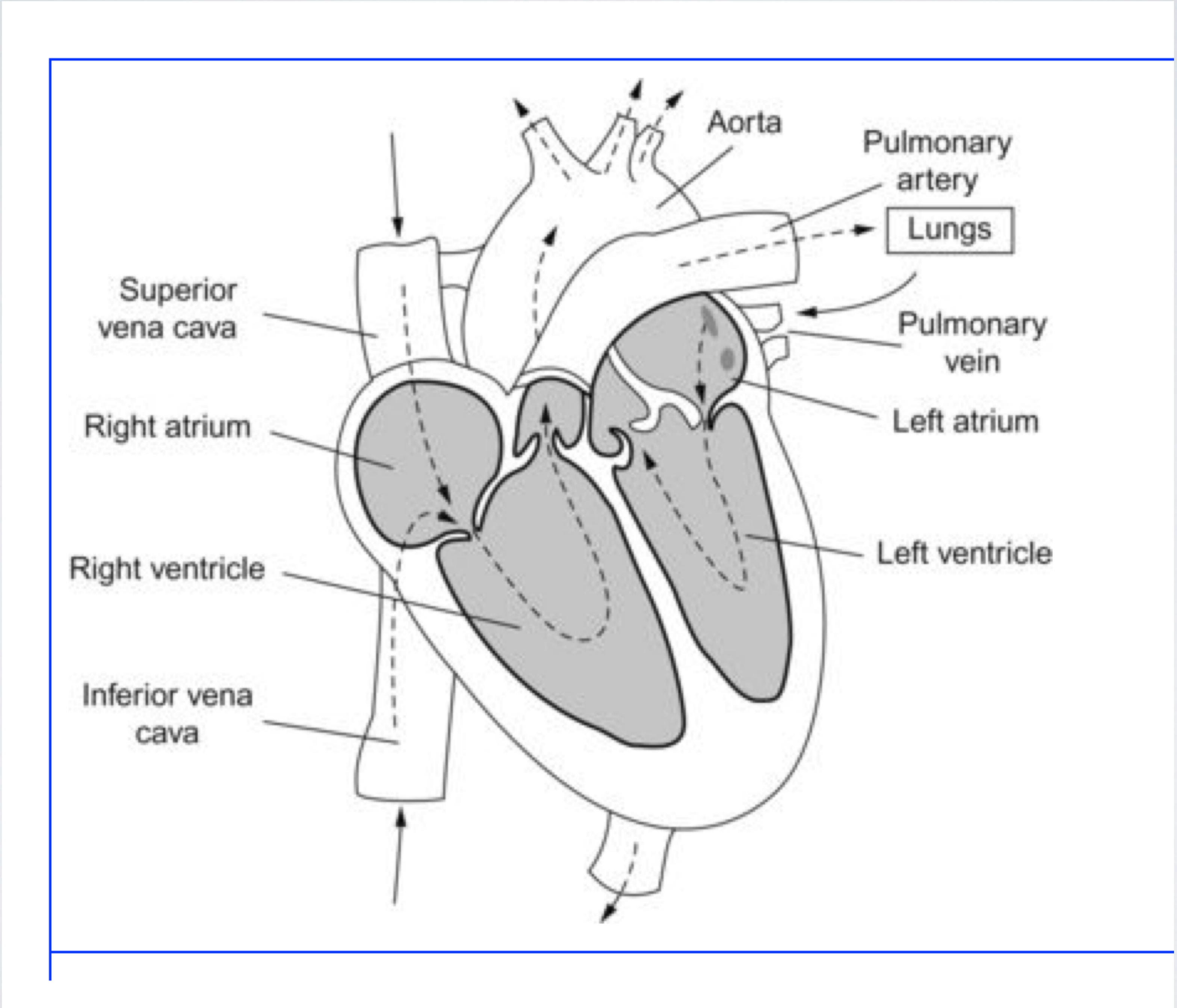


Mechanical Events in the Left (Right) Ventricle

Relaxation or *Diastole*

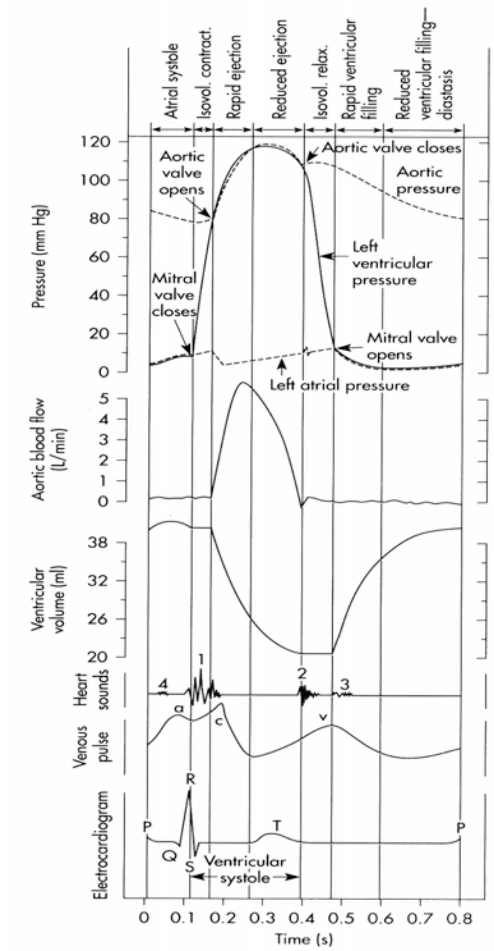
- Blood fills *ventricle* from *atrium* -- *mitral* (*tricuspid*) valve opens.

Atrial *Systole* or Contraction

- *Atrium* contracts to expel remaining blood and "prime" ventricular pump.

Contraction or *Systole*

- *Ventricle* contracts, *aortic* (*pulmonic*) valve opens, blood is ejected into the *aorta*.



Anatomy of the heart

The heart is the pump of the circulatory system, i.e. it is the source of energy that makes the blood flow.

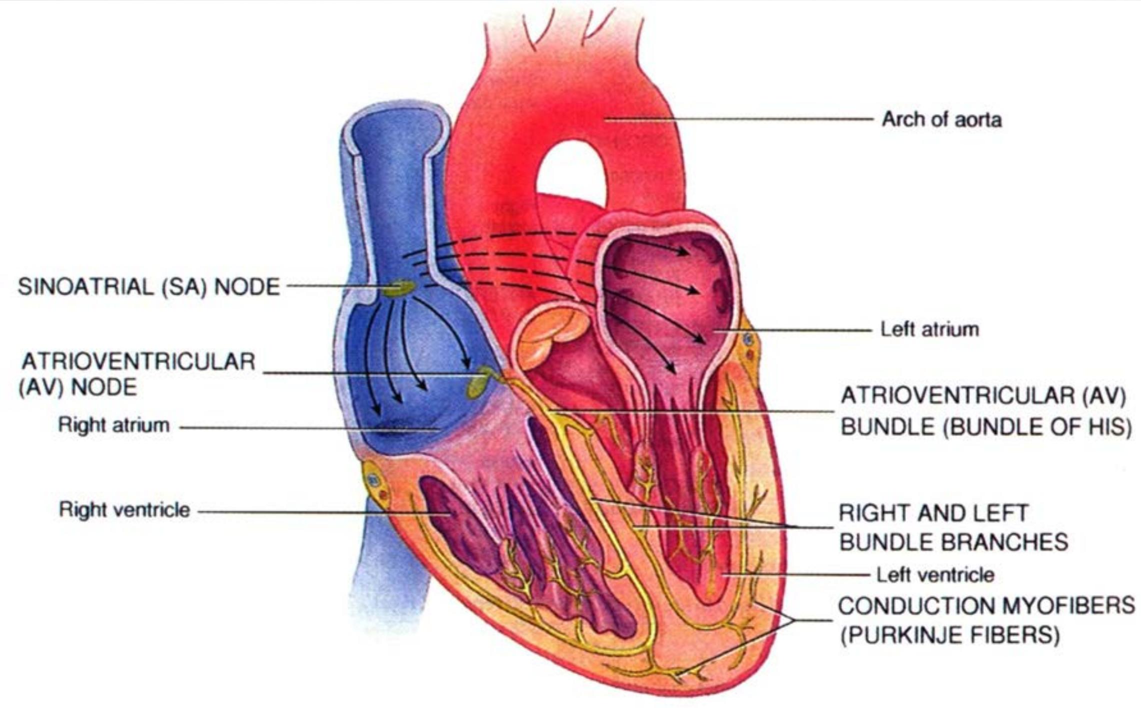


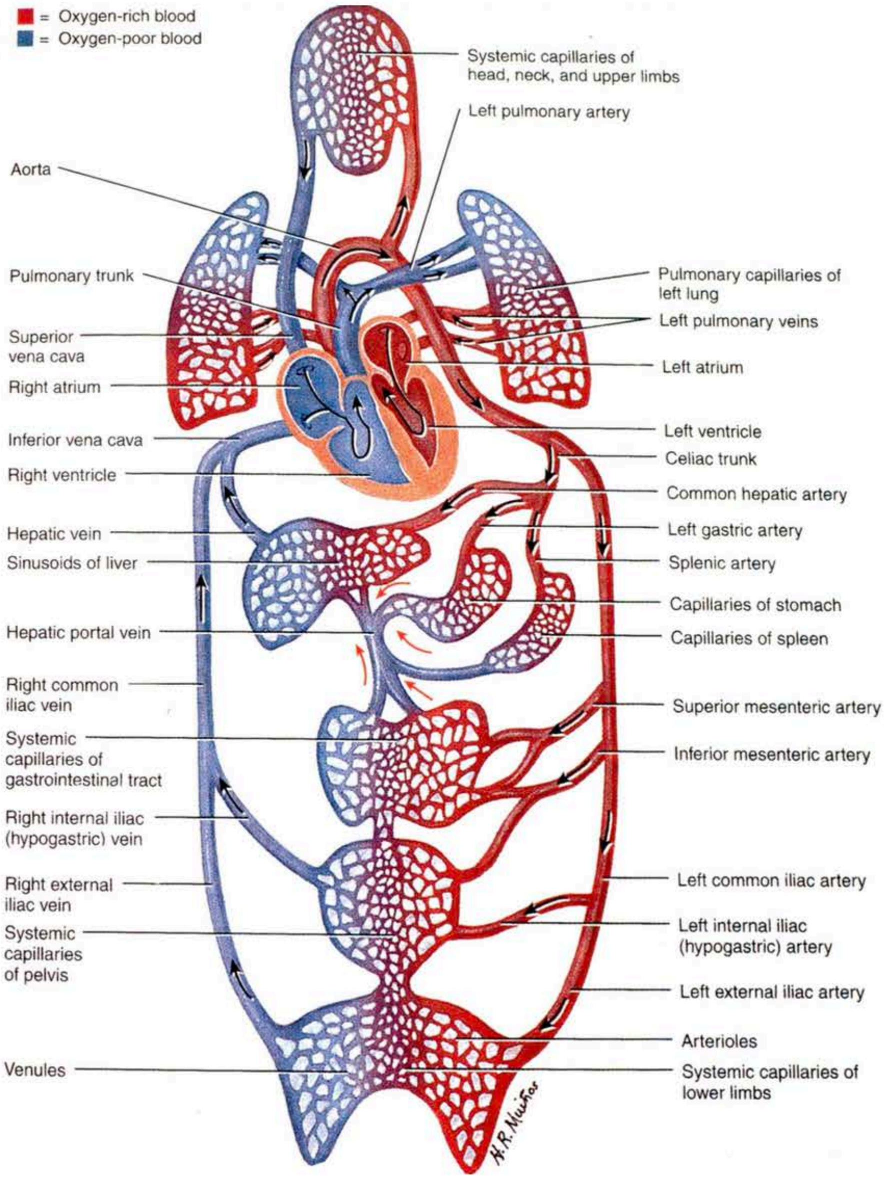
Figure: Diagram of heart, showing the major structures, by Ottesen *et al.*, 2004).

The heart may be thought of as two pumps in series. Blood passes . . .

- . . . from the venous system . . .
- . . . into the **atrium**[a] (low-pressure chamber), . . .
- . . . through a non-return valve . . .
- . . . into the **ventricle** (high-pressure chamber), . . .
- . . . and through another non-return valve . . .
- . . . into the arterial system.

[a] In these notes, I have tried to highlight in colour important technical terms that you should be familiar with. Green highlighting is used to emphasise terms that are defined elsewhere in these notes, while red highlighting emphasises terms as they are being defined.

The cardiovascular system



- For a blood particle that starts in the left side of the heart, its journey around the cardiovascular system is as follows:

  Left side of heart → systemic arteries → capillaries → systemic veins → right side of heart → pulmonary system (lungs) → left side of heart → . . . .

- Vessels:
  - systemic arteries, containing about 20% of the blood,
  - systemic veins, containing about 54% of the blood,
  - pulmonary circulation, containing about 14% of the blood,
  - capillaries, containing a small fraction of the blood,

  and the heart contains about 12% (varies during heart cycle) (Noordergraaf, 1978).

Figure: Sketch of the cardiovascular system (Ottesen, Olufsen & Larsen, *SIAM Mon. Math. Mod. Comp.*, 2004).

Cardiovascular System

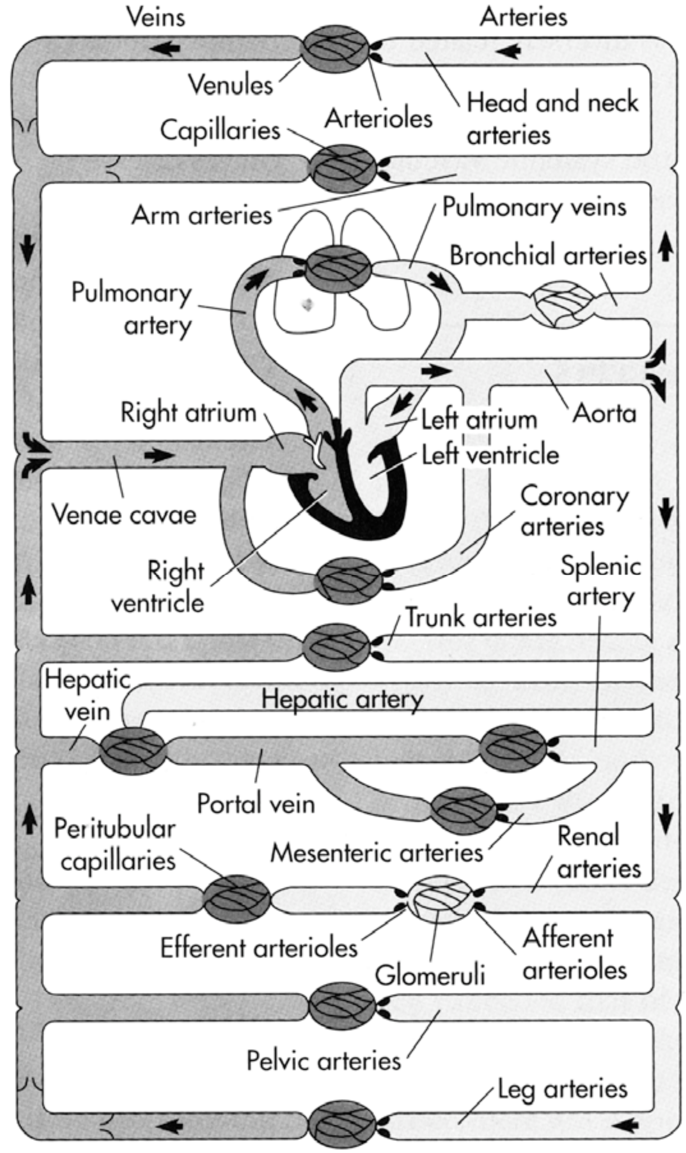
Heart: 2 Chambers in Series

Pulmonary circulation in between right heart and left heart.

Systemic circulation refers to remaining circulatory systems.

Left heart provides major component of work to drive blood through the systemic circulation.

Heart pulsation: *Systole* (Contraction) and *Diastole* (Relaxation/Filling)

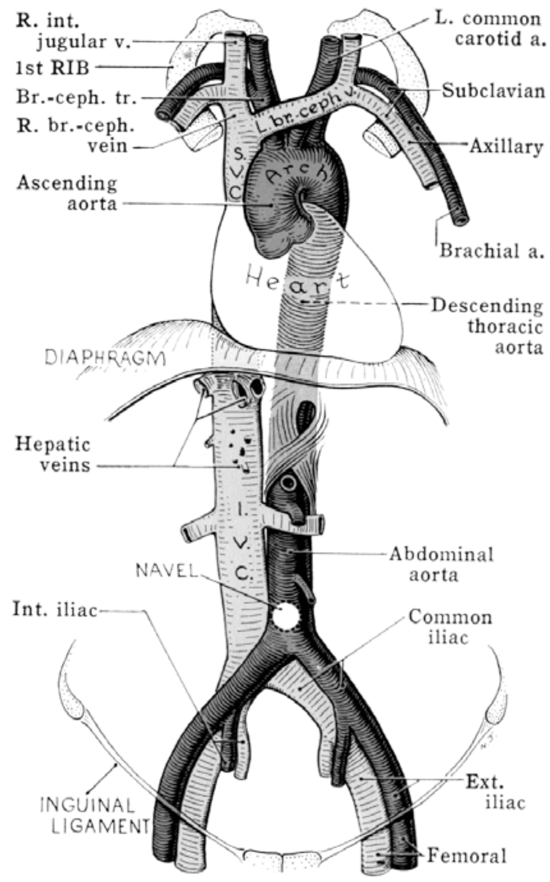


Major Arteries and Veins

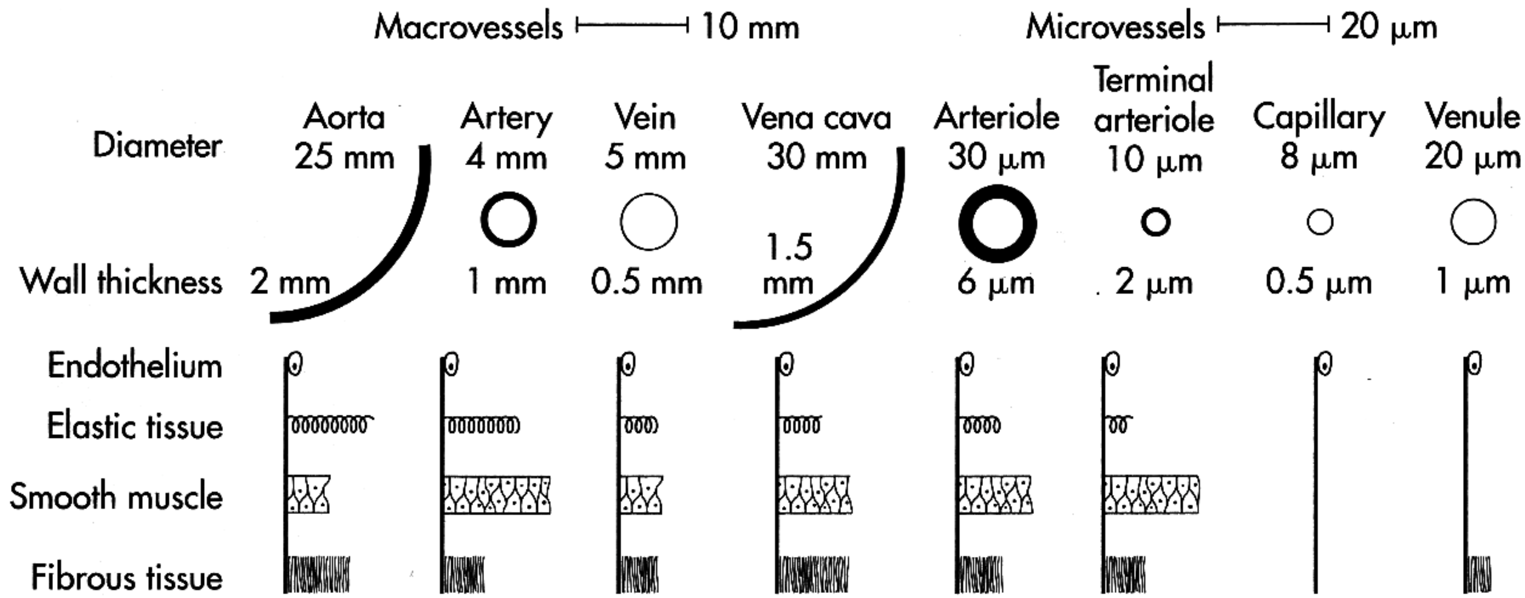
Arteries and Veins usually adjacent to each other.

Large arteries and veins: 25 - 30 mm in diameter.

Many interventional procedures (cardiac angiography, catheterization) use the femoral artery (left side) or femoral vein (right heart) as the origin for access to the heart.



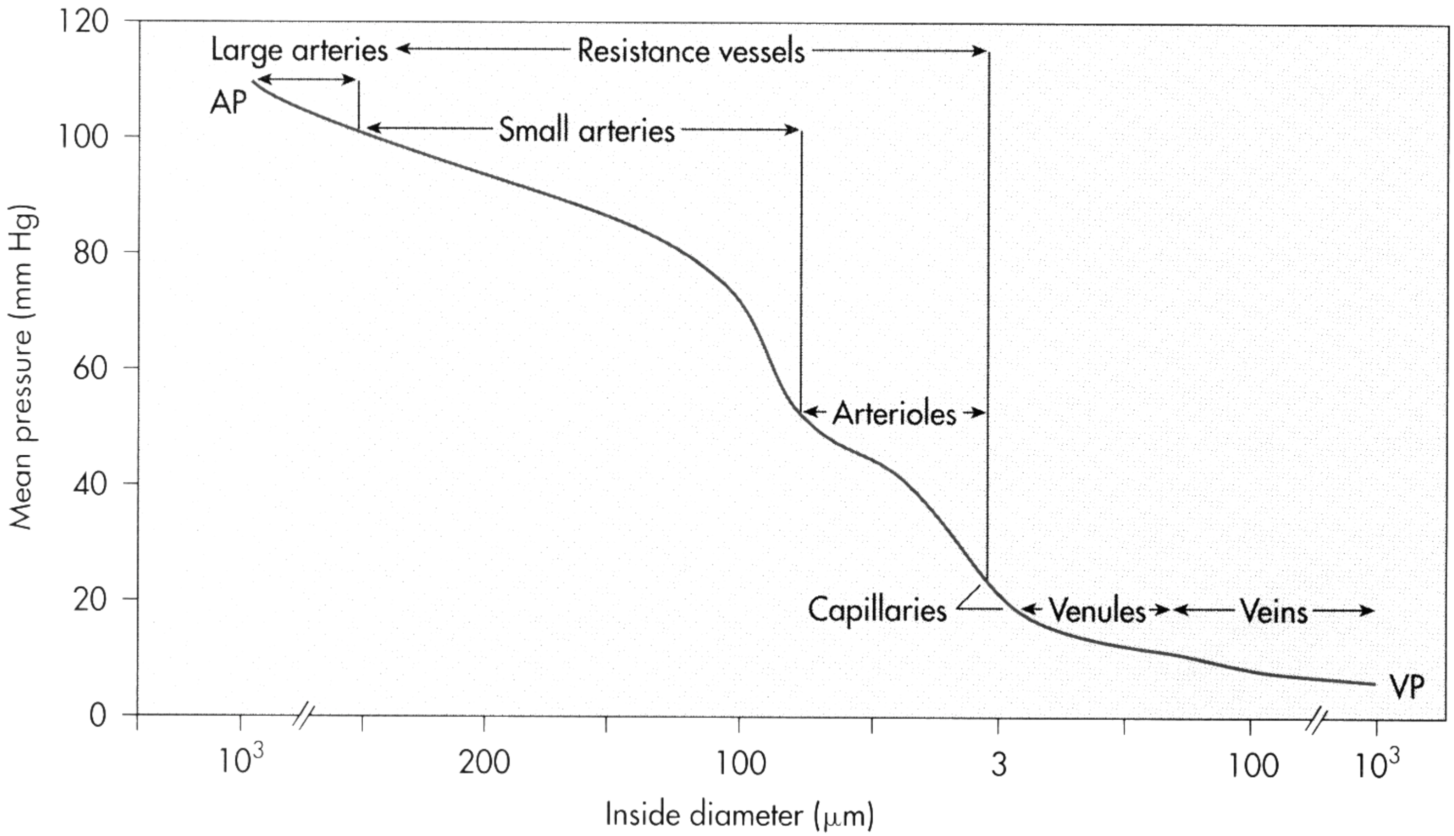
Major Arteries and Veins



Original contraction is pulsatile. However, flow in capillaries and veins is almost steady state, due to the elasticity of the large arteries.

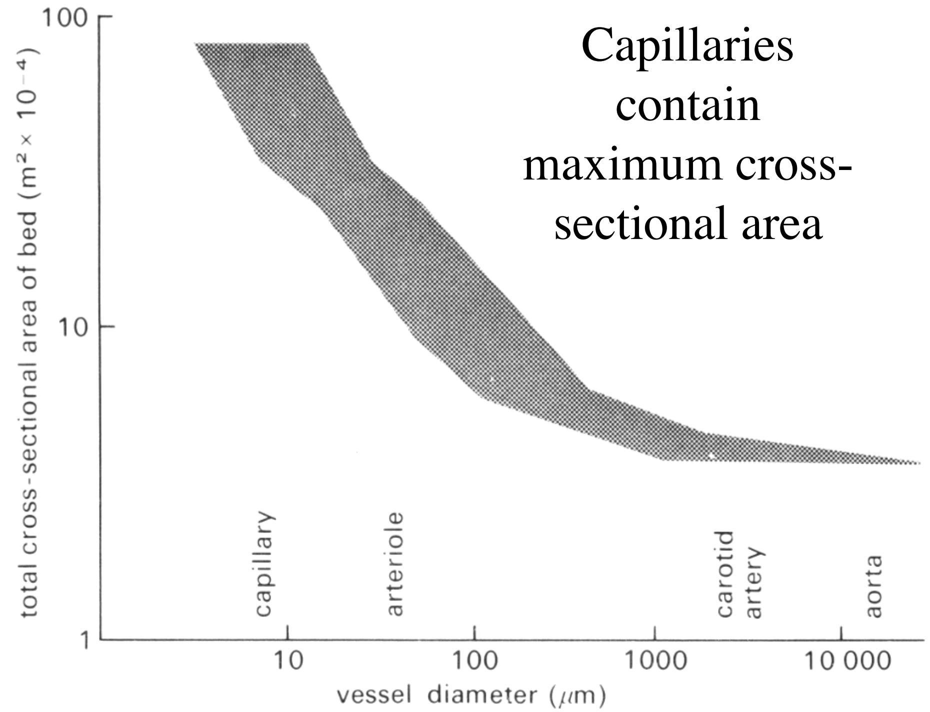
Pulse Pressure = Systolic Pressure - Diastolic pressure

Pressure Drop in Cardiovascular System



Small arteries produce the largest pressure drop

Functional Flow Area



Capillaries contain maximum cross-sectional area

WHAT KINDS OF ANALYSIS COULD WE DO?

- Allometric
- Calculations of fluid flow
- Examination of diagnostic signals
- Design — fabrication of models for surgeons or protheses for insertion

LOG-LOG PLOT OF HEART RATE AND ANIMAL SIZE

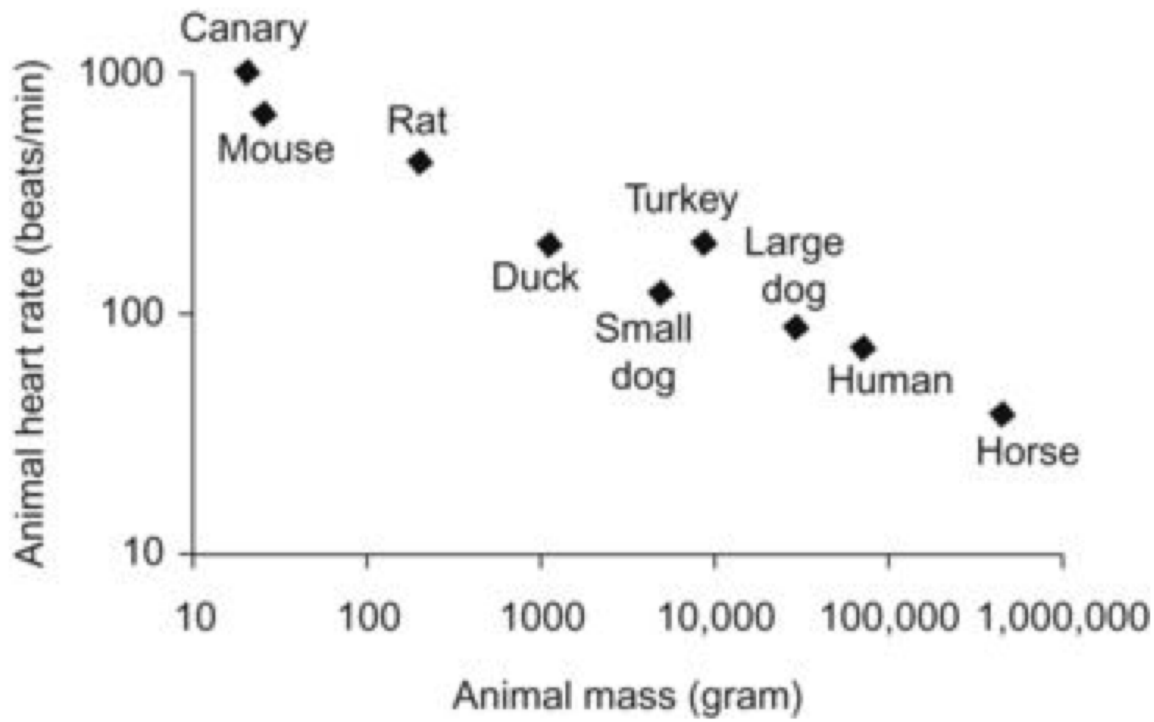


Figure 4.8 The relationship between animal mass and heart rate. As the mass of the animal increases, there is a general decrease in heart rate. The relationship between these two measurements can be correlated to many different properties of the animal, as described in the text.

ORIGIN OF THIS BEHAVIOR?

$f \sim M^{-.35}$

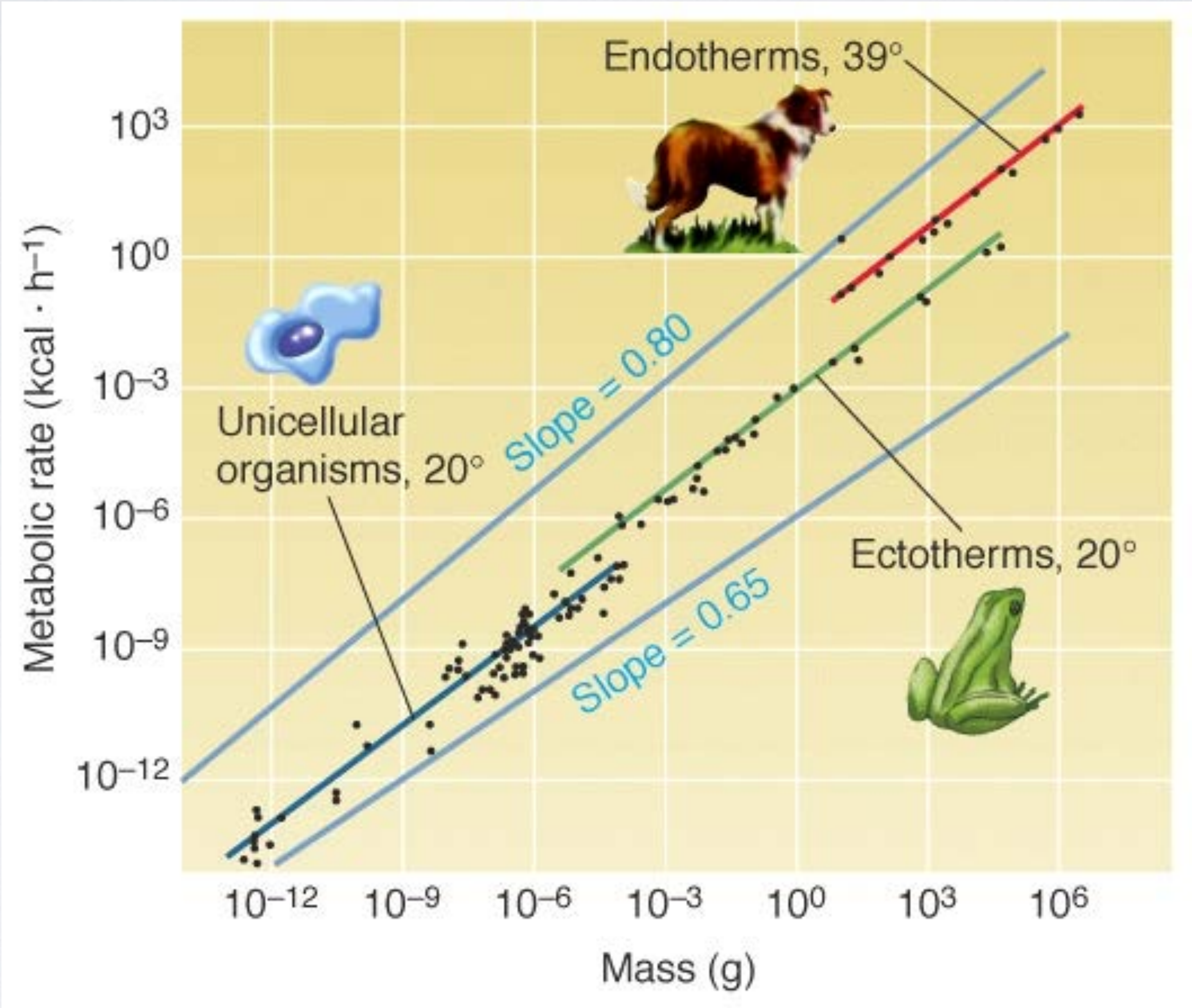
- The heart (attempts) to provide, in response to various stimuli, the flow rate of blood that is needed (at some instant) for all of a creatures needs
  - Flowrate to provide oxygen and other nutrients
  - To achieve this flowrate, “viscous losses” and gravity head must be overcome
- So the heart must simultaneously meet these two criteria

DIMENSIONAL REASONING

- flow rate times pressure gradient is "power"
- Flow rate will be a heart volume/time period
- Pressure gradient is caused by deceleration of "velocity squared"
- Heart power:
  - $(V_h * f)\ (\rho\ (V_h^{(1/3)} * f)^2 ==> \rho\ f^3 V_h^{5/3}$
- How does this power scale with animal size?

METABOLIC POWER(KLEIBER'S LAW)

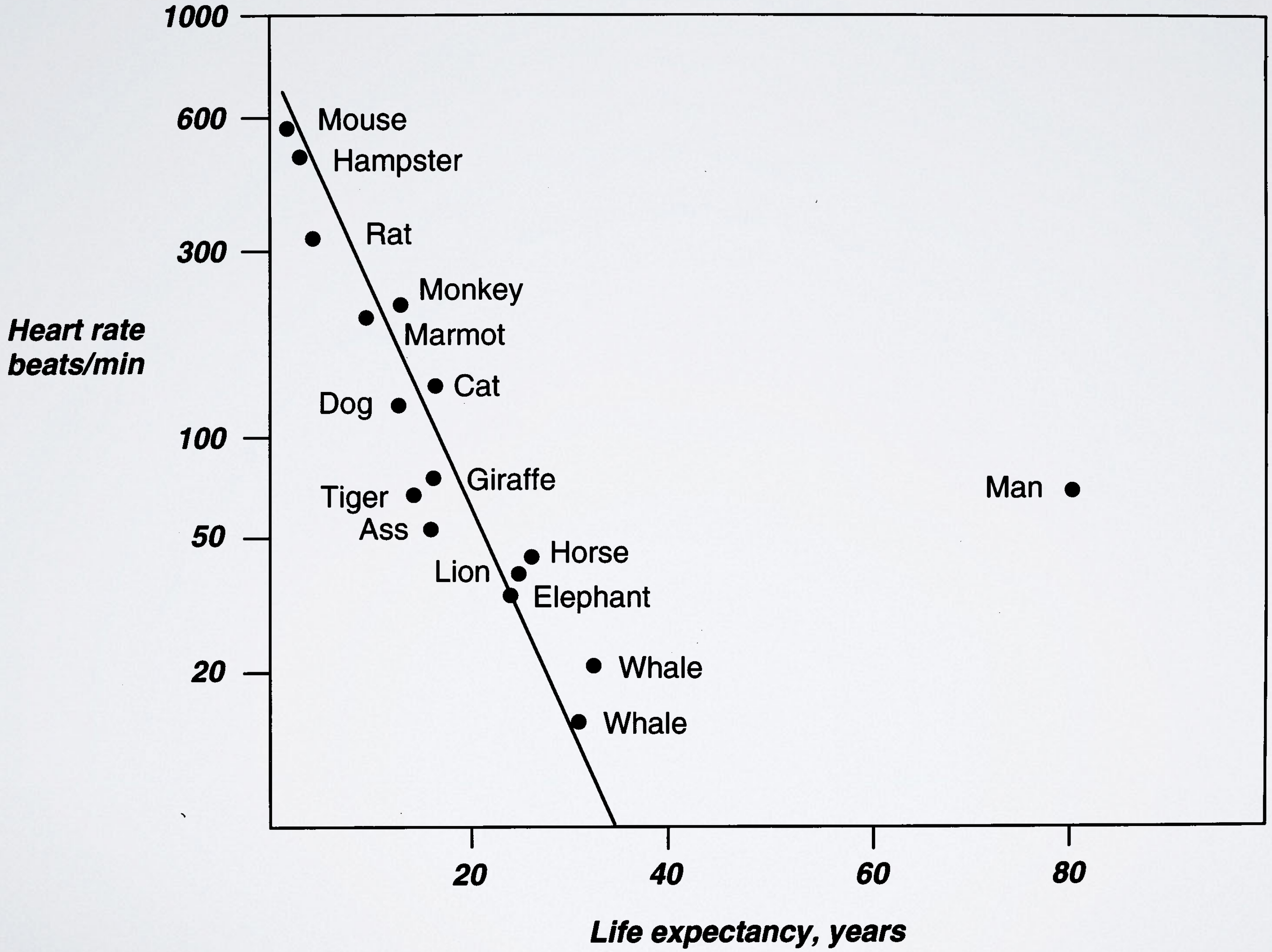
$P \sim M^{.75}$



HEART RATE — MASS

- $\rho f^3 V_h^{5/3} \sim M^{.75}$
- Further $V_h \sim M$
  - http://www.biologyreference.com/Re-Se/Scaling.html
- Which gives…
  - $f \sim M^{-.31}$
- Interesting… I don't know how "correct" it is
- There are other allometric observations….

HEART RATE — LIFE EXPECTANCY

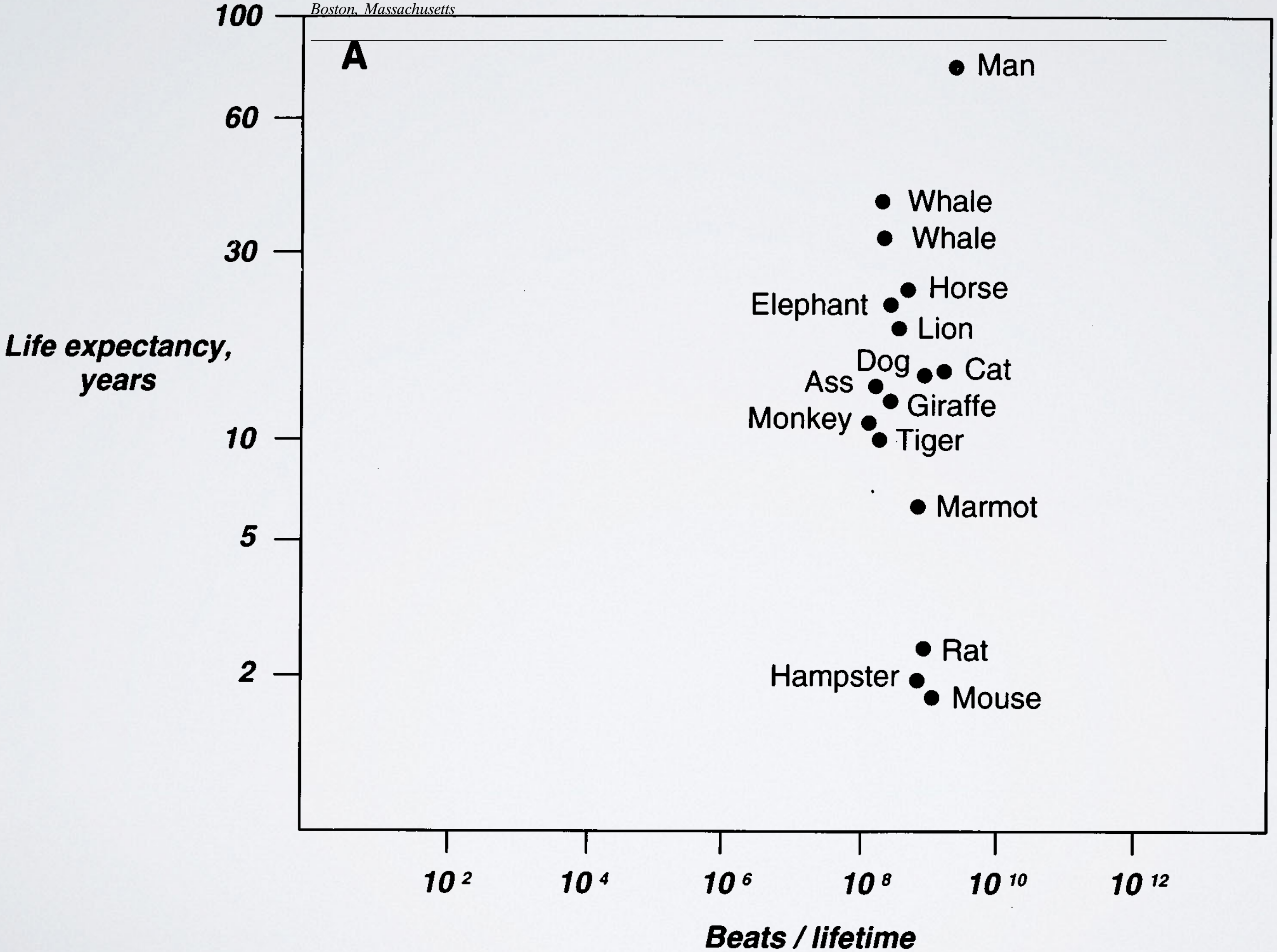


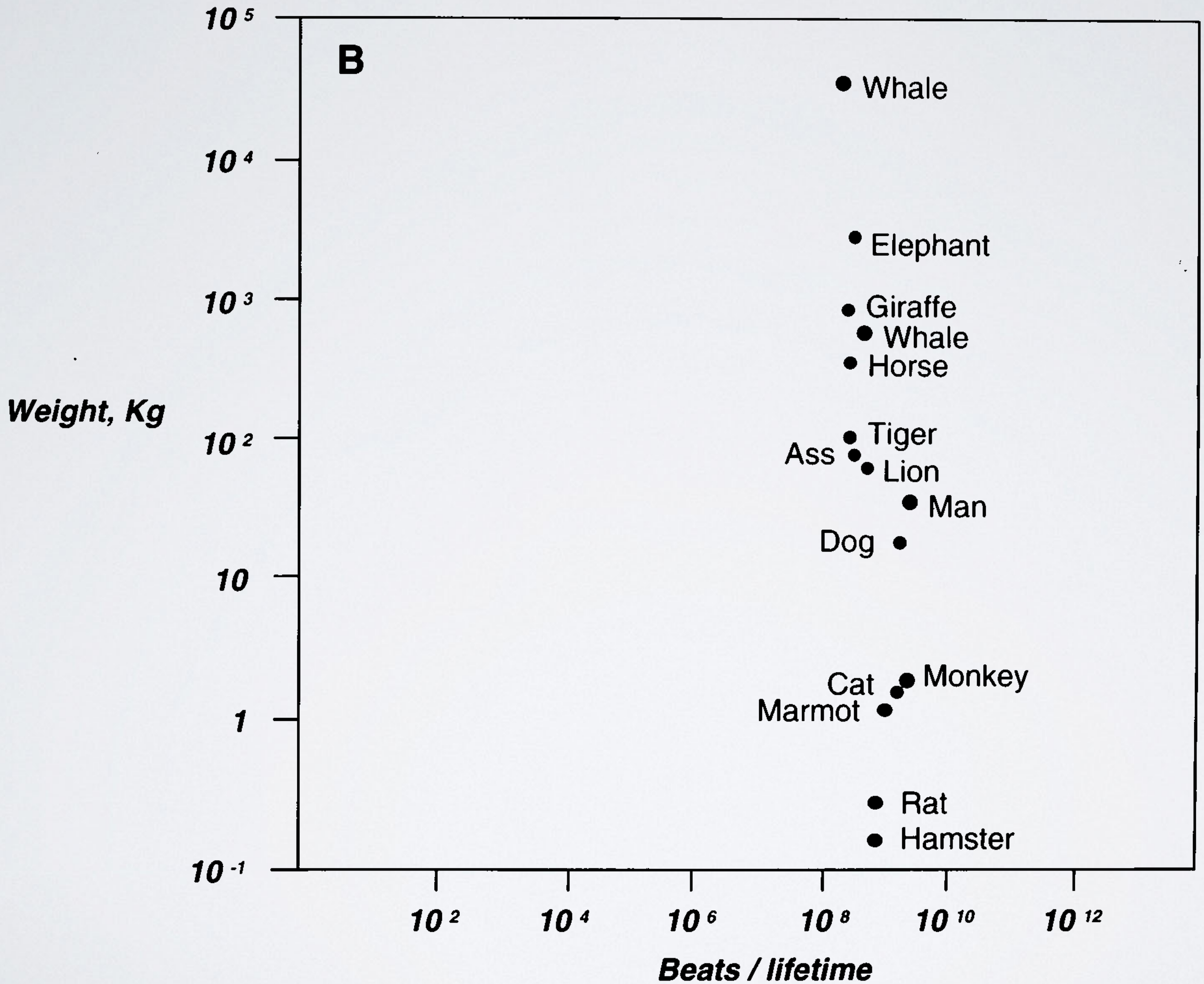
JACC Vol. 30, No. 4
October 1997:1104–6

EDITORIAL

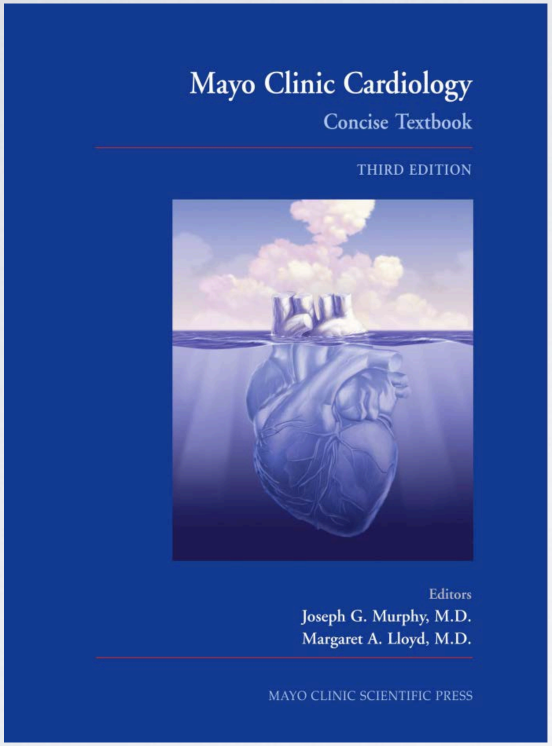
Rest Heart Rate and Life Expectancy

HERBERT J. LEVINE, MD, FACC
Boston, Massachusetts




B
Whale
Elephant
Giraffe
Whale
Horse
Tiger
Ass
Lion
Man
Dog
Cat
Monkey
Marmot
Rat
Hamster
Weight, Kg
10 5
10 4
10 3
10 2
10
1
10 -1
10 2
10 4
10 6
10 8
10 10
10 12
Beats / lifetime

A CALCULATION:



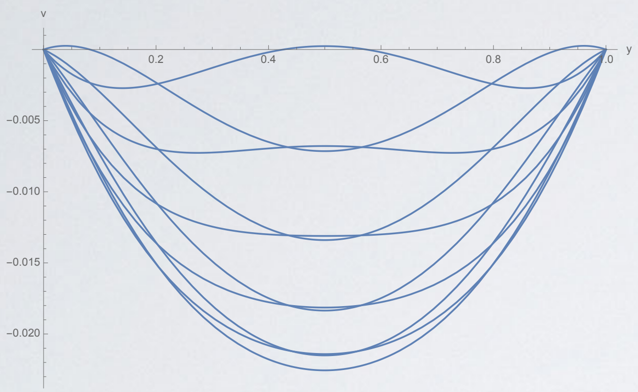
To make these measurements, it is essential to understand and use the modified Bernoulli equation (Equation 11 and Fig. 10), in which the decrease in pressure across a stenosis is equal to $4v^2$, and the concept of the time-velocity integral (TVI), or "stroke distance" (Fig. 11).

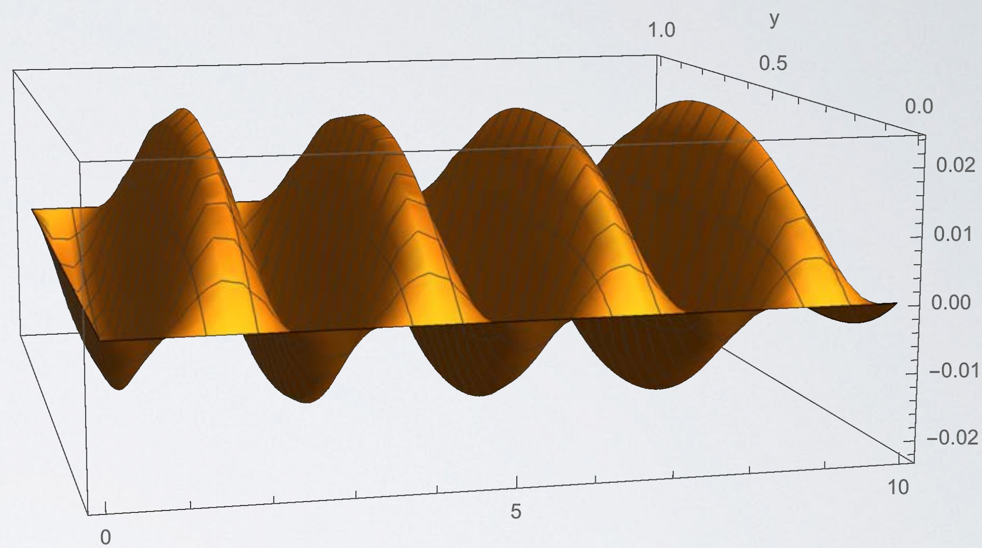
- …which is consistent with the Cardiologist saying that he had to do some calculations to be sure, but he thought that the blood pressure in the the pulmonary loop was OK.

(TRANSIENT) NAVIER-STOKES EQUATIONS

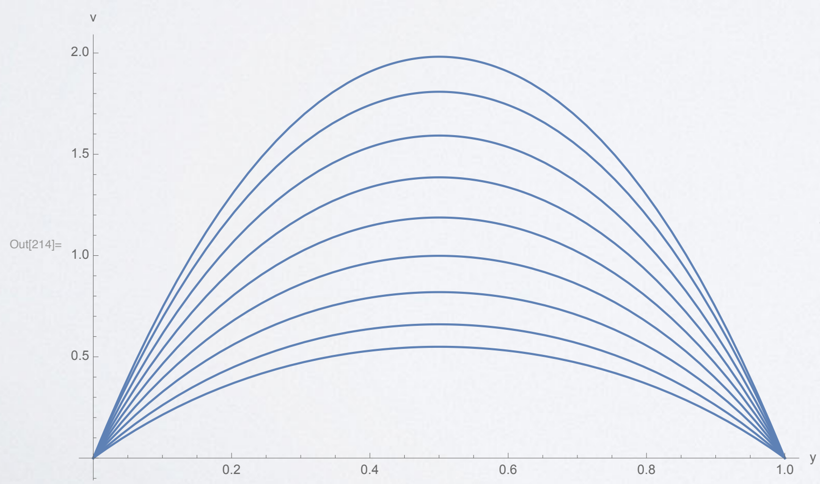
$$\frac{\partial v(t,y)}{\partial t} = -\frac{1}{\rho}\frac{\partial p(t)}{\partial x} + \frac{\mu}{\rho}\frac{\partial^2 v(t,y)}{\partial y^2}$$

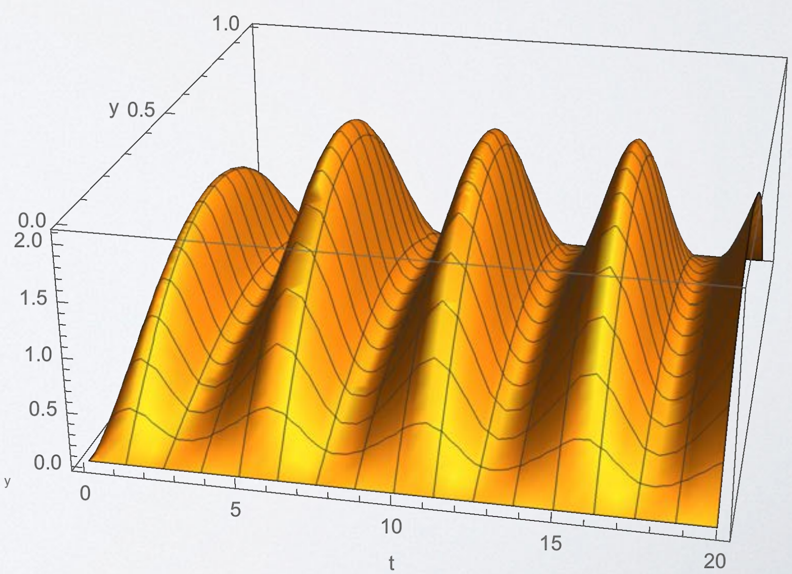
```
dpdx = + .05 Cos[2 ω t];
```



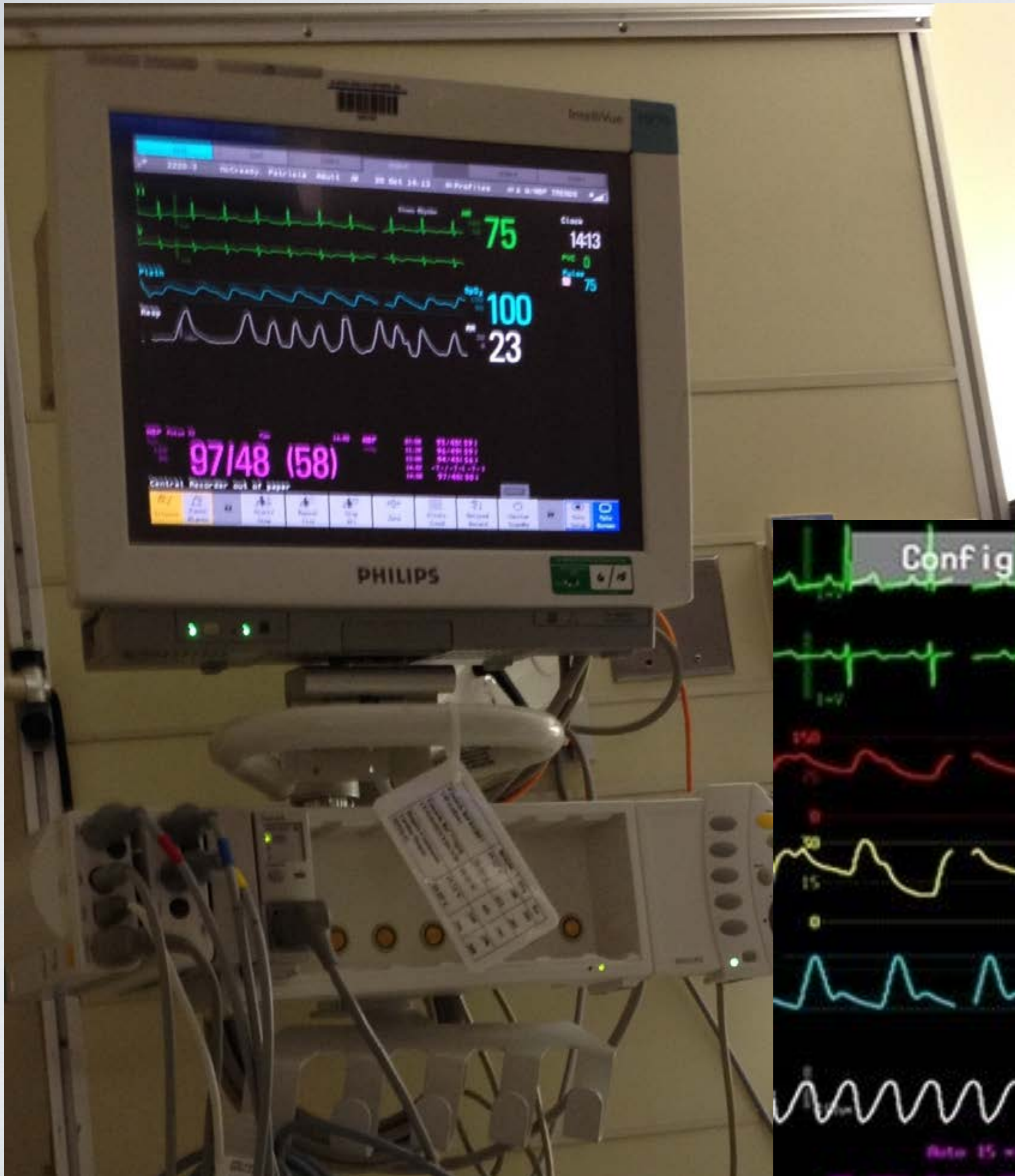


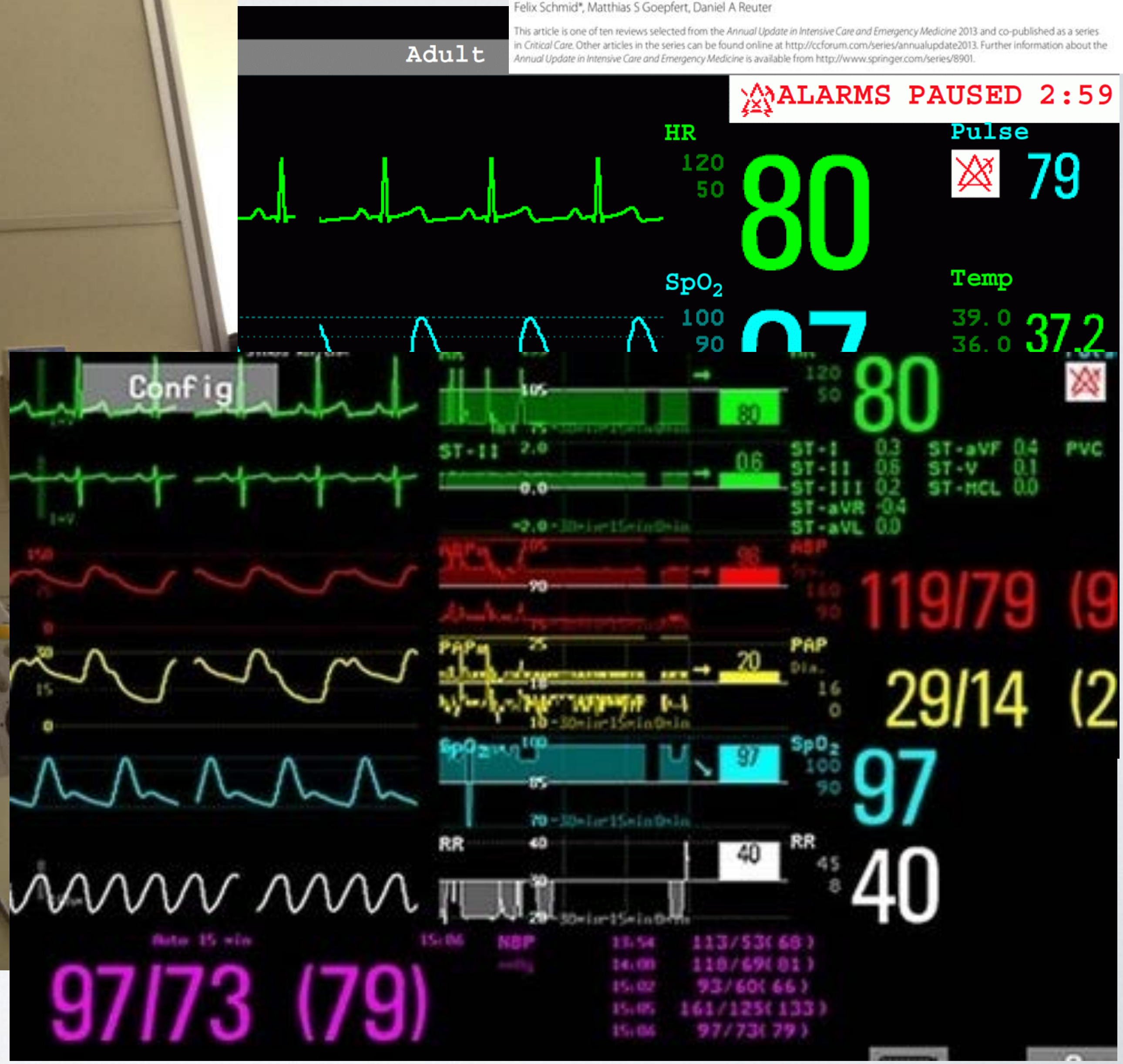

```
dpdx = -(1 + ( Cos[ω t] + .2 Cos[2 ω t - π / 2] + .05 Cos[4 ω t - π / 2] + .02 Cos[8 ω t - π / 2] + .01 Cos[16 ω t]));
```

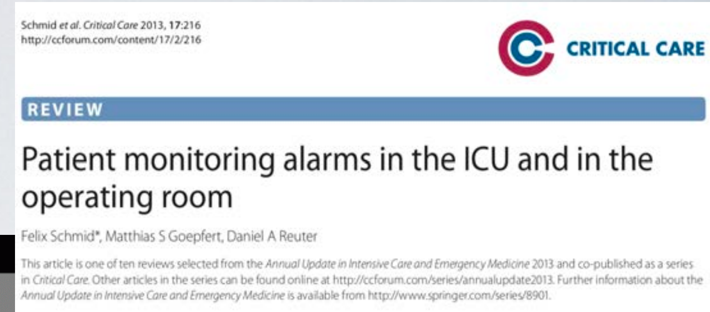




CARDIAC ICU







Schmid *et al. Critical Care* 2013, 17:216
http://ccforum.com/content/17/2/216

CRITICAL CARE

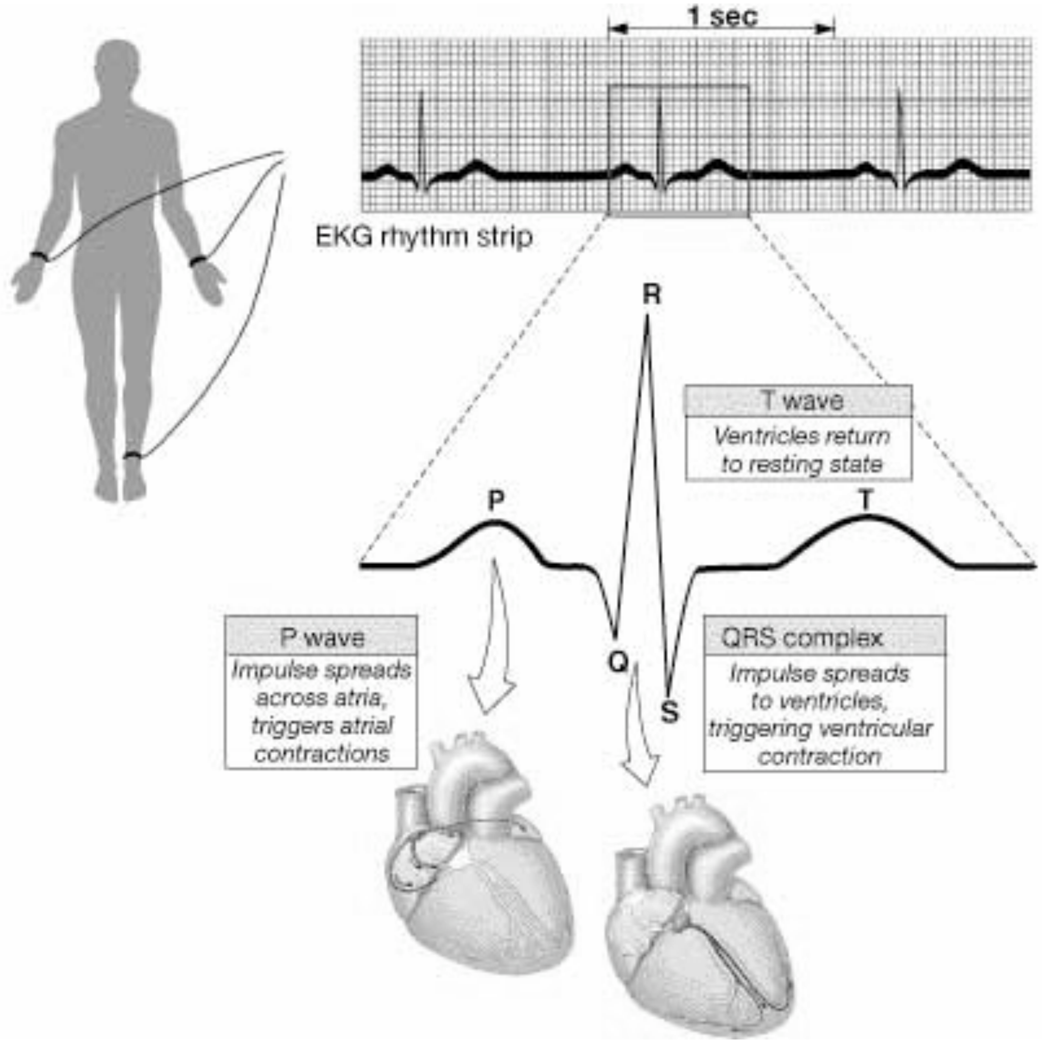
REVIEW

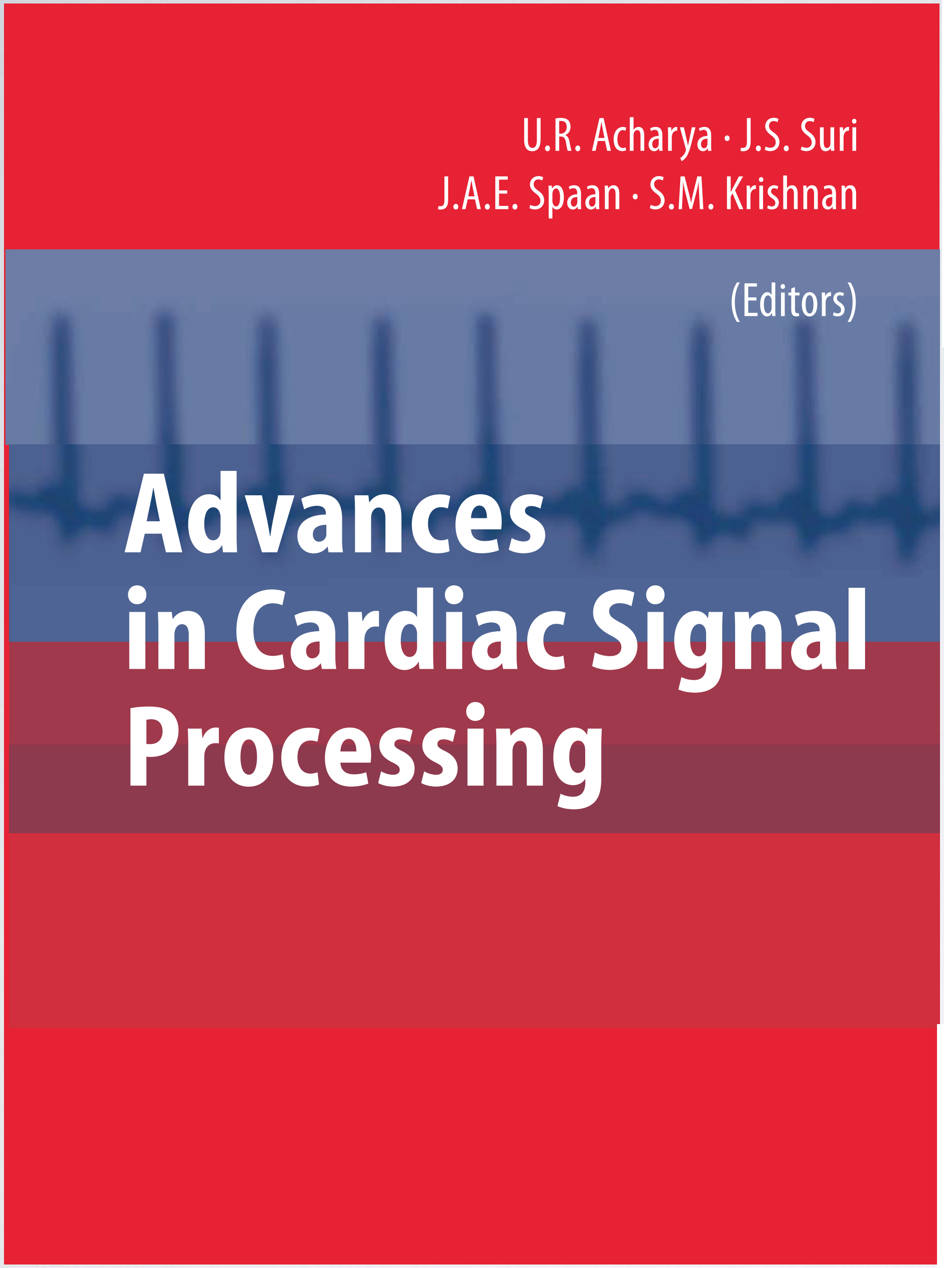
Patient monitoring alarms in the ICU and in the operating room

Felix Schmid*, Matthias S Goepfert, Daniel A Reuter

This article is one of ten reviews selected from the *Annual Update in Intensive Care and Emergency Medicine* 2013 and co-published as a series in *Critical Care*. Other articles in the series can be found online at http://ccforum.com/series/annualupdate2013. Further information about the *Annual Update in Intensive Care and Emergency Medicine* is available from http://www.springer.com/series/8901.

The Electrocardiogram (EKG)





5

Heart Rate Variability

Rajendra Acharya U, Paul Joseph K, Kannathal N, Lim Choo Min and Jasjit Suri S

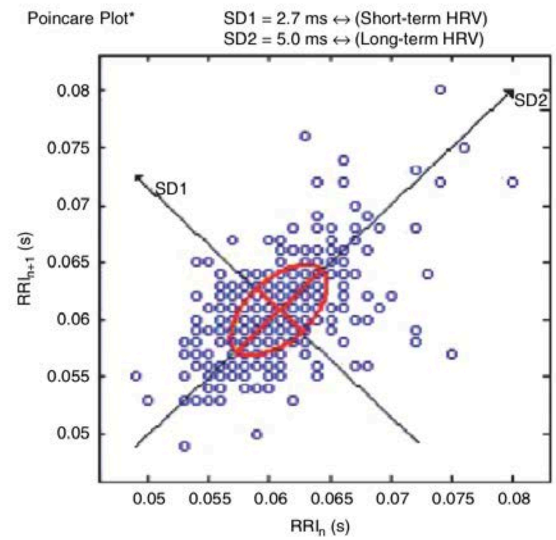


Fig. 5.2. Poincare plot of a normal subject

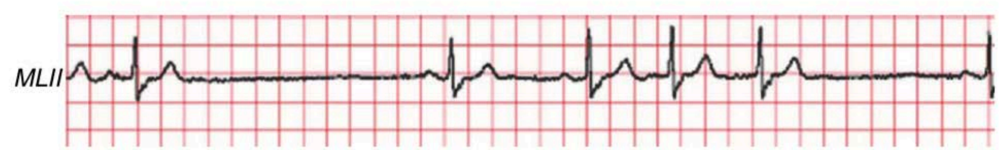
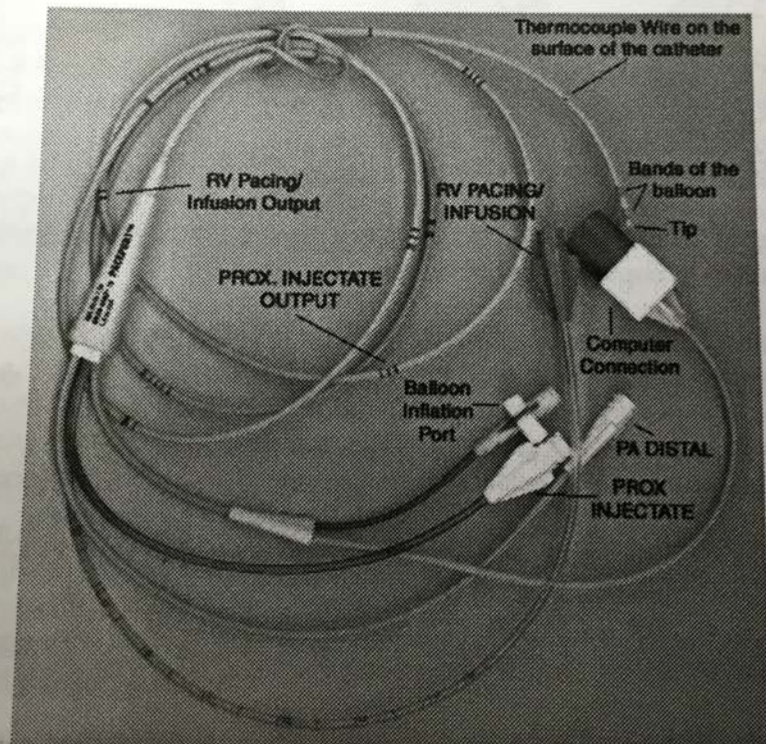


Fig. 1.73. In sick sinus syndrome (brady-tachy), the contractions of heart oscillates between fast & slow rates

HOW TO GET TRACINGS

- Usually inflate the balloon when you get to RA , which is about ~ 20cm mark, should see RA tracing on monitor
- Withdraw only with balloon deflated
- Make sure to print out tracings of RA, RV, and PA pressures as you advance the catheter. Can also perform oxygen saturations at each chamber to diagnose shunt.
- If inflation to < 1cc causes wedge then PA line is overly distal; deflate and withdraw slightly
- If inflation to 1.5cc cannot cause wedge, then PA line is overly proximal; advance carefully
- Confirm placement with CXR – tip should be in PA within the middle third of thoracic width
- Zero by nursing before taking measurements

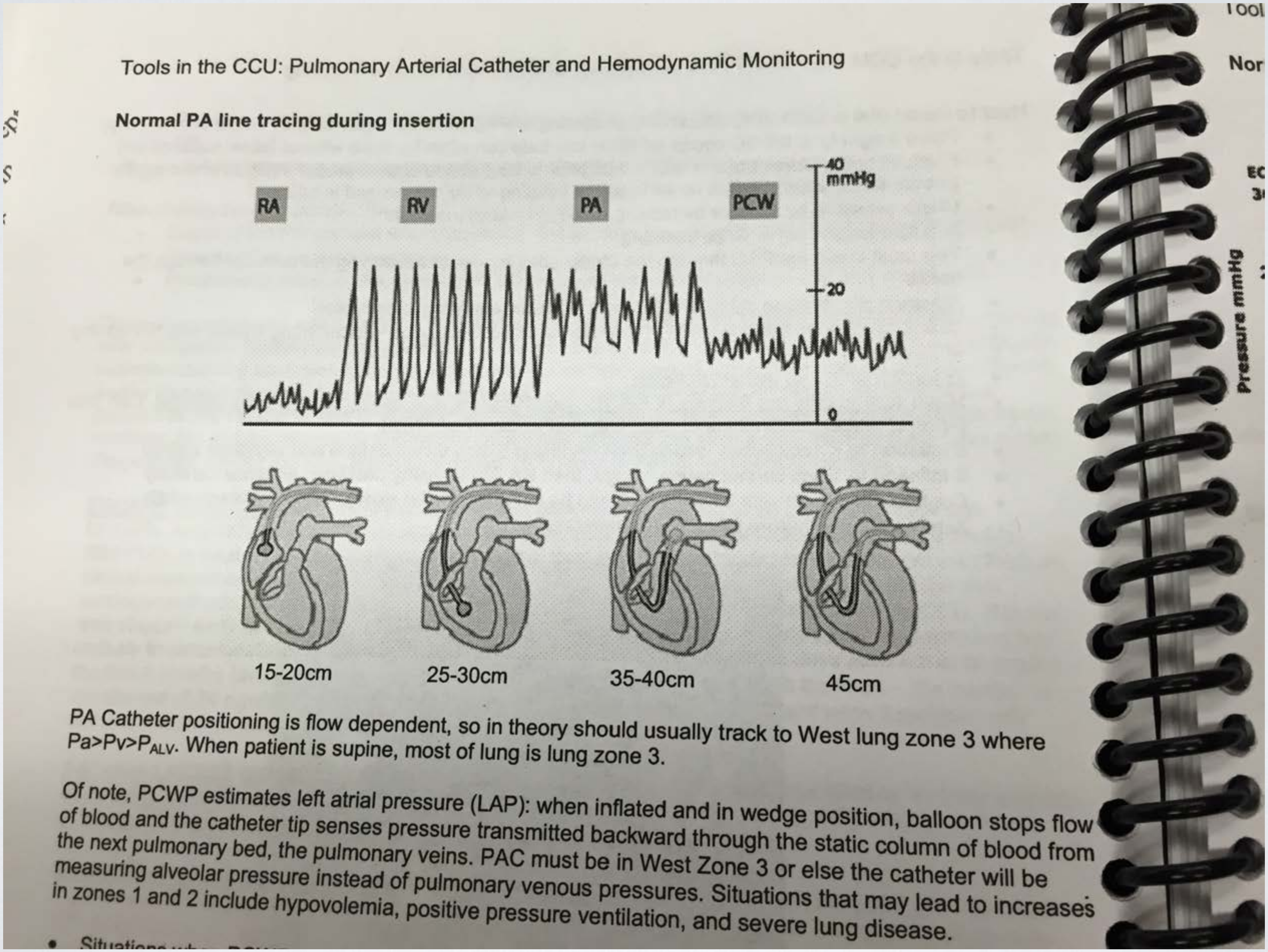


Tools in the CCU: Pulmonary Arterial Catheter and Hemodynamic Monitoring

How to insert (the actual manipulation and advancing of PAC must be supervised by fellow or attending):

- Place a right IJ or left SC cordis (at MGH this step can often be done without fellow supervision)
- Flush all ports, check balloon with 1.5 cc prior to insertion to ensure proper inflation and integrity. Ensure that the balloon has no air leaks by inflating while submerged in saline
- Check pressure transducer by moving catheter prior to insertion
- Maintain natural curve while inserting
- You must insert the PAC through the sterile sheath prior to advancing the catheter through the cordis
- Advance with balloon inflated ONLY with fellow or attending supervision
- Usually inflate the balloon when you get to RA , which is about ~ 20cm mark, should see RA tracing on monitor
- Withdraw only with balloon deflated
- Make sure to print out tracings of RA, RV, and PA pressures as you advance the catheter. Can also perform oxygen saturations at each chamber to diagnose shunt.
- If inflation to < 1cc causes wedge then PA line is overly distal; deflate and withdraw slightly
- If inflation to 1.5cc cannot cause wedge, then PA line is overly proximal; advance carefully
- Confirm placement with CXR – tip should be in PA within the middle third of thoracic width
- Zero by nursing before taking measurements

INSERTING CATHETER



Normal PA line tracing during insertion

PA Catheter positioning is flow dependent, so in theory should usually track to West lung zone 3 where $Pa > Pv > P_{ALV}$. When patient is supine, most of lung is lung zone 3.

Of note, PCWP estimates left atrial pressure (LAP): when inflated and in wedge position, balloon stops flow of blood and the catheter tip senses pressure transmitted backward through the static column of blood from the next pulmonary bed, the pulmonary veins. PAC must be in West Zone 3 or else the catheter will be measuring alveolar pressure instead of pulmonary venous pressures. Situations that may lead to increases in zones 1 and 2 include hypovolemia, positive pressure ventilation, and severe lung disease.

VASOACTIVE MEDICATIONS AND INOTROPES IN THE CCU

he acutely ill cardiac patient may require the continuous infusion of medications to support cardiovasc
ınction. These agents fall into four general categories:

1. vasopressors
2. inotropes
3. chronotropes
4. vasodilators

ıe physiologic effect of these medications is derived from their action upon catecholamine receptors
enerally α-1, β-1 and β-2 adrenergic receptors) or downstream signaling pathways. Most will exert a
ɔmbination of effects based on the pattern of receptor activation. Thus, knowledge of drug mechanisn
itical to understanding the resulting hemodynamic effects. Invasive monitoring including arterial line,
ɛntral venous catheter, and sometimes pulmonary artery catheter are often needed.

Class	**Drug**	**Dose**	**Mechanism**
Vasopressor	phenylephrine	10-1000 mcg/min	α-1 agonist
	vasopressin	0.01-0.04 units/min	V-receptor agonist
Mixed	norepinephrine	2-100 mcg/min	α-1, β-1 agonist
	dopamine	1-20 mcg/kg/min	D < β-1, β-2 < α-1 agonist
	epinephrine	0.05-10 mcg/min	α-1, β-1, β-2 agonist
Ionotrope	digoxin	1 mg IV in divided doses	Na^+/K^+ pump inhibitor
Inodilator	dobutamine	10-1000 mcg/min	β-1 > β-2 agonist
	digoxin	1 mg IV in divided doses	Na+/K+ pump inhibitor
Inodilator	milrinone	0.375-0.75 mcg/kg/min	PDE III inhibitor
Chronotrope	isoproterenol	0.1-20 mcg/min	β-1, β-2 agonist
Vasodilator	nitroprusside	5-800 mcg/min	NO
	nitroglycerin	10-500 mcg/min	NO donor

ıtecholamine receptors

- α-1 adrenergic receptors are predominantly located in the peripheral vasculature. Binding to tl

HEART ARTERIES

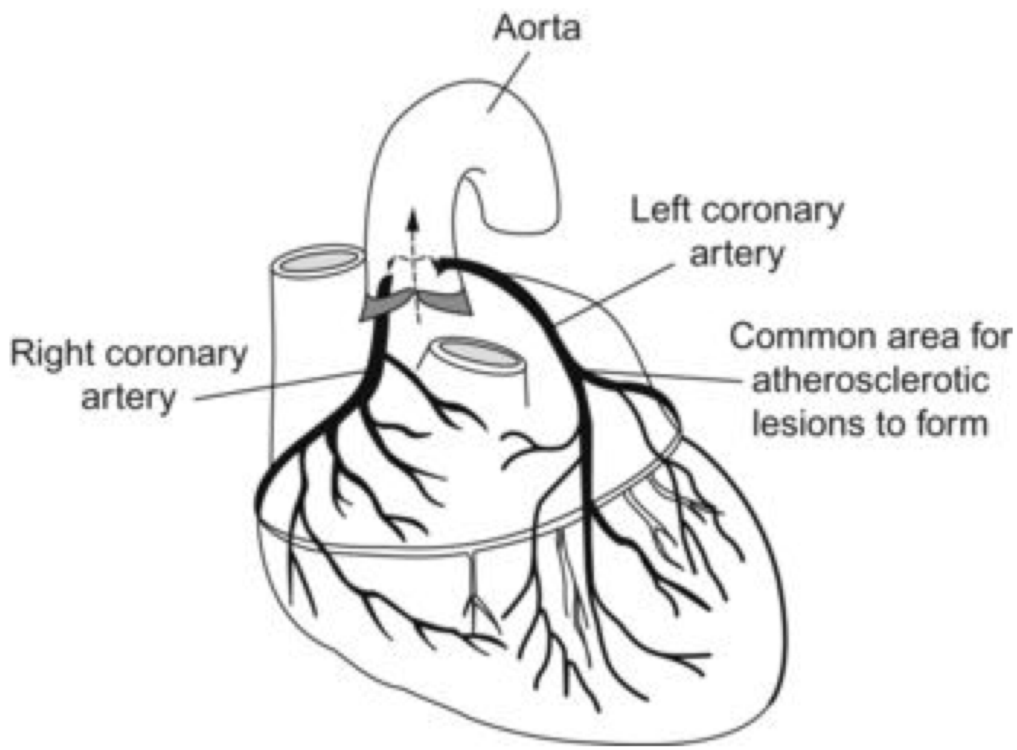


Figure 4.14 The coronary arteries that supply the entire cardiac muscle with blood. These vessels are principal locations for atherosclerotic lesions and other cardiac diseases. Depending on the severity of the damage to these vessels, blood flow to the cardiac muscle cells can be severely impaired.

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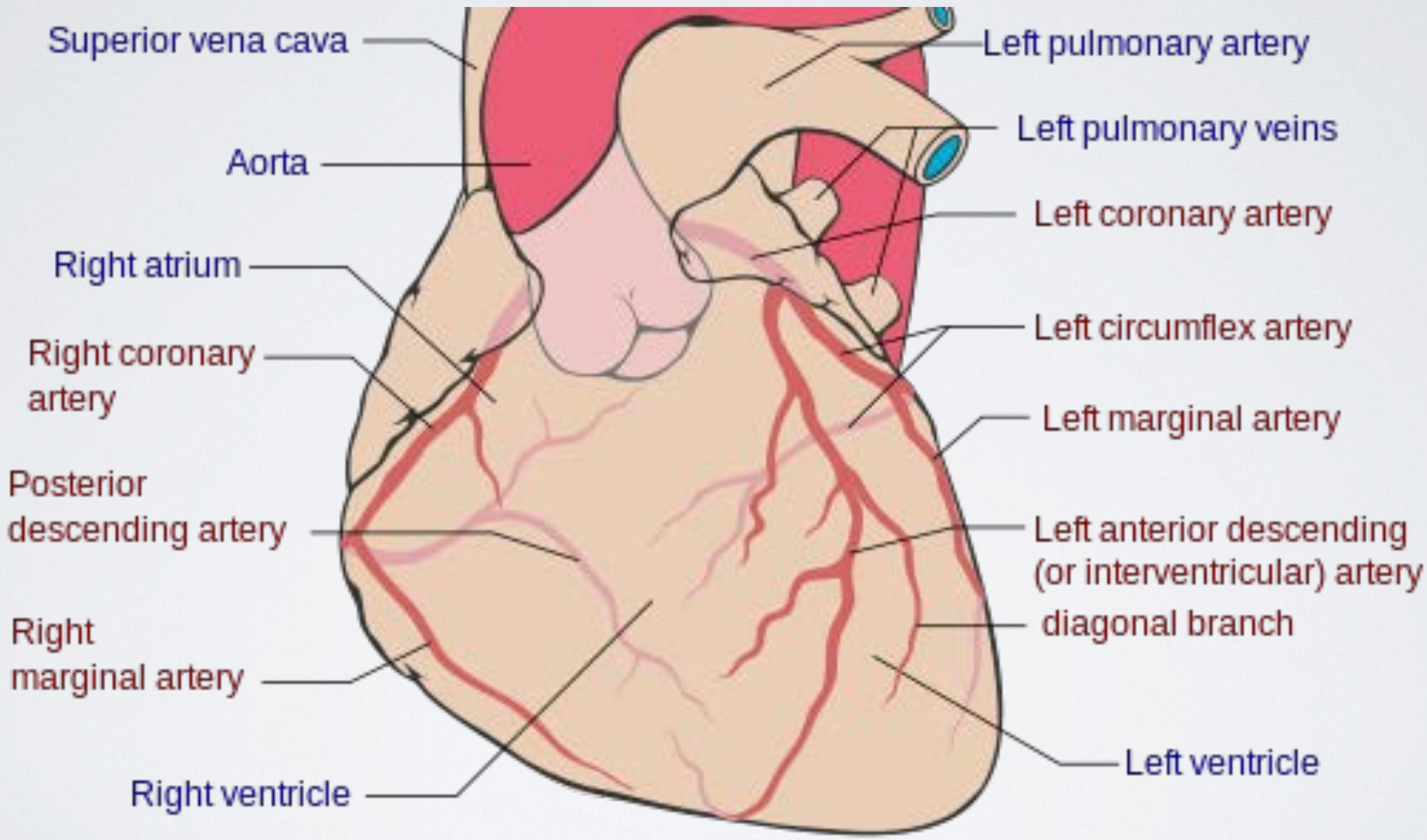


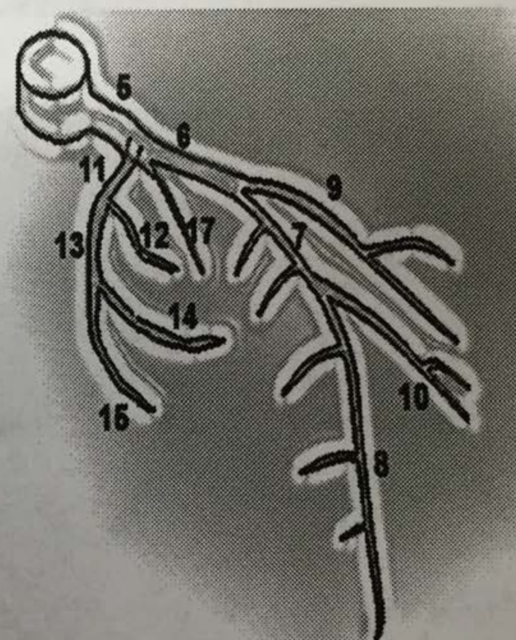
ARTERY DIAGRAM

The right coronary also gives rise to the posterior descending (PDA) in a right dominant system as
and the posterior left ventricular (PLV).

Dominance: Most individuals (85% of population) are right dominant. Dominance is determined by
artery gives off the PDA (RCA v. LCx).

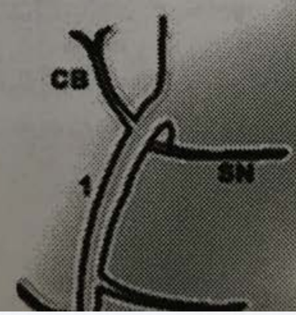
Left Coronary Artery Branches

5- Left main coronary artery
6- Proximal LAD
7- Mid LAD (after take off of D1)
8- Distal LAD
9- First diagonal branch (D1)
10- Second diagonal branch (D2)
11- Proximal Left Circumflex
12- First Obtuse Marginal (OM1)
13- Mid left circumflex (after OM1)
14- Second Obtuse Marginal (OM2)
15- Distal Left Circumflex
17- Ramus intermedius



Right Coronary Artery Branches

1- Proximal RCA
2- Mid RCA
3- Distal RCA
4- Posterior descending artery (PDA)
16- Posterior left ventricular branch (PLV)



BASICS IN CORONARY ANATOMY AND ANGIOGRAPHY

The gold standard for the evaluation of coronary arteries is invasive coronary angiography (often referred to as cath). The goal of coronary angiography is to identify coronary anatomy and atherosclerotic burden. You should review the angiography for each patient in the CCU. It is important to have an appreciation for the relevant anatomy involved in the clinical presentation of each patient. Knowledge of each patient's coronary anatomy will also allow you to be more prepared to deal with complications.

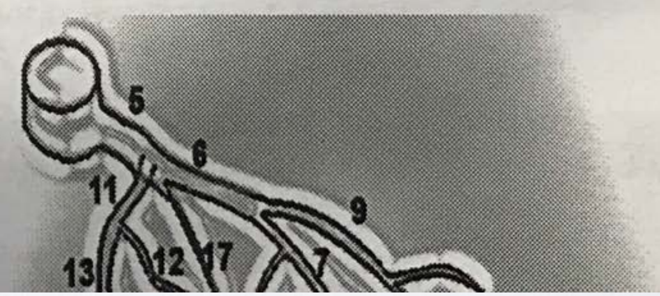
Left Coronary Artery: The left main coronary artery (LM) bifurcates early in its course into the left anterior descending artery (LAD) and the left circumflex artery (LCx). The LAD runs on the anterior part of the interventricular septum. It has two sets of branches – the diagonal branches, which feed the anteroapical and lateral wall, and the septal perforators, which branch from the LAD in straight angles and feed the septum. The left circumflex runs in the AV groove, toward the posterior aspect of the heart. Branches off the LCx are called obtuse marginal branches (OMs) and supply the lateral wall. In left dominant systems, the LCx also gives rise to the PDA (in right dominant systems, the PDA branches off the RCA). Some patients have a ramus intermedius branch, which is a 3^{rd} branch coming off the left main artery (in between the takeoffs of the LAD and LCx).

Right Coronary Artery (RCA): The first branch off the RCA is usually the conus branch, which feeds the right ventricular outflow tract. In roughly 50-60% of patients, the next branch of the RCA is the sinus node artery. The next branch off the right coronary artery is the acute marginal, which supplies the right ventricle The right coronary also gives rise to the posterior descending (PDA) in a right dominant system as well as and the posterior left ventricular (PLV).

Dominance: Most individuals (85% of population) are right dominant. Dominance is determined by which artery gives off the PDA (RCA v. LCx).

Left Coronary Artery Branches

5- Left main coronary artery
6- Proximal LAD
7- Mid LAD (after take off of D1)
8- Distal LAD



CATHETER FLOW REDUCTION!

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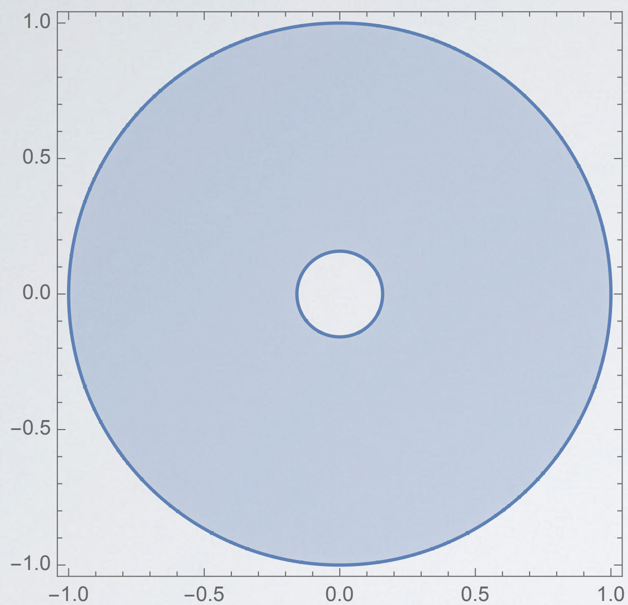

Flow domain

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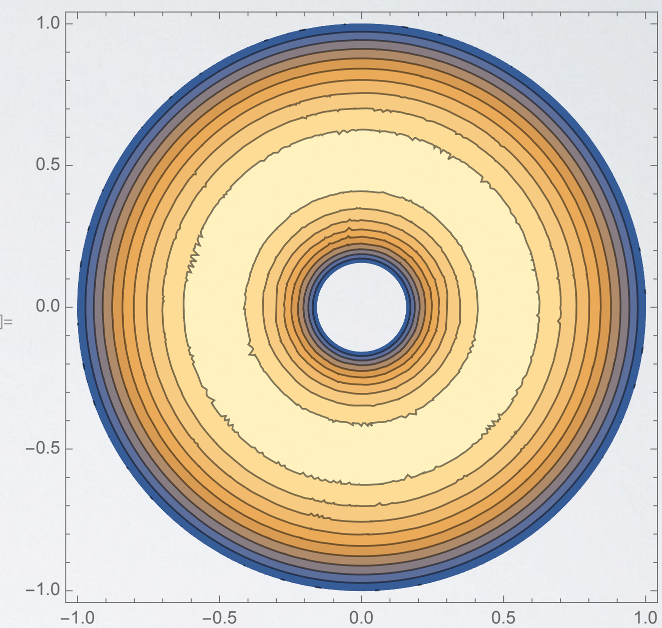


Contours of velocity

In[287]:= `ratiocenterp025 = flowratecenterp025 / flowrateopen`

Out[287]= `0.483903`

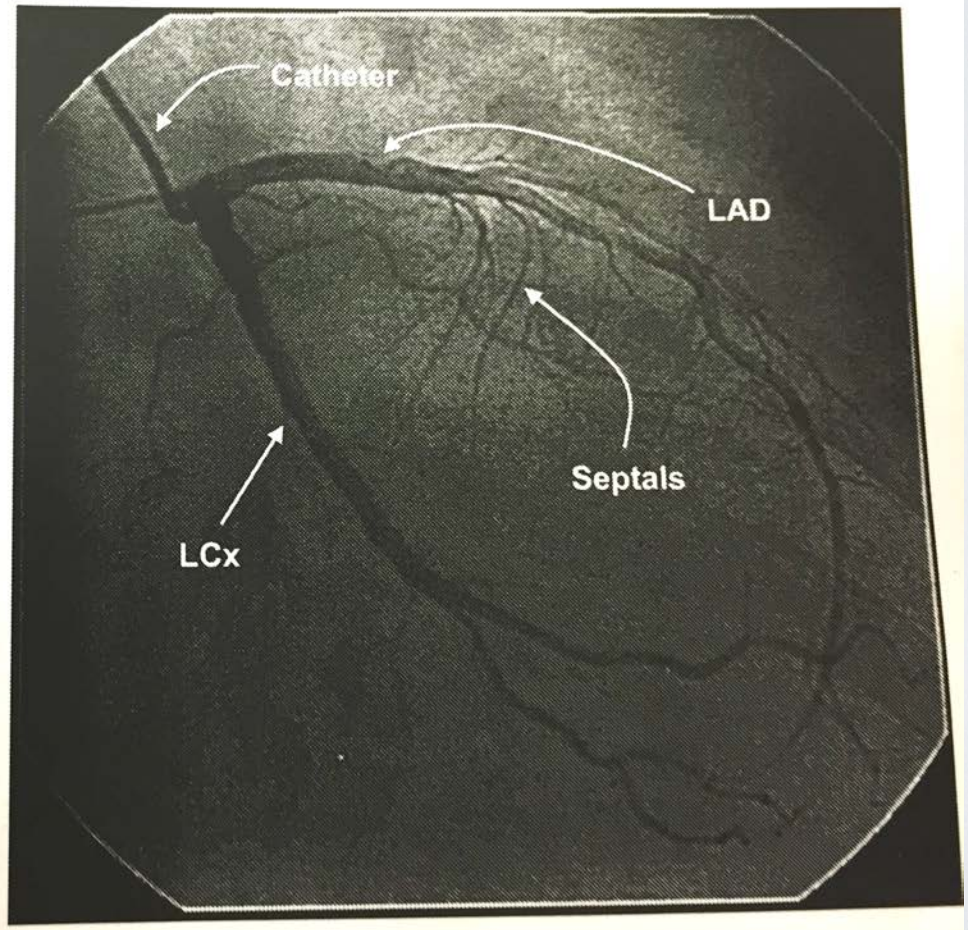
about 50% reduction in flow!

ANGIOIGRAPHY

Tools in the CCU: Basics in Coronary Anatomy and Angiography

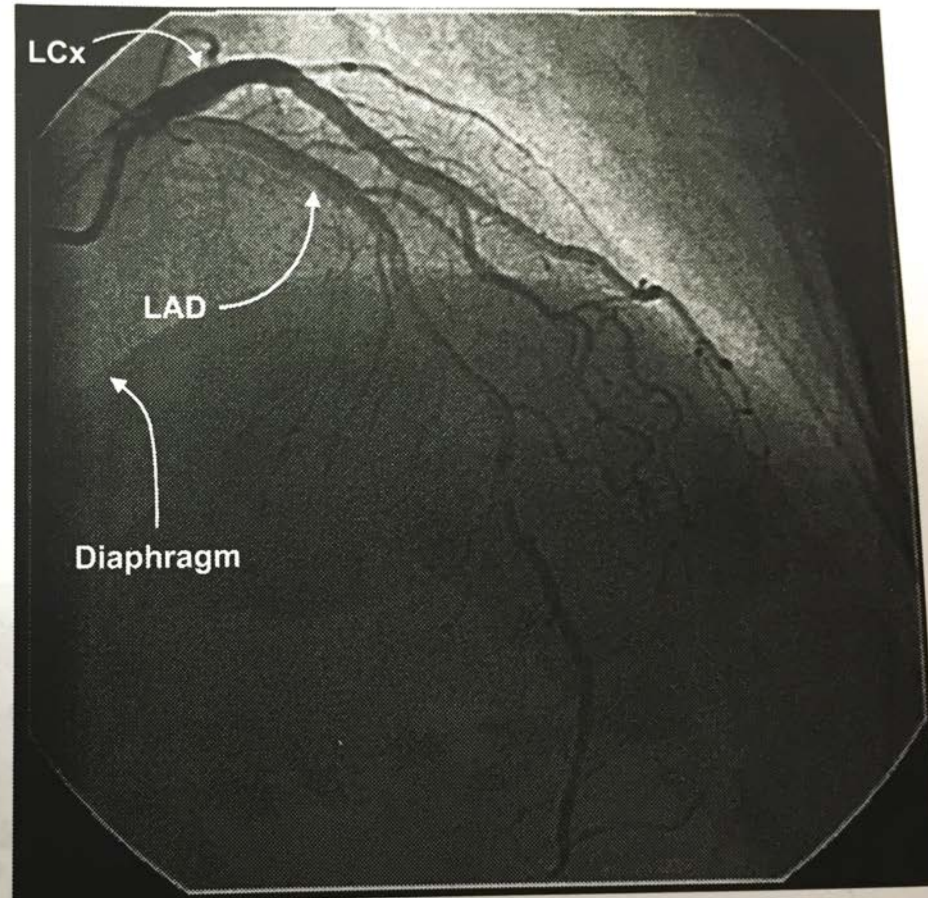
RAO Caudal: LCA view

The camera is on the right side of the patient, looking up from the abdomen. The heart is directed toward the right side of the image, and the catheter is on the left side of the image. The left circumflex is clearly delineated.



RAO Cranial: LCA View

The camera is again on the right side of the patient, now looking down towards the heart from the shoulders. The heart is again directed toward the right side of the image, and the catheter is on the left. The diaphragm is clearly seen. The LAD runs down towards the apex of the heart and ends with a shape that resembles "Salvador Dali's mustache."



“STENOSIS” (NARROWING)

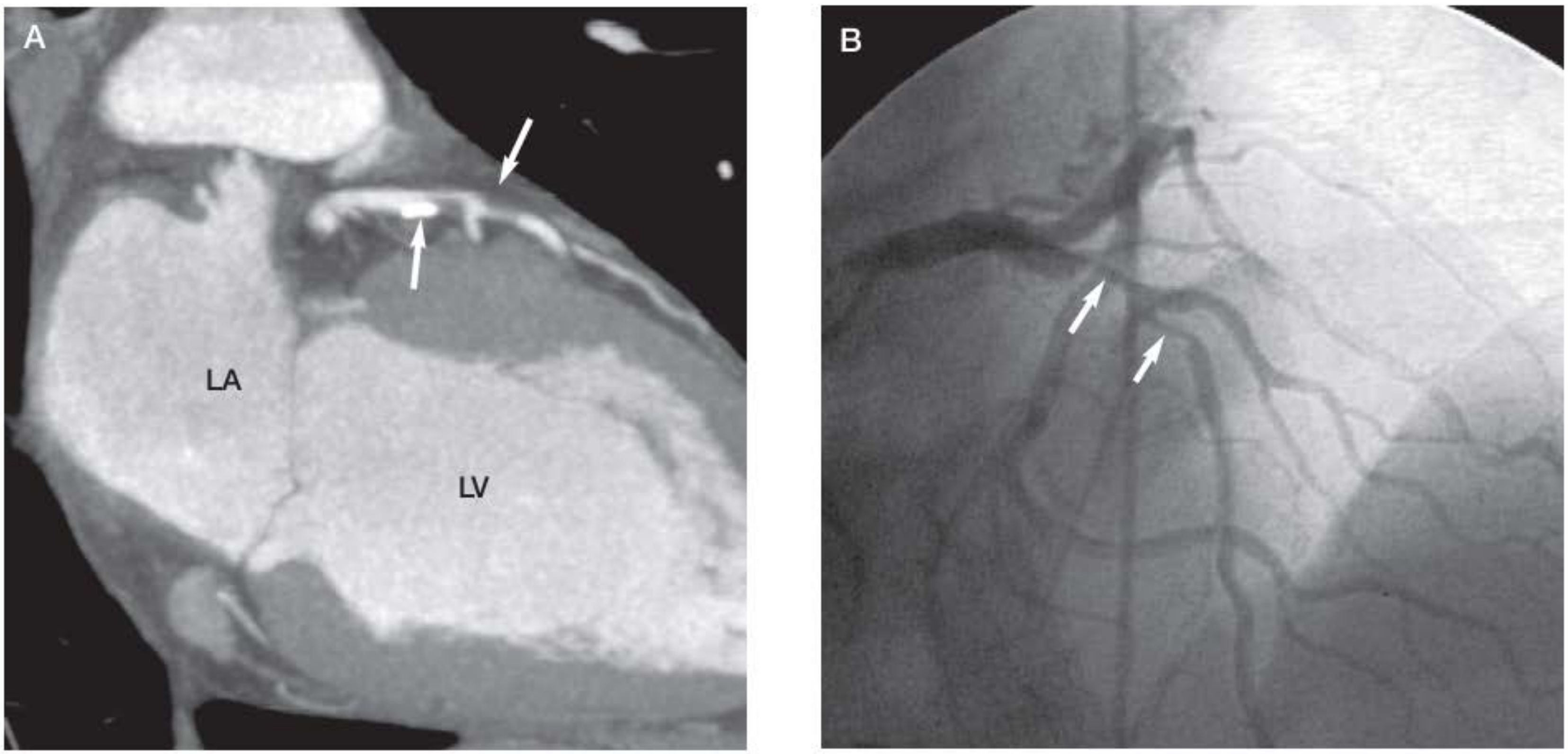


Fig. 6. Tandem high-grade coronary artery stenoses (*arrows*) in left anterior descending artery, proximal and distal to a diagonal branch. *A*, Coronary computed tomographic angiogram, reformatted in vertical long axis. *B*, Invasive selective coronary angiogram. LA, left atrium; LV, left ventricle.

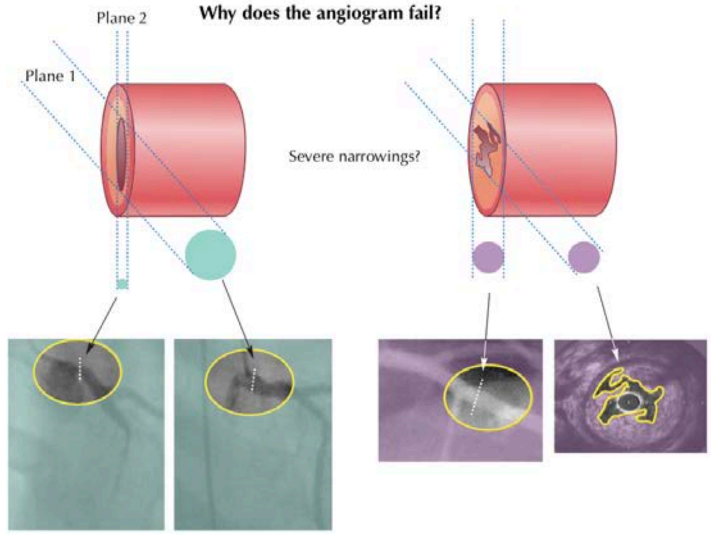


Figure 1 **Why Does the Angiogram Fail to Predict Physiology?**

The angiogram is a 2-dimensional image of 3-dimensional structures. Most intermediate lesions are oval shaped with 2 diameters, 1 narrow and 1 wide dimension. The angiogram of an eccentric lesion cannot reliably indicate flow adequacy. Other lesions **(lower right)** may appear hazy but widely patent, only to be responsible for angina due to plaque rupture, as demonstrated by intravascular ultrasound cross-section **(far right corner)**. Figure illustration by Rob Flewell.

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STATE-OF-THE-ART PAPER

Current Concepts of Integrated Coronary Physiology in the Catheterization Laboratory

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Irvine, California; and Atlanta, Georgia

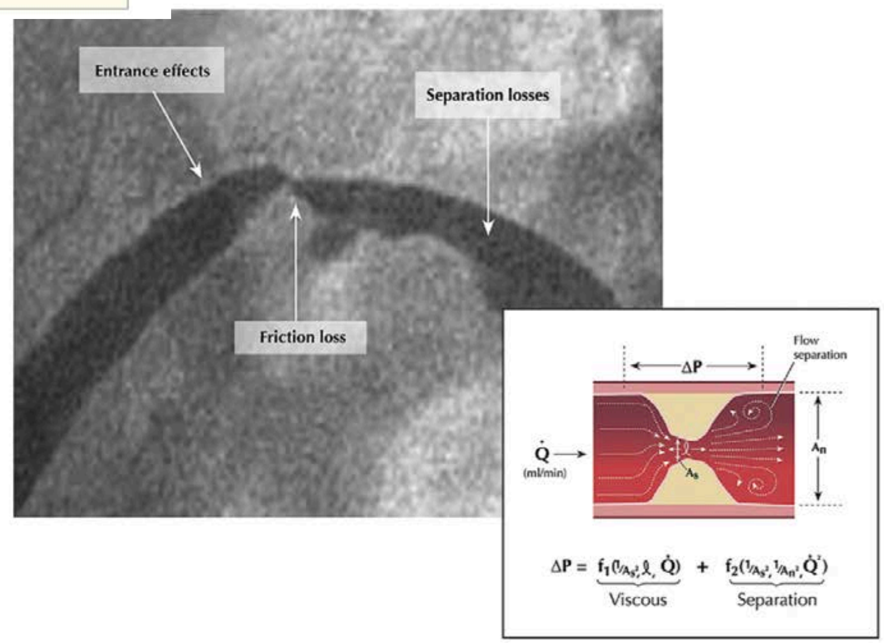
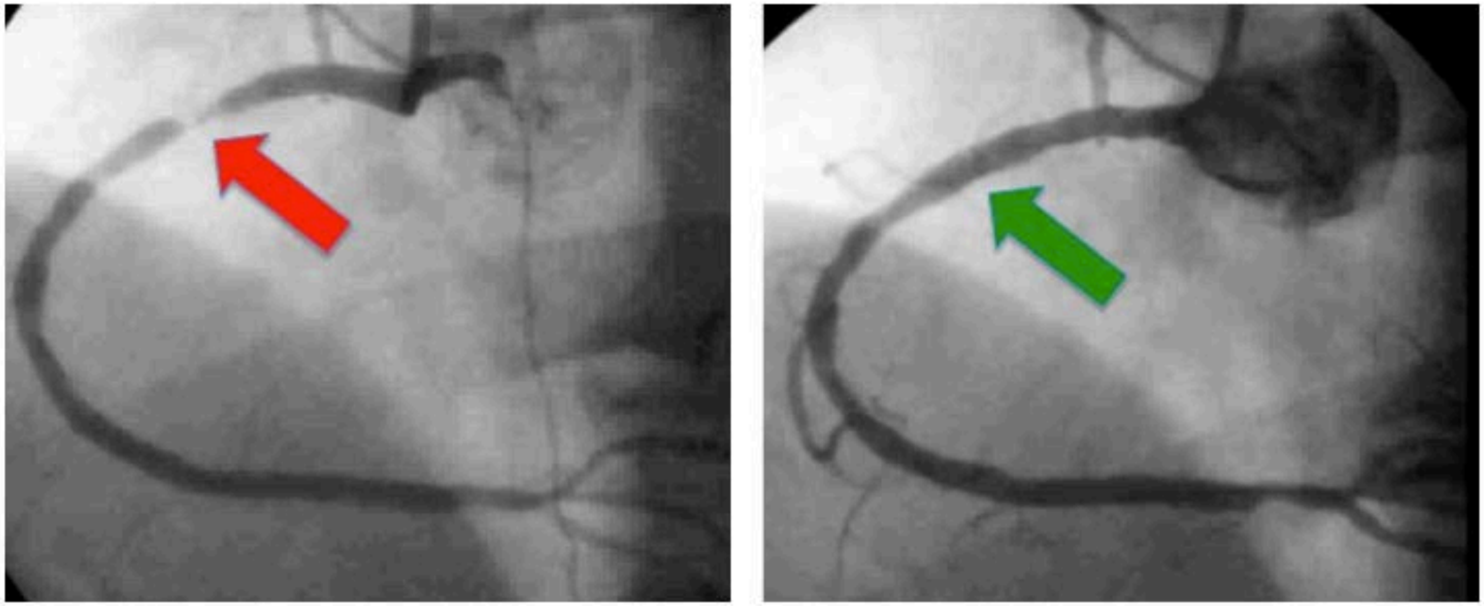
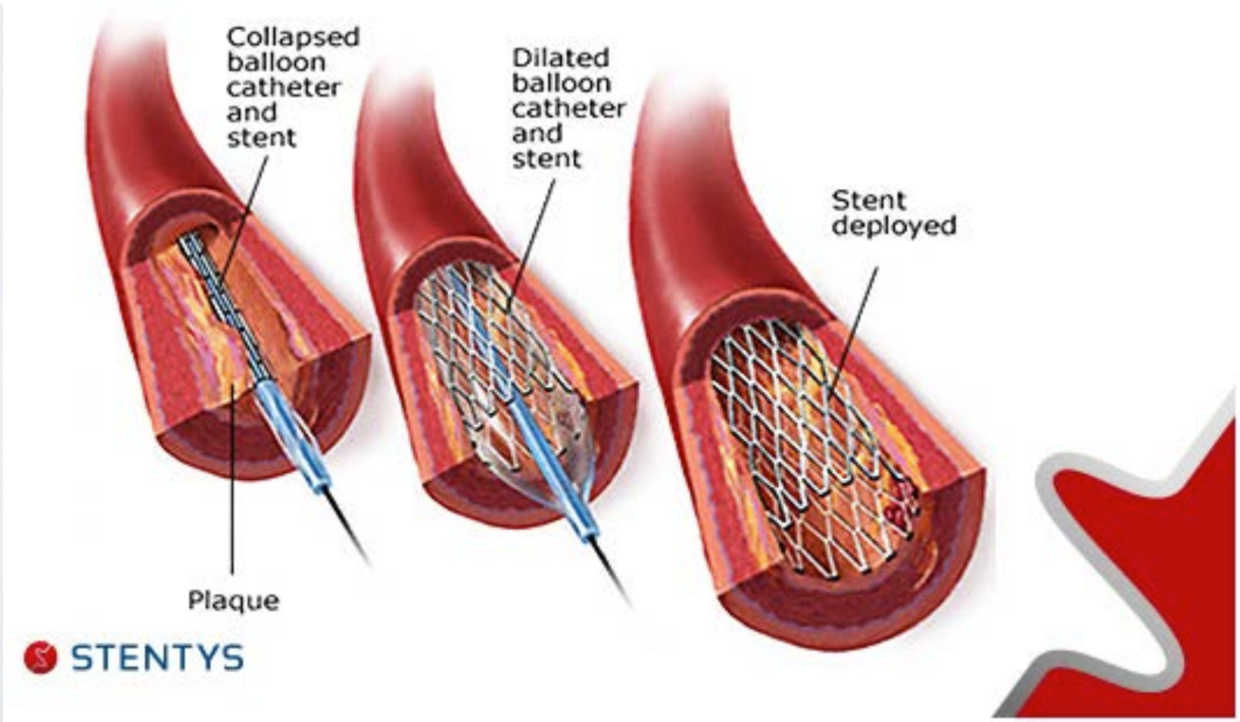


Figure 2 **Factors Producing Resistance to Coronary Blood Flow**

The angiographic 2-dimensional images cannot account for the multiple factors that produce resistance to coronary blood flow and loss of pressure across a stenosis. The eccentric and irregular stenosis **(upper panel)** shows **arrows** designating entrance effects, friction, and zones of turbulence accounting for separation energy loss. The calculation of pressure loss (ΔP) across a stenosis **(lower right panel)** incorporates length (l), areas stenosis (A_s), reference area (A_n), flow (Q), and coefficients of viscous friction and laminar separation (f_1 and f_2) as contributors to resistance and hence pressure loss. Figure illustration by Rob Flewell.



Example of coronary angiogram of a 55 year old gentleman with chest pain. Image on the left shows the right coronary artery with a severe narrowing (red arrow). Image on the right was taken after angioplasty and stenting showing resolution of the narrowing.



40

Valvular Stenosis

Rick A. Nishimura, MD

AORTIC VALVE

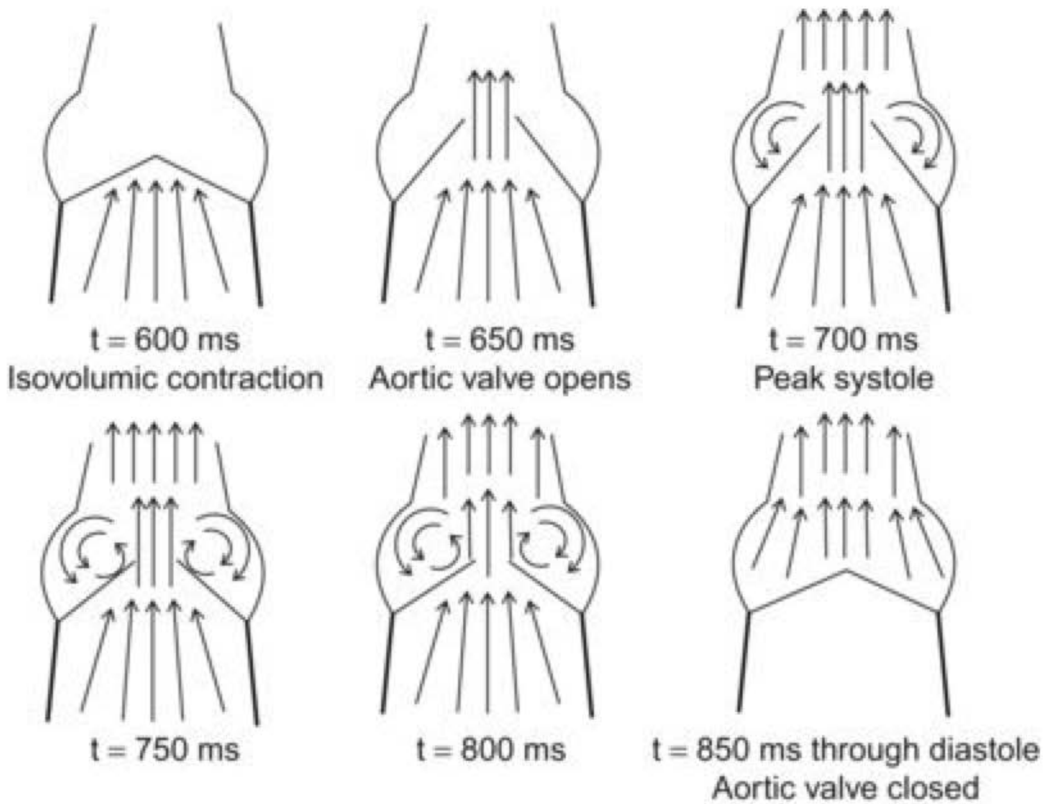
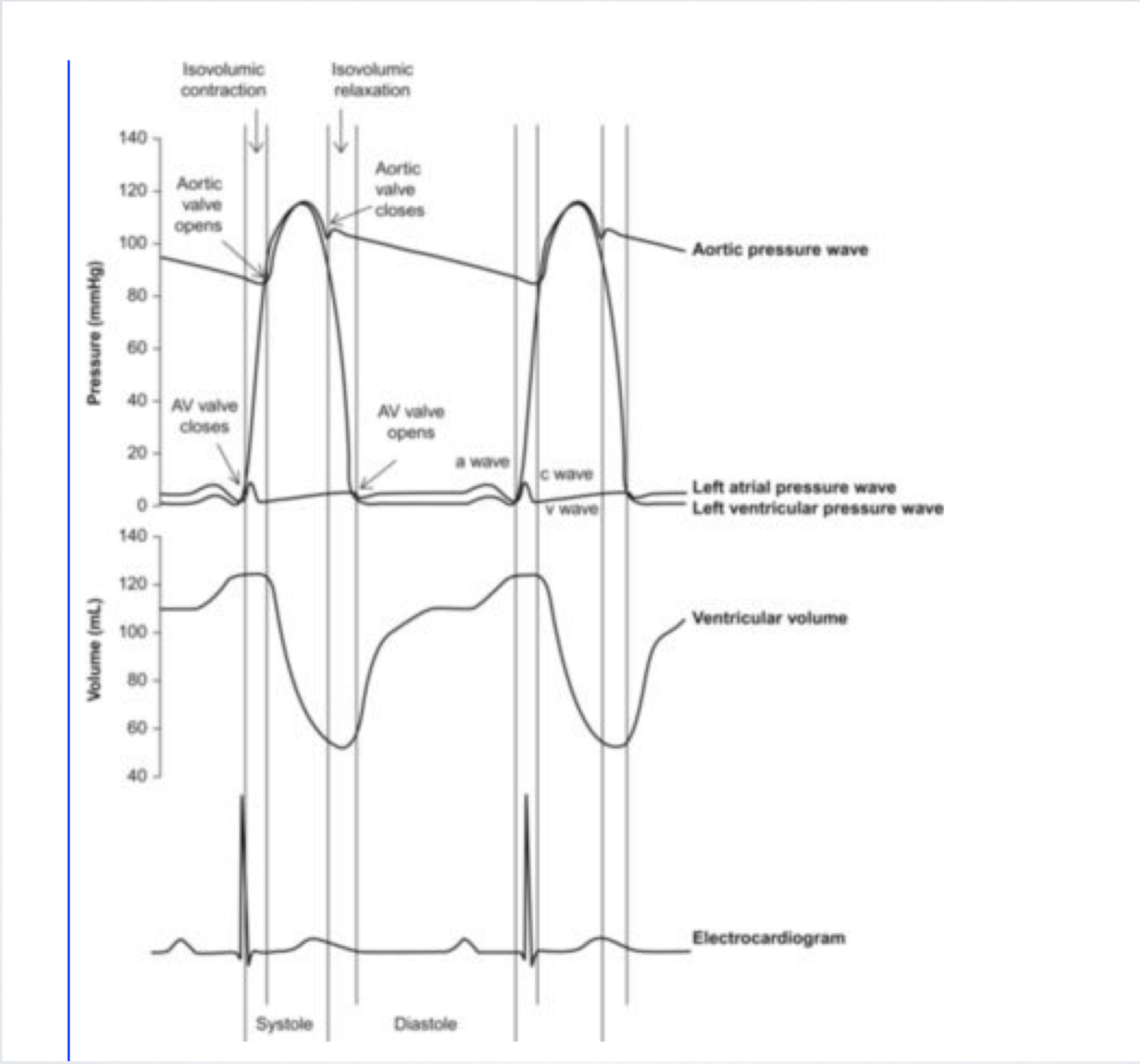


Figure 4.13 Schematic of blood flow through the aortic valve during the cardiac cycle (the times overlap and continue from Figure 4.12). Again, the times are relative, but give a general idea of blood flow through the aortic valve.

PRESSURE TRACINGS



gradients (particularly those with a low AV area but lower gradients than expected), are a particularly high risk group; the low gradients may not be reassuring if they are due to poor myocardial function. Conversely, a normal valve may appear to have a low area if the ejection fraction and the flow across it are low. In patients with low flow, low gradient AS, a dobutamine challenge during echo or cardiac catheterization is a Class II indication for determining whether the severity of aortic stenosis is due to true valvular disease or rather due to other causes of low ejection fraction.

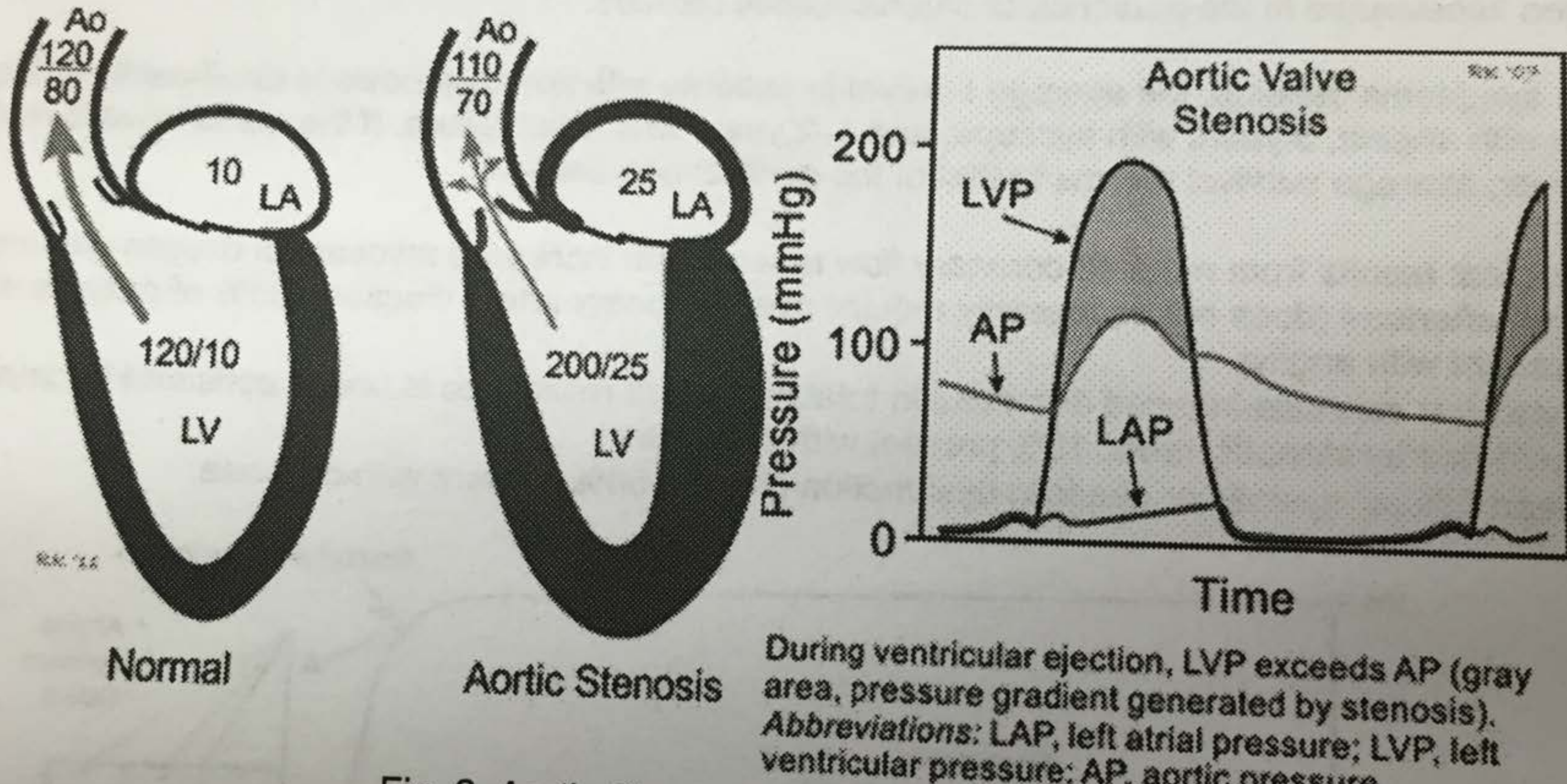


Fig. 2. Aortic Stenosis. CVPhysiology.com.

Acute Medical Management

Volume status can be difficult to optimize in hospitalized patients with severe aortic stenosis. Adequate preload is necessary to ensure sufficient cardiac output in the face of a stenotic aortic valve, and nitrates and diuretics should be used with caution to avoid decreasing preload too quickly and to avoid low diastolic blood pressure (as coronary flow occurs during diastole). Conversely, too high of an afterload can further potentiate high wall tension and diastolic dysfunction, resulting in flash pulmonary edema. Initial management of hypotension in patients with severe AS ...

AORTIC STENOSIS

Aortic stenosis (AS) is the most common cardiac valve abnormality in the United States. It is most caused by calcification of the aortic valve (age > 70) or by a congenital bicuspid valve (age < 70), rheumatic heart disease is responsible for the majority of cases in the developing world. The incide bicuspid valve varies between 0.5–2% worldwide; in a large observation cohort, 30% of adults nee aortic valve replacement (AVR) for AS after 20 years of follow-up. Calcific AS is more common in elderly, and persons with dyslipidemia, chronic kidney disease and atherosclerosis as the disease in part from an active inflammatory process.

	Valve area (cm^2)	Mean gradient (mm Hg)	Jet velocity (m/s)
Mild	> 1.5	< 25	< 3.0
Moderate	1.0–1.5	25–40	3.0–4.0
Severe	< 1.0	> 40	> 4.0
Critical	<0.75		>5.0

Table 1. 2008 ACC/AHA guidelines for grading of severity in aortic stenosis.

In general, there are minimal hemodynamic effects as the aortic valve area is reduced from norm cm^2) to 1.5–2 cm^2. Additional reduction in valve area from half its normal size to one quarter (< 1 gradually leads to LV outflow obstruction, increased afterload and concentric LV hypertrophy wh initially adaptive but becomes maladaptive with time. This process ultimately results in decrease reserve as well as first diastolic and later systolic heart failure. Following diagnosis, the aortic va generally decreases by 0.1 cm^2 per year, though this rate can vary significantly from patient to p is often accelerated in the presence of chronic kidney disease.

Once symptoms develop, the average survival in patients with aortic stenosis is significantly rec years with angina, 3 years with syncope, and 1–2 years with heart failure. If the aortic valve is r however, average survival returns to that of the general population.

CARDIOLOGY CALCULATION

Gorlin. In the cardiac catheterization laboratory, the AVA is calculated from the pressure gradient and an independent measure of cardiac output.

$$AVA = \frac{1{,}000 \times CO}{44 \times SEP \times HR \times \sqrt{\Delta P}}$$

where CO = cardiac output, HR = heart rate, P = pressure difference across the valve, and SEP = systolic ejection period.

Two-dimensional and Doppler echocardiography can also provide reliable estimations of aortic valve area by the continuity equation:

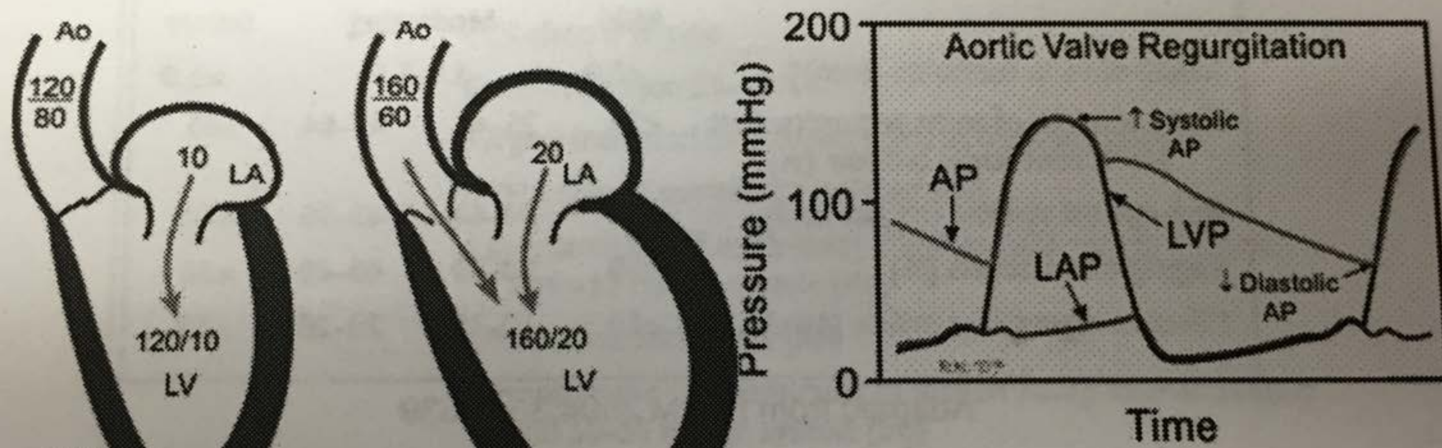
$$AVA = \frac{LVOT_{area} \times LVOT_{TVI}}{AV_{TVI}}$$

where AV = aortic valve flow velocity, LVOT = left amphetamines or dopamine)

- Aortic disease, e.g. annuloaortic ectasia, Marfan syndrome, aortitis due to giant cell, Takayasu's, or syphilis:

Pathophysiology

AR occurs when the aortic valve does not close completely and blood flows back into the LV from the aorta during diastole. As shown in Figure 1, this results in a quick fall in aortic diastolic pressure (AP tracing). Backflow from Aorta to LV → Increase in LVEDV → increased LV preload → increased SV and SBP Increased SBP and decreased DBP as described above lead to characteristic high pulse pressure.



During ventricular relaxation, blood flows backwards from aorta into the ventricle. Aortic systolic pressure increases, aortic diastolic pressure decreases, and pulse pressure increases; LAP increase. *Abbreviations:* LAP, left atrial pressure; LVP, left ventricular pressure; AP, aortic pressure.

Fig. 1. Aortic Regurgitation. CVPhysiology.com.

Acute: In the non-compensated heart with acute AR, the increase in LVEDV results in a large increase left-sided pressures leading to congestive heart failure. The volume overload of the LV pushes it to the

2006;107:421-428)

Echo evaluation: AR is quantified by measuring the vena contracta (the width of the regurgitant jet) on Doppler as well as the regurgitant orifice size and regurgitant volume. With severe AR, Doppler shows steep deceleration in the jet velocity due to equalization of aortic and LV pressure and prolonged diastolic flow reversal in the aorta.

Table 1. Classification of the Severity of Aortic Regurgitation.*

Variable	Aortic Regurgitation			
	Mild	Moderate‡		Severe
Width of vena contracta (mm)†	<3.0	3.0–5.9		≥6.0
Ratio of width of aortic regurgitant jet to left ventricular outflow (%)	<25	25–44	45–64	≥65
Regurgitant volume (ml per beat)	<30	30–44	45–59	≥60
Regurgitant fraction (%)	<30	30–39	40–49	≥50
Effective regurgitant orifice (mm²)	<10	10–19	20–29	≥30

Adapted from NEJM 2004;351:1539

Measurement of LV systolic and diastolic diameters, EF, and aortic width on TTE are also important as these are key parameters which influence management. Because chronic AR may be silent while the LV dilates, exercise testing may be indicated in asymptomatic patients, especially those with limited activity, to assess functional limitations and changes in LV function with stress.

Management (acute or decompensated AR if not surgical candidates or prior to surgery)

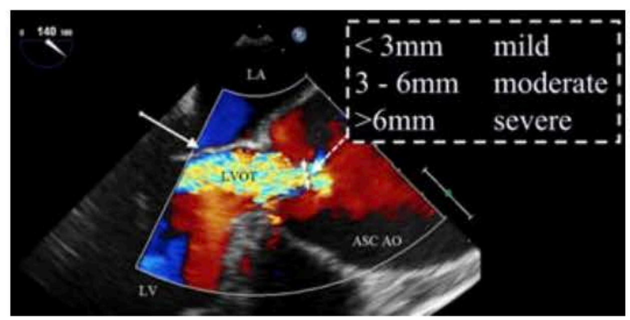
- IV afterload reduction such as nitroprusside (Class IIb)
- Inotropic support such as dobutamine as needed
- Further chronotropic support with overdrive pacing or isoproterenol to reduce diastolic regurgitation time
- Diuretics as needed to reduce fill

TEE Evaluaton of Aortic Regurgitation

Christopher A. Troianos, M.D.
Professor and Chair, Department of Anesthesiology
Western Pennsylvania Hospital
West Penn Allegheny Health System
Pittsburgh, PA

Indicator	Mild	Moderate	Severe	
Angiographic Grade	1+	2+	3+	4+
Jet width/LVOT width	<0.25	0.25-0.46	0.47-0.64	≥0.65
Vena contracta width (cm)	<0.3	0.3-0.6	>0.6	
Deceleration slope (m/sec^2)			>3	
Pressure half-time (msec)	>500	500-200	<200	
Regurgitant volume (ml/beat)	<30	30-59	≥ 60	
Regurgitant fraction, %	<30	30-49	≥ 50	
Regurgitant orifice area (cm^2)	<0.10	0.10-0.29	≥0.30	

The ME AV LAX also allows for measurement of the vena contracta. The vena contracta is slightly different than the jet width in that it is the smallest diameter of regurgitant color flow at the level of the aortic valve and is usually smaller than the jet width in the LVOT. Some consider the vena contracta measurement to be a stronger measurement than the jet width/LVOT ratio (8). Despite the simplicity of the vena contracta measurement in estimating the severity of AR, there are few limitations, such as the presence of multiple or abnormally shaped regurgitant jets and the



Fluid Mechanics of Blood Clot Formation

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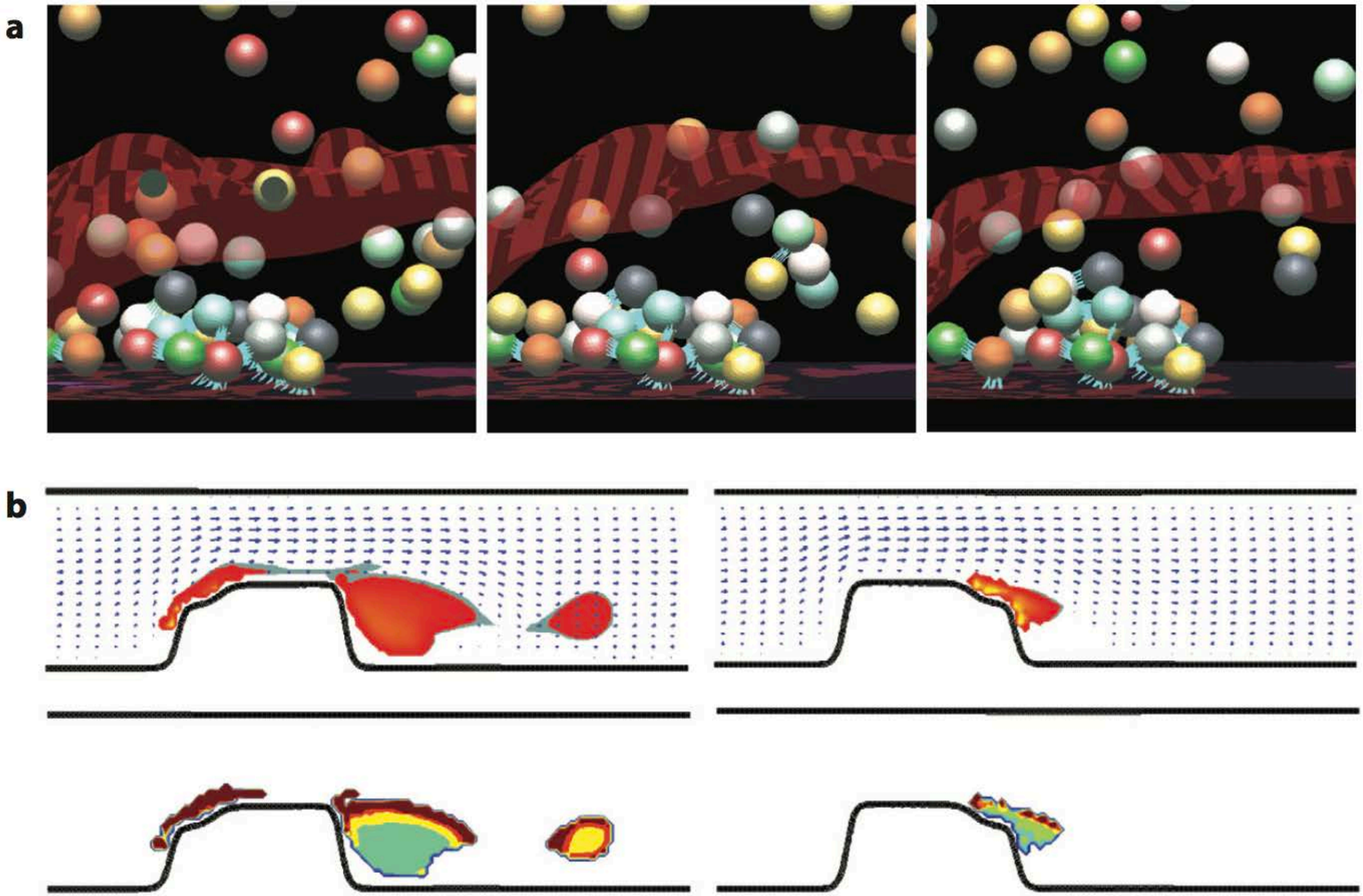
a
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Hemodynamics of Cerebral Aneurysms

Daniel M. Sforza,[1] Christopher M. Putman,[2,3] and Juan Raul Cebral[1]

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Review

Mechanics, mechanobiology, and modeling of human abdominal aorta and aneurysms

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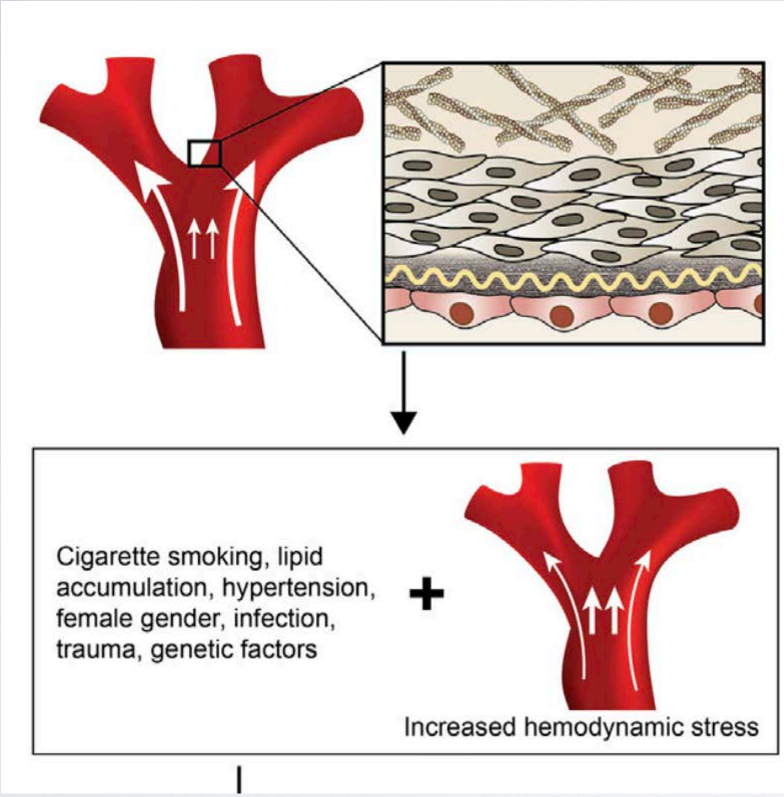
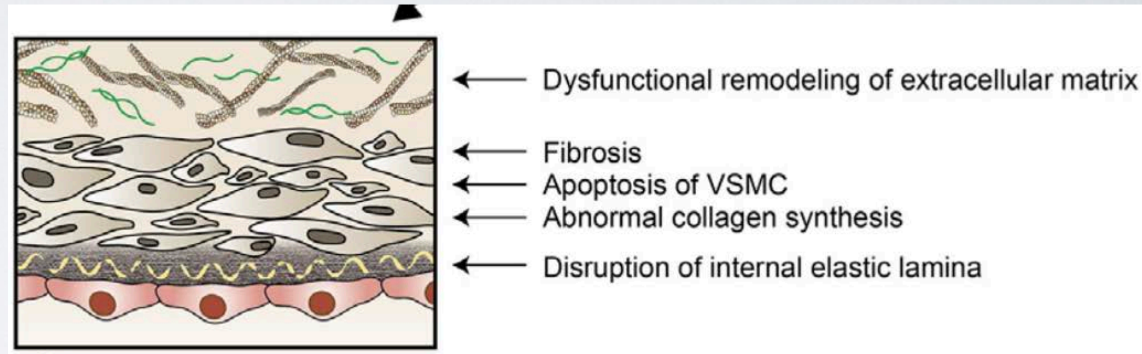
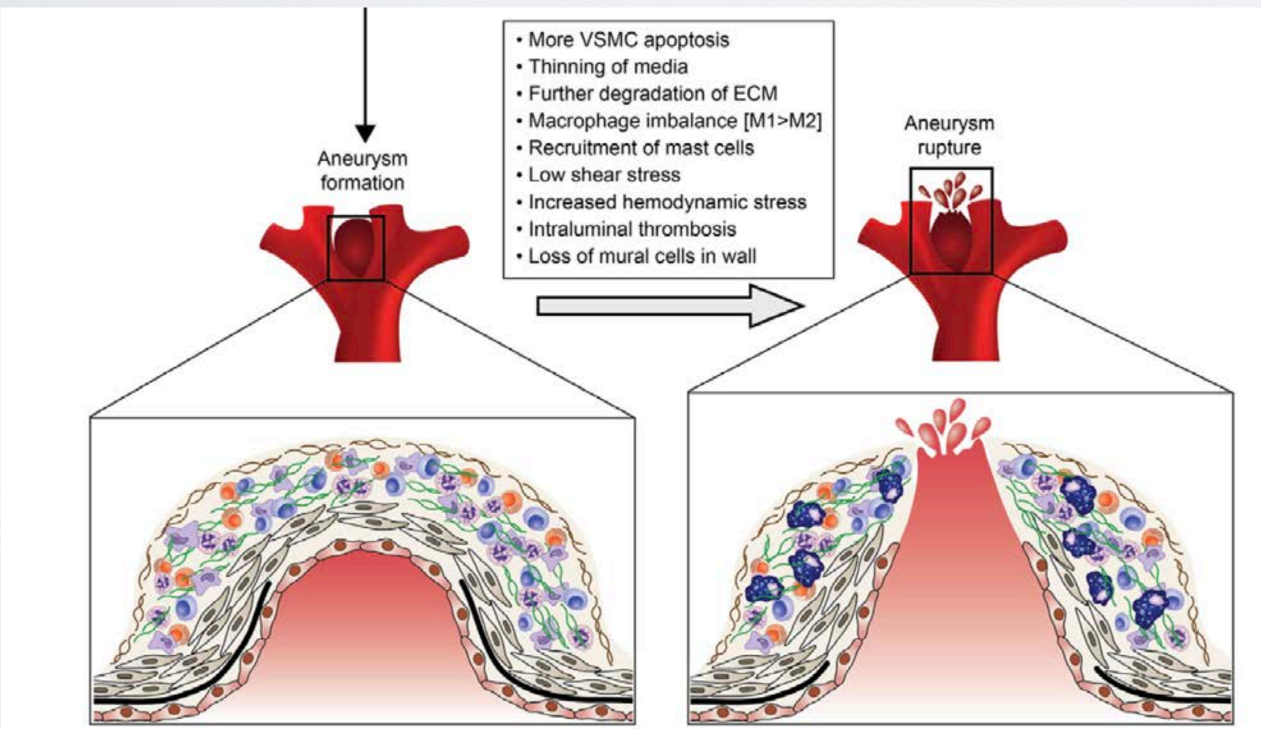
[b] *Institute of Biomechanics, Graz University of Technology, Graz, Austria*
[c] *Department of Solid Mechanics, Royal Institute of Technology (KTH), Stockholm, Sweden*


Cigarette smoking, lipid accumulation, hypertension, female gender, infection, trauma, genetic factors
+
Increased hemodynamic stress


Dysfunctional remodeling of extracellular matrix
Fibrosis
Apoptosis of VSMC
Abnormal collagen synthesis
Disruption of internal elastic lamina


Aneurysm formation
• More VSMC apoptosis
• Thinning of media
• Further degradation of ECM
• Macrophage imbalance [M1>M2]
• Recruitment of mast cells
• Low shear stress
• Increased hemodynamic stress
• Intraluminal thrombosis
• Loss of mural cells in wall
Aneurysm rupture

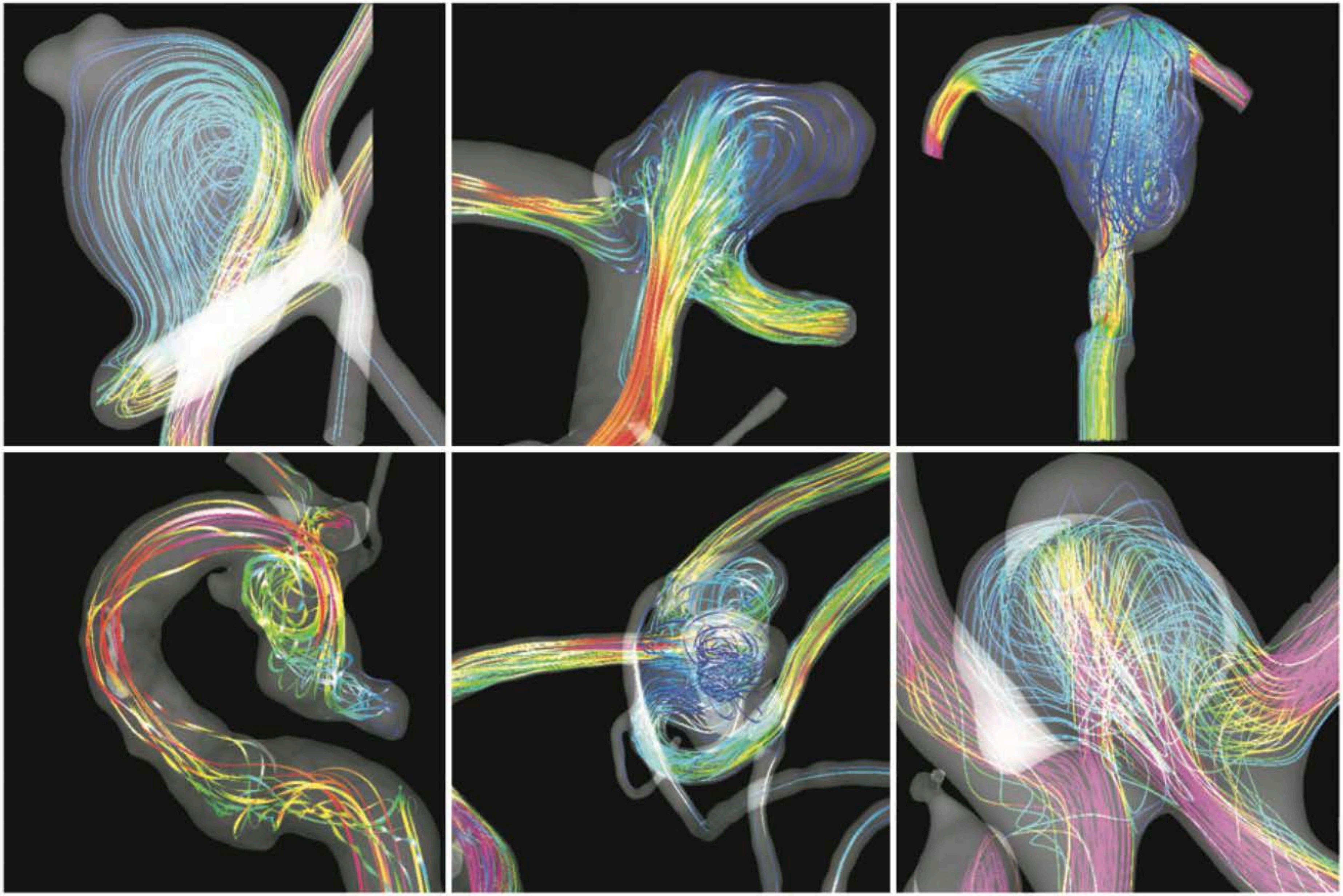


Figure 3

Intra-aneurysmal flow patterns, ranging from simple patterns with a single recirculation region (*top left*) to complex patterns with several vortical structures that can be stable, moving, or intermittent during the cardiac cycle.

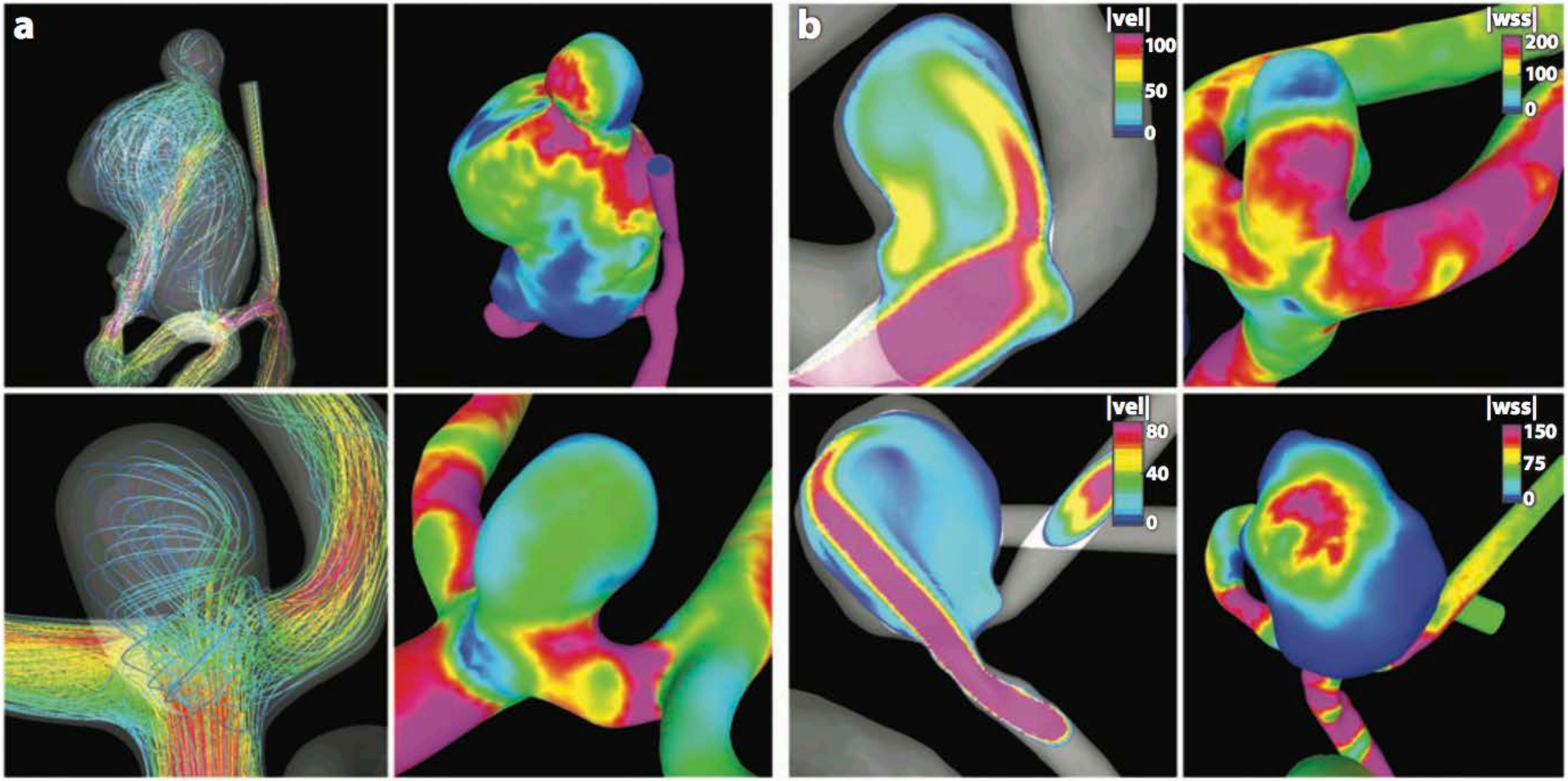


Figure 4

(*a*) Aneurysms with concentrated inflow jet and regions of locally elevated wall shear stress (WSS) (*top panels*) and with diffuse inflow jet and WSS uniformly lower than the parent artery (*bottom panels*). (*b*) Aneurysms with large (*top panels*) and small (*bottom panels*) impingement regions compared to the aneurysm size.

RAPID PROTOTYPE/3D PRINTING

3D printing trachea - Google Search | Ford's Tango With Trump -

SCHMIT PROTOTYPES

Home Capabilities Industries Vi

Medical Rapid Prototyping

Pre-Surgical Plan Modeling

Computer tomography (CT) scans are used to create a model of a fractured bone, such as a jawbone, which allows the surgery team to prebend fixation plates and more accurately choose screws and other accessories needed to complete the surgery. RP is beneficial in surgeries where there are anatomical abnormalities, creating a biomodel to prepare for non-traditional techniques.

Custom Surgical Implants

Instead of grinding a standard implant to fit a patient (before and during surgery), custom implants can be sized according to CT or x-ray images prior to the scheduled surgery. The patient and surgical team spend less time in the OR with a prepared implant sized to fit.

CONCLUSIONS

- Engineering: modeling, experimental tools, data analysis, has already made significant contributions to medicine.
- The need remains great and we can expect much more
  - Tissue/cell level measurements and treatments
    - artificial organs
  - More sophisticated prosthetic scaffolds and devices
- System level analysis for organizations as well as people
- “Big Data”… but less Doc typing while she is talking to you!