Haemodynamics in anaesthesia and intensive care

MUDr. Michal Horáček
Dept. of Anaesthesia/ICM
University Hospital Motol
Praha

Medicine
in vital functions disturbances

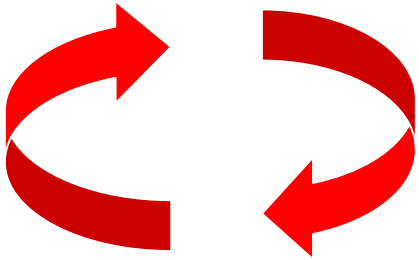
is

an applied physiology!

Medicine
in vital functions disturbances
(consciousness, breathing, circulation
and homeostasis)
is

applied physiology!

„Each adult has approximately 100 trillion cells, that must continuously exchange oxygen and nutrients with external millieu to stay alive.“



Paul L. Marino, MD, PhD, FCCM
The ICU Book, 3. vyd., 2006
Lippincott WIlliams Wilkins

How to manage critical states

- **A** – Airways
- **B** – Breathing
- **C** – Circulation
- **D** – Definitive diagnosis and treatment

To transport oxygen into cells!

prof. Peter Safar
1924-2003

Program

- basic physiology
- heart rhythm disturbances

In part 2

- circulation monitoring
- Shock
- cannulations
- circulatory support

Program

- **basic physiology**
- heart rhythm disturbances

In part 2

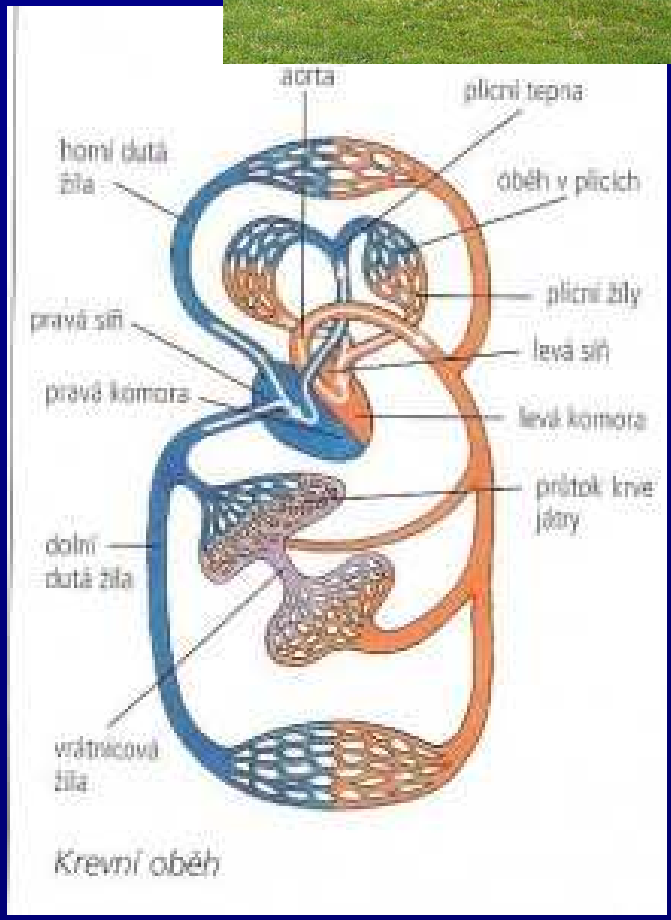
- circulation monitoring
- shock
- cannulations
- circulatory support

Circulation

- heart as a pump
- vessels as pipes
- blood
  (filling of the system)

Circulation

- heart as a pump
- vessels as pipes
  - distribution = arteries
  - exchanger = capillaries
  - capacitance = veins
- blood (filling the system)
  - heart 7 %
  - pulmonary circ. 9 %
  - systemic circ. 84 %
    - arteries 15%
    - capillaries 5%
    - veins 75%



Functions of the circulation

- transport
  - oxygen
  - CO_2 + metabolites
  - cells, hormones, enzymes, drugs
- regulatory
  - to maintain the homeostasis
  - to enable the coordination of organs

Circulatory failure

failure to provide transport and regulatory functions

- **heart failure:** failure to pump the amount of blood necessary to satisfy metabolic needs of tissues
  - injury of : myocardium, valves, arrhythmias, support tissues
  - myocardial failure: injury due to ischaemia/reperfusion, inflammation, trauma, metabolic disturbances, drugs + toxins, deposition (e.g. amyloid)
- **vessel failure:**
  - **macrocirculation:** failure to transport blood into tissues and to change the character of blood flow from pulsatile to continuous
  - **microcirculation:** exchange failure between blood and tissues
- **blood failure:** anaemia, thrombosis, bleeding

The NEW ENGLAND JOURNAL of MEDICINE

EDITORIAL

◂ Previous Volume 340:467-468 February 11, 1999 Number 6 Next ▸

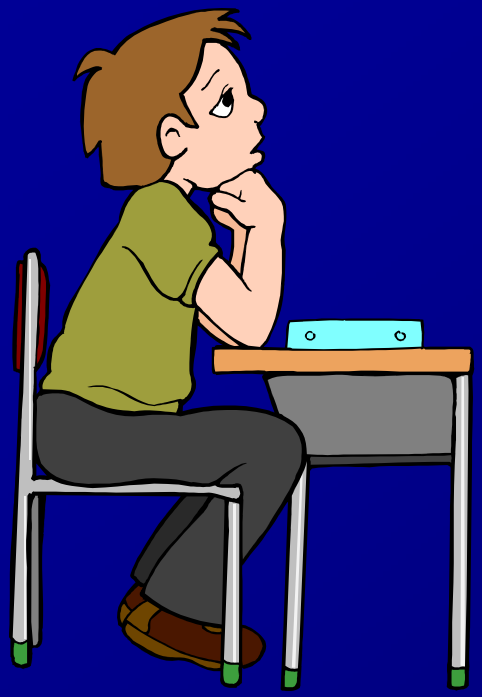
Transfusions in Critically Ill Patients

E. W. Ely and G. R. Bernard

„The art of fluid administration and hemodynamic support is one of the most challenging aspects of treating critically ill patients.“

The main
short-term function
of circulation
is
to transport
enough oxygen
into tissues!

Delivery of oxygen into tissues?



- cardiac output: 5 l/min
- Hb concentration: 150 g/l
- Hb saturation with O_2: 98 %
- O_2 binding to Hb: 1,34 ml/g
- O_2 dissolved in plasma

$DO_2 = 5 * 150 * 0,98 * 1,34 + 0,225 * 13,3$

$= 984,9 + 2,99$

$\equiv 1000$ ml/min

$DO_2I = 520\text{-}720$ ml/min/m^2

What does regulate the cardiac output?

a) brain?
b) heart?
c) metabolic needs?
d) a + b + c is correct?

Circulation

- Heart as a pump
- Vessels as pipes
- Blood (filling the system)

The heart does not regulate its output!

Heart does not regulate its output!

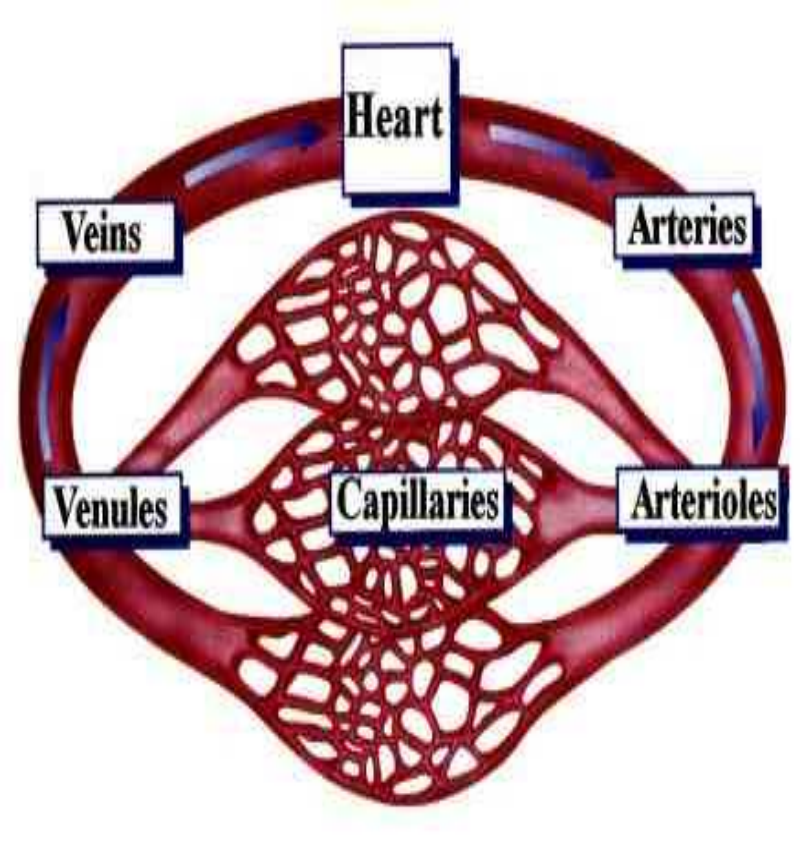
Evidence:

- atrial stimulation 60-160/min changes CO only slightly
  Stein et al. Circulation 1966:33:925

- patient on extra-corporeal circulation:
  CO is limited by venous return
  observed by ourselves as well as by others
  unpublished

What drives blood in the circulation?

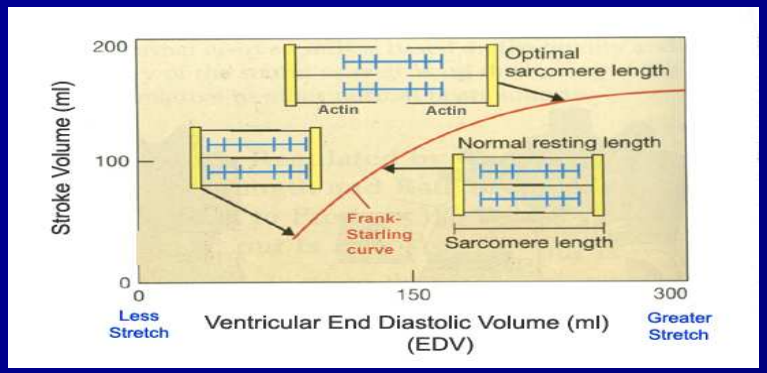
- Heart as a pump
  - competent
  - tolerant
- Vessels as pipes
  - diastolic recoil of arterial walls
  - muscle pump of veins
  - negative pressure in chest in inspirium during spont. ventilation



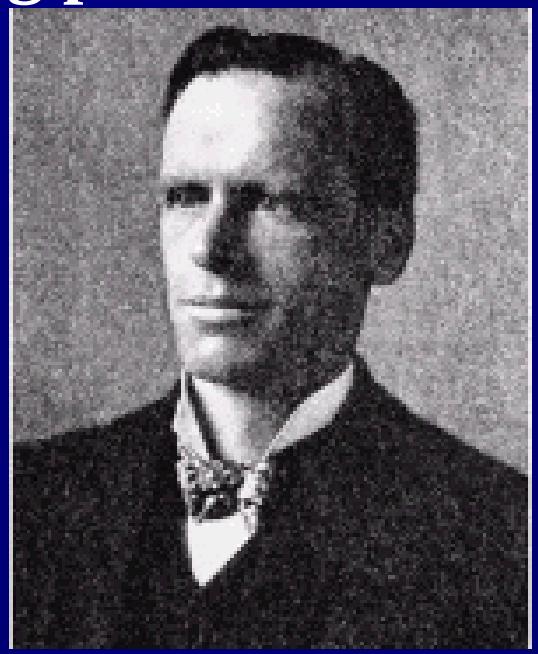
Heart as a pump

Competent and tolerant!

- **competent**
  - **able to pump all blood which returns back and with low filling pressure**



  - Bainbridge´s reflex
- **tolerant (permissive)**



Ernest Henry Starling
1866-1927

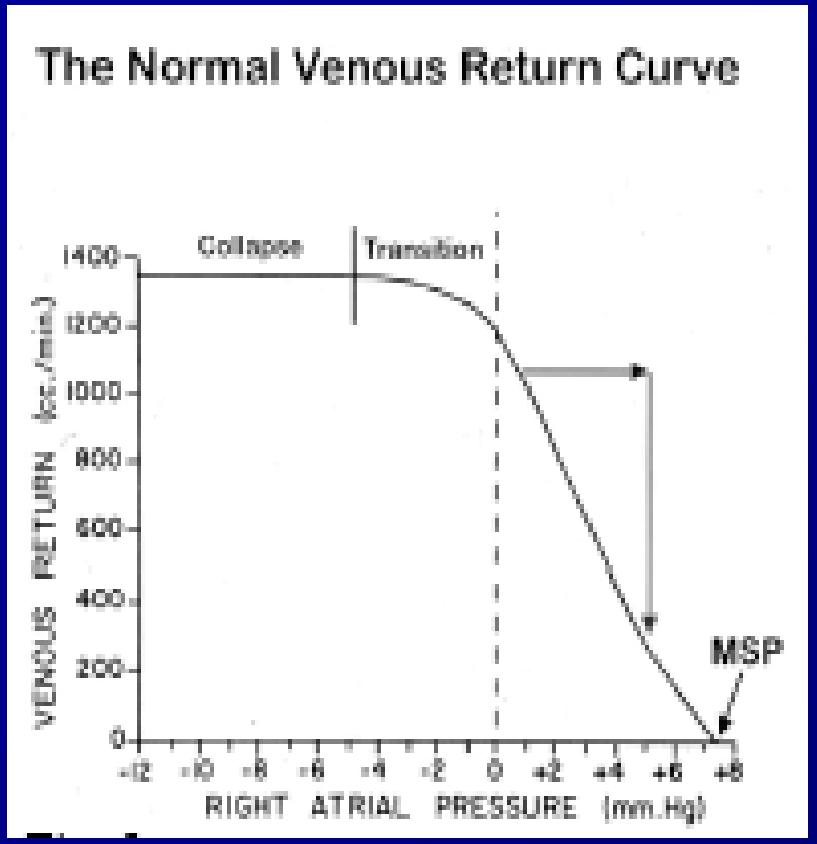
Heart as a pump

Competent and tolerant!

- **competent**
  - able to pump all blood which returns back and with low filling pressure
- **tolerant (permissive)**
  - **abble to pump only the blood, which returns (venous return)**

Arthur C. Guyton
1920-2003

Venous return (VR)

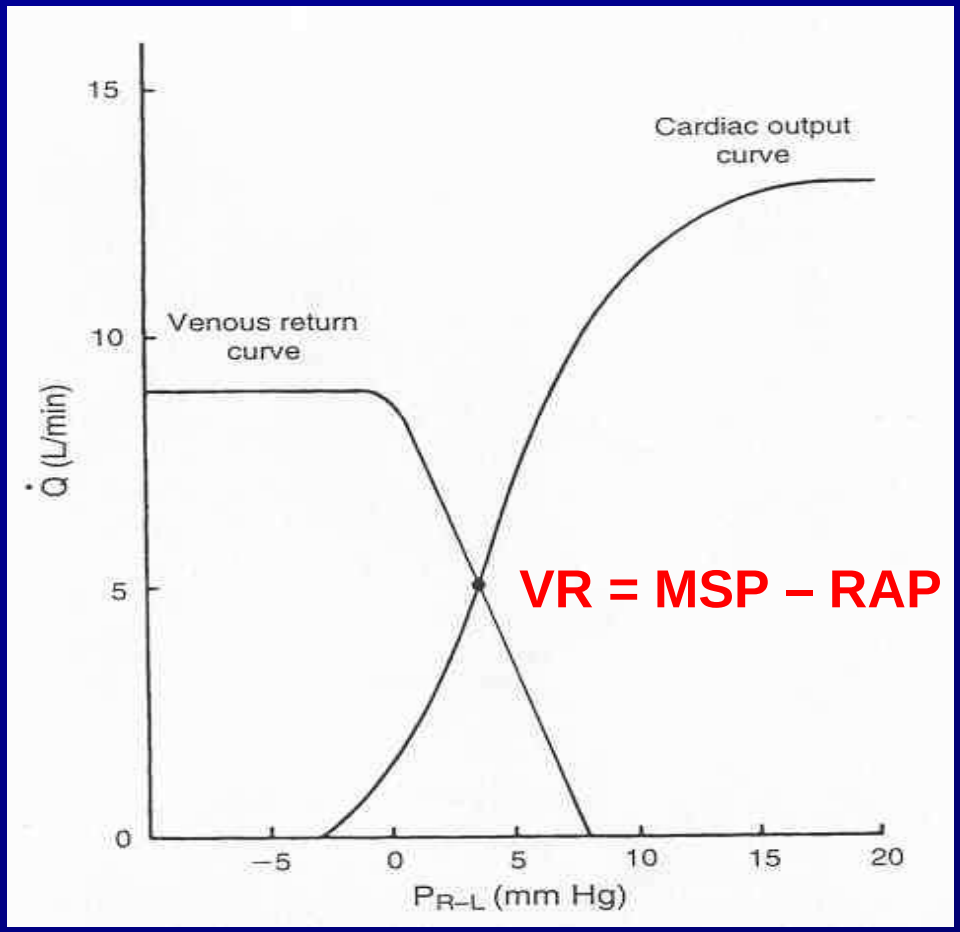


VR = MSP – RAP

MSP (mean systemic pressure)

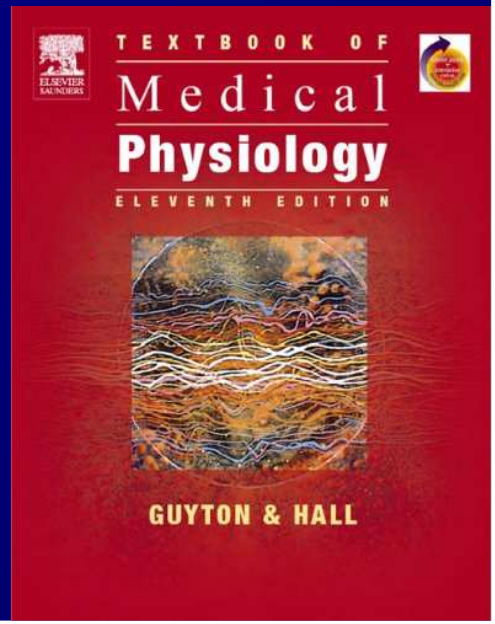
- blood volume
- vessel tone
- distribution of blood in circulation

Venous return



healthy heart

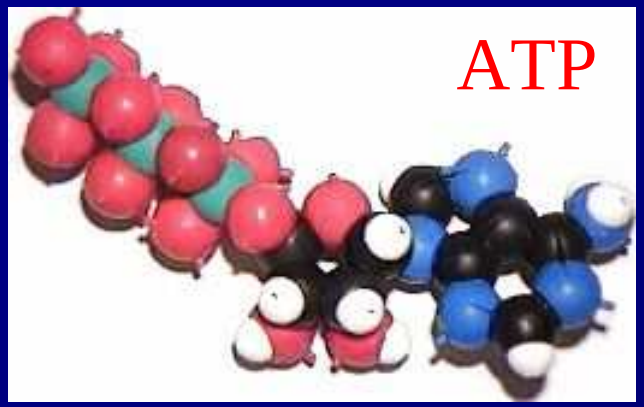
Arthur C. Guyton
1920-2003



Heart as a pump

Competent, tolerant and omnivorous

pump is driven by ATP



- 350 g = 0,5 % b.w.
- 5% of cardiac output
- 10% body O_2 consumption
- synthesis and consumption 35 kg ATP daily

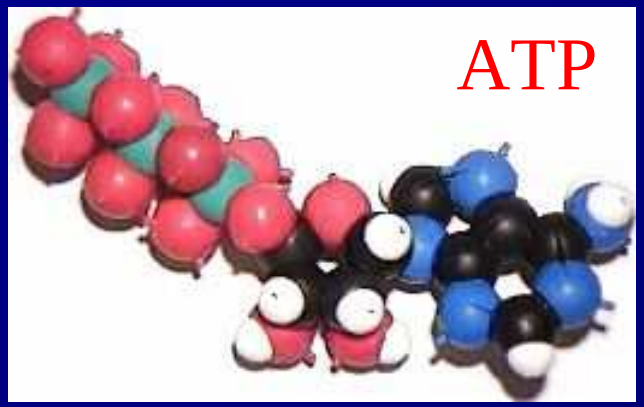
  = > 100 000 x amount of stores

  (calculation according to O_2 consumption in myocardium)
- estimated efficiency ≈ 20-40%

Heart as a pump

Competent, tolerant and omnivorous

pump is driven by ATP



- fatty acids (70%/20%)*
  1 mol 135 mol ATP
  100 g 53 mol ATP
- sugars (60-70%/50-75%)*
  1 mol 38 mol ATP
  100 g 21 mol ATP
- lactate (10%/30%)
- other

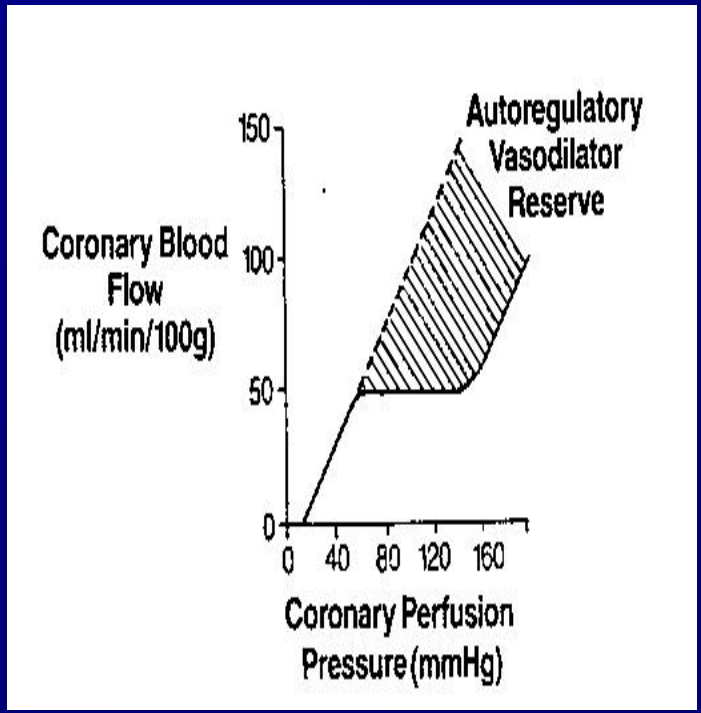
**Fuel share on O_2 consumption in fasted /fed state*

Is the heart always omnivorous?

Vessels

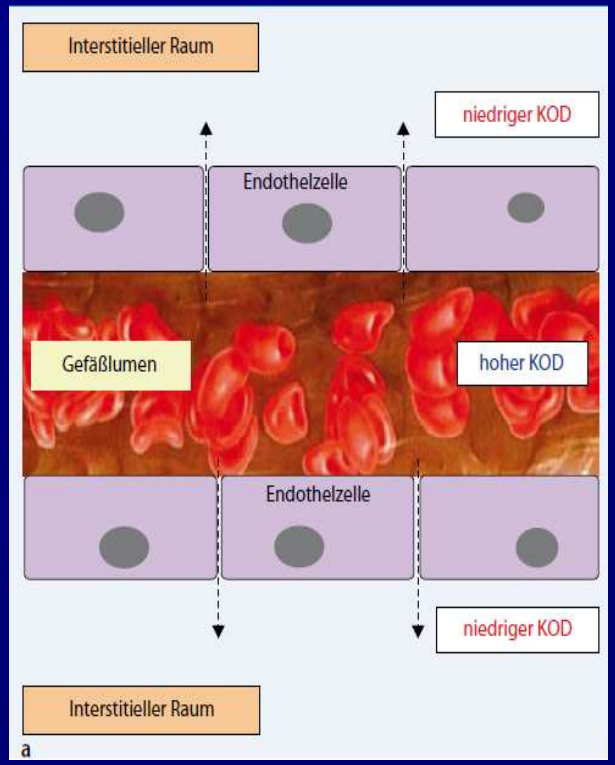
- compliant arteries
  - elastic
  - resistive (arterioles)
- capillaries
- veins
  - resistive (venules)
  - collapsing
- flow autoregulation

micro-circul.

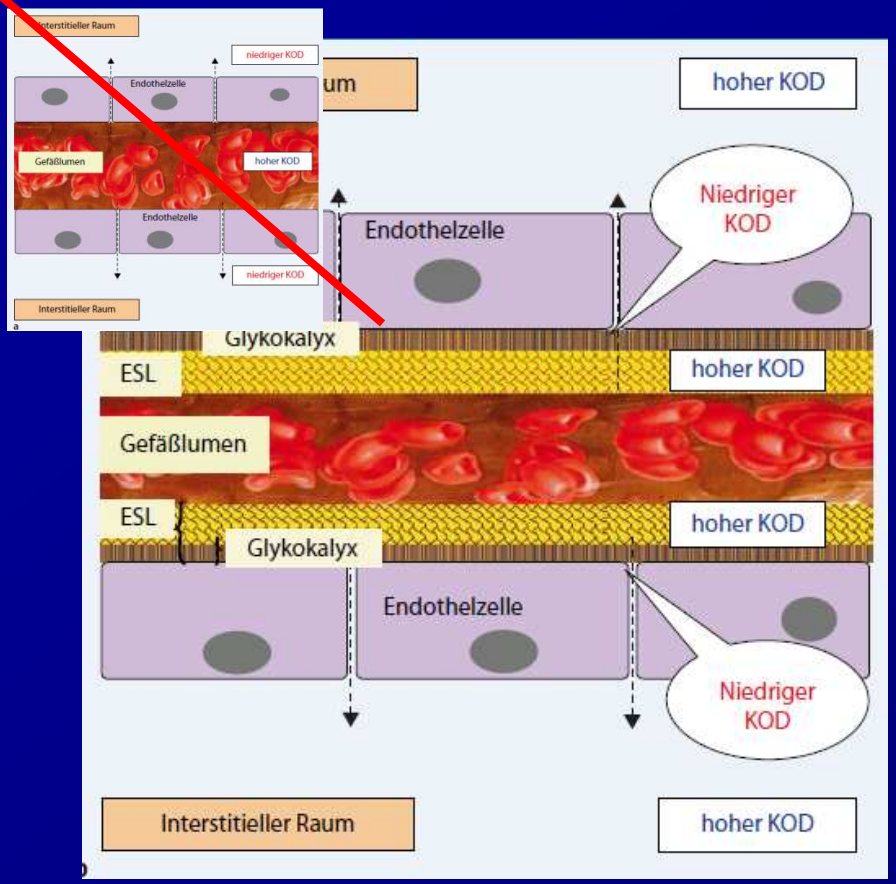


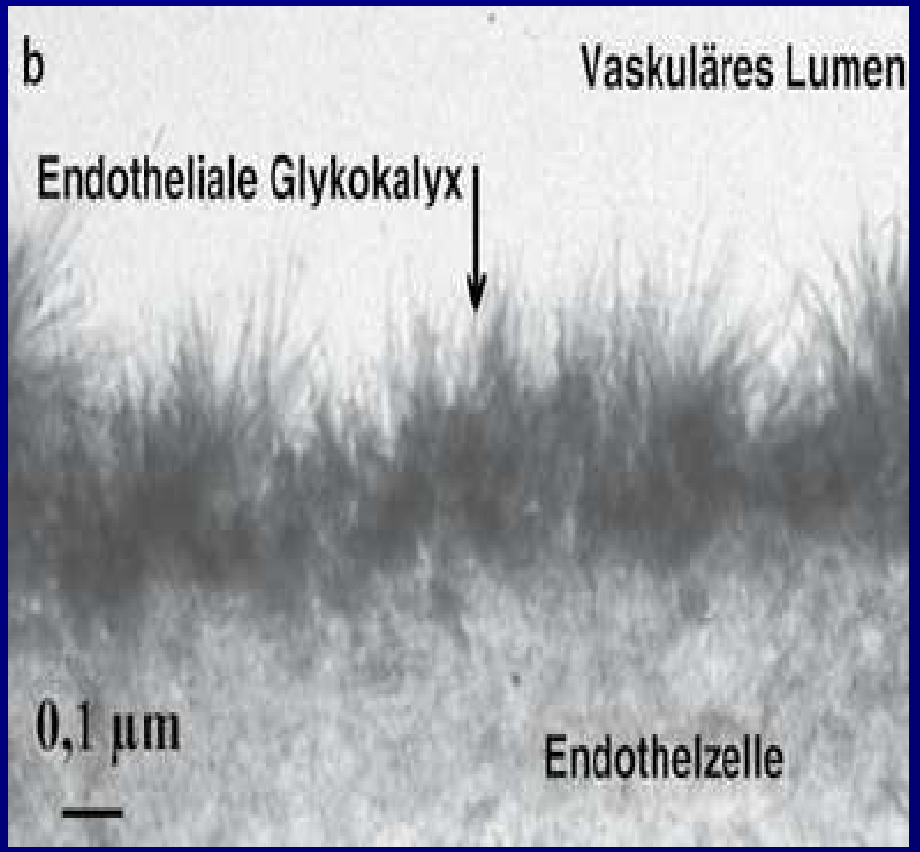
Vessels have endothelium!

- 10^{13} cells, 1 kg, 4000-7000 m^2
- function
  - vasomotor tone
  - fluido-coagulation balance
  - transport of nutrients and cells
  - local balance of mediators
  - formation of new vessels



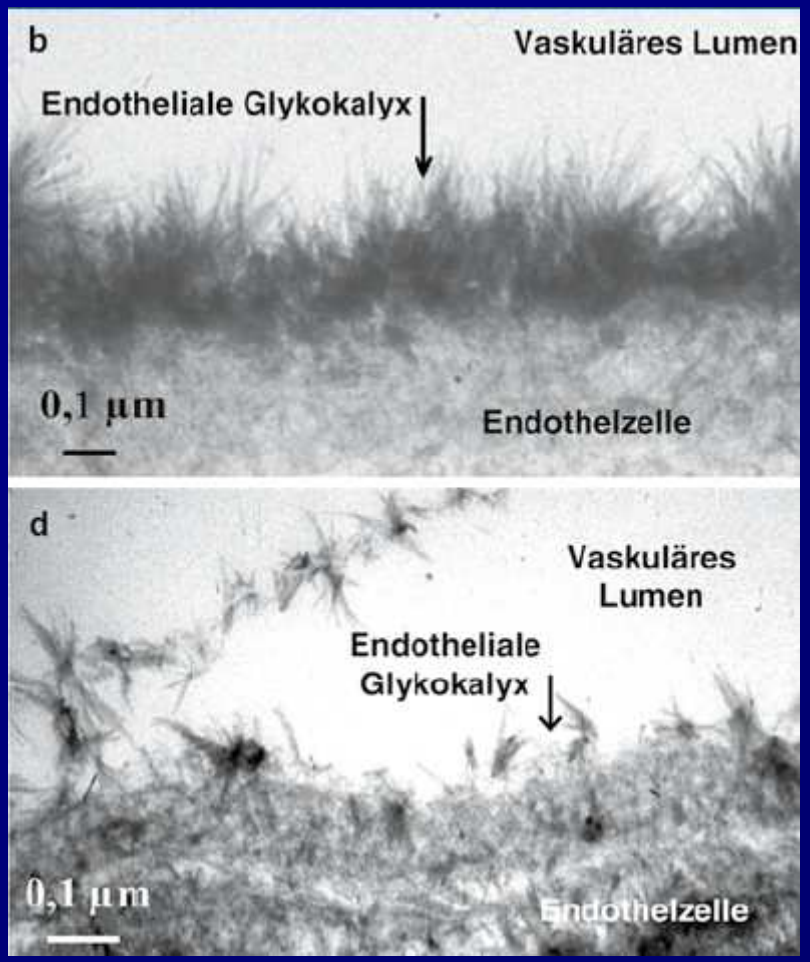
Vessels have endothelium!
Endothelium has glykokalyx!





Glykokalyx functions

- filtration function
- fragile glykokalyx destroyed by ANP, TNF-α
  - surgery, trauma
  - sepsis
- effects of its destruction
  - leucocytes adhesion
  - thrombocytes aggregation
  - escape of substances into interstitial space

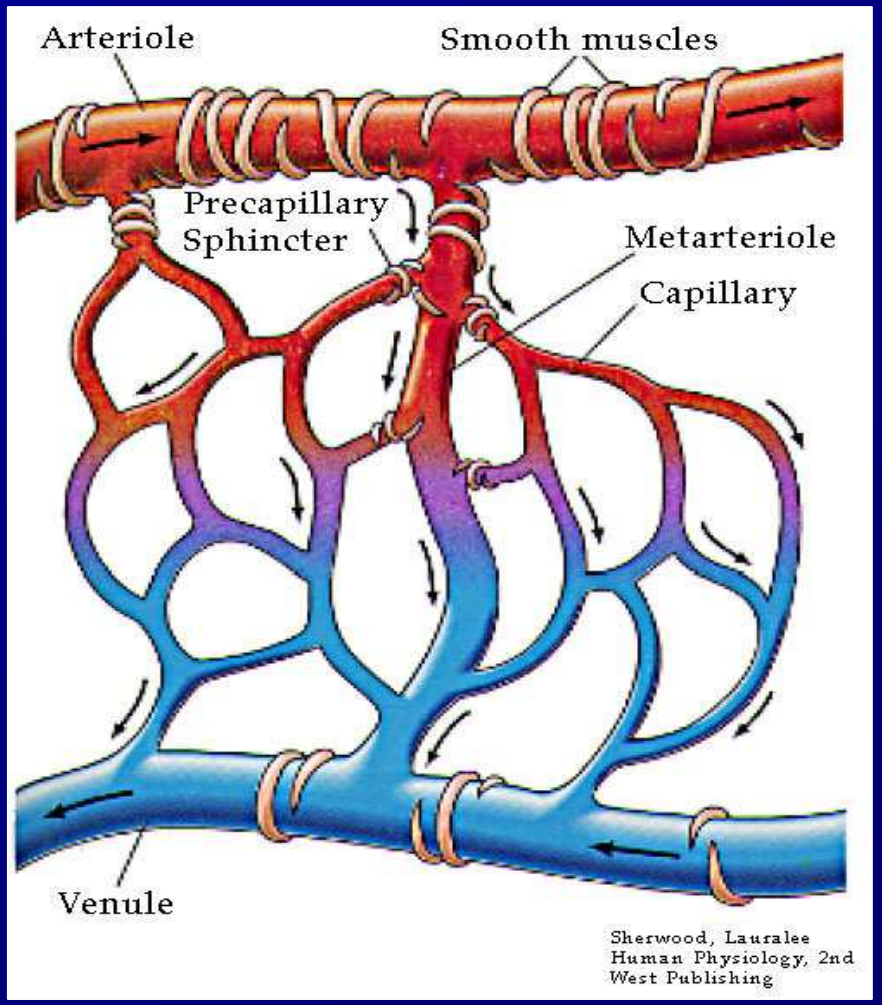


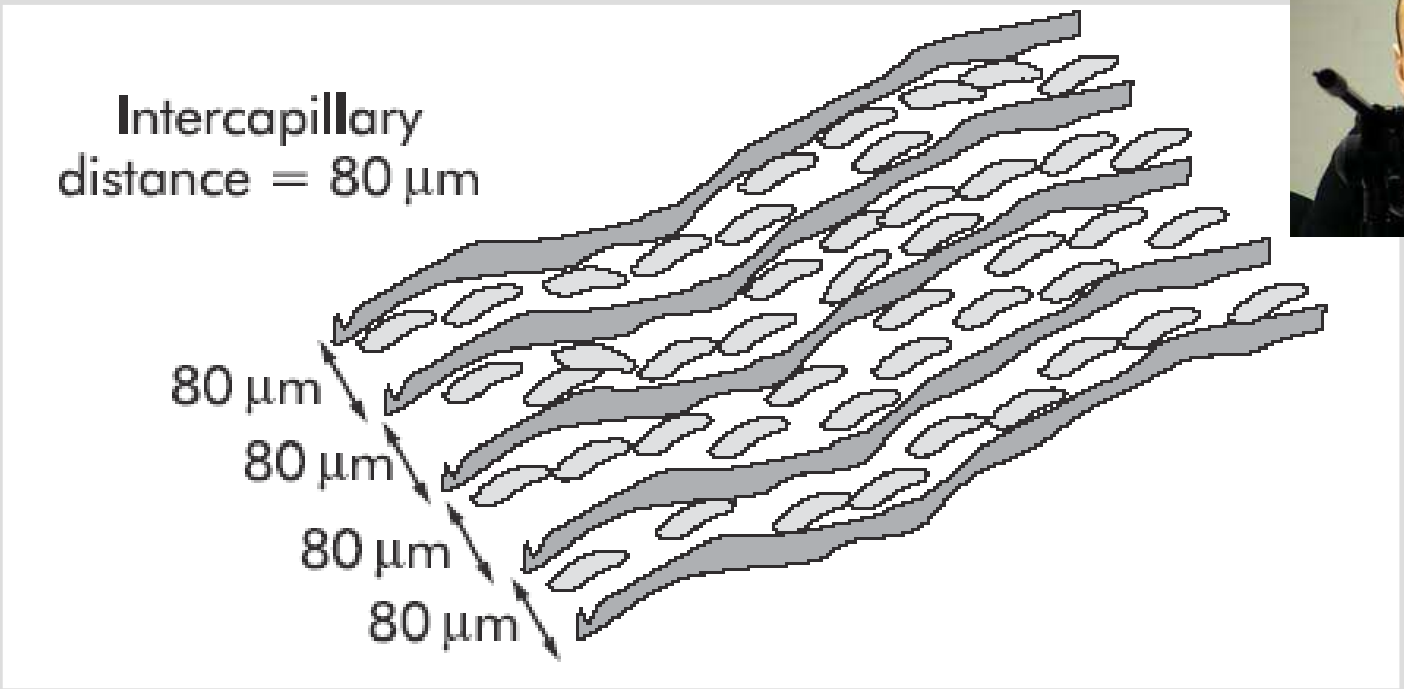
Microcirculation – basic facts

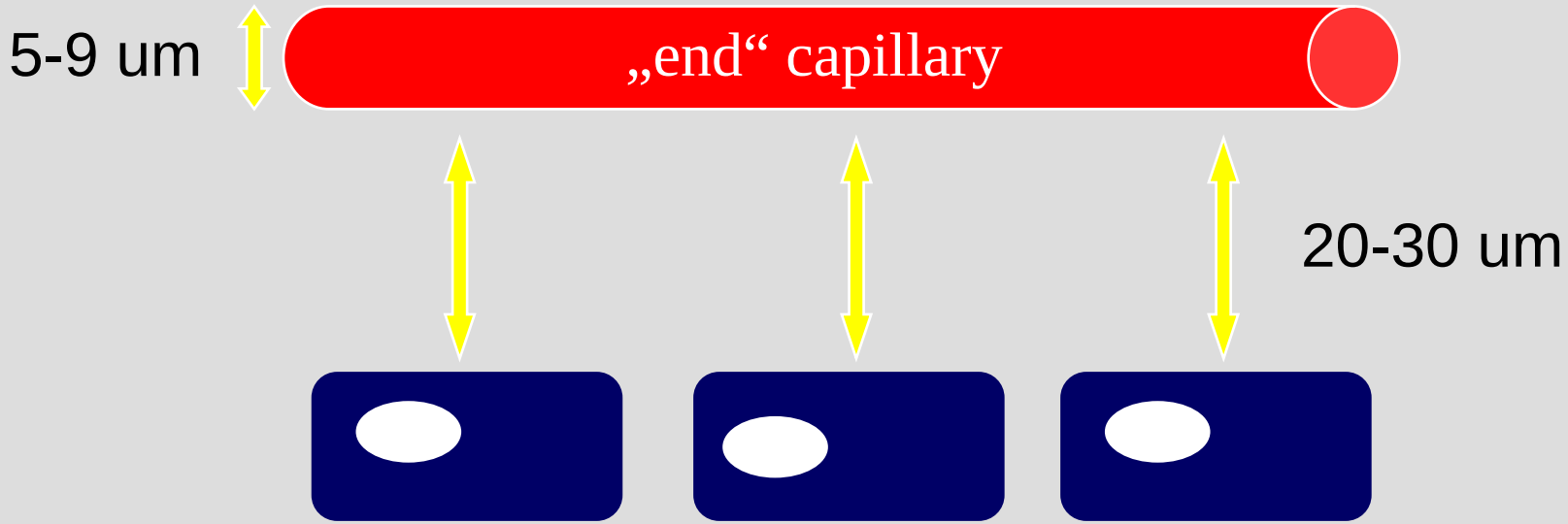
structure

- arterioles
- capillaries ≤ 300 um
  - 2000 capillaries/mm^2
  - small (< 25 um)
  - middle (25-50)
  - large (> 50)
- venules
- A-V anastomoses

Lymphatic system




Intercapillary
distance = 80 µm
80 µm
80 µm
80 µm
80 µm


5-9 um
„end“ capillary
20-30 um

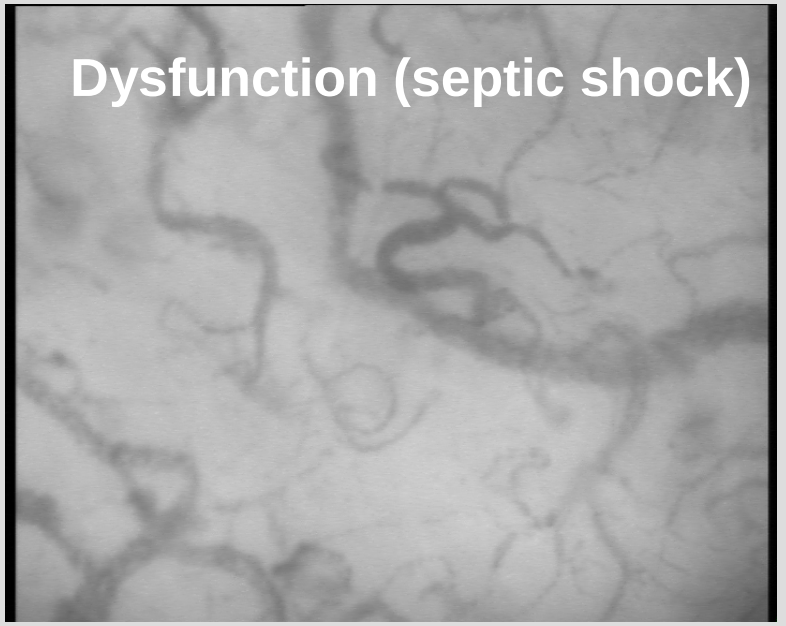
Microcirculation – basic facts

- structure
  - different in different organs and tissues
  - intraorgan heterogenity

- flow regulation
  - systemic-regional-local-myogennic level
    - not a continual flow, but **„on-off“**
    - „vasomotion“ phenomenon (~ ptO_2)
    - actual metabolic needs of tissues
    - vascular reactivity and its changes (homeostasis changes, drugs)

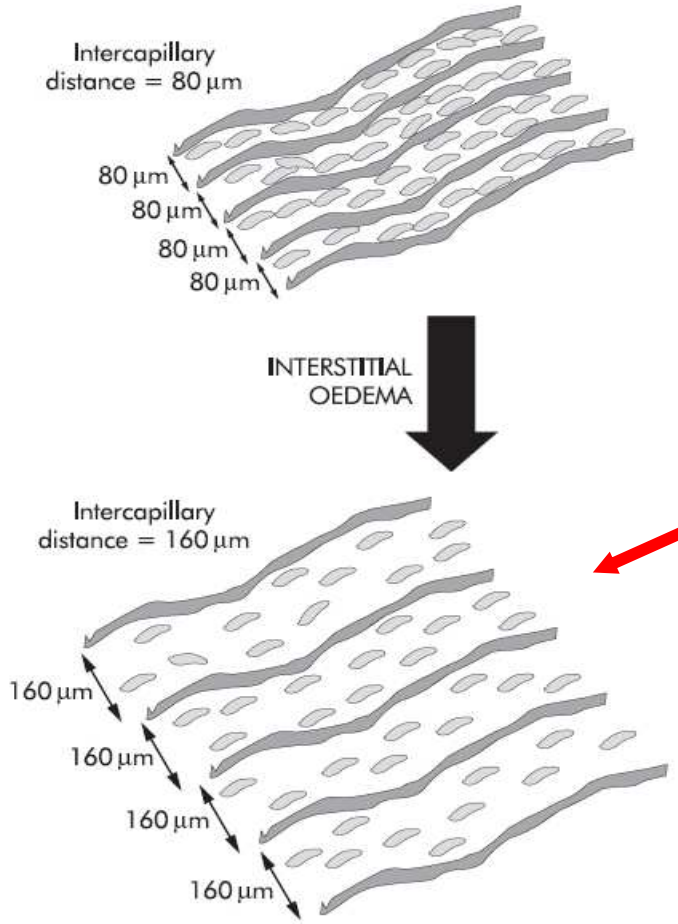
Microcirculatory dysfunction

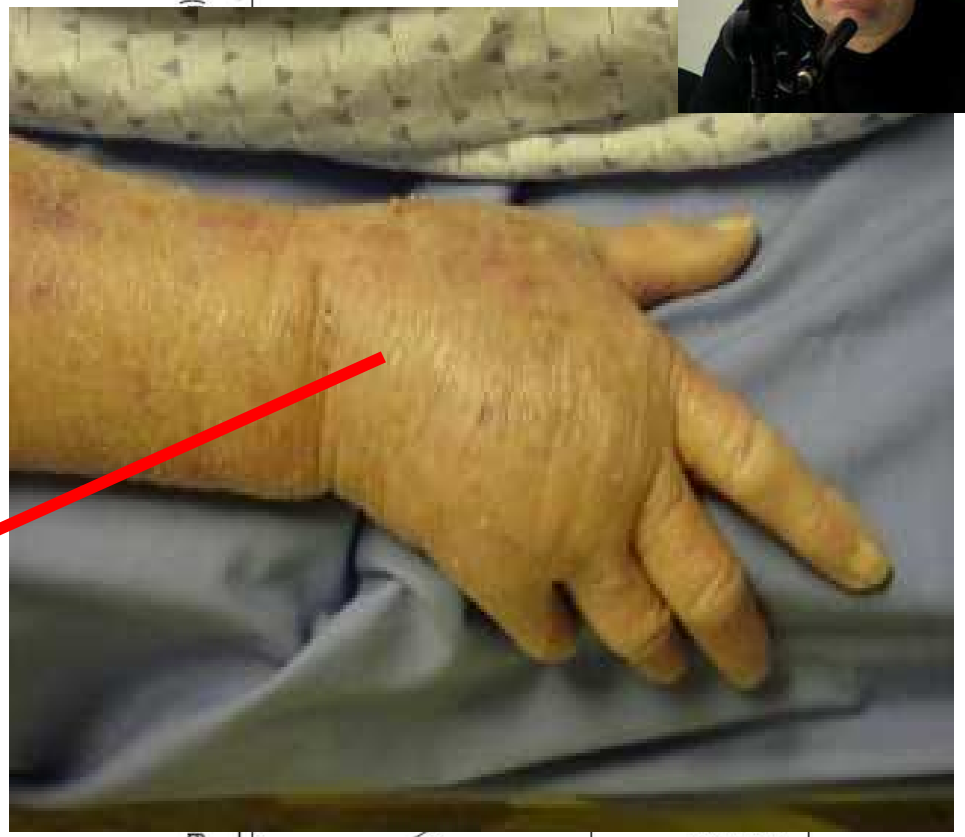
1. Decresased capillary density
2. Increased number of „non-perfused“ or „intermitenttly perfused“ capillaries
3. Restoration of normal state after topical application of acetylcholine



Own data – sublingual mucous membrane, SDF method

Oxygen delivery and consumption in the critically ill





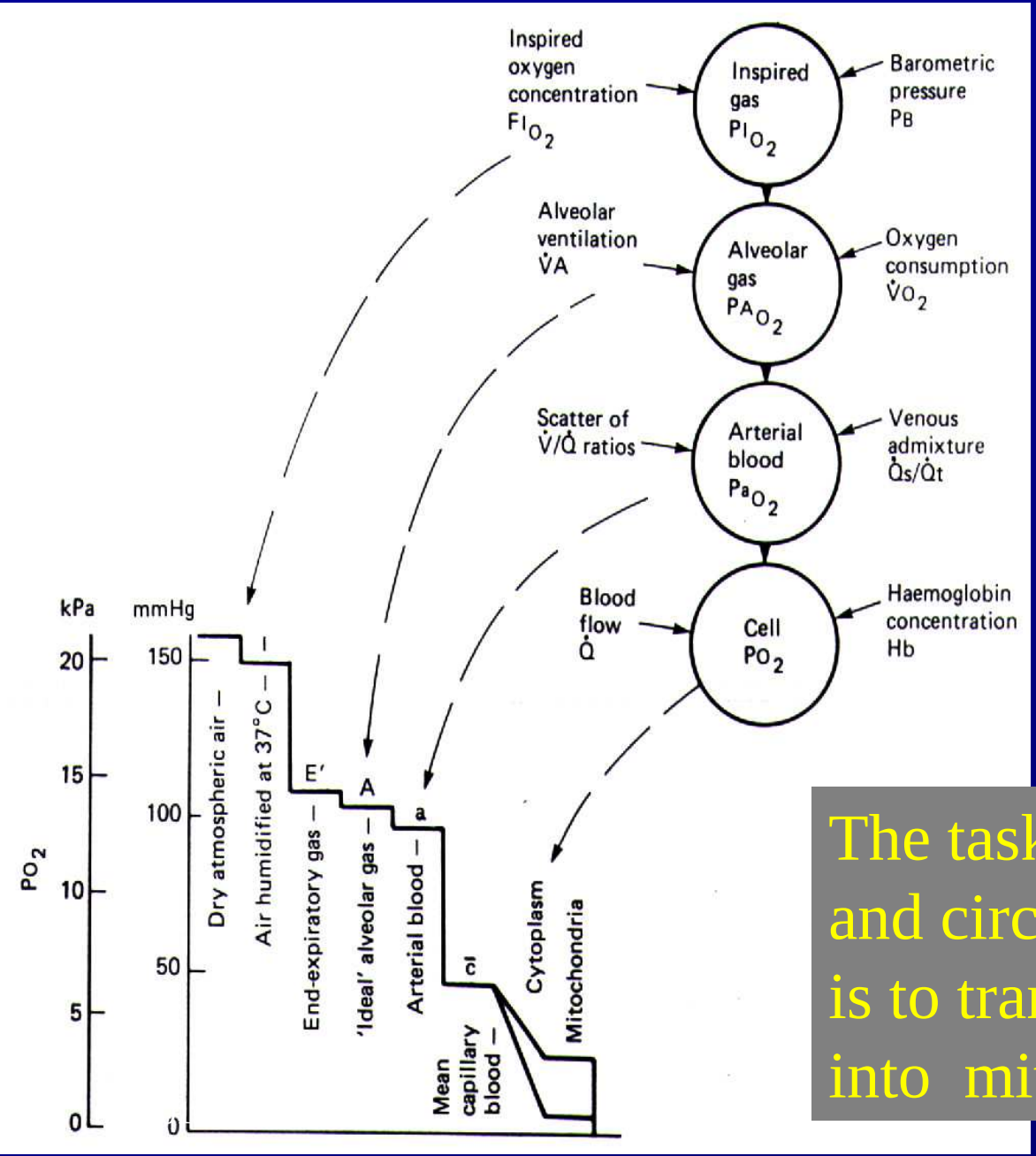
The pulmonary physician in critical care • 2: Oxygen delivery and consumption in the critically ill

R M Leach and D F Treacher

Thorax 2002;57;170-177
doi:10.1136/thorax.57.2.170

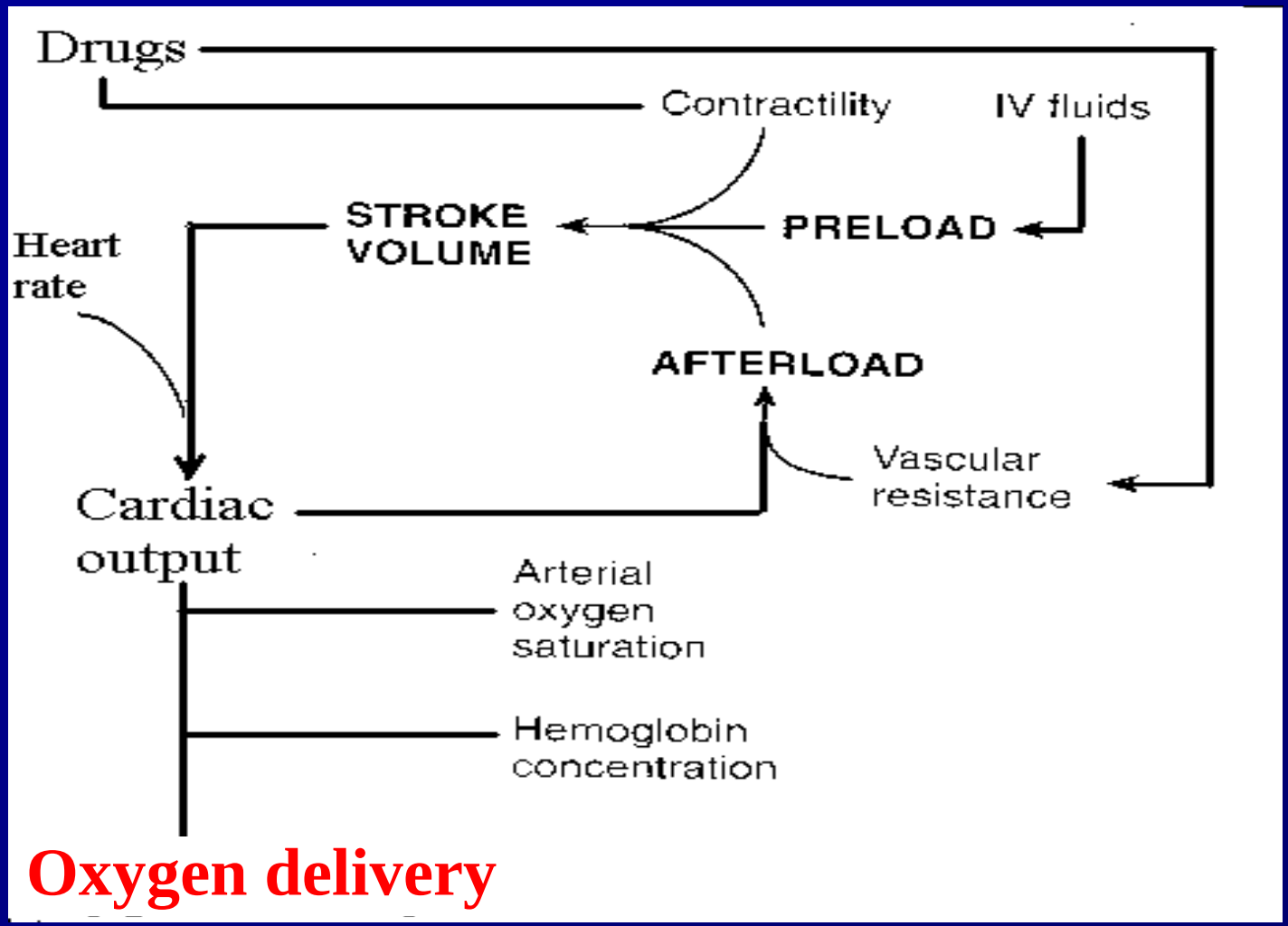
Oxygen delivery, cardiac output and its components

Oxygen cascade



The task of respiratory and circulatory systems is to transport enough of O_2 into mitochondrias.

Oxygen delivery



Oxygen delivery

$C_aO_2 = Hb \times 1{,}34 \times S_aO_2 + p_aO_2 \times 0{,}225$

$DO_2 = CO \times C_aO_2$

520 - 720 ml/min/m^2

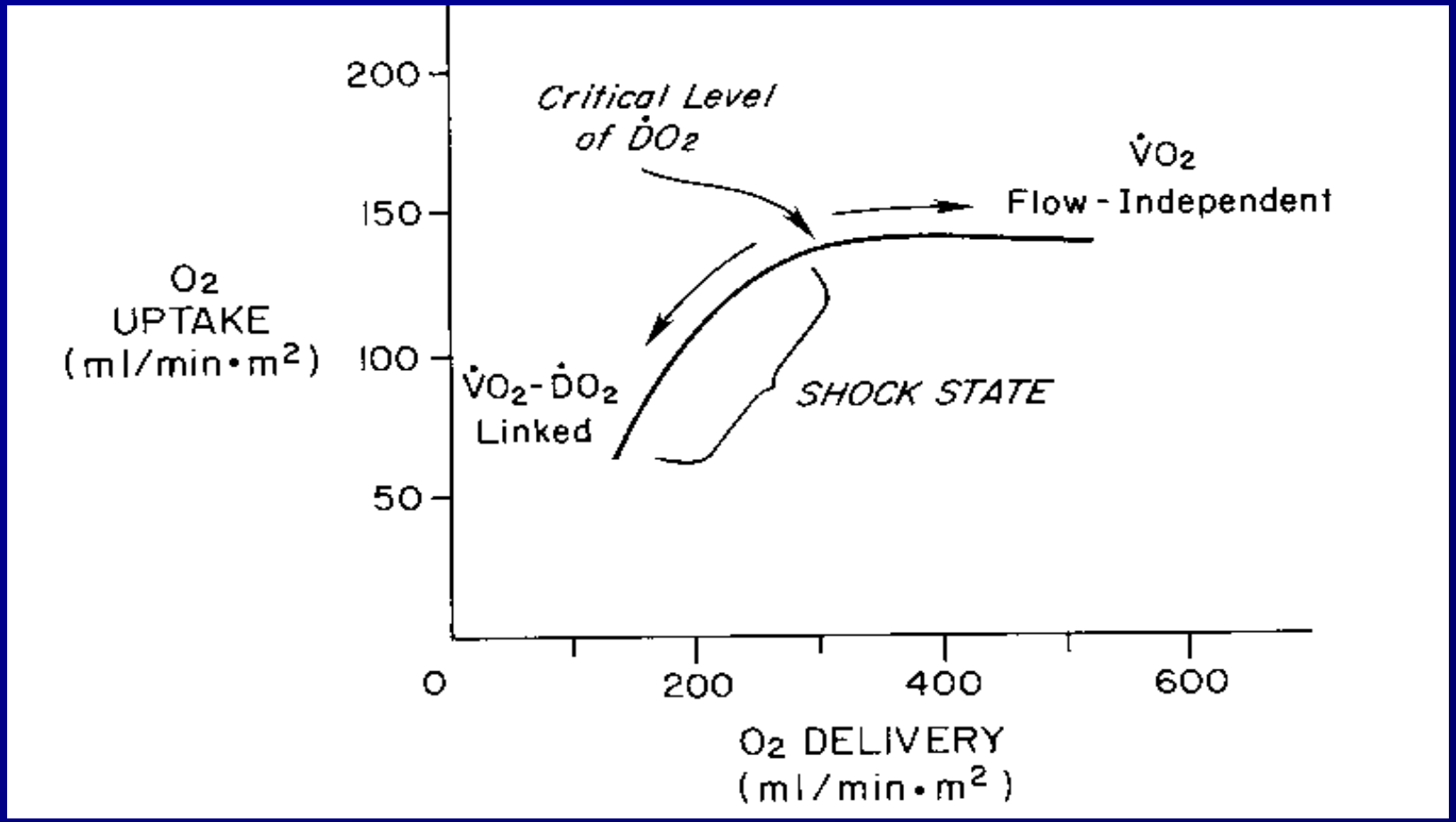
$VO_2 = CO \times (C_aO_2 - C_vO_2)$

110 - 160 ml/min/m^2

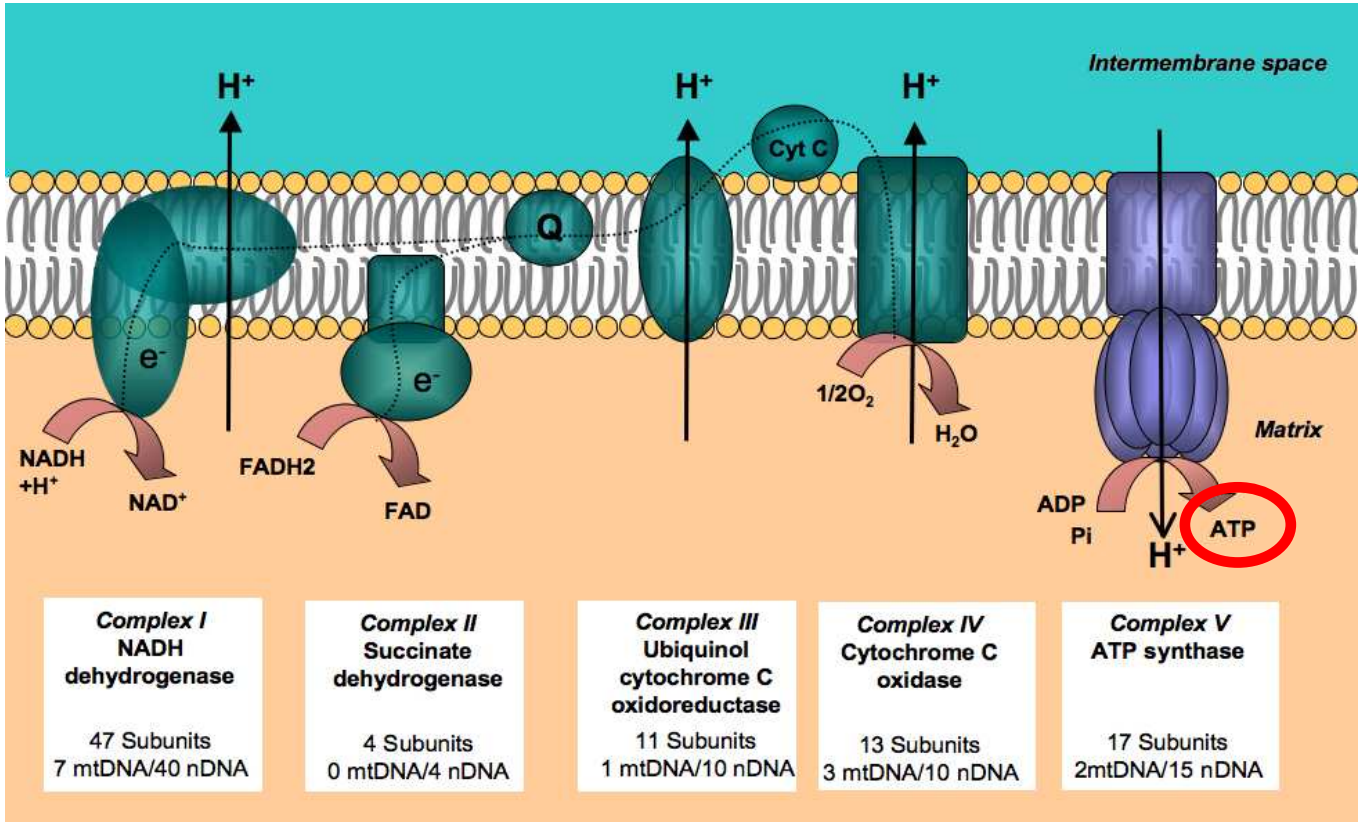
$O_2ER = DO_2 / VO_2$

0,22 - 0,32

DO_2 - VO_2 ratio



Electron respiratory chain in mitochondria



Frontiers in Bioscience 14, 4015-4034, January 1, 2009

Oxygen delivery

Inadequate oxidative fosforylation in mitochondria
is the main factor
of MODS, MOF a death
in critically ill.

Cardiac output

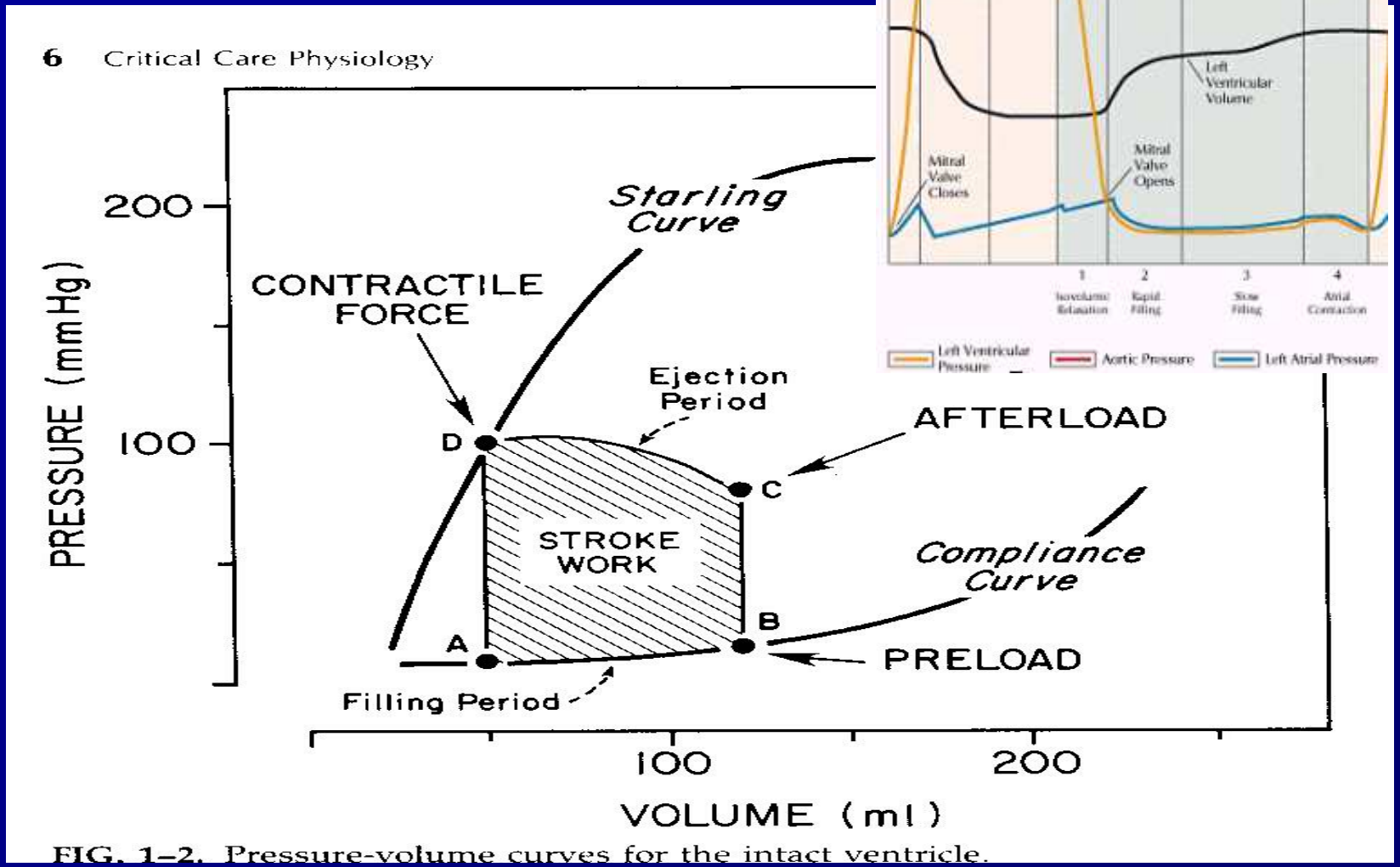


FIG. 1–2. Pressure-volume curves for the intact ventricle.

Cardiac output

- preload
- contractility
- afterload
- rate
- rhytm & synergy of contraction
- compliance

Parameters of the heart as a pump

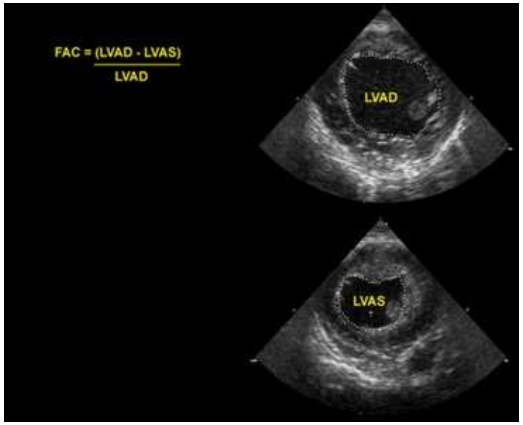
- chronotropy
- inotropy
- dromotropy
- bathmotropy
- lusitropy
- plecotropy

- heart frequency
- contractility
- action potential conductivity
- irritability
- relaxation
- rotation

ejection = longitudinal shortening + compression + rotation

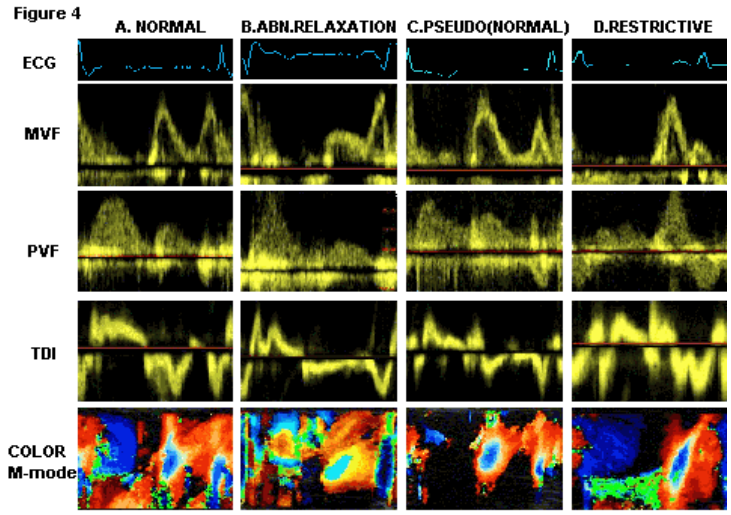
Cardiac output

- preload = force strretching fibers before contraction = ED fiber lenght = EDV = EDP
  - blood volume, venous tone, ventricle compliance, contractility, afterload
- contractility = ability of the myocardium to contract and a to eject from the left/right ventricle = work, that the heart can do on the given level of the load
- afterload = force acting against fiber shortening during ejection = wall tension
  - systolic pressure, wall thickness a ventricle radius (T = Pr/2h)
  - arterial impendance
    - compliance: against velocity of changes of blod flow (pulsatile comp.) compontent)
    - resistance: against mean velocity of blood flow (non-pulsatile c.)



Heart function

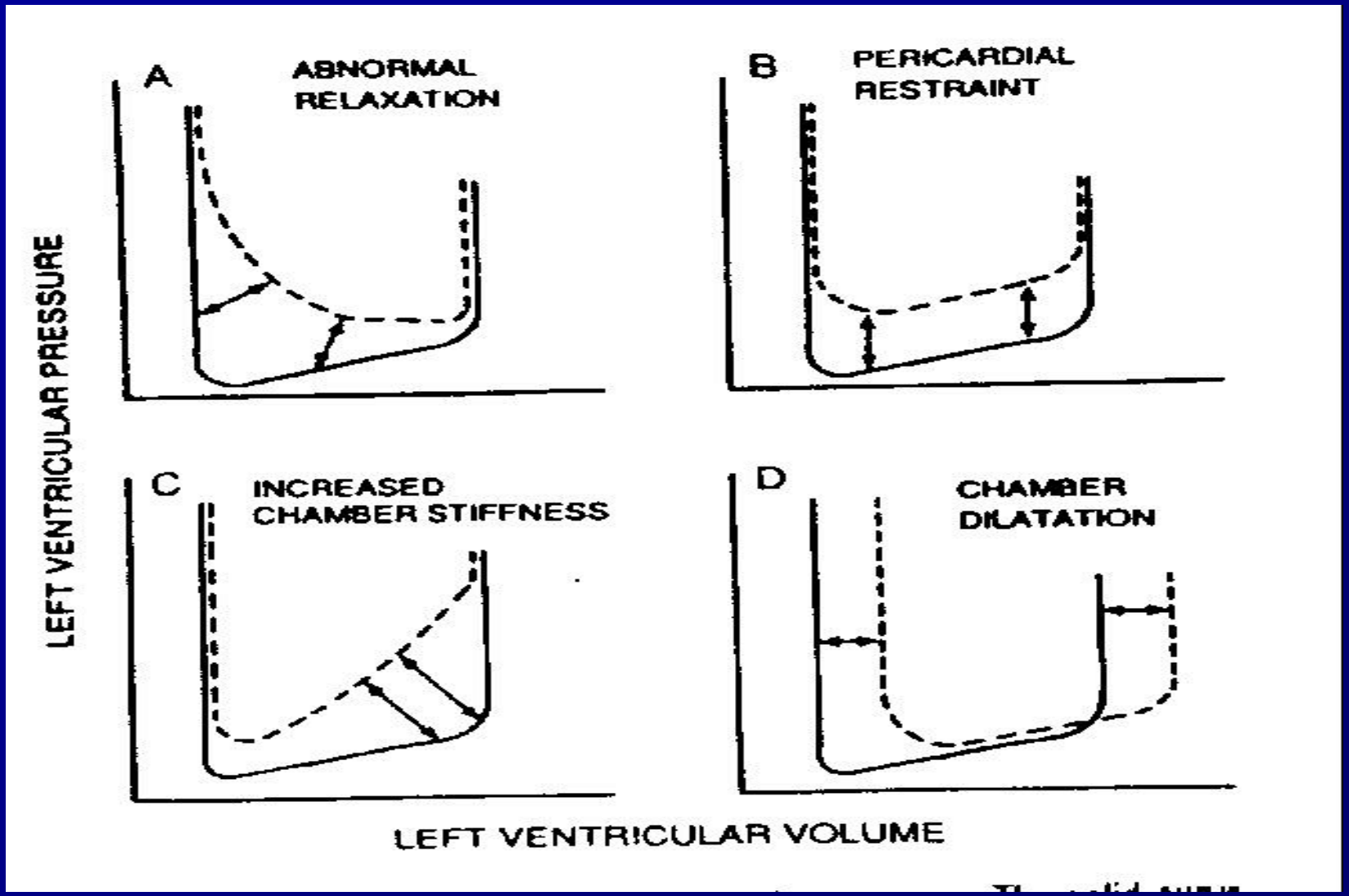
- systolic dysfunction
  - ejection fraction EF = (EDV – ESV)/EDV
  - ventricle stroke work (MAP – PCWP) * SV * 0,0136
- diastolic dysfunction
  - compliance
    = ΔEDV/ ΔEDP
  - distensibility
  - relaxation



Diastolic dysfunction

- compliance ΔEDV/ ΔEDP
  - increased stiffness of the ventricle – AS, hypertension
  - increased stiffness of myocardium – restrictive cardiomyopathy, hemochromatosis
- distensibility = ↑ EDP with given EDV
  - internal factors: ischaemia
  - external factors: limited expansion of the ventricle in diastoly – tamponade
- relaxation

Diastolic dysfunction



Cardiac output and blood pressure

$$CO = \frac{(MBP - RAP)}{SVR}$$

Which is more important, pressure or flow?

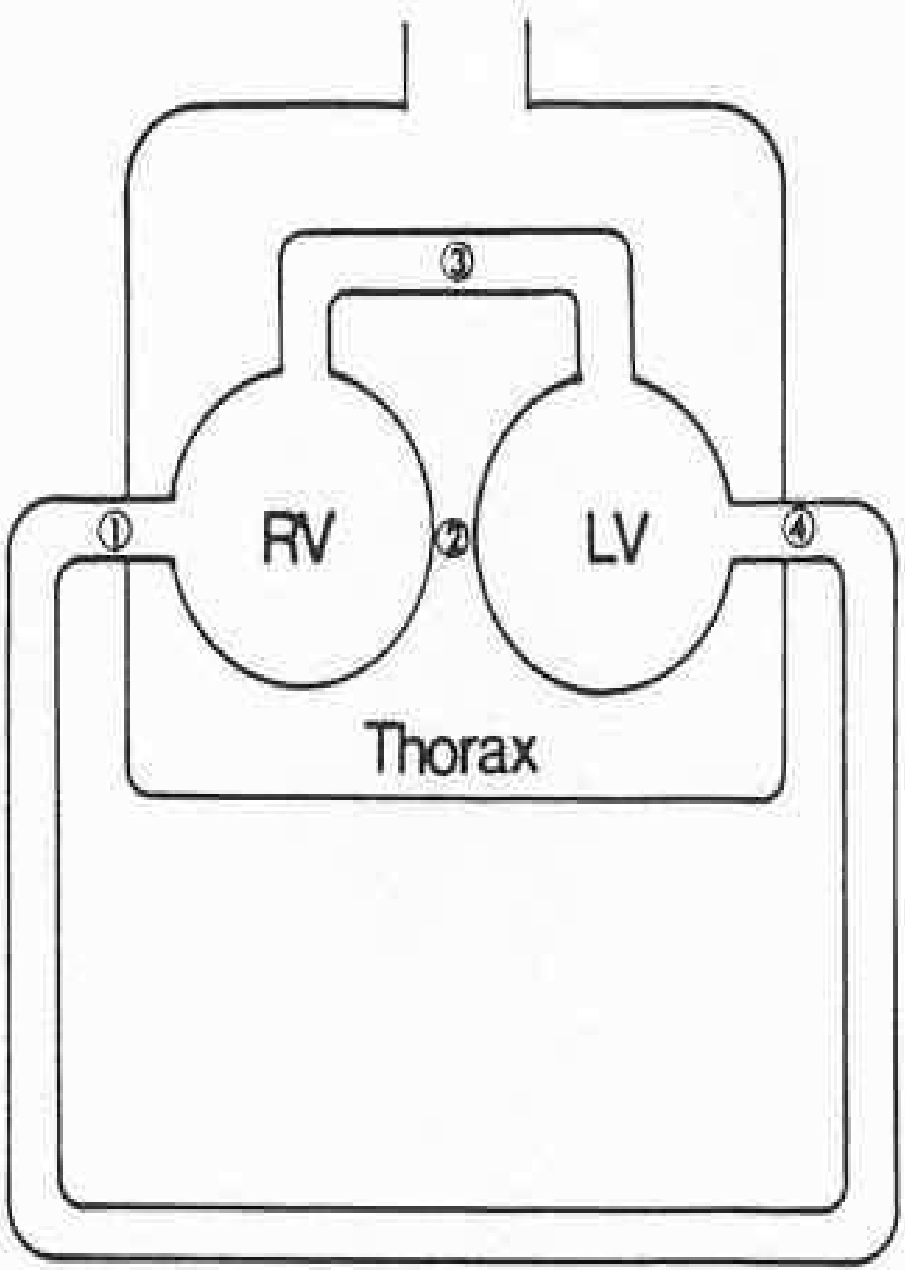
Flow transport oxygen!

$DO_2 = CO \cdot Hb \cdot Sa \cdot 1{,}34$

Pressure enables flow!

$$Q = \frac{\pi r^4}{8L\mu} \times \Delta P$$

- diameter of vessel´s lumen
- viscosity



Main aspects of cardio-pulmonary interactions:

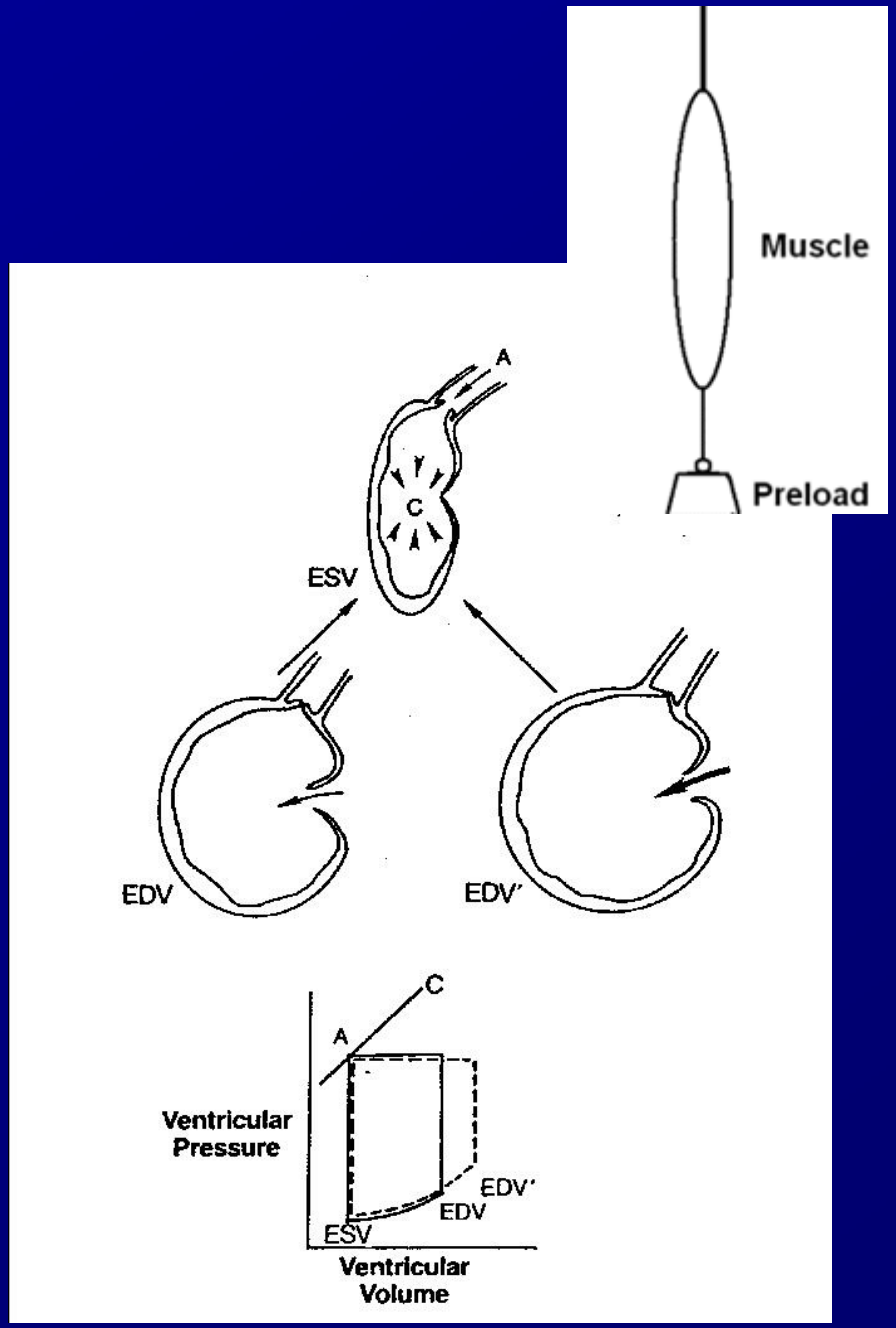
1. ~Ppl → ~RAP → ~ gradient for venous return
2. Interdependence of ventricles due to the shift of septum
3. ~lung volume affects PBF, PVR and PAPsyst.
4. ~Ppl → ~transmural Ao pres. → ~LV afterload

1. MV + PEEP limit VR
2. NEEP increases VR inspirium through a resistor in trauma

Preload

Preload

- force stretching fibers at the end of diastoly
- EDV, EDP, CVP or LAP či PCWP



Hypovolemia

Hypovolemia is common in surgical, trauma and ICU patients.

Boldt, J: New Light on Intravascular Volume Replacement Regimens:
What Did We Learn from the Past Three Years? Anest Analg 2003;97:1595–604

Hypovolemie can lead to:

- organ dysfunction
- increased morbidity
- lenghtening of stay
- death.

Gan TJ. et al. Goal-directed intraoperative fluid administration reduces length of hospital stay after major surgery. Anesthesiology 2002 Oct;97(4):820-6

Blood loss in closed fractures

• forearm	• 50-400 ml
• arm	• 100-800 ml
• shank	• 100-1000 ml
• femur	• 300-2000 ml
• pelvis	• 500-5000 ml

according to Burri & Henkemeyer
In: Drábková et al.: Basics of resuscitation, Avicenum, Praha 1982

Hypotension a hypoperfusion

The most important cause is hypovolemia!

Treatment = to replenish volume!

- crystalloids, kolloids, hypertonic solutions
- transfusion

  Anemia is better tolerated than hypoxia!
- volume challenge

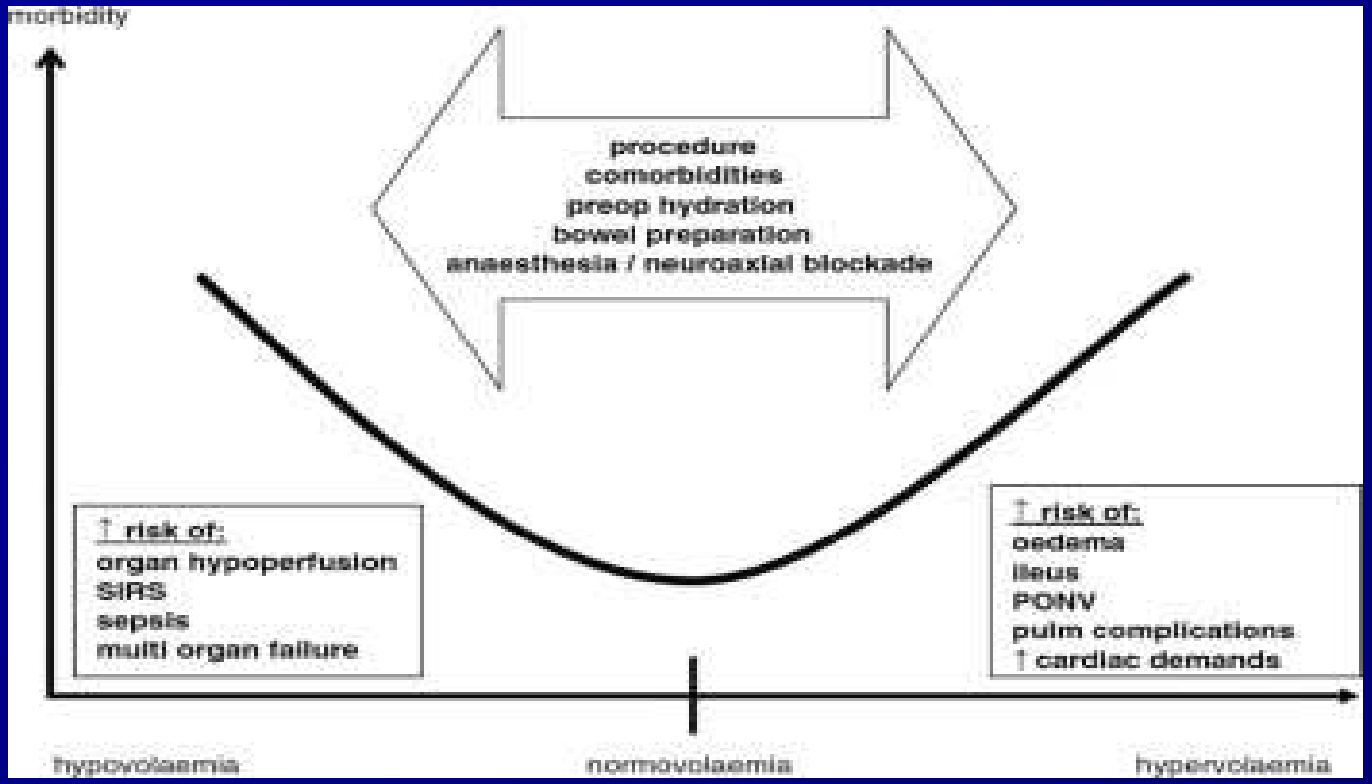
Hypervolemia

- srdce: ↓ LVSV, ischaemia, failure
- plíce: interstitial lung eodema, atelectases, pneumonia, respiratory failure
- ledviny: ↑ Load on renal function, ↑ risk of urine retention
- GIT: gut oedema, nutrition intolerance, longstanding ileus, endotoxin and bacteria translocation
- koagulace: hyper- (crystalloids), hypo- (colloids)
- hojení rány: ↓ diffusion of O_2, ↓ wound healing

Holte K, Sharrock NE, Kehlet H: Pathophysiology and cklinical implatiations of perioperative fluid excess. Brit J Anaesth 2002:89:622-32

Some fluid, any fluid ... please!!

Grocott MPV, Hamilton, MA: Resuscitation fluids. Vox sanquinis 2002:82:1-8

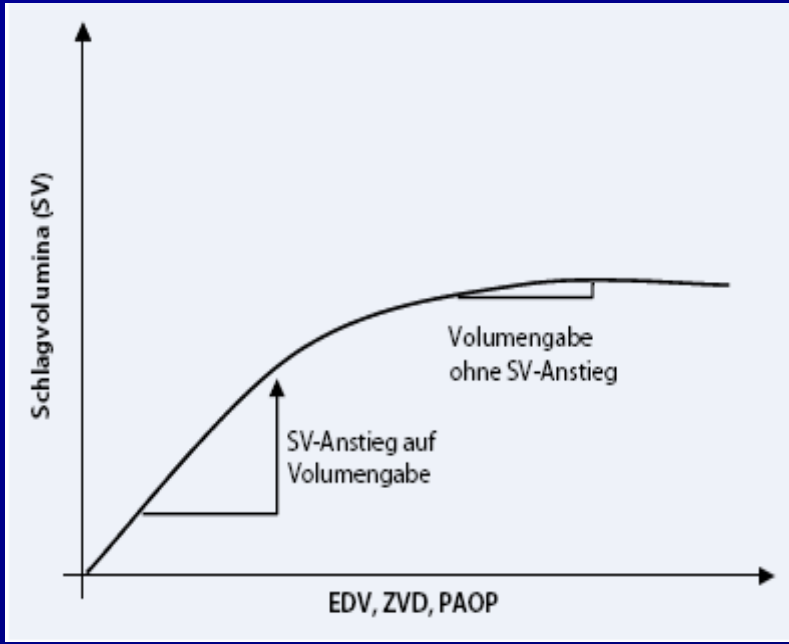


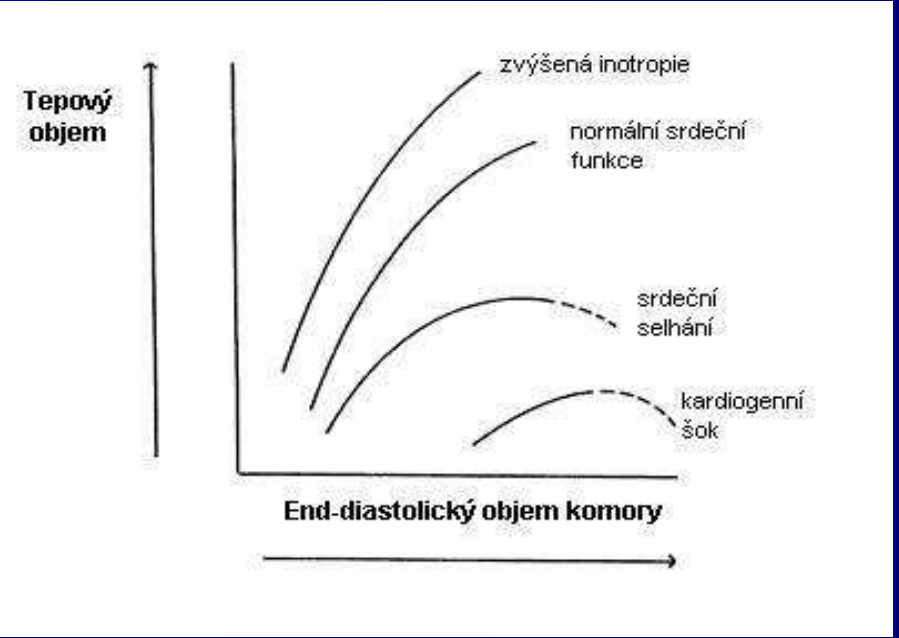
Don’t be generous with fluid!

Oh MS, Kim HJ: Basic rules of parenteral fluid therapy. Nephron 2002:92 (suppl 1):56-59

Is hypotension caused by hypovolemia?

volume challenge: dif. dg. of hypovolemia

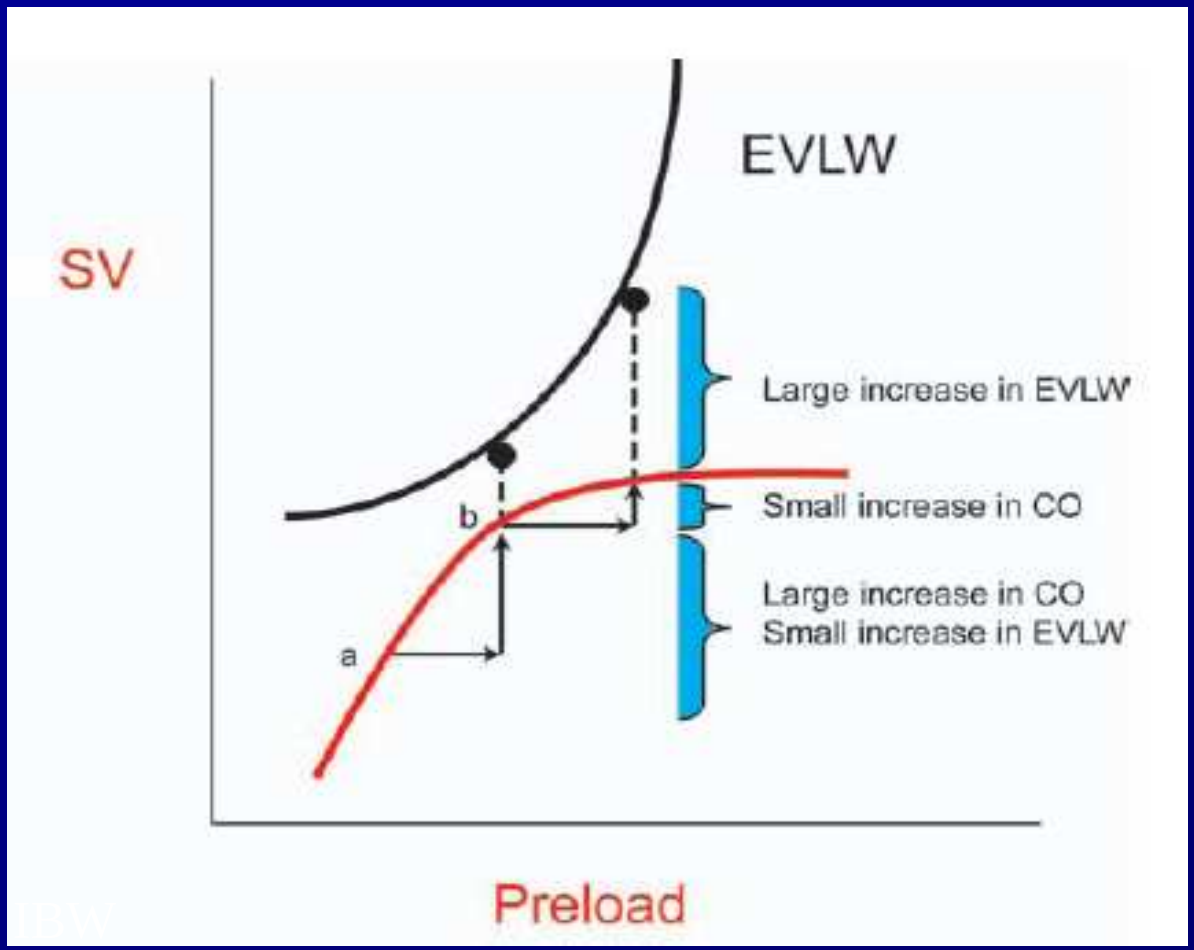




Frankova-Starlingova křivka x Marikova-Phillipsova křivka

Paul Ellis Marik

norma EVLW 5-7 ml/kg IBW



How to replenish volume loss?

- crystalloids
- colloids
- hypertonic solutions
- blood

Optimal strategy remains unknown,
is probably different in defferent patients.

Crystalloids or colloids?

crystalloids – solutions of ions and small organic molecules in water

- solutions of ionts
- solutions of glucose

colloids – homogenic dispersions of large molecules in crystalloid solution (F 1/1, hypertonic, balanced solution, glucose)

- natural x synthetic
- monodispersive x polydispersive

Colloids

- natural
  - albumin
- synthetic
  - dextrans
  - gelatine
    - modified
    - urea-linked (Haemaccel)
    - succinyl-linked (Gelofusin)
  - hydroxyethylstarch (HES)
    - potato
    - waxy maze

Characteristics of colloids

- size and duration of volume effect
- haemorrheology
- hemostatic effects
- interactions with endothelium and inflammatory cells
- adverse effects
- price

Size and duration of volume effect

Solution	Alb. 5 %	Alb. 20%	**HES 130/ 0,4**	HES 200/ 0,5	HES 200/ 0,5	HES 450/ 0,7	Dex. 60	Dex. 40	Gel
Conc. (%)	5	20	**6%**	6	10	6	6	10	3,5-5,5
Volume effect (%)	80 %	130 - 150 %	**100 %**	100%	130 - 150%	100%	100%	150 - 200%	80%
Volume effect (h)	2-3	2-3	**3-4**	3-4	3-4	5-6	5	3-4	1-2
M. weight (kD)	66	66	**130**	200	200	450	60	40	30-35

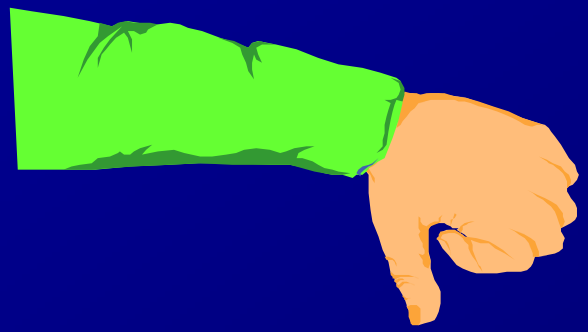
Boldt, Priebe: Intravascular Volume Replacement Therapy with Synthetic Colloids: Is There an Influence on Renal Function? Anesth Analg 2003;96:376-382

Niemi TT et al.: J Anesth 2010: 24:913–925

Haemorrheology

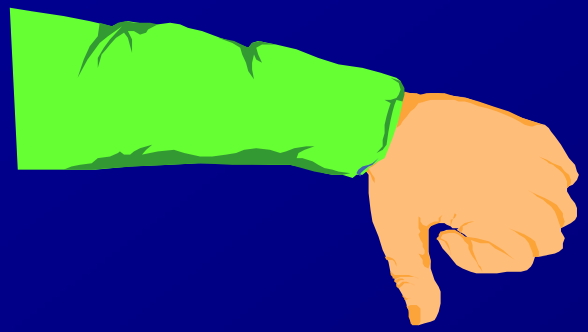
- all colloids change blood rrheology
  (physics of flow a substance deformation)
- ↓ viscosity, mainly colloids with small molecule
- low-molecular dextrans ↓ erythrocyte aggregation

Hemostatic effects



- all colloids disturb haemostasis
- Coagulation factos dilution
- gelatine
  - ↓ activity of thrombo, ↓ vWf, impairs polymeration of fibrin monomers
- dextrany, mainly high-molecular
  - ↓ thrombo function, ↓ f VIII, ↑ fibrinolysis
- HES, mainly high-molecular
  - ↓ thrombo function, ↓ vWf

Colloids and kidneys



- dextrans
  - hyperoncotic renal failure
  - tubular obstruction
  - direct toxic effect
- **gelatine** – no effect
- HES – cautiously in patients in risk of or with ARF

Boldt, Priebe: Intravascular Volume Replacement Therapy with Synthetic Colloids: Is There an Influence on Renal Function? Anesth Analg 2003;96:376-382

Anaphylaxis risk

Colloid	Number of administrations	Anaphylaxis	Risk
Gel	9 424	32	0,345
Dextran	1 861	5	0,273
HES	5 231	3	0,058
Albumin	3 073	3	0,099
Total	19 593	43	0,219

Laxenaire MC, Charpentier C, Feldman L. Anaphylactoid reactions to colloid plasma substitutes: incidence, risk factors, mechanisms. A French multicenter prospective study. Annales Francais d'Anesthesie et Reanimation 1994;13:301–10

Crystalloids or colloids?

Christiane Hartog
Konrad Reinhart

CONTRA: Hydroxyethyl starch solutions are unsafe in critically ill patients

Intensive Care Med (2009) 35:1337–1342

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The article arguing for this proposition is available at:
doi:10.1007/s00134-009-1520-6.

C. Hartog · K. Reinhart (✉)
Department of Anaesthesiology and Intensive Care Medicine,
Friedrich Schiller University,
Erlanger Allee 101, 07747 Jena, Germany
e-mail: konrad.reinhart@med.uni-jena.de
Tel.: +49-3641-9323101
Fax: +49-3641-9323102

Abstract *Purpose:* To describe the risk–benefit profile of hydroxyethyl starch (HES).
Methods: Narrative review.
Results: (1) Efficacy: no single clinical study or systemic review has shown that administration of any HES solution confers a clinically relevant benefit compared to crystalloids in critically ill patients or surgical patients in need of volume replacement. Contrary to beliefs expecting a ratio of 4:1 or more for crystalloid to colloid volume need, recent studies of goal-directed resuscitation observed much lower ratios of between 1 and 1.6. (2) Safety: HES administration is associated with coagulopathy, nephrotoxicity, pruritus and increased long-term mortality. Clinical studies claiming that modern HES 130/0.4 is safe have serious methodological drawbacks and do not adequately address the safety concerns. *Conclusions:* Given the complete lack of superiority in clinical utility studies and the wide spectrum of severe side effects, the use of HES in the ICU should be stopped. The belief that four times as much crystalloid as colloid fluid volume is needed for successful resuscitation is being seriously questioned.

Keywords Colloids · Crystalloids · Hydroxyethyl starch · Efficacy · Safety · Critically ill

EUROPEAN MEDICINES AGENCY
SCIENCE MEDICINES HEALTH

19 December 2013
EMA/809470/2013

Hydroxyethyl-starch solutions (HES) no longer to be used in patients with sepsis or burn injuries or in critically ill patients

HES will be available in restricted patient populations

On 23 October 2013, the Coordination Group for Mutual Recognition and Decentralised Procedures – Human (CMDh)*, endorsed by majority the recommendations of the European Medicines Agency's Pharmacovigilance Risk Assessment Committee (PRAC), which concluded that HES solutions must no longer be used to treat patients with sepsis (bacterial infection in the blood) or burn injuries or critically ill patients because of an increased risk of kidney injury and mortality.

HES solutions may continue to be used in patients to treat hypovolaemia (low blood volume) caused by acute (sudden) blood loss, where treatment with alternative infusions solutions known as 'crystalloids' alone are not considered to be sufficient. In order to minimise potential risks in these patients, HES solutions should not be used for more than 24 hours and patients' kidney function should be monitored

Capillary leakage

- CNS: disturbed consciousness
- srdce: decreased compliance, diastolic dysfunction
- lung: oedema, disturbed gas exchange
- gut: ↓ absorption, ↑ bacteria translocation
- tissues: disturbed O_2 diffusion, impaired wound healing

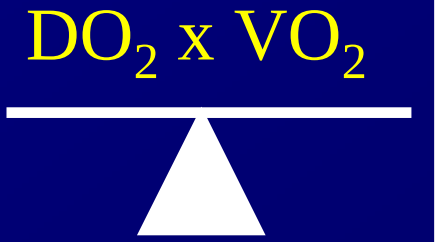
Traylor, R.J., Pearl, R. G.: Crystalloid Versus Colloid Versus Colloid: All Colloids Are Not Created Equal. Anest Analg 1996;83:209-12

Gudelines for perioperative infusion therapy

- no universally accepted guidelines
- to replenish perioperative deficit
  - effect of illness or treatment (e.g. diuretics)
  - preoperative fasting?
  - gut preparation (e.g. laxatives)
- to maintain normovolemia
- to maintain concentration of haemoglobin and coagulation factors

Conclusion

- low RAP makes venous return easier
  (VR = MSP – RAP)
- hypervolemia releases ANP, which destroys glycocalyx → escape into interstitium
- low ESV represents low afterload
  (T = P . r / 2h)
- oxygen delivery/consumption in balance



- Maintain homeostasis!
  (diseased heart can be choosy, not omnivorous)

Program

- basic physiology
- **heart rhythm disturbances**

In part 2

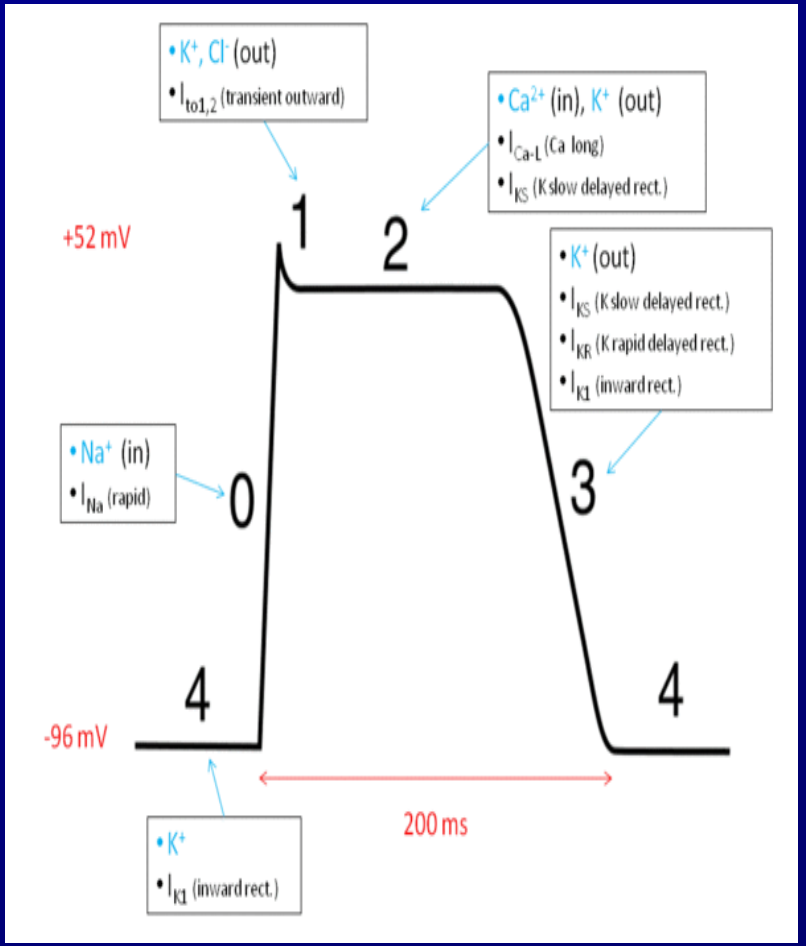
- circulation monitoring
- shock
- cannulations
- circulatory support

Myocyte electrophysiology

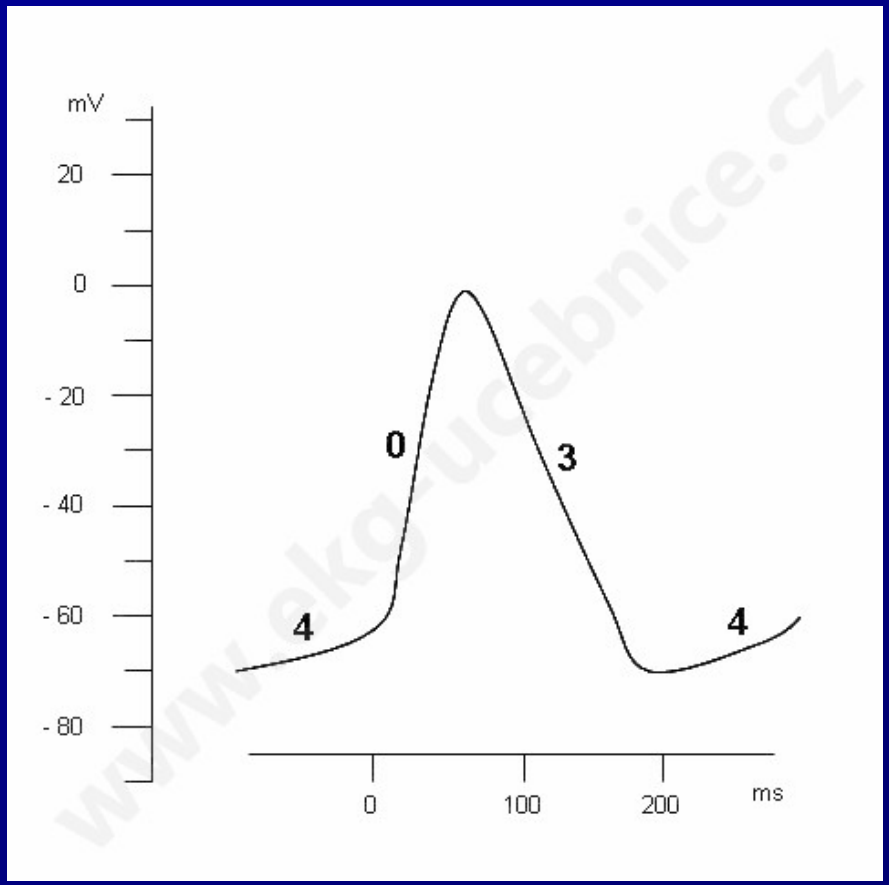
- resting transmembrane potential
  Nernst, resp.
  Goldman-Hodgkin-Katz equation

$$E = \frac{RT}{zF} \ln \frac{[\text{ion outside cell}]}{[\text{ion inside cell}]}$$

- action potential
  - 0 rapid depolarisation
  - 1 early rapid repolarisation
  - 2 plateau
  - 3 rapid repolarisation
  - 4 resting transmembrane. p.

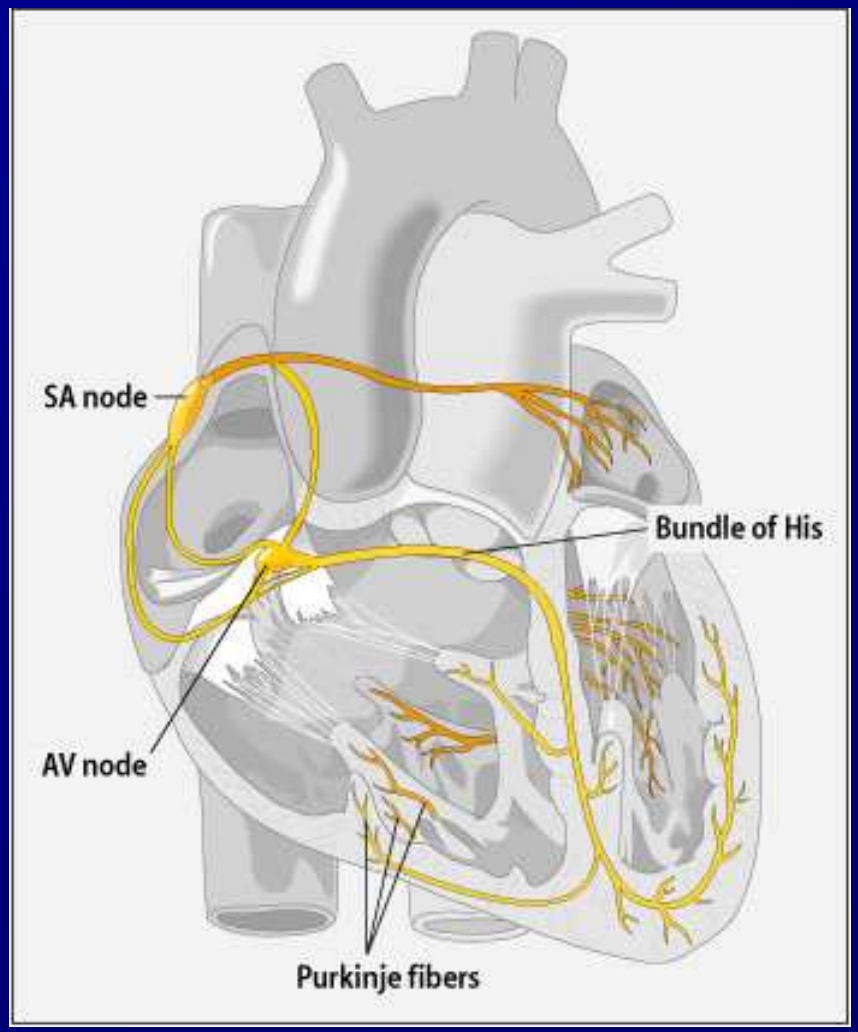


Spontaneous diastolic depolarisation



Conduction system anatomy

- sinus node
  - > 5 channels,
    e.g. I_f, Ca^{2+} channels L, T
- AV node
  - Ca^{2+} channels L, T
- Hiss bundle
  - Na^{+} channels
- Tawara´s branches
  - Na^{+} channels



Blood flow into conduction system

- Sinus node
- AV node
- Hiss bundle

- Right Tawara´s br.
- Levé Tawara´s br.
  - anterior bundle
  - posterior bundle

- ACD (60 %, RCX 40 %)
- ACD (90 %, RCX 10 %)
- ACD (AV nodal branch) + rr. septales RIA
- rr. septales RIA + ACD/ RCX

  - rr. septales RIA
  - AV nodal branch ACD + rr. sept.

Zimetbaum, Josephson: Use of the ECG in AMI. NEJM 2003:348:933-940

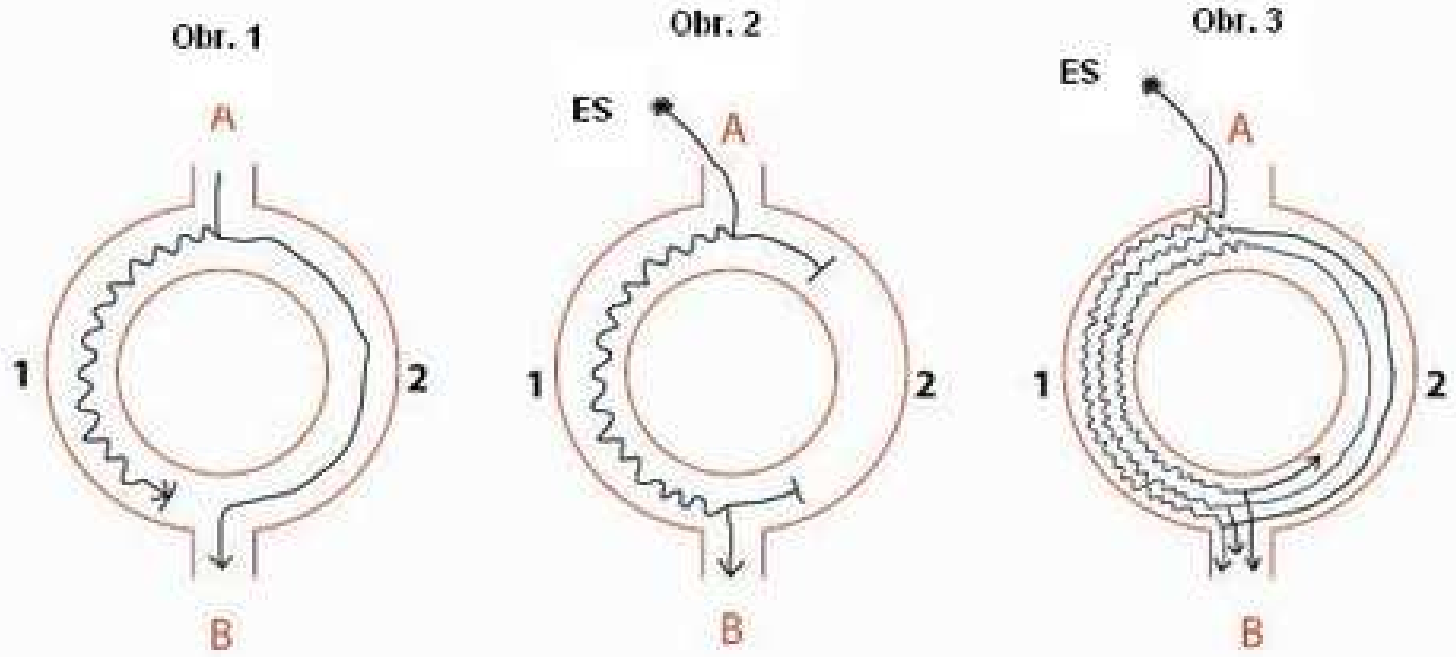
Heart rhythm disturbances

- **substrate:**
  infarction, scar, fibrosis, infiltration
- **trigger:**
  change of heart rate, extrasystole
- **modulating factors:**
  hypoxia, ischaemia, acidosis, ion concentration changes, changes in fiber stretch

Heart rhythm disturbances

- **disturbed automaticity**
  - disturbed normal automaticity
  - abnormal automaticity
  - triggered activity = early, late afterdepolarisation
- **conduction disturbances**
  - blockades: SA, AV, Tawara´s branches
  - re-entry (macro-reentry and micro-re-entry):
    2 places connected by 2 pathways with different conduction velocity and refractoriness
    + one-way block in one of the pathways

Re-entry



1 – slow pathway with short refractory period
2 – fast pathway with long refractory period
ES - extrasystole

Effects of heart rhythm disturbances

- **classical:**
  - electric instability
  - haemodynamic: reduction of cardiac output
  - prognostic
- **new:**
  - electrical remodelation
  - mechano-electric feedback

How to diagnose arrhythmias? RRR

- **R**ate?
  - bradycardia x tachycardia
- q**R**s duration > 0,12 s?
  - Action potential formation above/below AV node (cave aberrant conduction)
- **R**egularity of the R-R interval?
  - regular x irregular

Tachycardias

- **with narrow QRS complex (< 0,12 s)**
  - regular
    - sinus, atrial, atrial flutter, junctional
  - irregular
    - atrial fibrillation, multifocal atrial, atrial flutter or tachycardia with variable AV blockade
- **with wide QRS complex (> 0,12 s)**
  - SVT with aberrant conduction
  - ventricular tachycardia
  - torsade de pointes

Tachyarytmie according to frequency

narrow QRS (< 0,12 s)

- sinus tachycardia
- atrial fibrillation
- atrial fĺutter
- AV nodal reentry
- accessory pathway

re-entry

- multifocal atrial t.
- junctional tachycardia

ectopic

wide QRS (≥ 0,12 s)

- ventricular tachycardia and fibrillation
- SVT with aberrant conduction
- pre-excitation (WPW)
- ventricular stimulated rhythms

Heart rhythm distrubances – clinical causes

V
- ventilation (hypoxia, hyper- / hypocapnia) vegetative dysbalance

A
- anaesthetics and drugs (adverse effects, interactions)

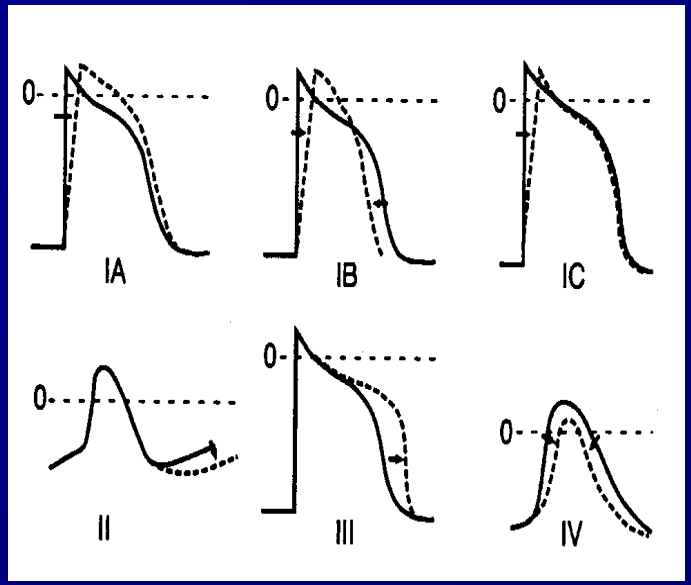
I
- irriation - mechanical, thermal

I
- ischaemia, ions a acid-baze balance

Cardioversion a defibrillation

- cardioversion = treatment of other rhythm disturbances than VF
  - synchronised
    - unstable SVT 50 J – 100 J
    - unstable atrial fibrillation 100-200 J
    - unstable atrial flutter 50 J – 100 J
    - unstable monomorph VT 200 J
  - unsynchronised
    - polymorph VT, torsade de pointes
- defibrillation

Antiarrhythmics

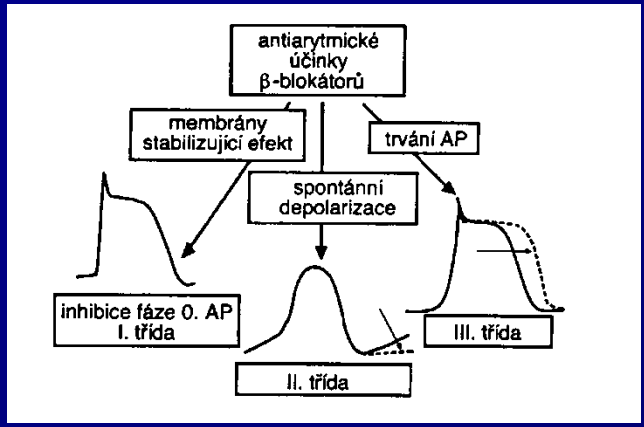


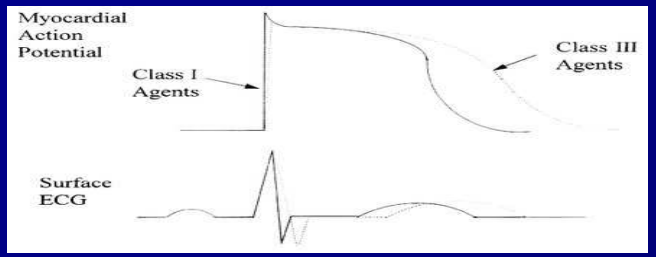
- I Sodium channel blockers
  - Ia chinidin, prokainamid, ajmalin
  - Ib lidokain, mexiletin, phenytoin
  - Ic encainid, flecainid, propafenon
- II Beta-blockers
- III Prolonging action potential - amiodaron
- IV Calcium channel blockers - verapamil, diltiazem
- other – digoxine, adenosine

elektrophysiological classification by Vaughan-Williams 1984

Guidelines for practice

- remove irritation
- correct homeostasis (mainly K^+, Mg^{2+})
- beta-blockers
  - cave: compensatory tachycardia, heart failure, WPW sy, asthma bronchiale
- amiodaron





	Duration	Dose	Rate
Day 1			
Phase 1	10 min	150 mg	15 mg/min
Phase 2	6 h	360 mg	1.0 mg/min
Phase 3	18 h	540 mg	0.5 mg/min
Subsequent days	24 h	720 mg	0.5 mg/min
Breakthrough episodes	10 min	150 mg	15 mg/min

Coding of cardiostimulators

- 1. letter – stimulated part
  A = atrium, V = ventricle, D = dual, both
- 2. letter – sensing
  A = atrium, V = ventricle, D = dual, both, 0 = nothing
- 3. písmeno = reaction to sensed signal
  I = inhibition, T = trigger, D = dual, 0 = nothing
- 4. letter = programmable functions
  P, M, 0, R = rate-responsive
- 5. letter = specific anti-tachycardic functions

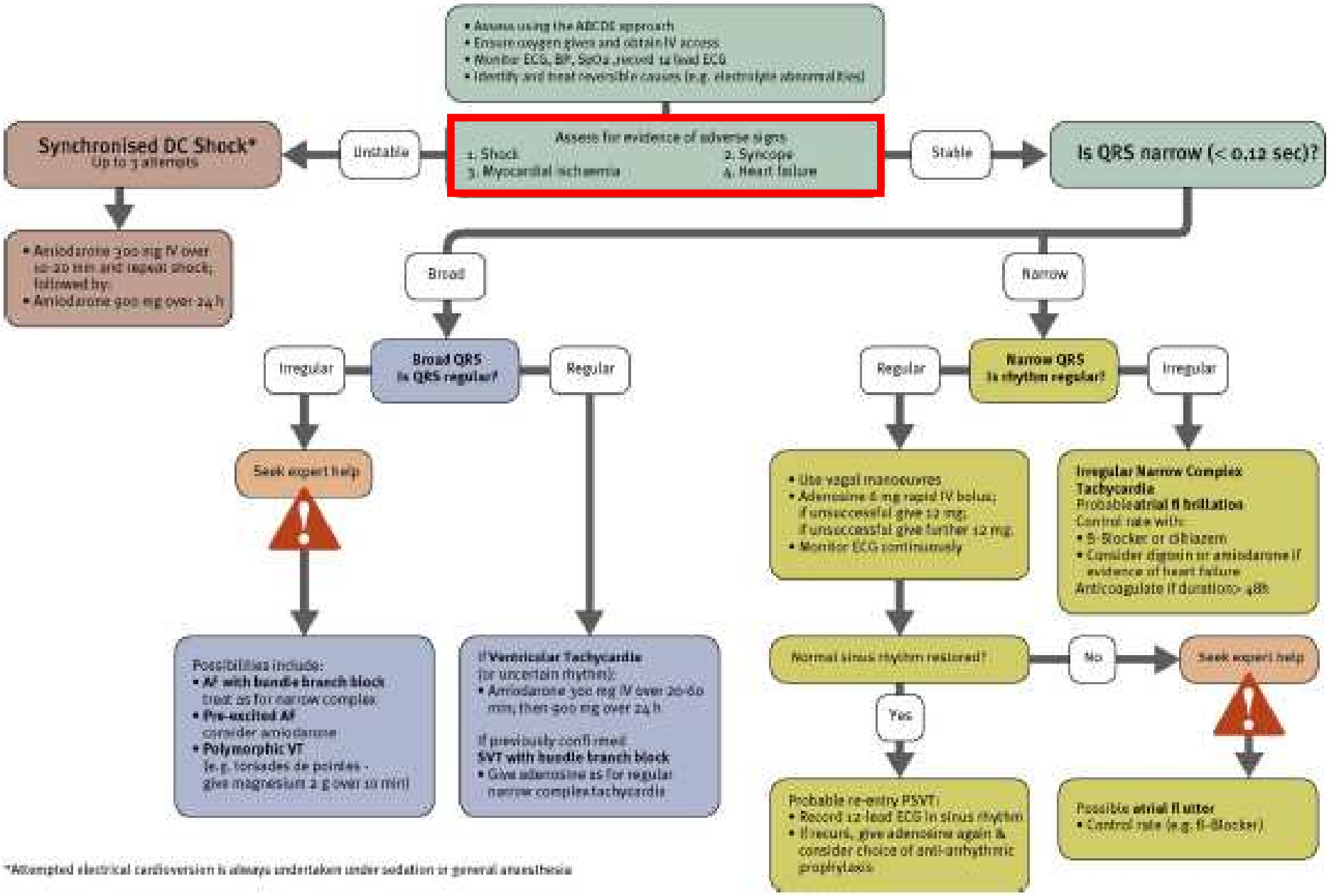
Indications for cardiostimulation

- sick sinus sy
- AV blockades
- bi- nebo trifascicular blocks
- neurogennic syncope
- cardiomypathy
- heart failure → ventricular resynchronisation =
  = hemodynamic indikation (biventricular CS)

Anesthesiological approach in patients with pacers

- check before surgery
- reason, regime a stimulation parameters
- dependence on stimulation - by ECG
- anaesthesia technique does not depend on stimulation
- electrocautery
- magnet?
- CS failure – isoprenaline 0,05-0,2 mg i.v. bolus, then infusion
- check after surgery, setting higher HR?

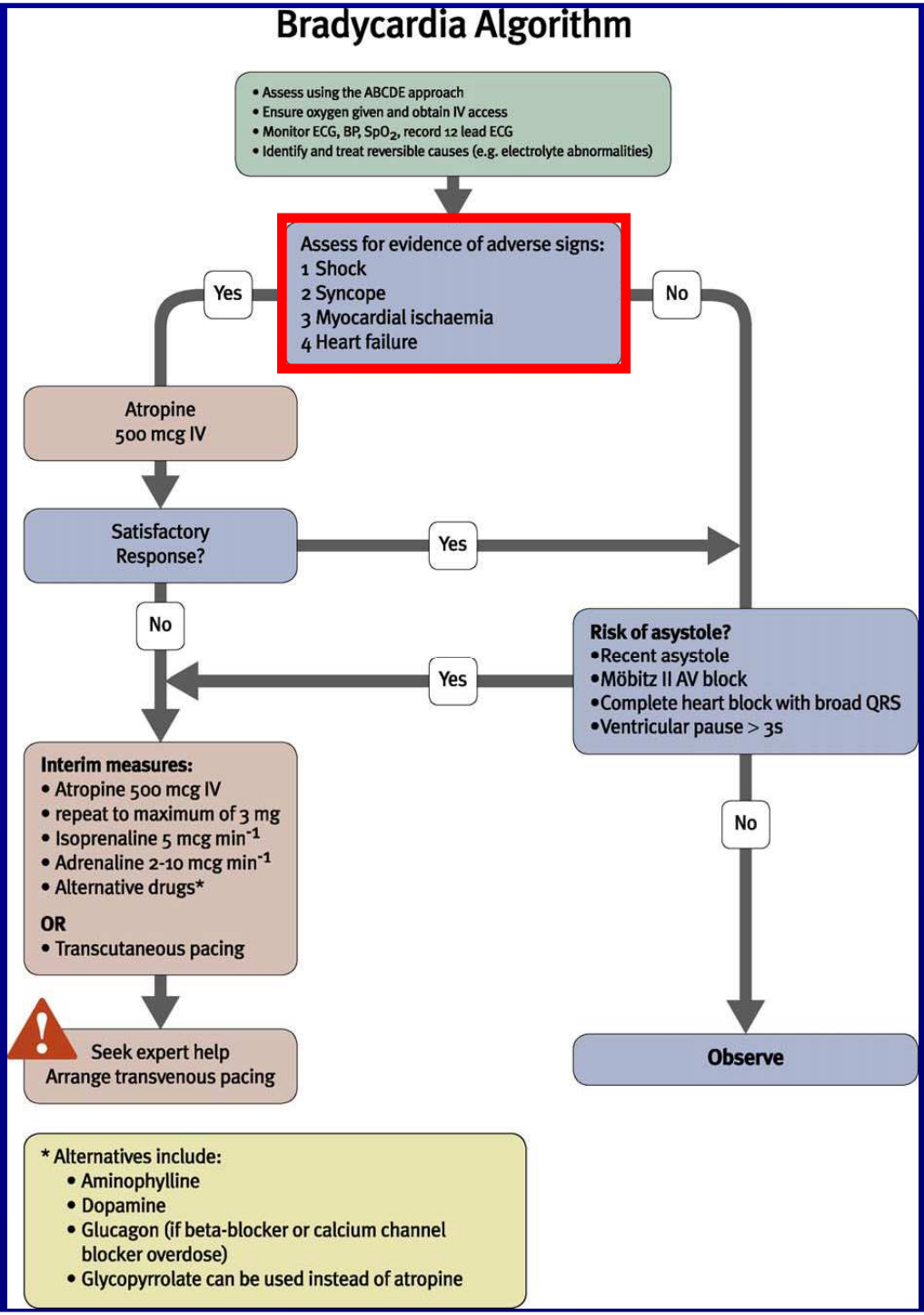
Tachycardia Algorithm (with pulse)



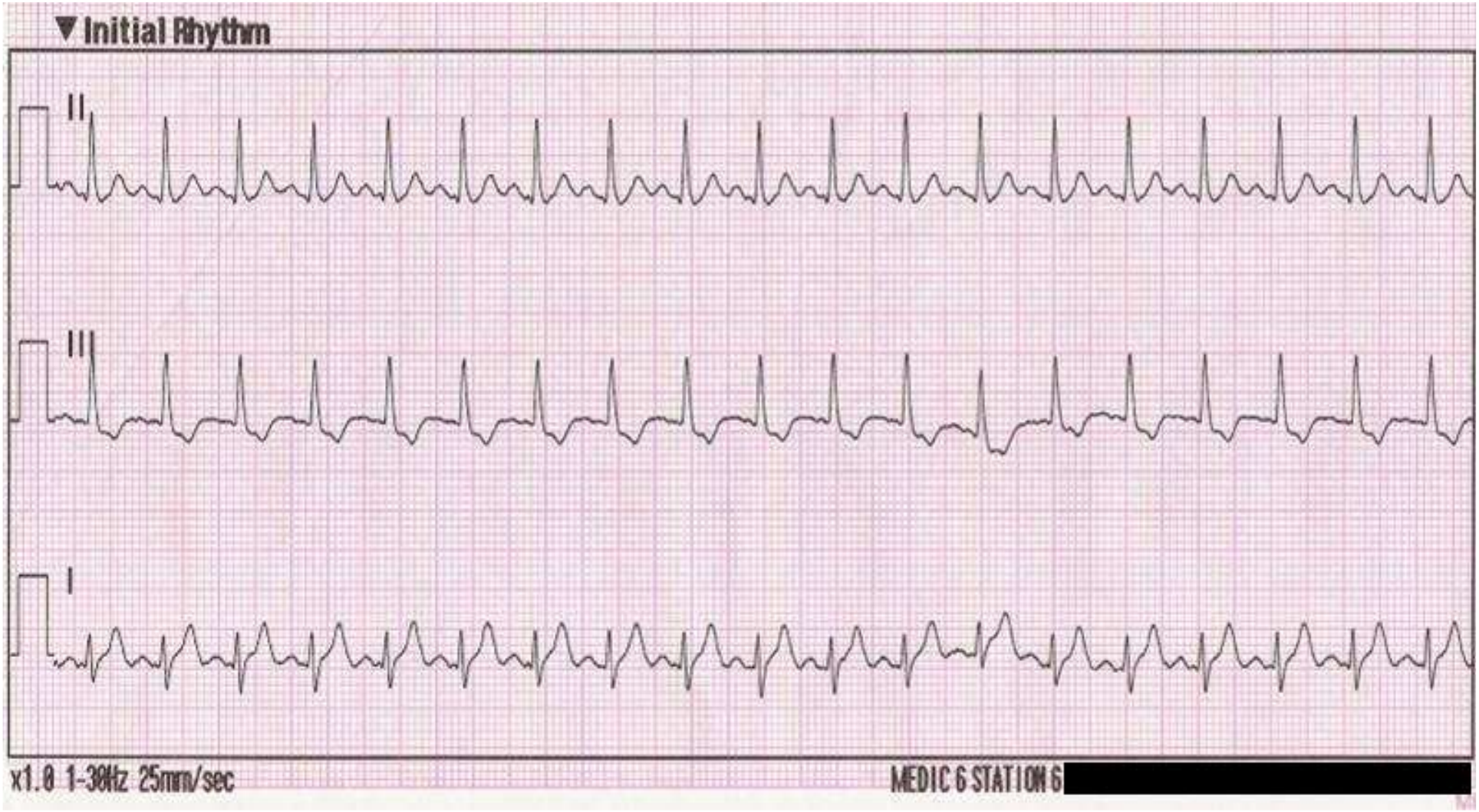
*Attempted electrical cardioversion is always undertaken under sedation or general anaesthesia

Fig. 4.11. Tachycardia algorithm. © 2010 ERC.

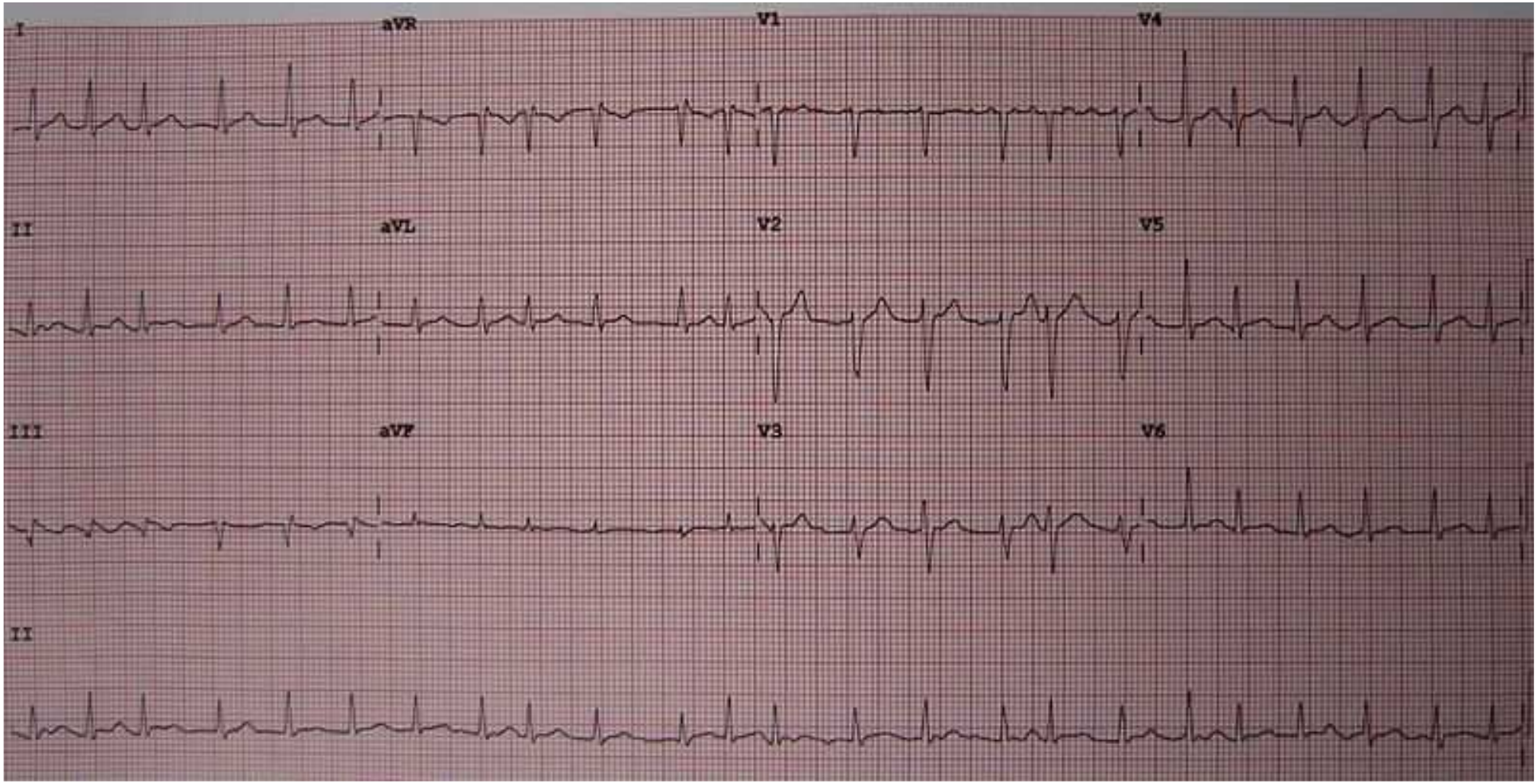
Bradycardia Algorithm



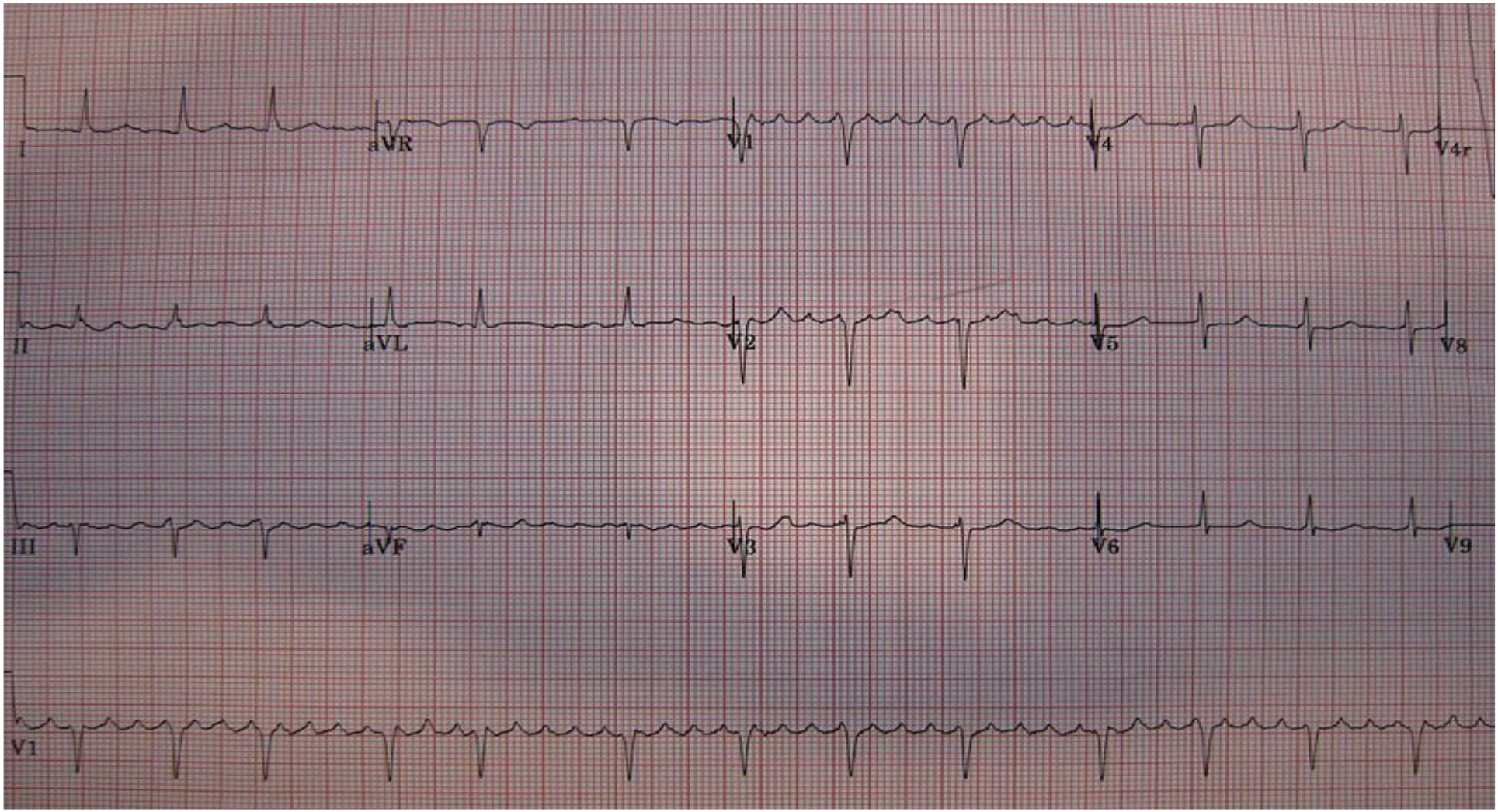
Sinus tachycardia



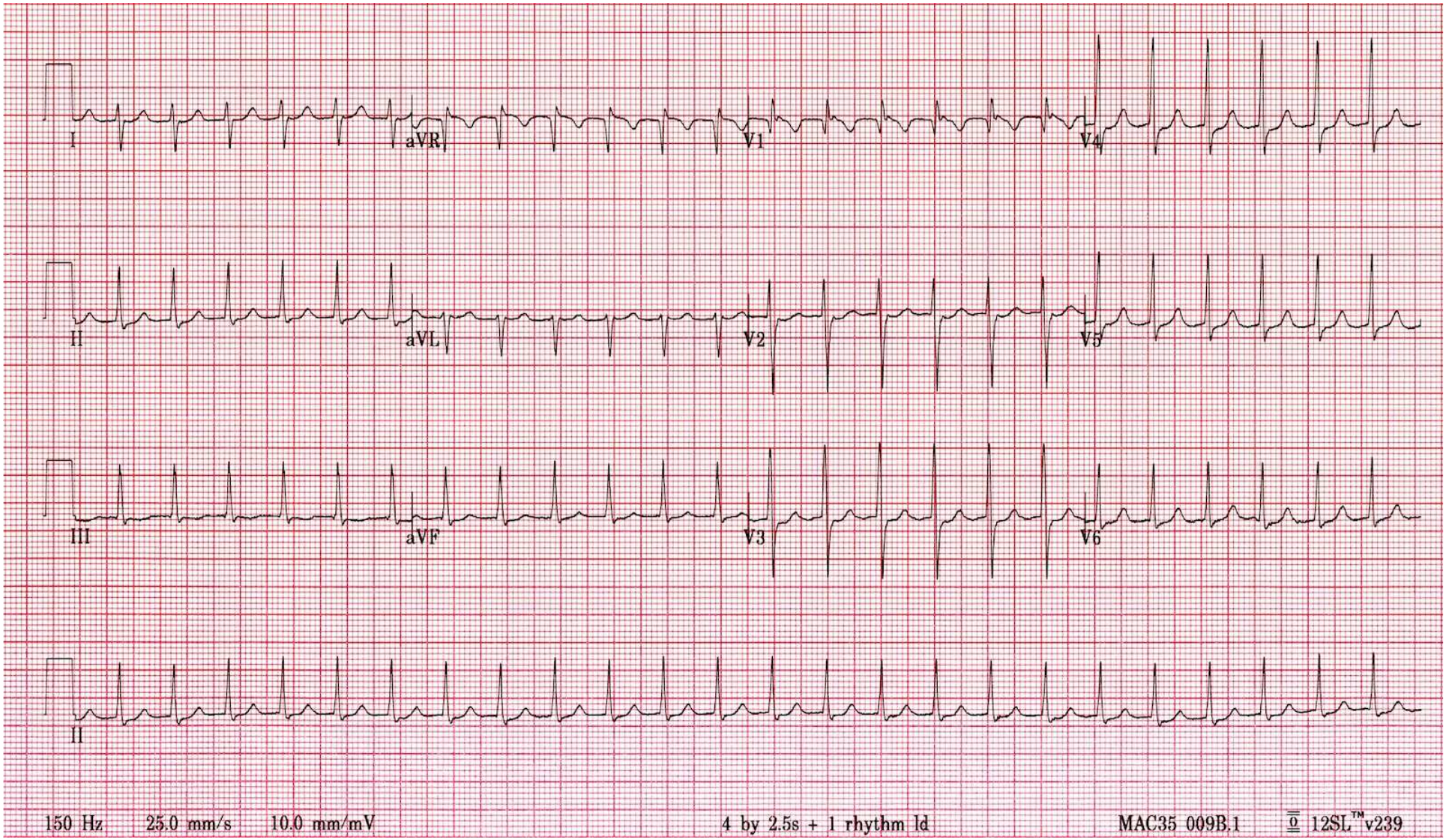
Atrial fibrillation



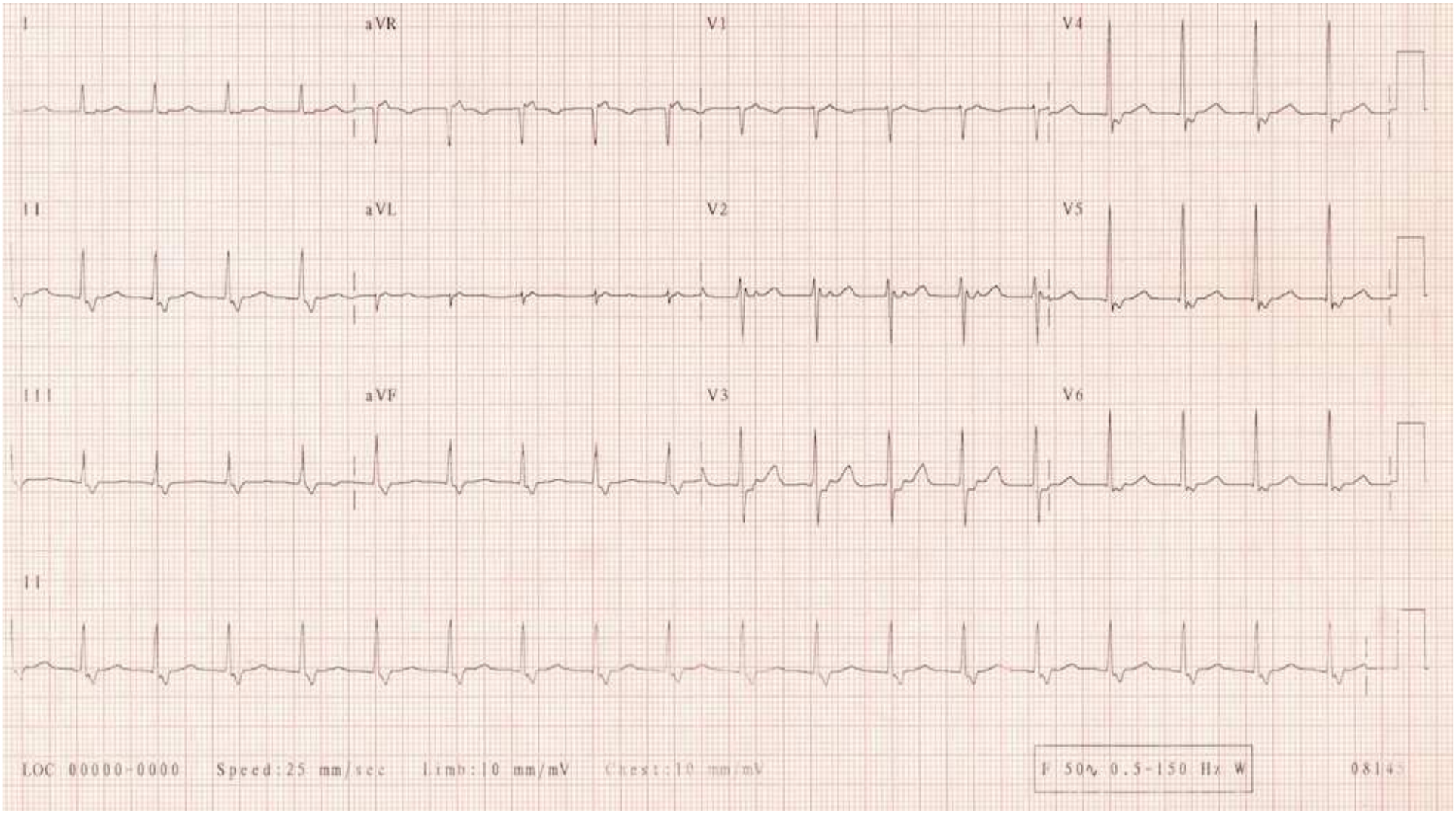
Atrial flutter



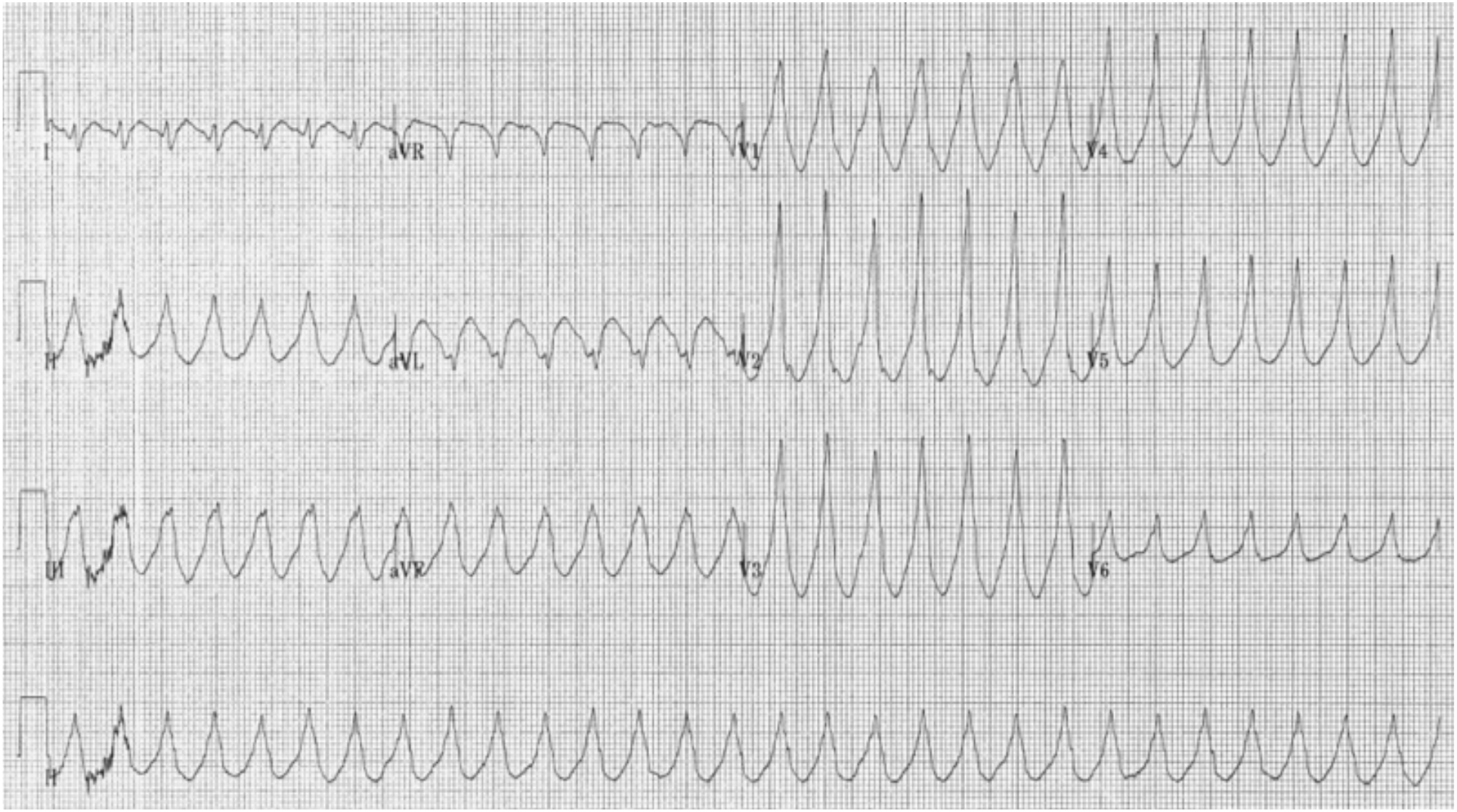
AV nodal reentrant tachycardia slow-fast



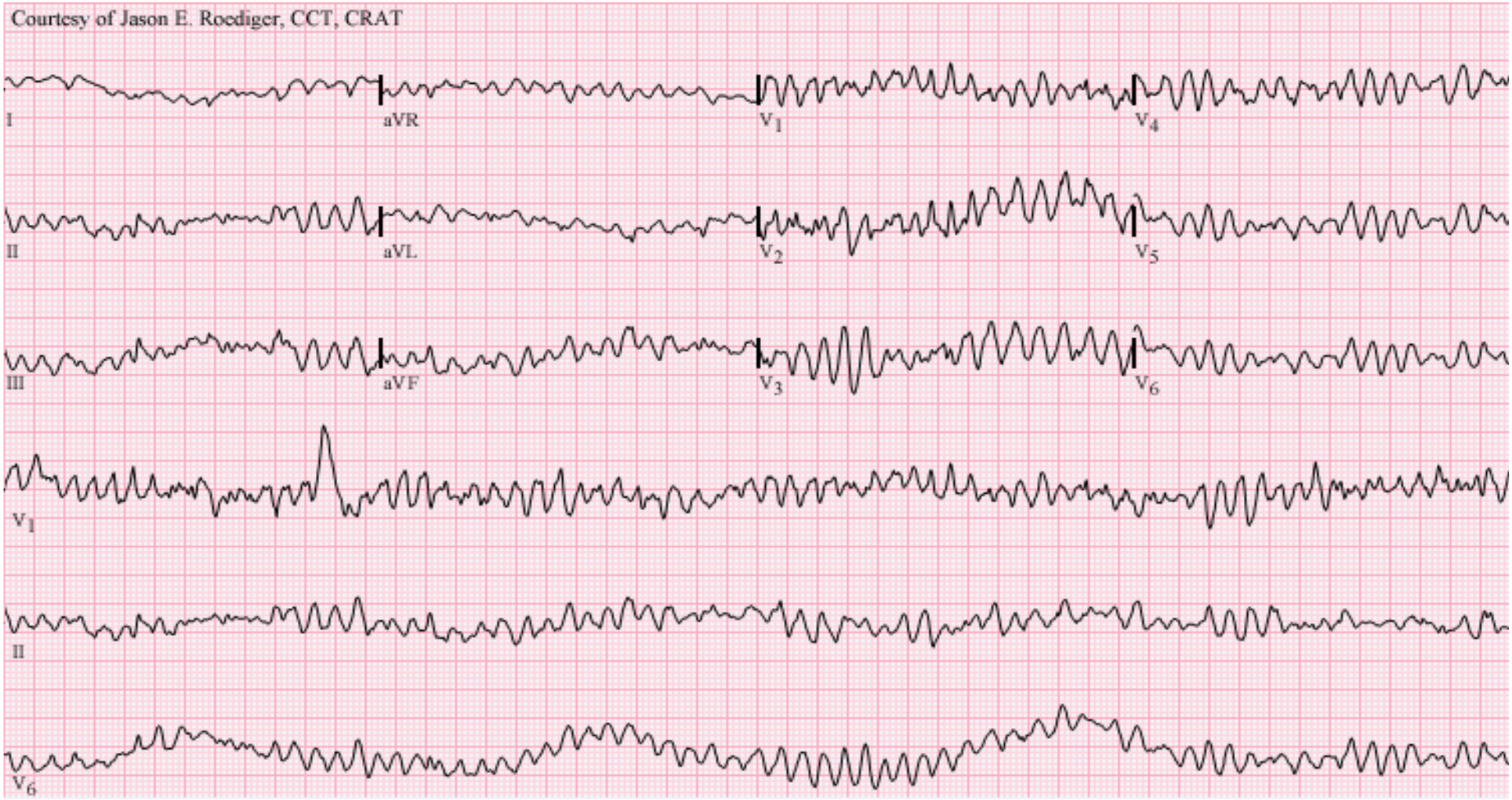
AV nodal reentrant tachycardia fast-slow



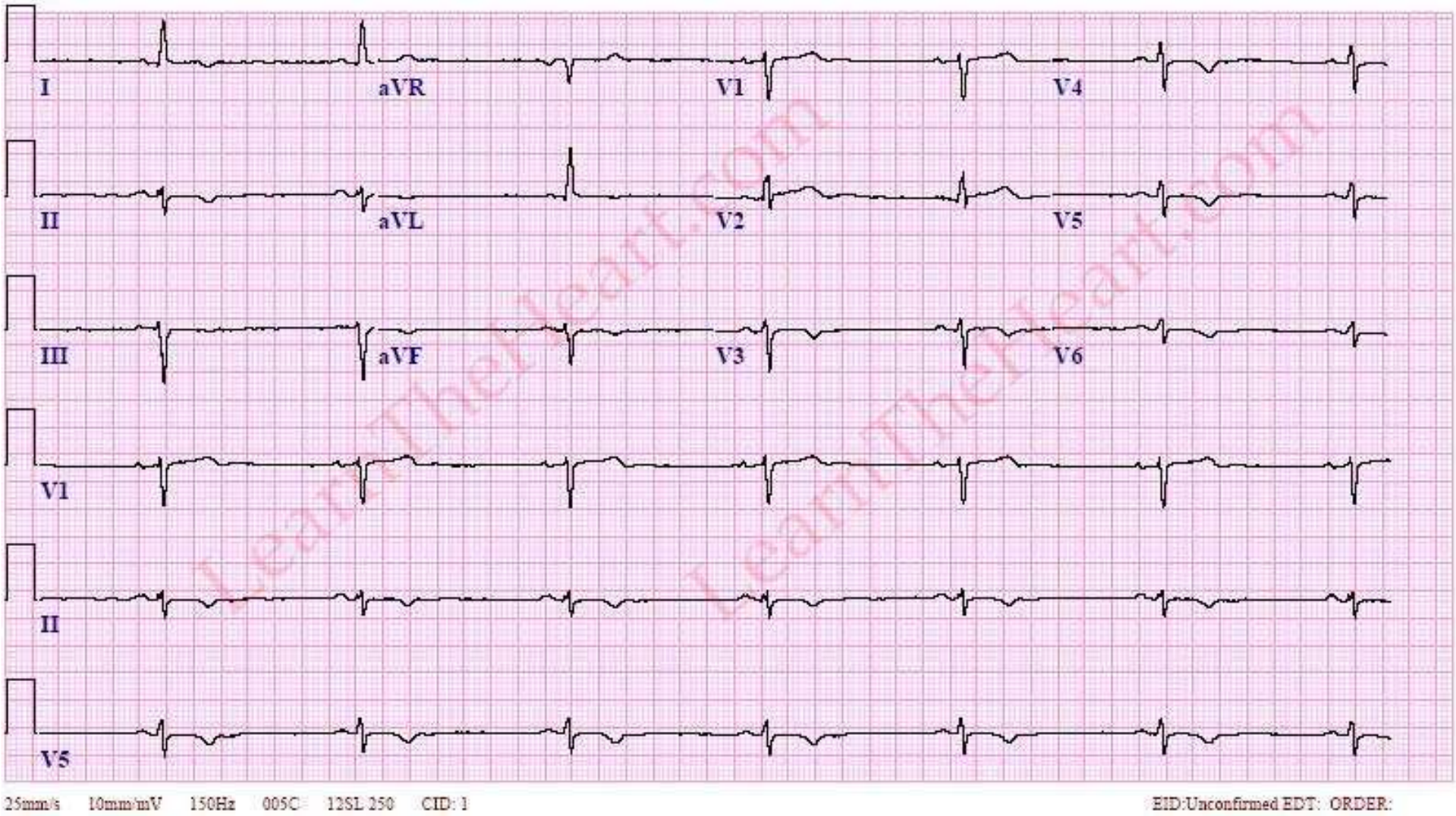
Ventricular tachycardia



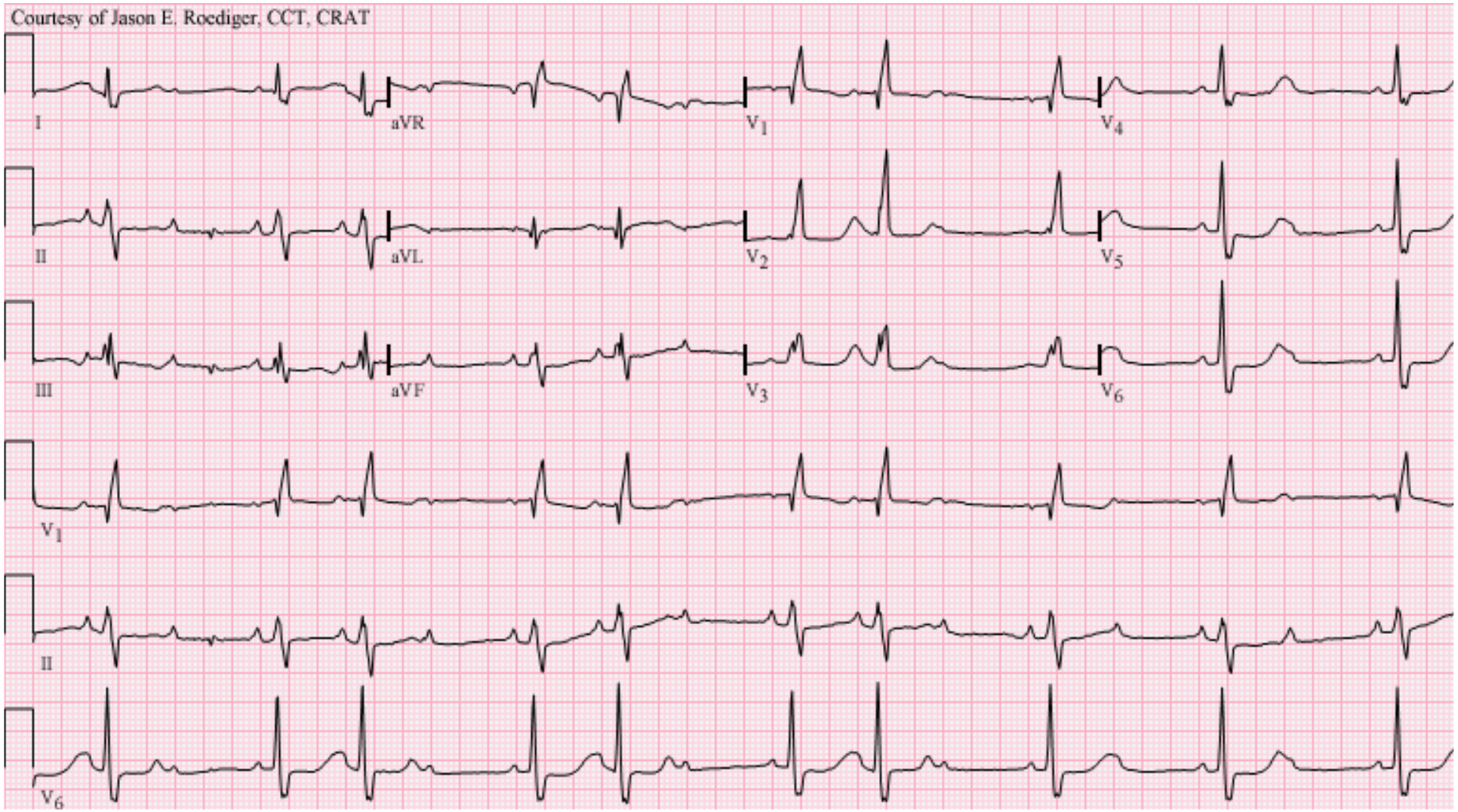
Ventricular fibrillation

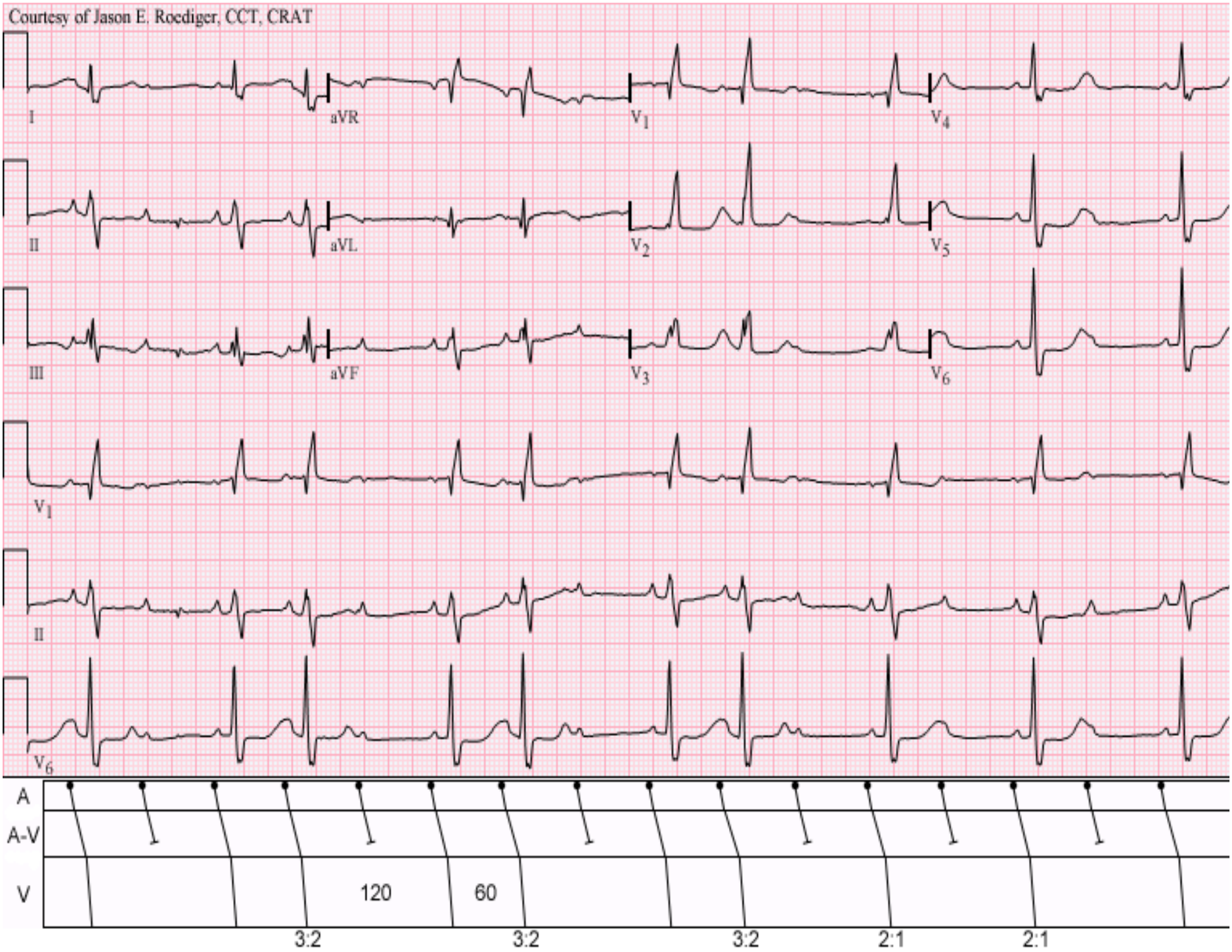


Sinus bradycardia



AV block degree II, type 2
3:2 and 2:1




Courtesy of Jason E. Roediger, CCT, CRAT
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V6
A
A-V
V
120
60
3:2
3:2
3:2
2:1
2:1

Program

- basic physiology
- heart rhythm disturbances
- **In part 2**
- circulation monitoring
- shock
- cannulations
- circulatory support