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AD-COR Program inovativ de formare in domeniul cardiologiei pediatrice POSDRU/179/3.2/S/152012

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MODUL TEORETIC

PAEDIATRIC CARDIAC CRITICAL CARE

Imputernicit: Prof. Dr. Tammam Youssef

Activitate prestata de I.R.C.C.S. POLICLINICO SAN DONATO – MILANO, ITALIA in baza contractului nr. 18/22144/29.07.2015

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Beneficiar: Universitatea de Medicină și Farmacie „Carol Davila” București

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*A4 - Planificarea, organizarea si desfasurarea activitatilor de formare a **medicilor** in domeniul cardiologiei pediatrice*

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PAEDIATRIC CARDIAC CRITICAL CARE

Bucharest, October 19, 2015

Matthias Angrés, MD, PhD
RobinAid Foundation
Hamburg / Germany

A4 - Planificarea, organizarea si desfasurarea activitatilor de formare a ***medicilor*** *in domeniul cardiologiei pediatrice*

Declaration of Disclosure

I have no actual or potential conflict of interest in relation to this presentation.

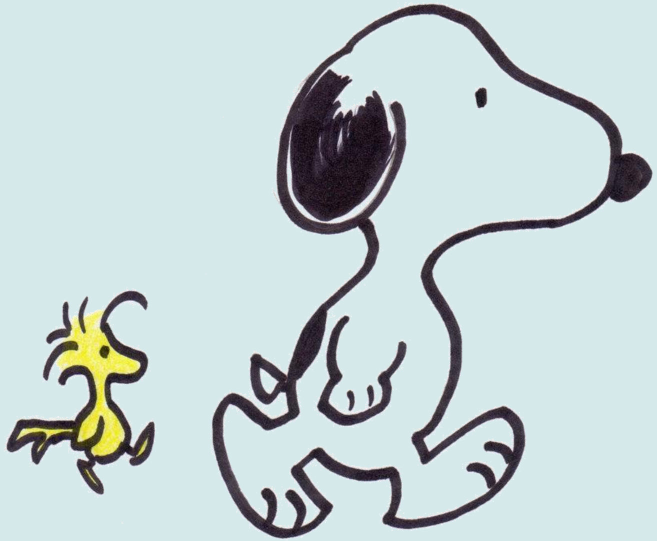
Today‘s agenda

- Short flashback
- Two lectures
- 15 minutes break between both parts

Let‘s continue our walk

Important topics in Paediatric Cardiac Critical Care

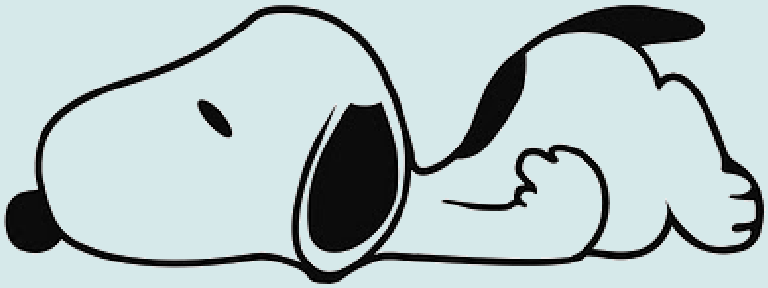
14 important topics



1. *Inotropic agents in paediatric cardiac critical care √*
2. Human factors in paediatric cardiac critical care
3. Challenges in setting up a new Paediatric Cardiac Critical Care Unit
4. *The effects of cardiopulmonary bypass during paediatric cardiac surgery √*
5. *Importance of pain management in paediatric cardiac critical care √*
6. *Respiratory assessment and management / respiratory complications*
7. *Cardiac arrhythmias: assessment and management / temporary pacemaker*

14 important topics

8. Medistinal bleeding and cardiac tamponade / blood transfusion, coagulation disorders, and postoperative anticoagulation
9. Post-cardiac surgery pulmonary hypertension: identification and management
10. Infections in the cardiac intensive care unit
11. Fast-track extubation in pediatric cardiac surgery
12. Basics of echocardiography in monitoring the postoperative pediatric cardiac surgery patient.
13. Mechanical circulatory support in paediatric cardiac surgery
14. One hundred useful references in paediatric cardiac intensive care

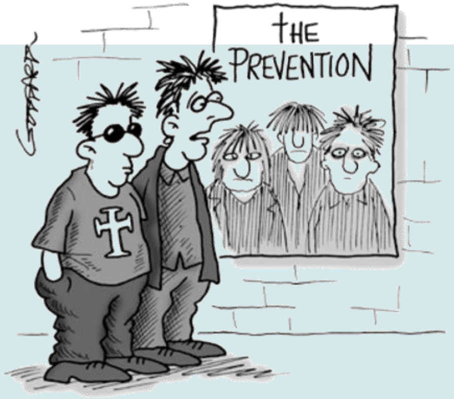


As you may remember

The highly complex paediatric patients with congenital or acquired heart disease require **interprofessional teamwork** and collaboration **to ensure high quality** outcomes **with low mortality and morbidity.**

As you may remember

Anticipation and prevention are the most important **keys for recognition, diagnosis, and successfull management** of all potential risks and complications in the postoperative critical care of paediatric cardiac patients.



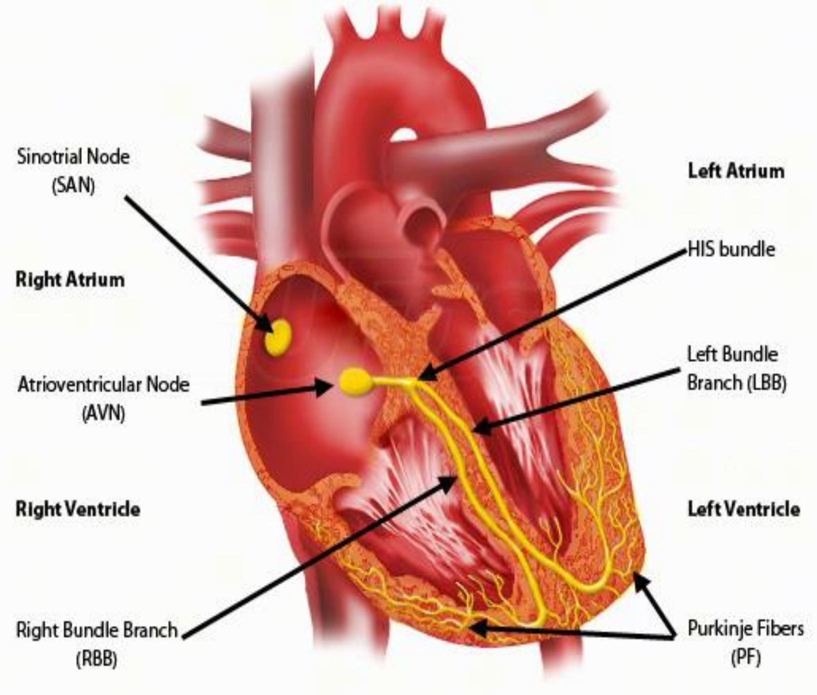
"Apparently they're better than The Cure"

Let‘s continue with the next topic

Arrhythmias in postoperative paediatric cardiac critical care and care of patients with temporary pacemaker

The cardiac conduction system

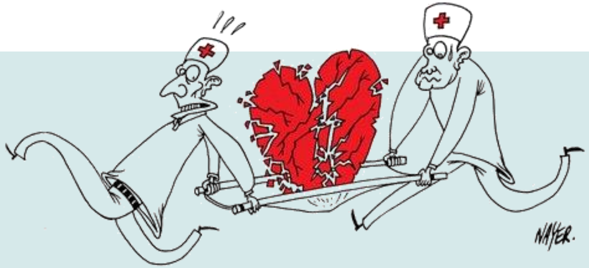
- The Sinoatrial (SA) node is the natural pacemaker of the heart
- The SA node releases electrical stimuli at a regular rate which is dictated by the needs of the body
- The electrical stimulus from the SA node eventually reaches the AV node by the internodal pathways
- After this the stimulus passes through the AV node and Bundle of His into the Bundle branches and Purkinje fibres



Arrhythmias in paediatric cardiac surgery

- Early postoperative arrhythmias are known complications in paediatric cardiac surgery
- **Incidence between 15 - 45 %**
- Transient and treatable in most cases
- Haemodynamically instable arrhythmias are **related to increased mortality**

Delaney JW et al. J Thorac Cardiovascu Surg 2006
Valsangiacomo E et al. Ann Thorac Surg 2002
Pfammatter JP et al. Ped Crit Care Med 2001
Rekawek J et al. J Thorac Cardiovasc Surg 2007
Hoffman TM et al. Ped Cardiol 2002
Lan YT et al. Curr Opin Cardiol 2003



Triggering factors

- Lower body weight (5 to 9 kg)
- Younger age (6 to18 months)
- Longer C.P.B.P. and aortic crossclamp times
- Use of deep hypothermia and circulatory arrest
- **Inflammatory response**
- Type of intervention

- Residual lesions
- Postoperative cardiac dysfunction
- **Scar and sutures**
- Electrolyte disturbances
- Catecholamine stimulation
- **Pain, anxiety**
- Stress response

Most important arrhythmias

Annals of Pediatric Cardiology
Official publication of the Pediatric Cardiology Society of India

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Ann Pediatr Cardiol. 2014 Jan-Apr; 7(1): 19–24.
doi: 10.4103/0974-2069.126543
PMCID: PMC3959055

Anticipation and management of junctional ectopic tachycardia in postoperative cardiac surgery: Single center experience with high incidence

Osama Abdelaziz and Salem Deraz[1]

Early post. operative arrhythmia	Number of patients	% of whole cohort (194)
JET	53	27
Junctional rhythm	14	7.2
Transient heart block	12	6.2
Permanent heart block	3	1.5
JET alternating with heart block	5	2.5
SVT	5	2.5
Atrial flutter	2	1
Atrial tachycardia	2	1

JET: Junctional ectopic tachycardia, SVT: Supraventricular tachycardia

Abdelaziz O et al. Ann Pediatr Cardiol 2014 Jan-Apr; 7(1): 19–24.

Most important arrhythmias

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Atrial tachycardia	2	1

JET: Junctional ectopic tachycardia, SVT: Supraventricular tachycardia

- **Junctional ectopic tachycardia**
- **Transient heart block**

Junctional ectopic tachycardia

- A **narrow complex tachycardia** with atrio-ventricular dissociation

- JET is documented most commonly following Tetralogy of Fallot (TOF) repair, AVSD repair, and Senning operation
- JET can be a result of a combination of factors, including underlying heart disease, type of surgical procedure, haemodynamic instability, and electrolyte imbalance, specially hypomagnesemia
- The pathological mechanism in detail is still unclear; is believed to be a result of direct trauma to the AV node and bundle of His

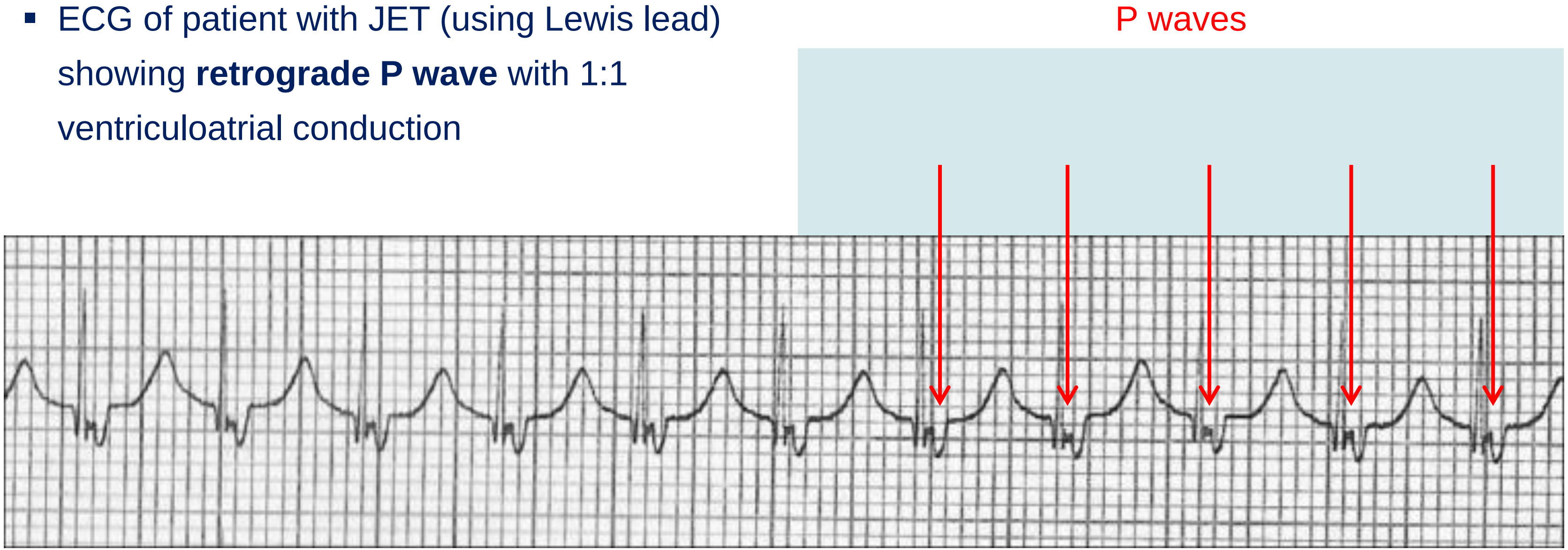
Junctional ectopic tachycardia

- ECG of patient with JET (using Lewis lead) showing the **P waves dissociated from QRS** with atrial rate less than the ventricular rate

P waves

Junctional ectopic tachycardia

- ECG of patient with JET (using Lewis lead) showing **retrograde P wave** with 1:1 ventriculoatrial conduction

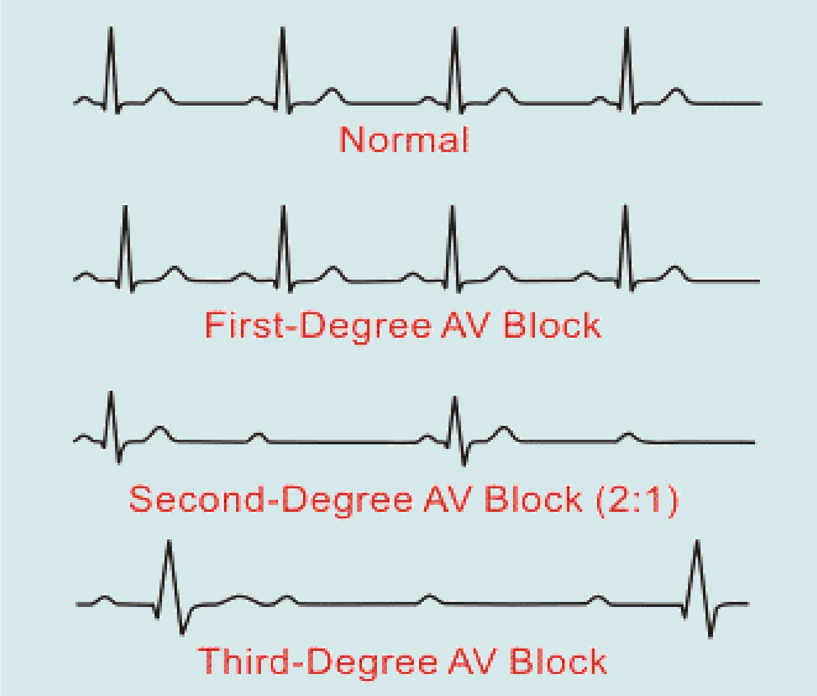


Therapeutic interventions

- **Step 1**:
  – avoid hyperthermia
  – Optimize pain management and anxiolysis
  – optimize electrolytes
  – give magnesium sulfate bolus of 30 mg/kg
- **Step 2**:
  – decrease the catecholamine dose if possible
  – especially stop Dopamine
- **Step 3**:
  – Amiodarone is the first line anti-arrhythmic drug for all patients with haemodynamically compromising JET
  – bolus dose of 5 mg/kg over 2 h followed by continuous infusion of 10-20 mg/kg/24 h.
- **Pacing therapy** can be used for overdrive or sequential pacing **in selected cases**

Transient heart block

- Atrioventricular conduction block (**AV block**) following open-heart surgery for congenital heart disease is uncommon but a well-known complication
- Its incidence ranges between **1 and 3%**
- Early-presenting heart block usually appears during surgery or shortly thereafter



Gross GJ et al. Heart Rhythm 2006

Lin A et al. J Thorac Cardiovasc Surg 2010

Transient heart block

- The greatest **risk for complete AV block** is associated with corrective surgical procedures for:
  - ventricular septal defects (**VSD**), usually as part of more complex congenital heart disease
  - atrioventricular septal defects (**AVSD**),
  - left ventricular outflow tract obstruction
  - L-transposition of the great arteries
  - Tetralogy of Fallot (**TOF**)
  - discordant atrioventricular connections

Transient heart block

- Most children who require pacing for surgically induced AV block are **less than 1 year old**
- **Spontaneous resolution** of complete AV block in the early postoperative period most often occurring **between 7 and 14 days**

- Complete AV block may resolve spontaneously **in 43 - 92% of children** (Gross et al. 2006)
- Factors associated with a spontaneous recovery of AV nodal function are currently not known

Therapeutic interventions

- Atrial and ventricular **pacing wires are essentials** in every open heart surgery
- Having a sequential pacemaker available, ready, and **knowing how it functioned** are also basic essentials in paediatric cardiac critical care

Temporary pacemakers

- Objectives:
  - explain the situations when temporary pacemakers are indicated
  - describe the **principles of pacing**
  - illustrate normal and abnormal pacemaker behavior
  - discuss the steps to be taken in **troubleshooting** a temporary pacemaker

Indications for temporary pacing

- Bradyarrhythmias
- **AV conduction block**
- Slow sinus or junctional rhythm
- Suppression of ectopy
- Drugs, electrolyte imbalances (Sick Sinus Syndrome)
- Secondary to pronounced atrial stretch
- Old TGA s/p Senning or Mustard procedure

Electrical concept

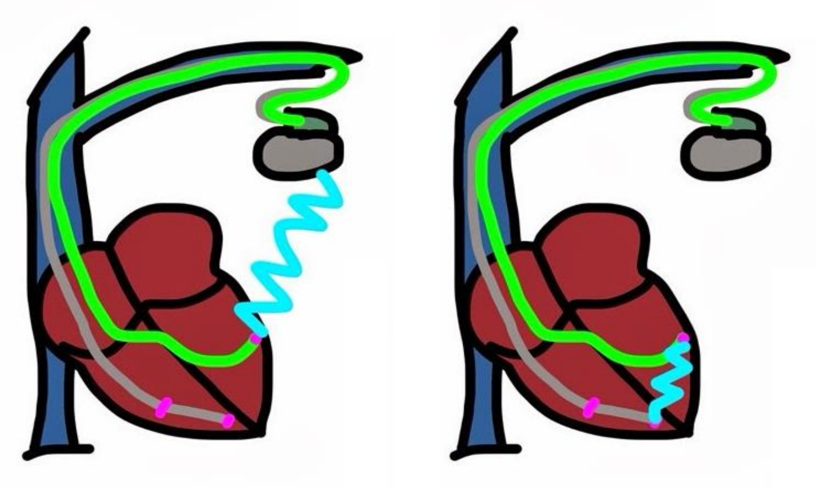
- **Electrical circuit**
  - pacemaker to patient, patient to pacemaker
- **Current**
  - the flow of electrons in a completed circuit; measured in milliamperes (mA)
- **Voltage**
  - a unit of electrical pressure or force causing electrons to move through a circuit; measured in millivolts (mV)
- **Impedance**
  - the resistance to the flow of current

Temporary pacing types

- **Transcutaneous**
  - emergency use with external pacing / defib unit
- **Transvenous**
  - emergency use with external pacemaker
- **Epicardial**
  - wires sutured to right atrium & right ventricle
  - atrial wires exit on the right of the sternum
  - ventricular wires exit on the left of the sternum

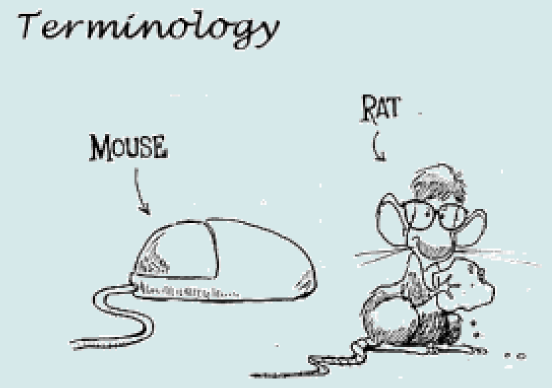
Wiring systems

- **Unipolar**
  – one electrode on the heart (-)
  – signals return through body fluid and tissue to the pacemaker (+)
- **Bipolar**
  – two electrodes on the heart (- / +)
  – signals return to the ring electrode (+) above the lead (-) tip



Overview of terminology

Pace	to deliver an electrical impulse
Sense	ability of the pacemaker to detect intrinsic electrical activity
Pacing Spike	stimulus from the pacemaker recorded on the ECG, a short narrow deflection
Capture	depolarization of the heart by an artificial stimulus; patients myocardial cells capture the impulse delivered by the pacemaker; pacer spike followed by a QRS associated with a pulse

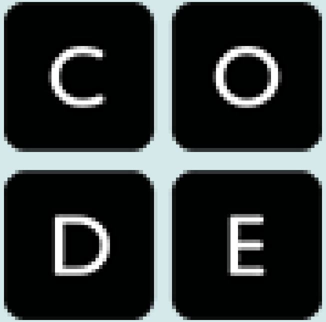


Modes of pacing

- **Atrial pacing**
  – intact AV conduction system required
- **Ventricular pacing**
  – loss of atrial kick
  – discordant ventricular contractions
  – sustains cardiac output
- **Atrial / Ventricular pacing**
  – natural pacing
  – atrial-ventricular synchrony

3-letter NBG temporary code

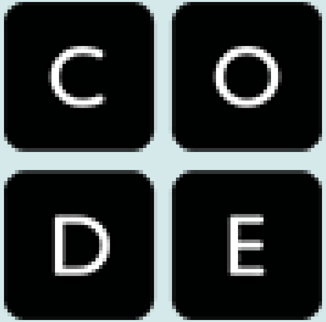
- **First Letter:**



- **Chamber Paced**
  - V - Ventricle
  - A - Atrium
  - D - Dual (A + V)
  - O - None

3-letter NBG temporary code

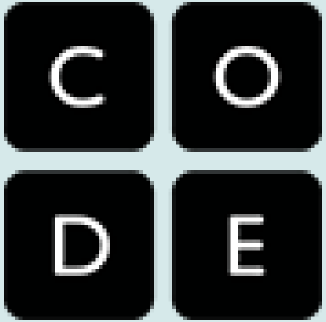
- **Second letter:**



- **Chamber Sensed**
  - V - Ventricle
  - A - Atrium
  - D - Dual (A & V)
  - O - None

3-letter NBG temporary code

- **Third letter:**



- **Sensed Response**
  - T - Triggers Pacing
  - I - Inhibits Pacing
  - D - Dual
  - O - None

Commonly used modes

- **AAI**
  – atrial demand pacing
- **VVI**
  – ventricular demand pacing
- **DDD**
  – atrial/ventricular demand pacing, senses and paces both chambers; trigger or inhibit
- **AOO**
  – atrial asynchronous pacing

Settings

- **Atrial and ventricular output**
  - milliamperes (mA)
  - typical atrial: mA 5
  - typical ventricular: mA 8 - 10
- **AV Interval**
  - milliseconds (msec)
  - time from atrial sense/pace to ventricular pace
  - synonymous with “PR” interval

- **Atrial and ventricular sensitivity**
  - millivolts (mV)
  - typical atrial: 0.4 mV
  - typical ventricular: 2.0mV

Settings

- **Atrial/ventricular rate**
  – set at physiologic rate for individual patient
  – AV Interval, upper rate, + PVARP automatically adjust with set rate changes
- **Upper rate**
  – automatically adjusts to 30 bpm higher than set rate
  – prevents pacemaker mediated tachycardia from unusually high atrial rates
  – Wenckebach-type rhythm results when atrial rates are sensed faster than the set rate

- **Refractory period**
  – PVARP: Post Ventricular Atrial Refractory Period
  – time after ventricular sensing/pacing when atrial events are ignored

Important

- **Electrical Safety**
  - microshock
  - accidental de-wiring
  - taping wires
  - securing pacemaker
- **Removal of pacing wires**
  - potential myocardial trauma
  - bleeding
  - pericardial effusion / tamponade
  - haemothorax
  - ventricular arrhythmias

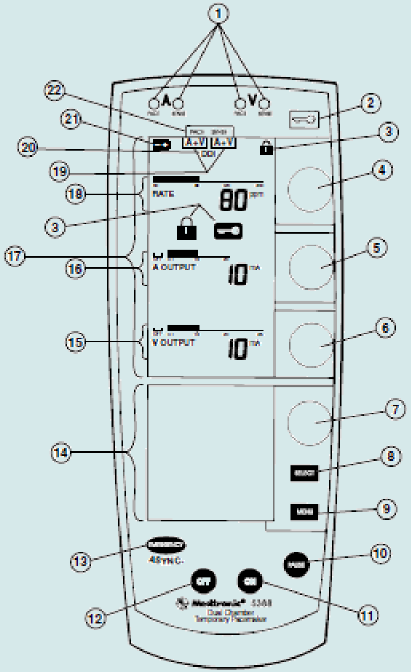
- **Pacemaker care & cleaning**
  - batteries
  - bridging cables
  - pacemakers

- **Medtronic 5388 Dual Chamber (DDD)**

Pacemaker

- **Medtronic 5388 Dual Chamber (DDD)**

1. Pace/Sense LEDs
2. Lock/Unlock Key
3. Lock Indicators
4. Rate Dial
5. Atrial Output Dial
6. Ventricular Output Dial
7. Menu Parameter Dial
8. Parameter Selection Key
9. Menu Selection Key
10. Pause Key
11. Power On Key
12. Power Off Key
13. Emergency/Asynchronous Pacing Key
14. Lower Screen
15. Ventricular Output Graphics
16. Atrial Output Graphics
17. Upper Screen
18. Rate Graphics
19. Setup Indicators
20. DDI Indicator
21. Low Battery Indicator
22. Setup Labels



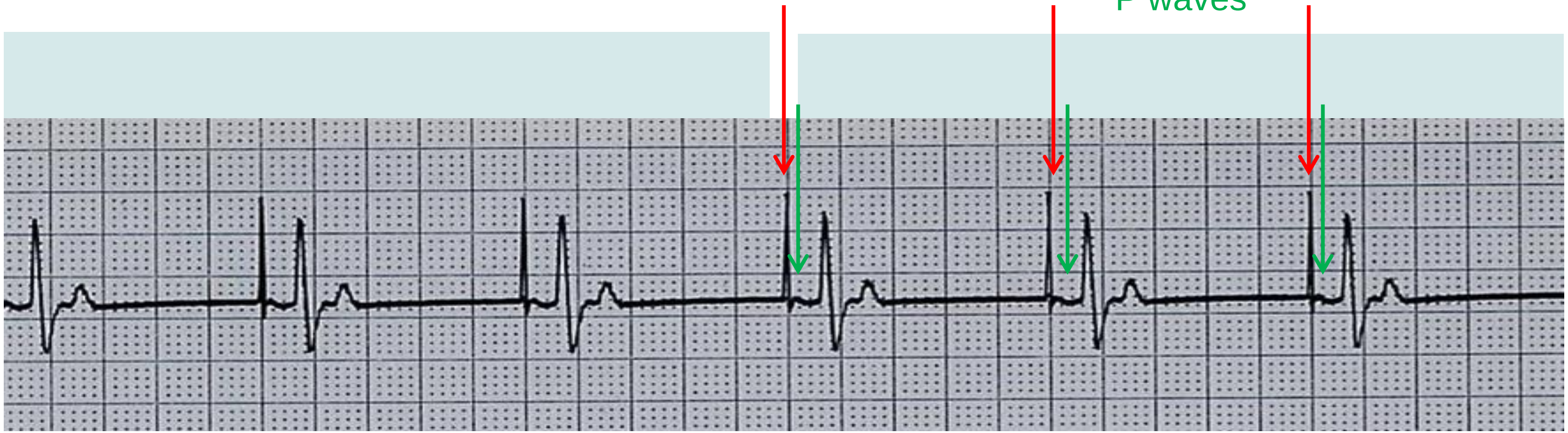
Pacemaker ECG

- Assessing paced ECG
- Identify intrinsic rhythm and clinical condition
- Identify pacer spikes
- Identify activity following pacer spikes
- Failure to capture
- Failure to sense

- Every pacer spike should have a P - wave or QRS complex following it!

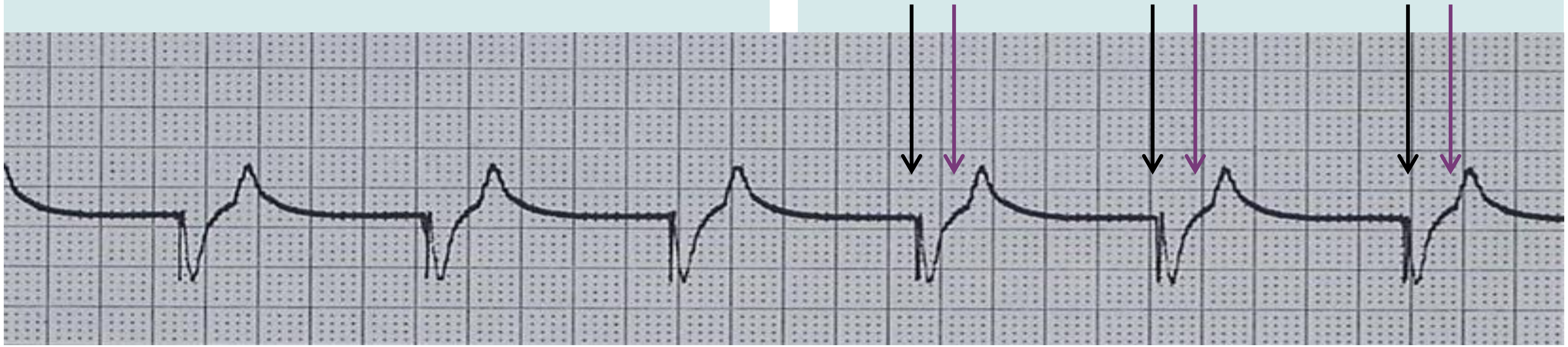
Normal pacing

- **Atrial pacing:**
  – atrial pacing spikes followed by P waves



Normal pacing

- **Ventricular pacing:**
  – ventricular pacing spikes followed by wide, bizarre QRS complexes

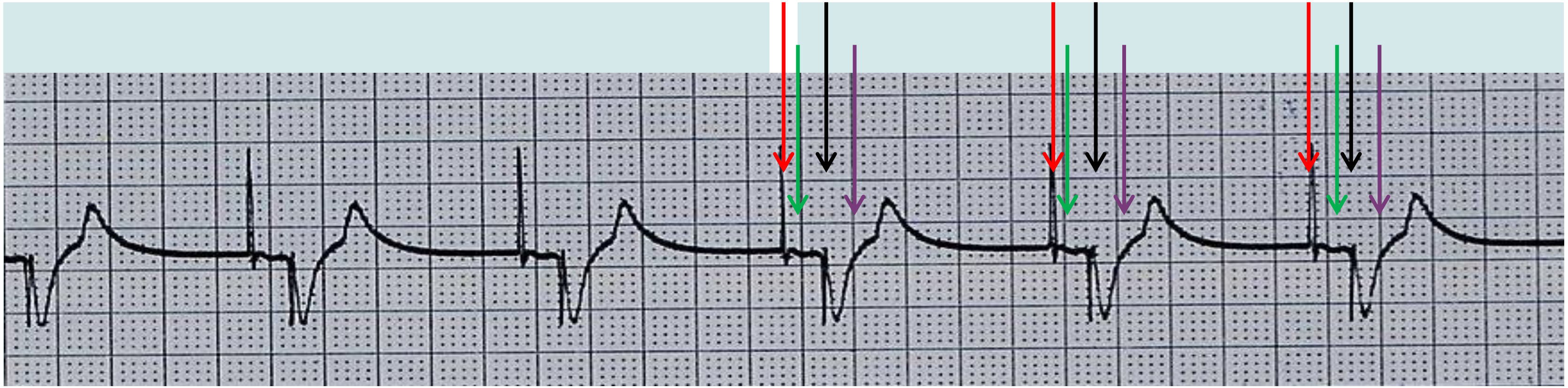


Normal pacing

- **A-V Pacing:**
- atrial + ventricular pacing spikes followed by atrial + ventricular complexes

Atrial + Ventricular pacing spikes

Atrial + QRS complexes

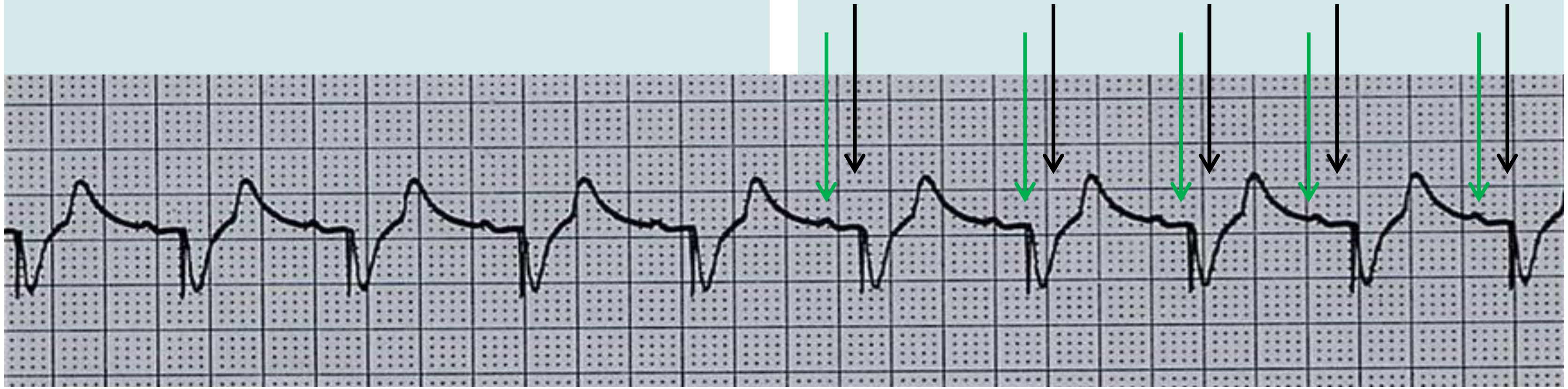


Normal pacing

- **DDD mode of pacing:**
  – ventricle paced at atrial rate

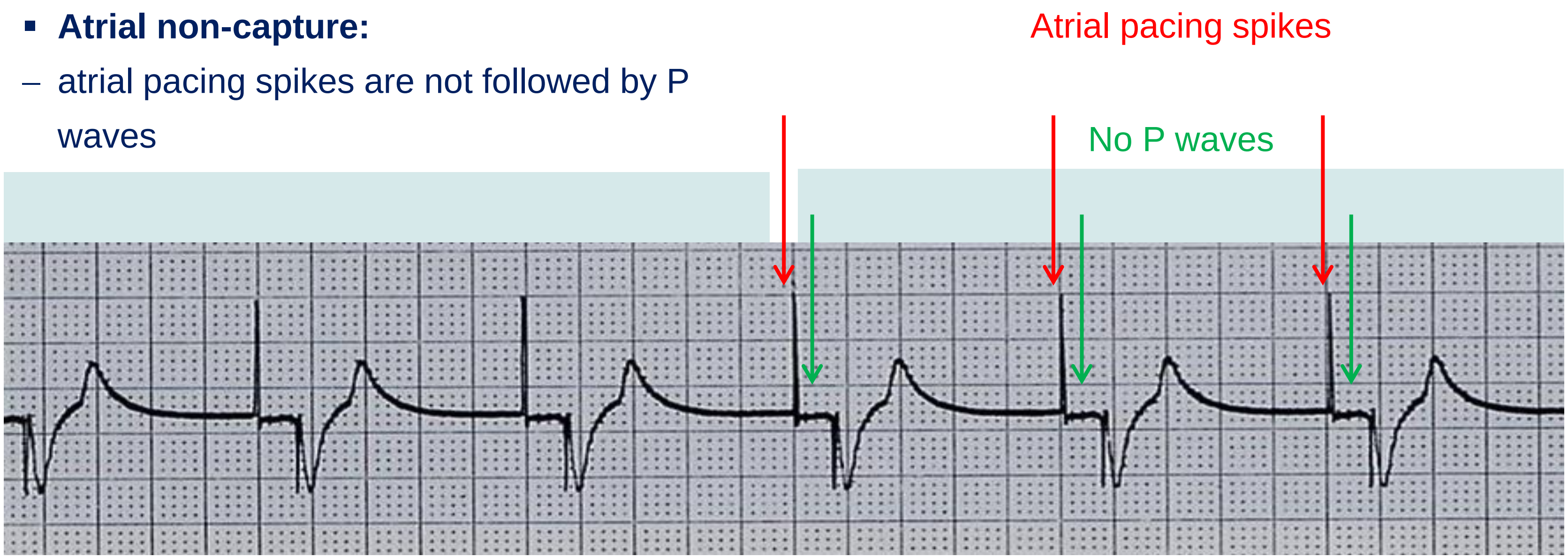
Ventricular pacing spikes

P waves



Abnormal pacing

- **Atrial non-capture:**
  – atrial pacing spikes are not followed by P waves

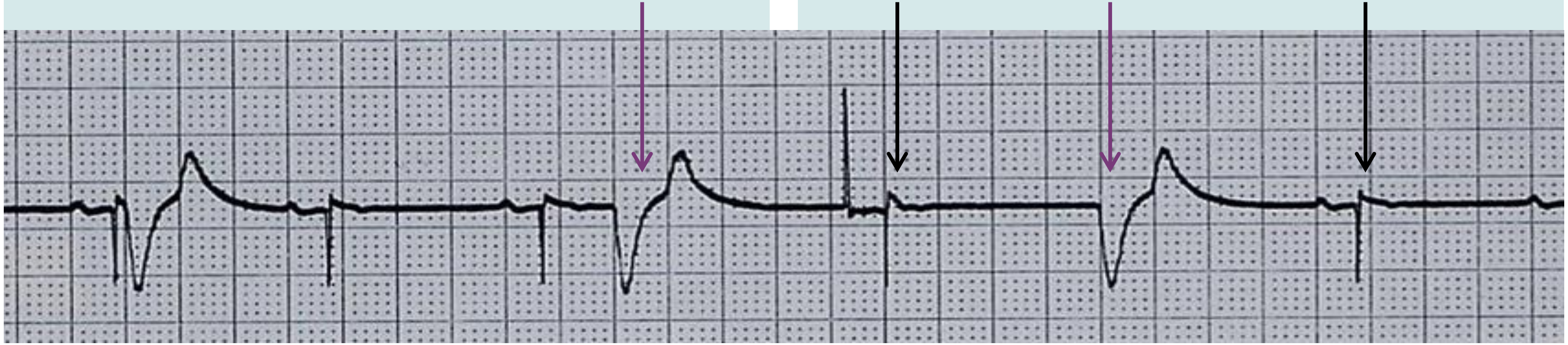


Abnormal pacing

- **Ventricular non-capture:**
  – ventricular pacing spikes are not followed by QRS complexes

Ventricular pacing spikes

QRS complexes



Failure to capture

- **Causes:**
  - insufficient energy delivered by pacer
  - low pacemaker battery
  - dislodged, loose, fibrotic, or fractured electrode
  - electrolyte abnormalities
    - acidosis
    - hypoxemia
    - hypokalemia

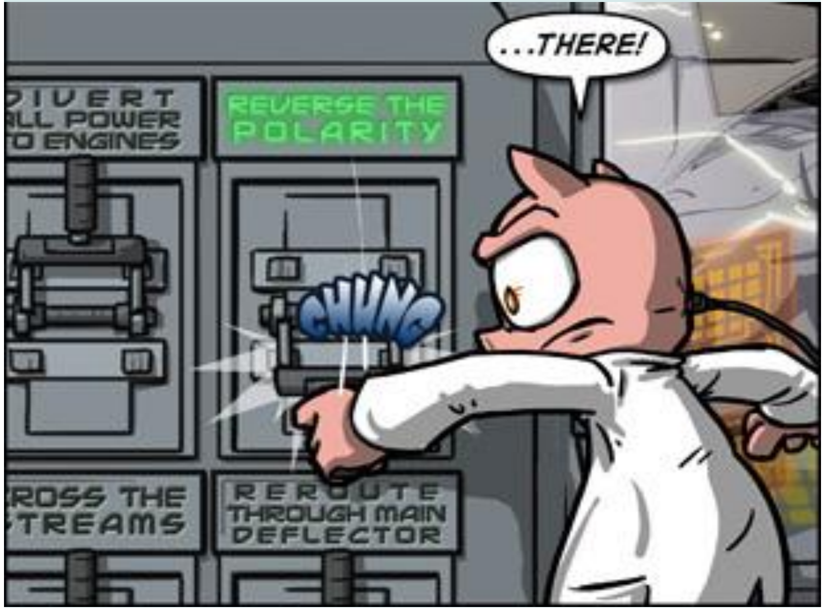
- **Danger:**
  - poor cardiac output

Failure to capture

- **Solutions:**
  - view rhythm in different leads
  - change electrodes
  - check connections
  - **increase pacer output (↑mA)**
  - change battery, cables, pacer
  - reverse polarity

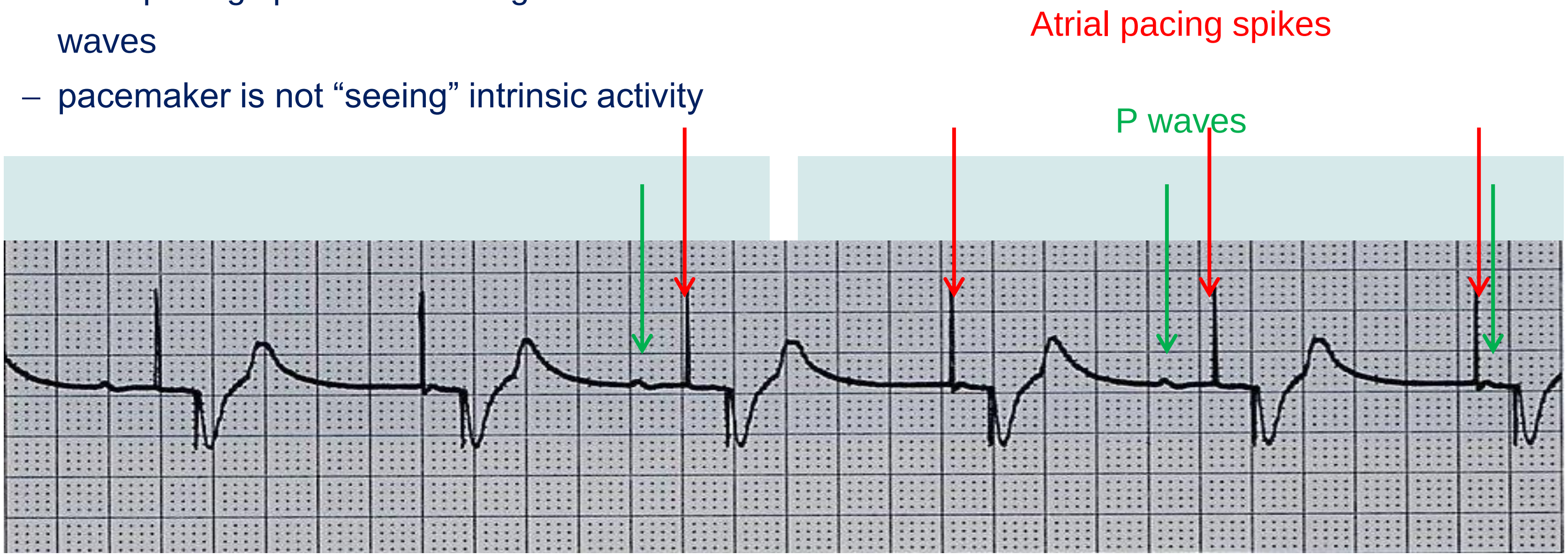
Reversing polarity

- **Changing polarity:**
  - Requires bipolar wiring system
  - Reverses current flow
  - Switch wires at pacing wire/bridging cable interface
  - Skin “ground” wire



Abnormal pacing

- **Atrial undersensing:**
  - atrial pacing spikes occur irregardless of P waves
  - pacemaker is not “seeing” intrinsic activity

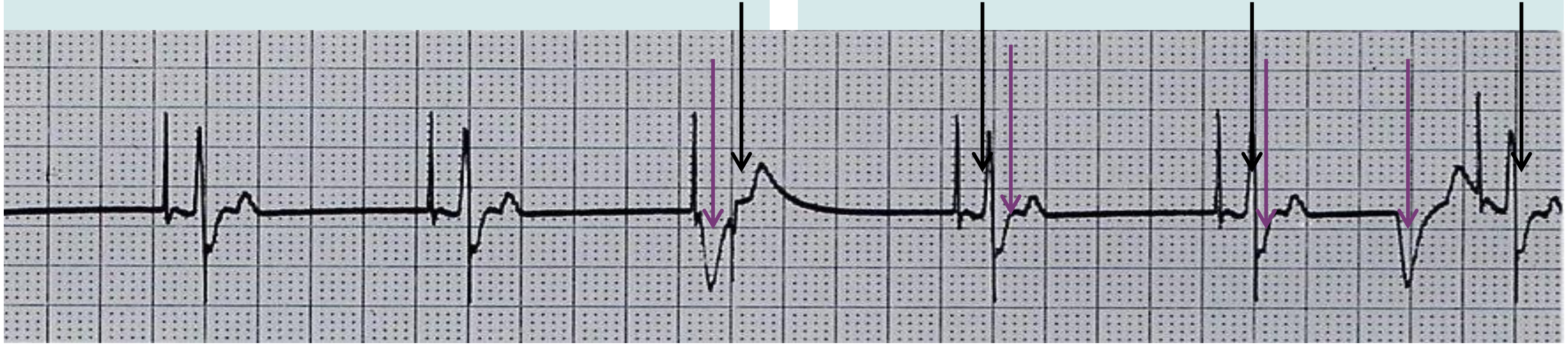


Abnormal pacing

- **Ventricular undersensing:**
  - ventricular pacing spikes occur regardless of QRS complexes
  - pacemaker is not “seeing” intrinsic activity

Ventricular pacing spikes

QRS complexes



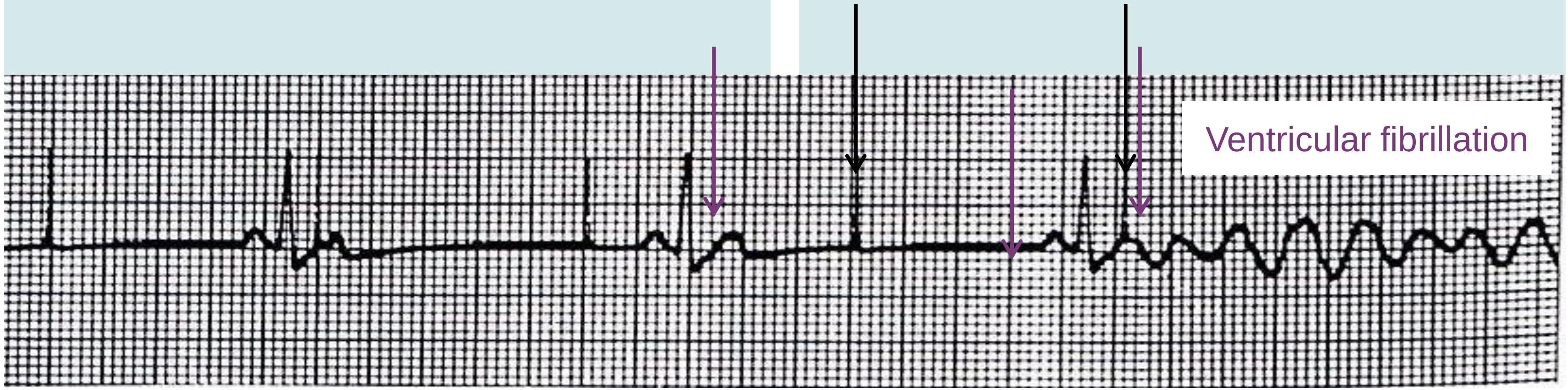
Failure to sense

- **Causes:**
  - pacemaker not sensitive enough to patient’s intrinsic electrical activity (mV)
  - insufficient myocardial voltage
  - dislodged, loose, fibrotic, or fractured electrode
  - electrolyte abnormalities
  - low battery
  - malfunction of pacemaker or bridging cable

Failure to sense

- **Danger:**
  - potential (low) for paced ventricular beat to land on T wave

  R- on -T phenomenon

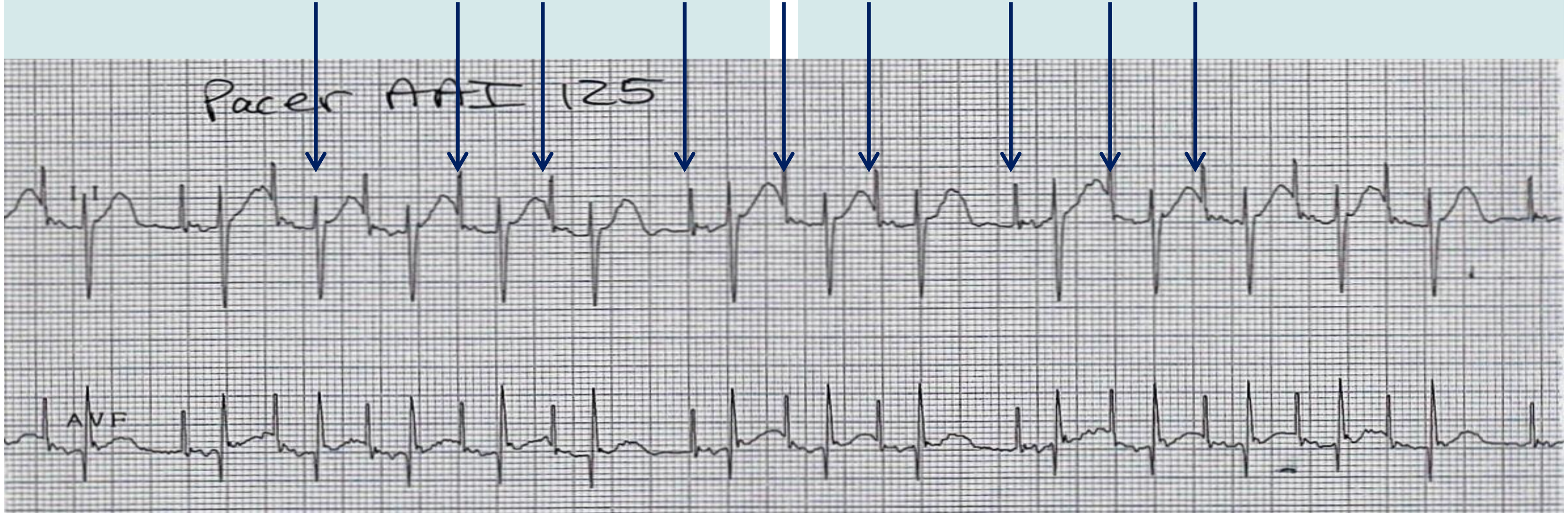


Failure to sense

- **Solution:**
  - view rhythm in different leads
  - change electrodes
  - check connections
  - **increase pacemaker’s sensitivity (↓mV)**
  - change cables, battery, pacemaker
  - reverse polarity
  - check electrolytes
  - unipolar pacing with subcutaneous “ground wire”

Oversensing

- Pacing does not occur when intrinsic rhythm is inadequate



Oversensing

- **Causes:**
  - pacemaker inhibited due to sensing of P - waves + QRS complexes that do not exist
  - pacemaker too sensitive
  - possible wire fracture, loose contact
  - pacemaker failure

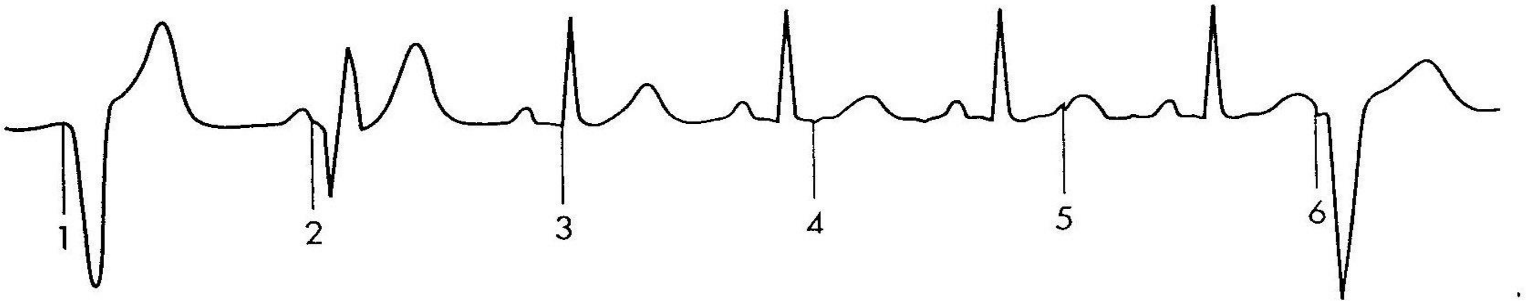
- **Danger:**
  - heart block, asystole

Oversensing

- **Solution:**
  - view rhythm in different leads
  - change electrodes
  - check connections
  - **decrease pacemaker sensitivity (↑mV)**
  - change cables, battery, pacemaker
  - reverse polarity
  - check electrolytes
  - unipolar pacing with subcutaneous “ground wire”

Competition

- **Assessment:**
  - pacemaker + patient's intrinsic rate are similar
  - unrelated pacer spikes to P wave, QRS complex
  - fusion beats



Competition

- **Causes:**
  - asynchronous pacing
  - failure to sense
  - mechanical failure: wires, bridging cables, pacemaker
  - loose connections

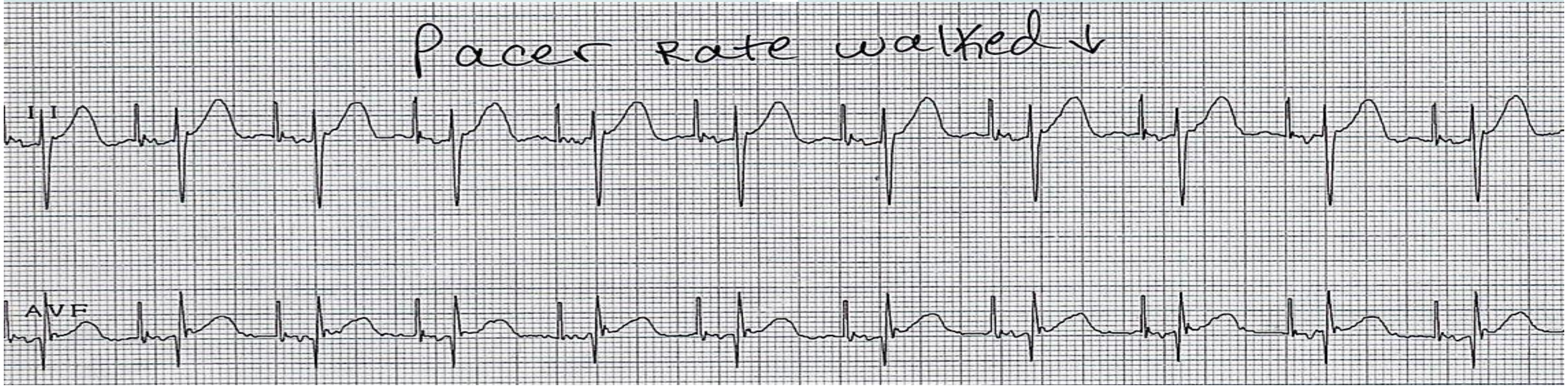
- **Danger:**
  - impaired cardiac output
  - potential (low) for paced ventricular beat to land on T wave

Competition

- **Solution:**
  - Assess underlying rhythm
  - slowly turn pacer rate down
  - troubleshoot as for failure to sense
  - **increase pacemaker sensitivity (↓mV)**
  - increase pacemaker rate

Assessing underlying rhythm

- Carefully assess underlying rhythm:
– right way: slowly decrease pacemaker rate

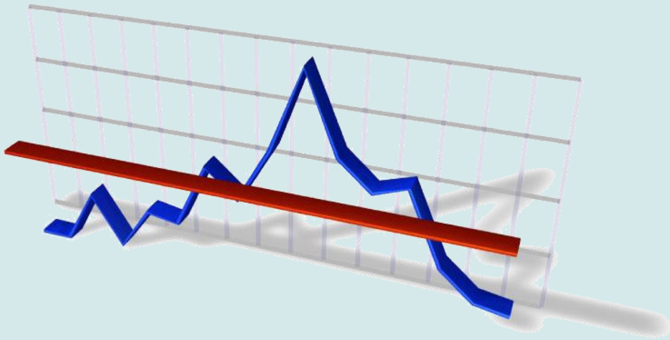


Stimulation threshold

- **Definition:**
  - minimum current necessary to capture + stimulate the heart

Stimulation threshold

- **Testing:**
  - set pacer rate 10 ppm faster than patient’s HR
  - decrease mA until capture is lost
  - increase output until capture is regained (threshold capture)
  - output setting to be 2x’s threshold capture



Sensitivity threshold

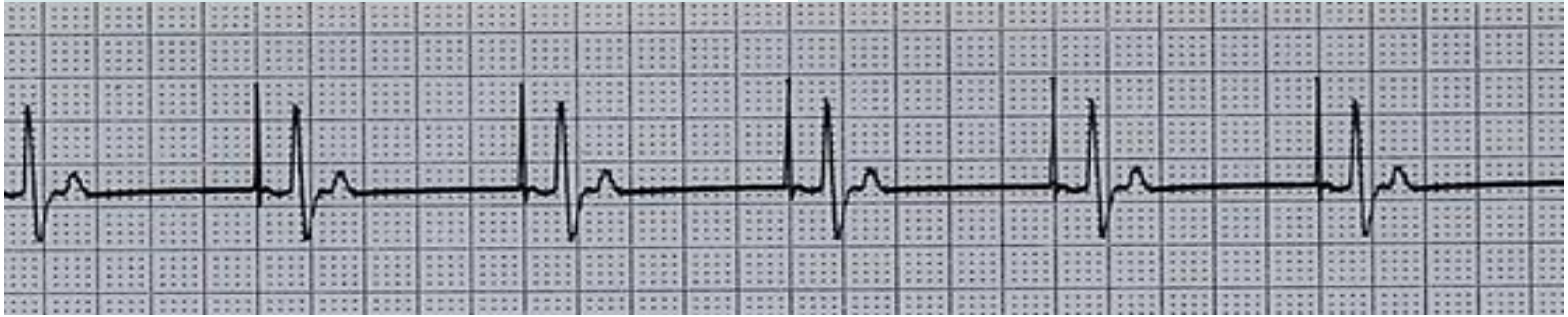
- **Definition:**
  - minimum level of intrinsic electric activity generated by the heart detectable by the pacemaker

Sensitivity threshold

- **Testing:**
  - set pacer rate 10 ppm slower than patient's HR
  - increase sensitivity to chamber being tested to minimum level (0.4mV)
  - decrease sensitivity of the pacer (↑mV) to the chamber being tested until pacer stops sensing patient (orange light stops flashing)
  - increase sensitivity of the pacer (↓mV) until the pacer senses the patient (orange light begins flashing).
  - this is the threshold for sensitivity
  - set the sensitivity at ½ the threshold value
  - example: Set sensitivity at 1mV if the threshold was 2mV

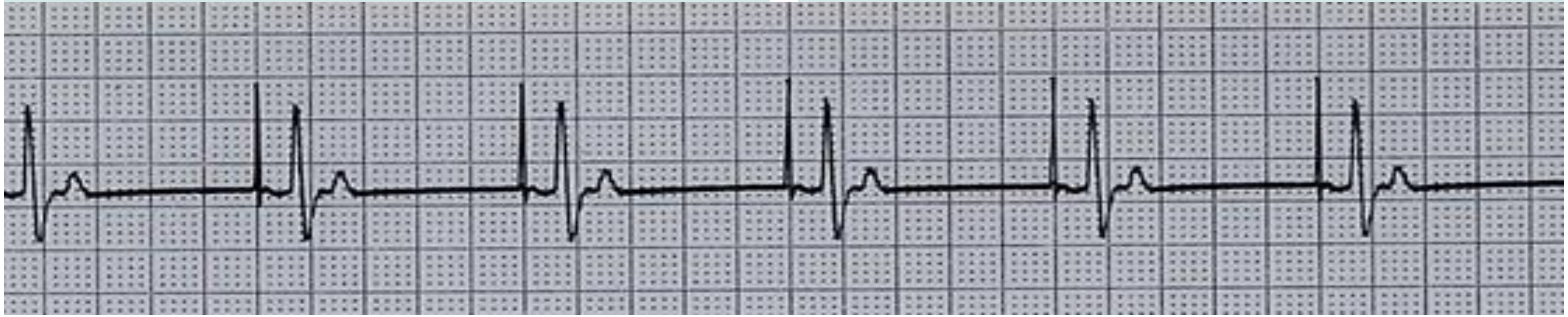
Mode of pacing:
rhythm / problem / solution

What does it show?



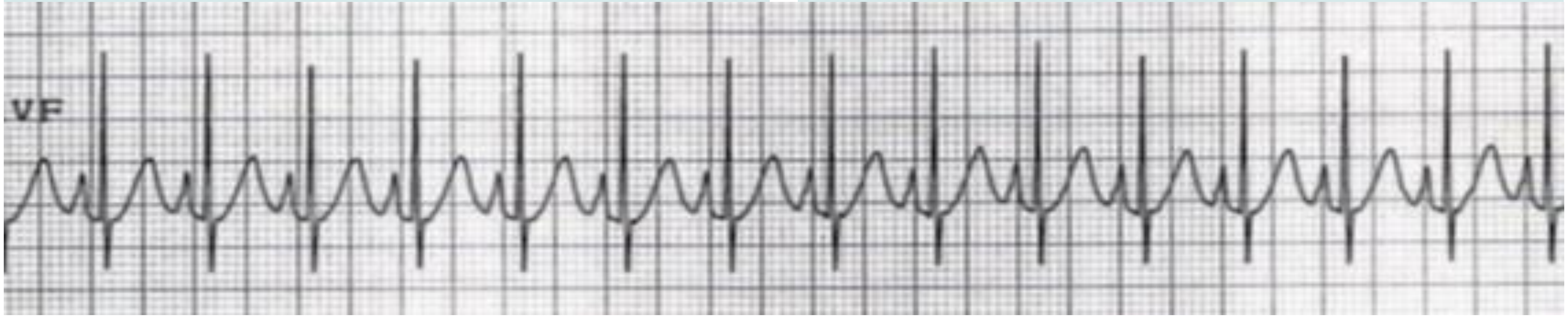
Mode of pacing:
rhythm / problem / solution

- **AAI**
  – normal atrial pacing



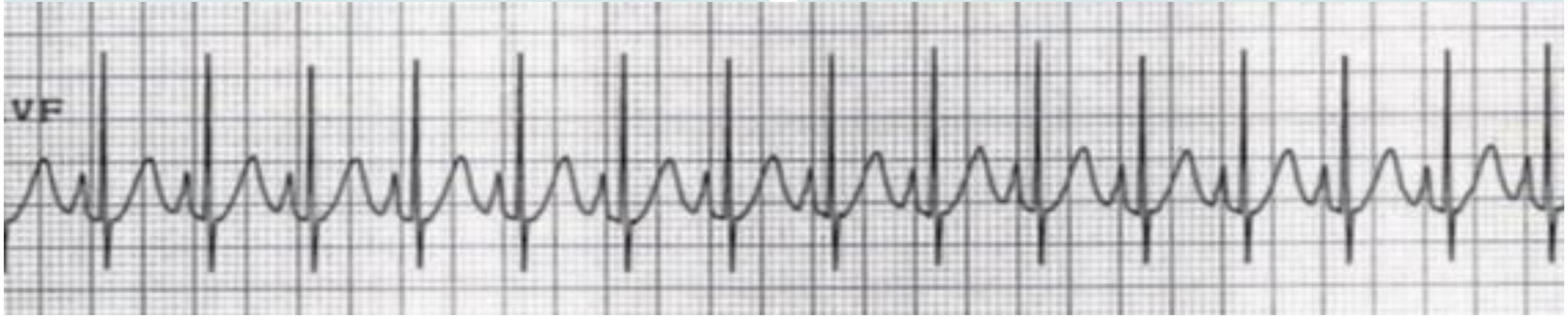
Mode of pacing:
rhythm / problem / solution

What does it show?



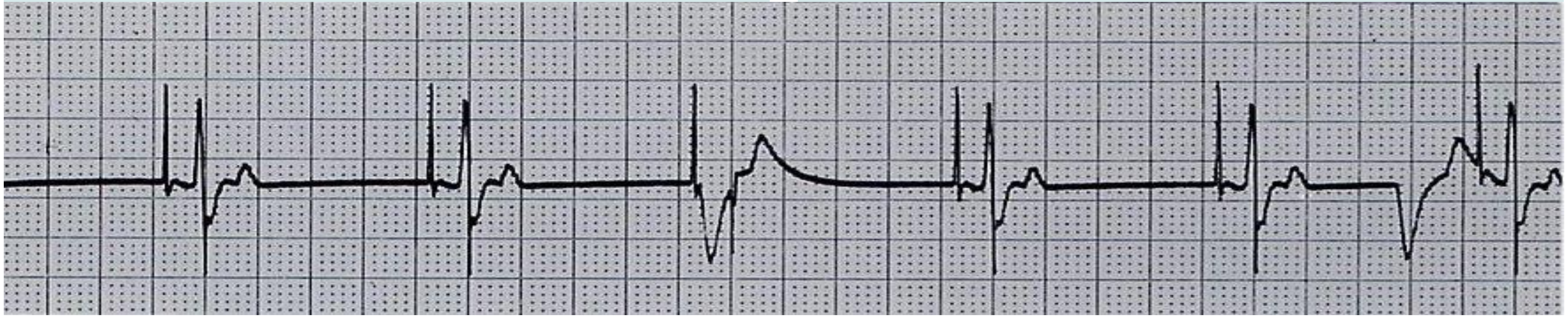
Mode of pacing:
rhythm / problem / solution

- **Sinus rhythm**
  - no pacing
  - possible back-up setting AAI, VVI, DDD



Mode of pacing, rhythm/problem, solution

What does it show?

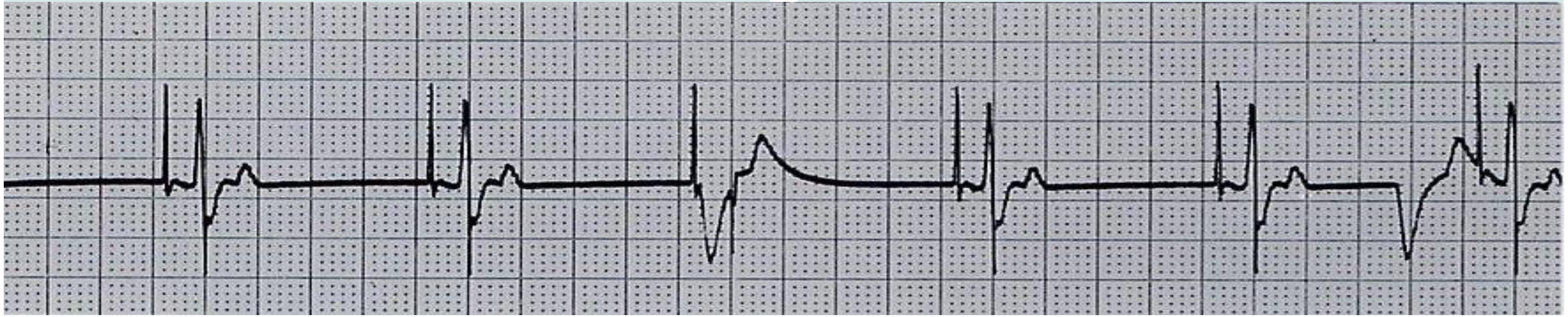


Mode of pacing, rhythm/problem, solution

- **DDD**

– failure to sense ventricle

– increase ventricular mA



Mode of pacing, rhythm/problem, solution

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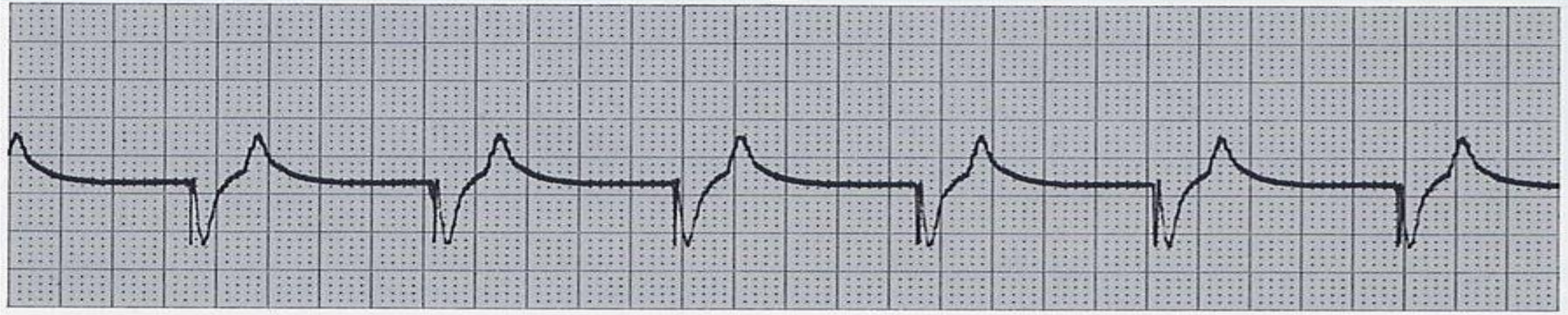
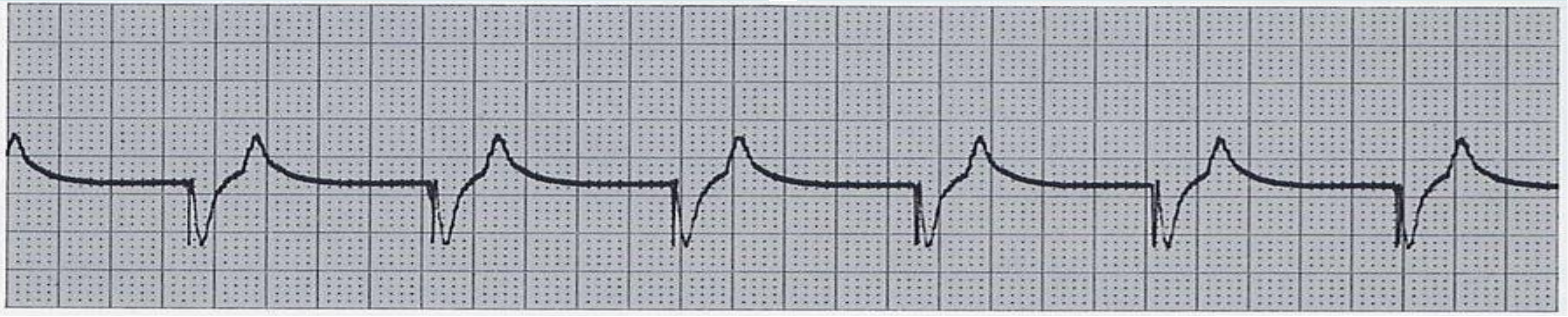


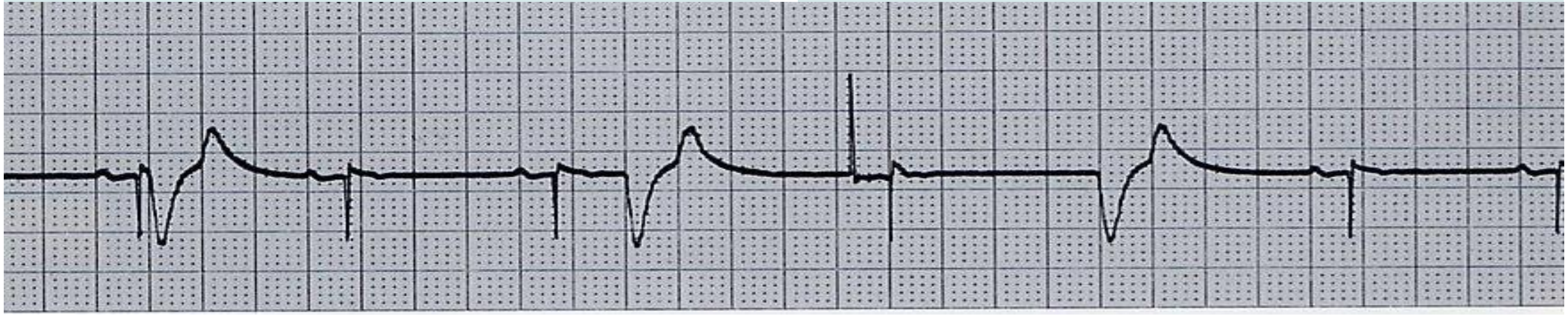
Mode of pacing, rhythm/problem, solution

- **VVI**
  - ventricular pacing



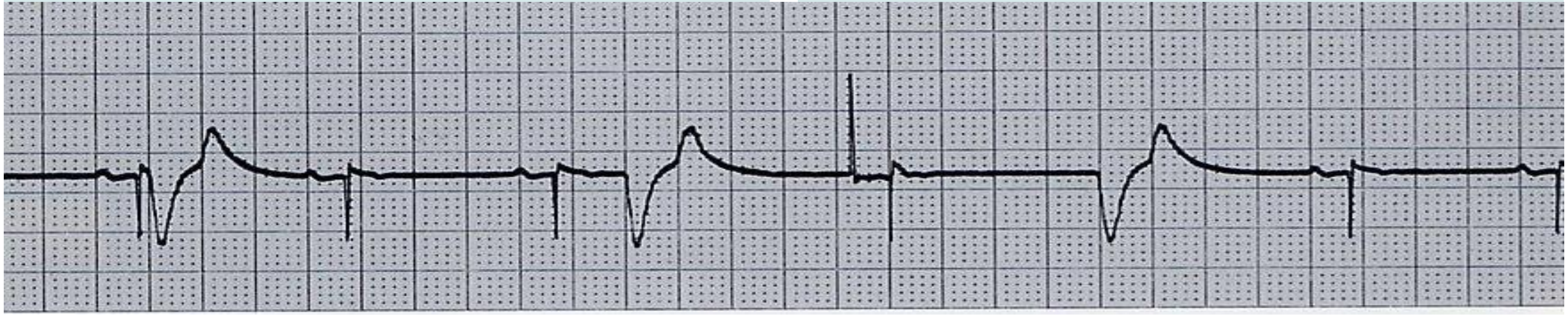
Mode of pacing, rhythm/problem, solution

What does it show?



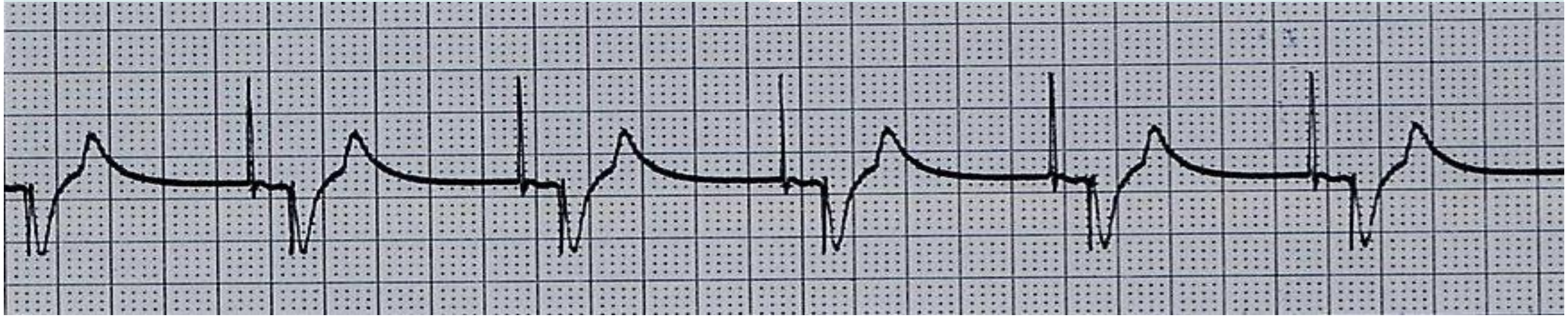
Mode of pacing, rhythm/problem, solution

- **DDD**
- – failure to capture atrial or ventricle
- – increase atrial & ventricular mA



Mode of pacing, rhythm/problem, solution

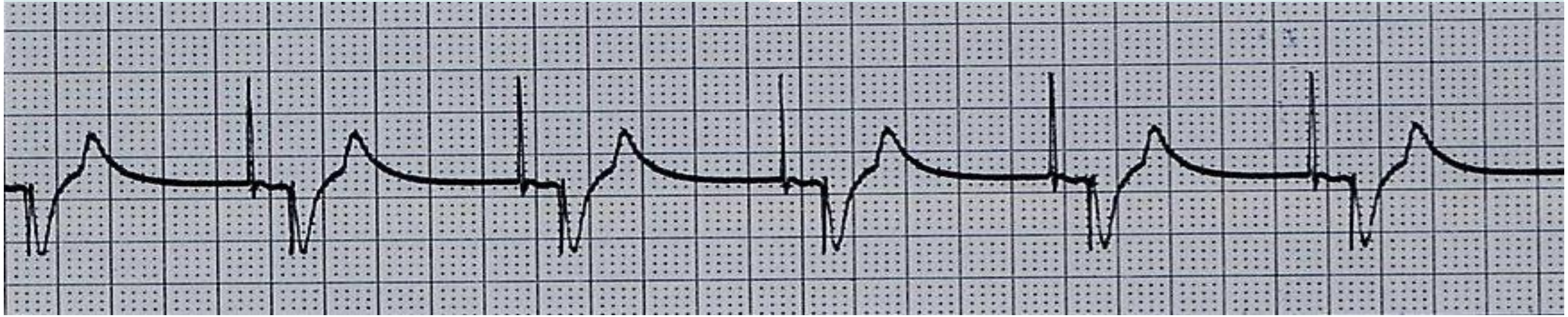
What does it show?



Mode of pacing, rhythm/problem, solution

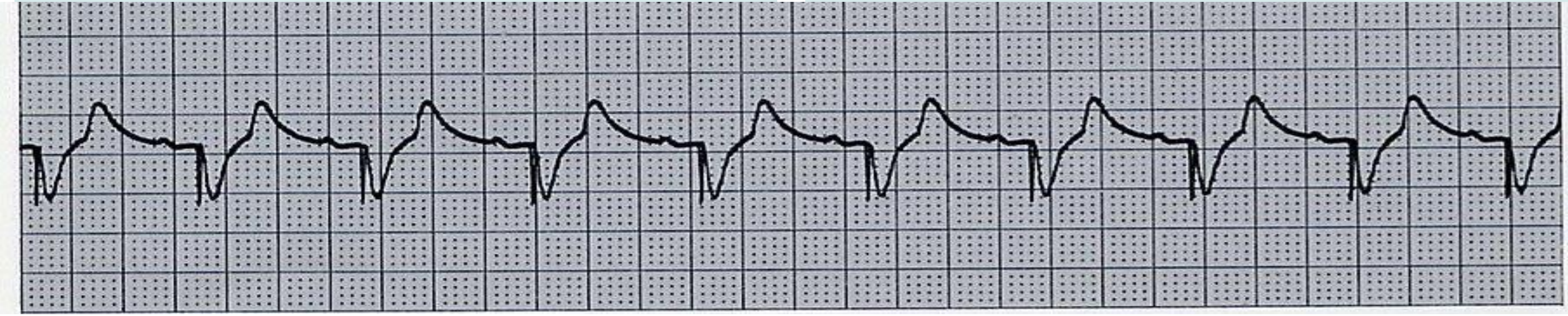
- **DDD**

– normal atrial + ventricular pacing



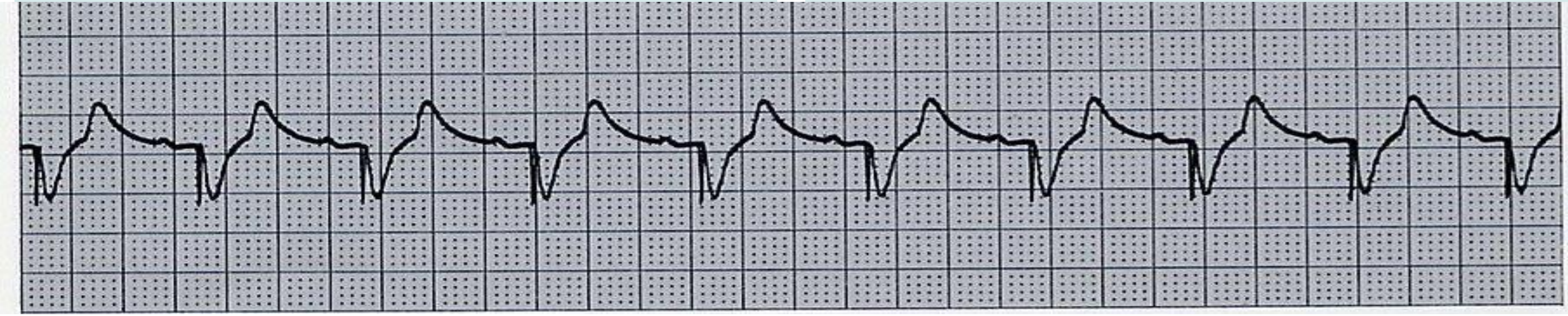
Mode of pacing, rhythm/problem, solution

What does it show?



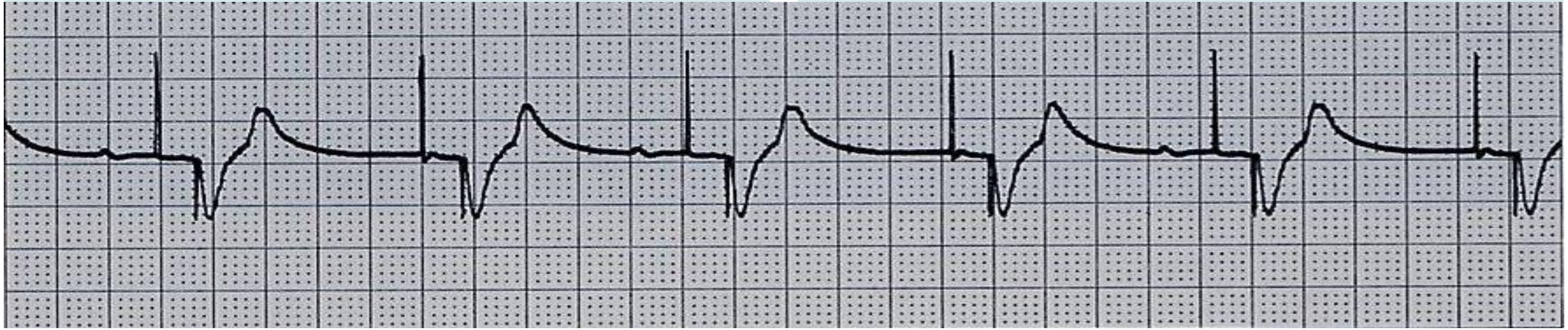
Mode of pacing, rhythm/problem, solution

- **DDD**
– normal atrial sensing
– ventricular pacing



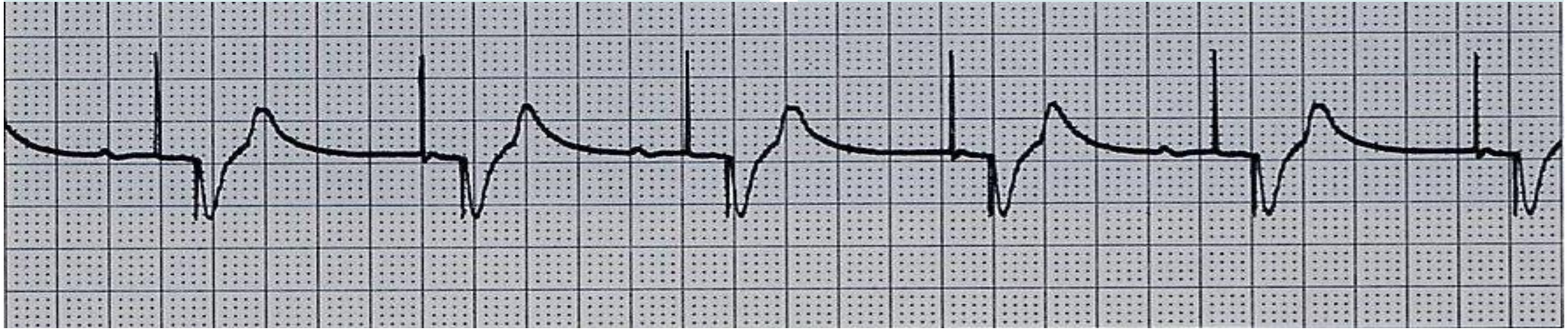
Mode of pacing, rhythm/problem, solution

What does it show?



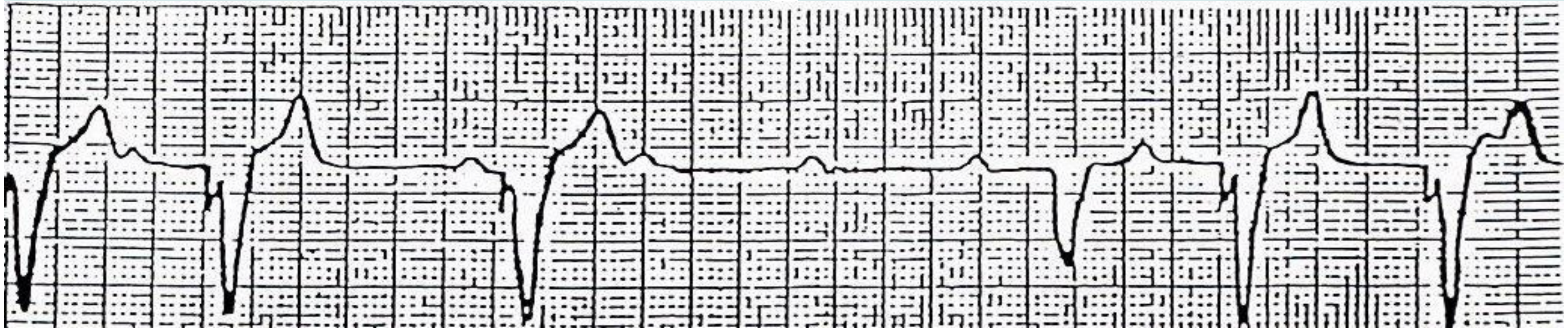
Mode of pacing, rhythm/problem, solution

- **DDD**
– failure to capture atria
– increase atrial mA



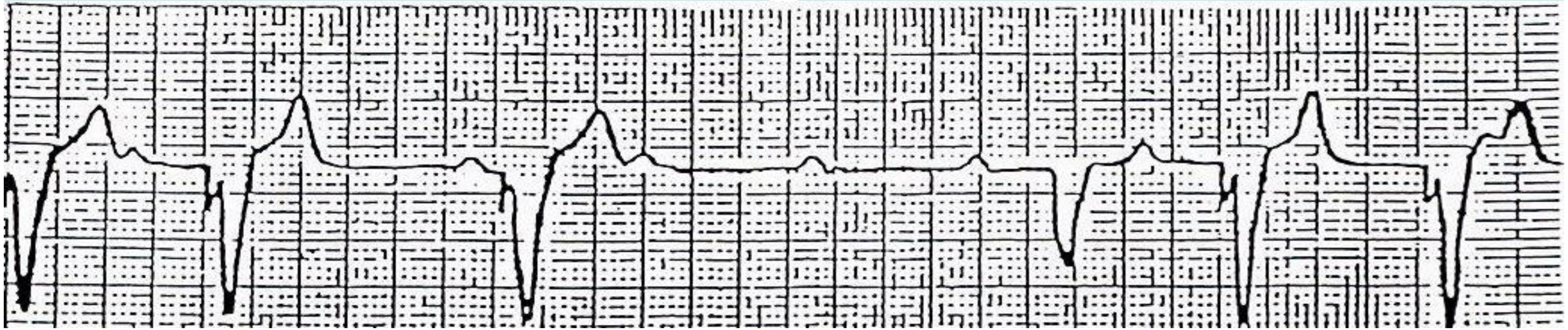
Mode of pacing, rhythm/problem, solution

What does it show?



Mode of pacing, rhythm/problem, solution

- **DDD**
  - Oversensing
  - decrease ventricular sensitivity



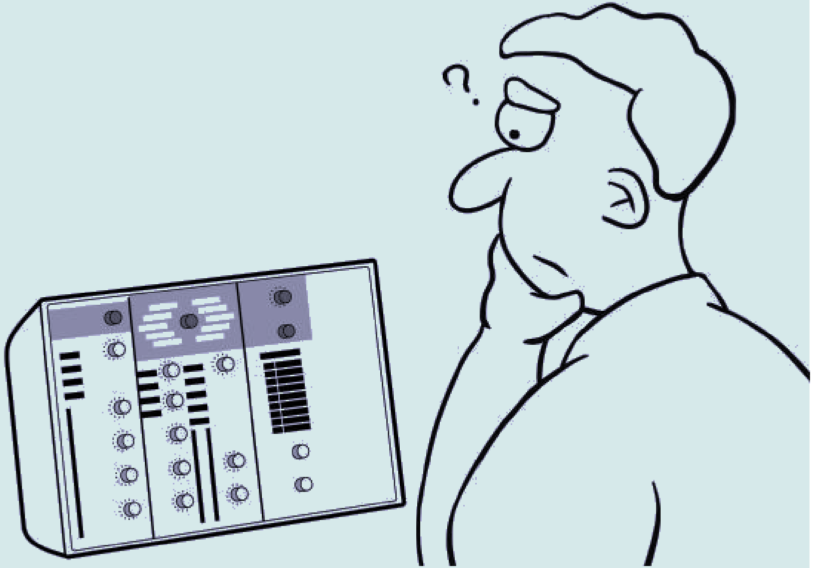
Near to the end

Thank you for your attention

Let‘s take a break

Let‘s continue with the next topic

Principles of mechanical ventilation in postoperative paediatric cardiac critical care



Small remark at the beginning

Lots of monitors and knobs to turn.

Small remark at the beginning

Some are more complicated than others.

Introduction

- Ventilating a child or newborn in the postoperative course after cardiac surgery requires a solid **basic understanding** of **respiratory system mechanics** and **cardiopulmonary physiology**.

- Furthermore, careful attention has to be paid to **avoid damaging the lungs** by potentially injurious mechanical ventilation.

- **Optimizing ventilator settings** during controlled and assisted ventilation, allowing **as early as possible** for **spontaneous ventilation** by still assisting mechanically the patient's respiratory efforts are important features for **lung protection**, for **minimizing potential hemodynamic side effects** of positive pressure ventilation, and for **early weaning** from mechanical ventilation.

Overview

- Anatomical and physiological aspects
- Modes of mechanical ventilation
- Cardiopulmonary interactions
- Lesion specific approaches
- Approach to extubation

Overview

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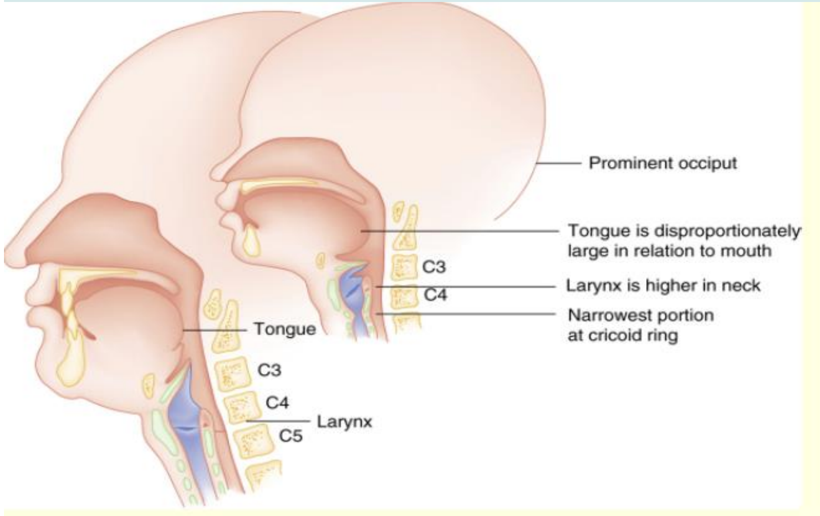
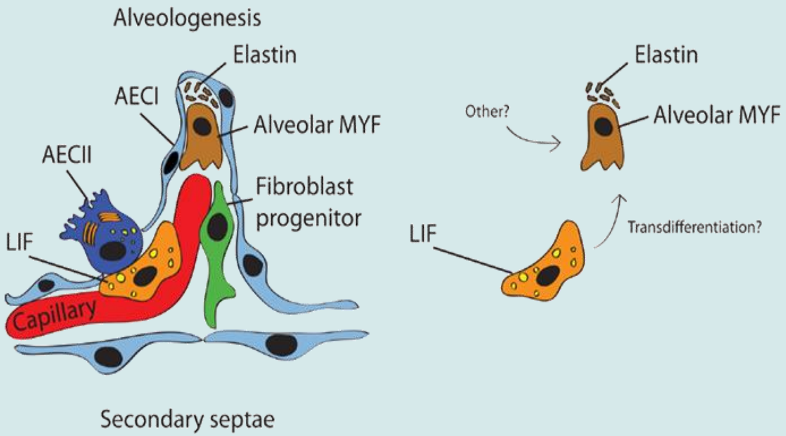
Infant versus adult airways

- Anterior and cephalad
- Floppy U-shaped epiglottis
- Subglottic area narrowest
- **Increased resistance**
- **Compliant chest wall**
- Increased WOB (work of breathing)
- **Increased VO_2 (8ml/kg/min)**



Developmental anatomy

- Lung and chest wall development (2 - 8 yrs)
- The process of alveolization continues beyond the infant age
- 20 - 50 million alveoli at birth in a term infant
- 300 million by the age of 8 years
- The increase in alveoli parallels the increase in **alveolar surface area**



Lung elastic recoil

- The lung matrix of a neonate contains only small amounts of collagen
- The elastin-to collagen ratio changes during the **first months and years of life** and affects **lung stiffness** and potential for overdistension and recoil

- **Lung recoil increase** in children over 6 years of age (more elastin)

Compliance of the lung and chest wall

- During the first year of life, the **compliance of the respiratory system increases** by as much as 150% (mainly lung)

- The **infant chest wall is remarkably compliant** and compliance decreases with increasing age.
- The **elastic recoil of an infant's chest wall is close to zero** and with age increases because of the progressive ossification of the rib cage and increased intercostal muscle tone

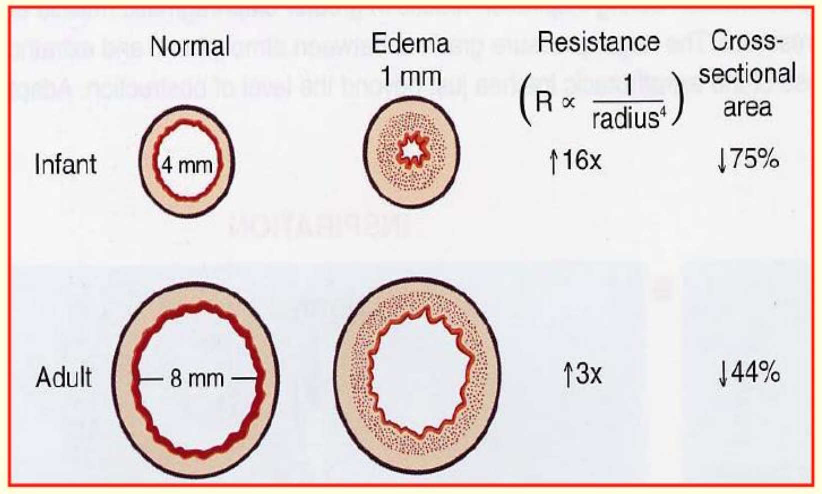
Clinical implications

- Decreased recoil of the infant chest wall increases **the possibility of lung collapse** in the setting of lung disease
- The excessive compliance of the infant chest wall requires the infant to perform more work than an adult chest to move a similar tidal volume

- During respiratory distress, a significant portion of the **energy** generated from diaphragmatic contraction **is wasted through the distortion of the highly compliant rib cage**
- Diaphragms could fatigue and infants could become apneic

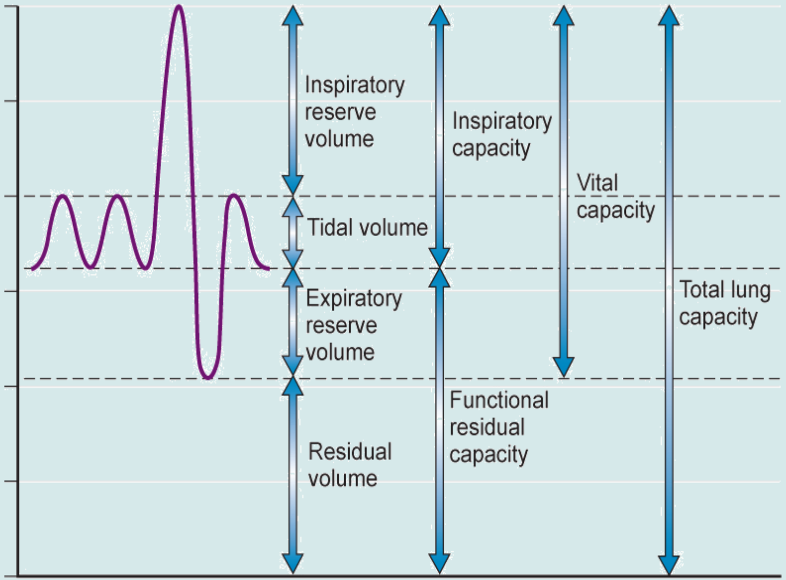
Resistance

- The main site of airway resistance in the adult is the upper airway
- It has been shown **that peripheral airway resistance in children younger than 5 years** of age **is four times higher** than adults
- The major site of resistance is the **medium-sized bronchi**



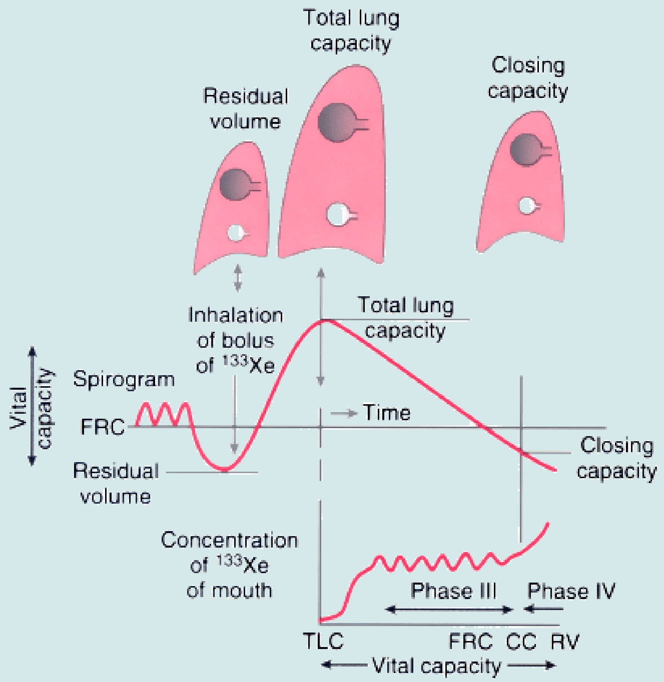
Lung volume

- Functional residual capacity (FRC) is determined by the static balance between the outward recoil of the chest wall and the inward recoil of the lung
- In infants, the outward recoil is quite small
- This leads to a **low ratio of FRC to TLC** (low reserve)



Closing capacity

- Definition: The volume of gas that remains in the lung when small alveoli and airways in dependent regions of the lung are collapsed or considered closed.
- The closing capacity is composed of residual volume and closing volume
- **Children** younger than 6 years of age have a **closing capacity greater than FRC** in the supine position (V/Q mismatch)



Developmental of the pulmonary circulation

- Development of the lungs and development of the pulmonary vasculature are closely related
- The arterial tree undergoes **complex remodeling** in the peripheral portions of the pulmonary circulation **following changes in wall stress**

- Muscularization of the pulmonary vasculature occurs throughout infancy and reaches adult levels by adolescence.
- Pulmonary vascular muscle thickness is a function of gestational age and blood flow

Developmental of the pulmonary circulation

- **Pulmonary Hypertension**

- The **ability of the pulmonary vasculature to constrict is not age dependent**
- Even newborns with only a small amount of arterial muscularization are able to induce significant pulmonary vasoconstriction, as noted in neonates with persistent pulmonary hypertension (PPHN)

Overview

- Anatomical and physiological aspects
- **Modes of mechanical ventilation**
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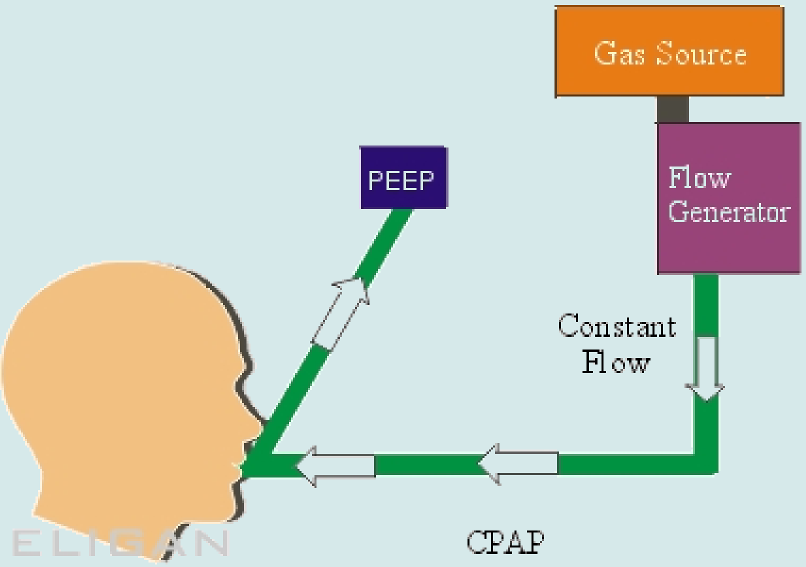
Mechanical ventilation

- **Oxygenation:**
  - determined by inspired oxygen and sufficient mean airway pressure
  - FIO_2
  - PIP, PEEP, I-time, flow
- **Carbon dioxide removal:**
  - determined by (minute ventilation - dead space ventilation) = alveolar ventilation
  - rate, tidal volume

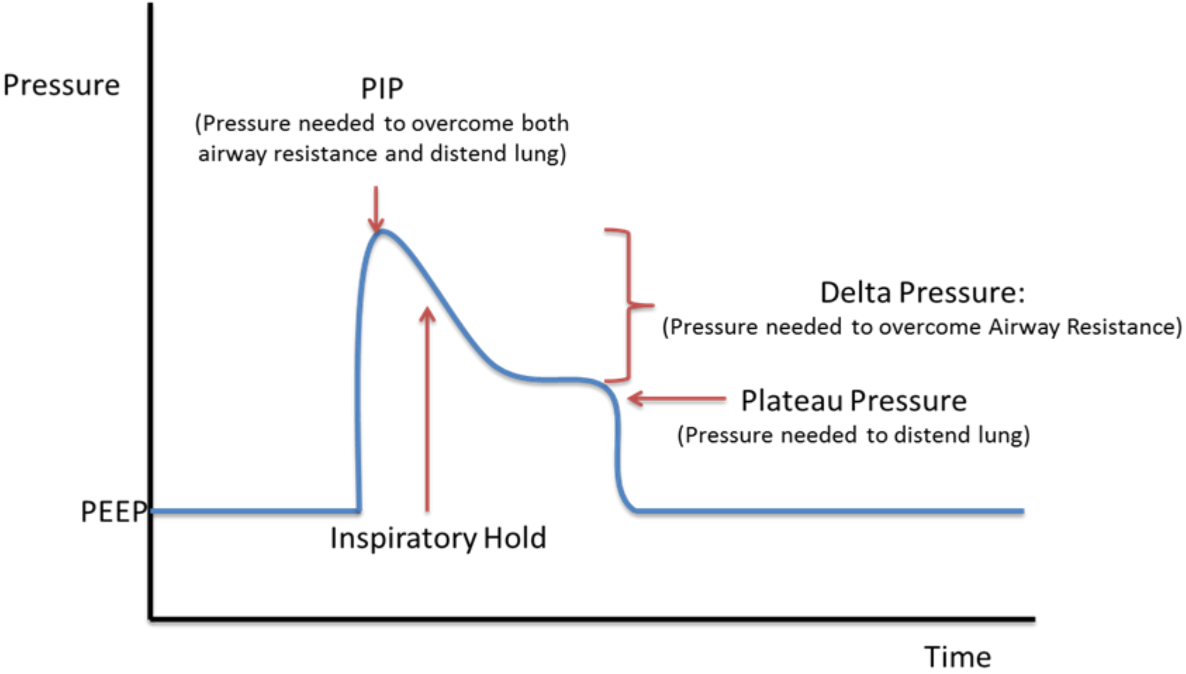


Important terms

- **Time**
  - i - Time: amount of time spent in inspiration
  - e - Time: amount of time spent in expiration
- **Volume**
  - amount of tidal volume that patient receives
- **Pressure**
  - measure of impedance to gas flow rate
- **Flow**
  - measure of rate at which gas is delivered

- **PEEP** = **p**ositive **e**nd **e**xpiratory **p**ressure
  - pressure maintained in the airways at the end of exhalation
  - keeps alveoli from collapsing
- **PIP** = peak inspiratory pressure
  - point of maximal airway pressure
- **Delta P**
  - difference between PIP – PEEP
- **MAP** = mean airway pressure

Important terms



Important terms

- **CMV** = **C**ontrolled **M**echanical **V**entilation
- **SIMV** = **S**ynchronized **I**ntermittent **M**andatory **V**entilation
- **BIVENT** = Biphasic Positive Airway Pressure
- **AC** = **A**ssist **C**ontrol
- **CPAP** = **C**ontinuous **P**ositive **A**irway **P**ressure
- **PRVC** = **P**ressure **R**egulated **V**olume **C**ontrol

Ventilatory support

- **Assisted breaths determined by**

- **Trigger**
  - time, flow, pressure
- **Cycle**
  - time cycled breaths
- **Limit**
  - volume or pressure

Modes of ventilation

- **Volume limited (SIMV):**
  - constant flow during inspiration
  - square flow wave pattern
  - **set parameters: TV, rate, PEEP, i-time**
  - PIP--dependent variable
  - fixed minute ventilation
  - PaCo2 and pH remain stable

- **Pressure limited (A/C):**
  - **set parameter: PIP, PEEP, rate, IT**
  - pressure constant throughout
  - tidal volume dependent variable
  - decelerating flow pattern
  - lung compliance and airway resistance determine gas delivery
  - theoretically **less barotrauma**

Modes of ventilation

- **Pressure support ventilation:**
  - decelerating inspiratory flow
  - patient triggers breath
  - constant pressure delivered
  - better patient-ventilator synchrony
  - used with volume or pressure mode or **weaning mode**

- **Pressure Regulated + Volume Control (PRVC)/APV:**
  - pressure limited
  - tidal volume targeted
  - decelerating flow waveform
  - achieve **TV goals without barotrauma**, mean airway pressure maintained

Initial ventilator settings

- **Neonate < 5.0 kg:**
  - iT 0.4 - 0.7 sec
  - I : E ratio 1:1.5 – 2.0
  - PEEP 5 cm H_20
  - PS + 6 - 8 cm H_20
  - TV 8 - 10 ml/kg
  - rate 25 - 40/min
  - PIP max 20 cm H_20

- **Infant / Child:**
  - IT 0.7 - 1.0 sec
  - I : E ratio 1:2 – 3.0
  - PEEP 5 cm H_20
  - PS + 5 cm H_20
  - TV 10 ml/kg
  - Rate 15 – 40 / 10 - 20
  - PIP max 30 cm H_20

Overview

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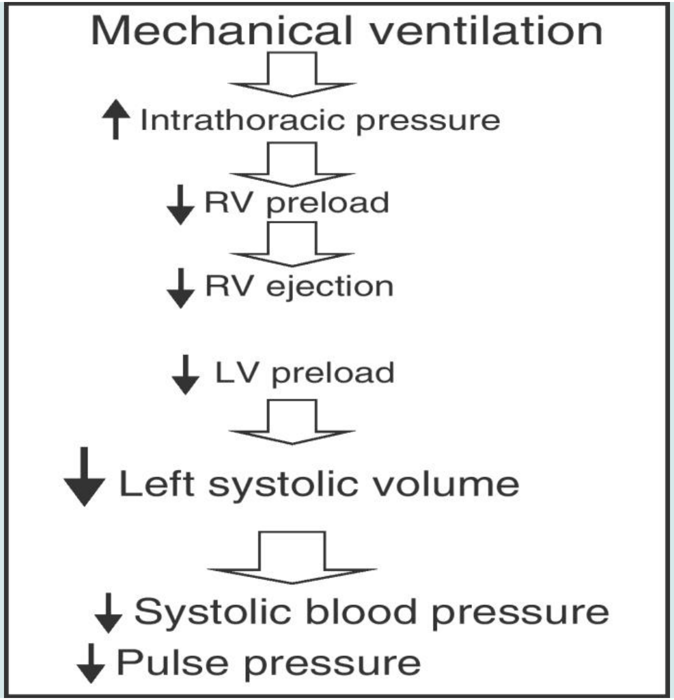
Effects of mechanical ventilation

- Alteration of lung volume
- Changes in ITP (intrathoracic pressure)
- Altered acid-base balance
- Altered PaO_2
- Changes in neurohormonal activity

- **All can affect cardiac function**

Intrathoracic pressure changes

- Venous return affects RV preload
- Pressure gradient between CVP and P_{RA}
- Respiratory induced changes in ITP directly effect P_{RA}



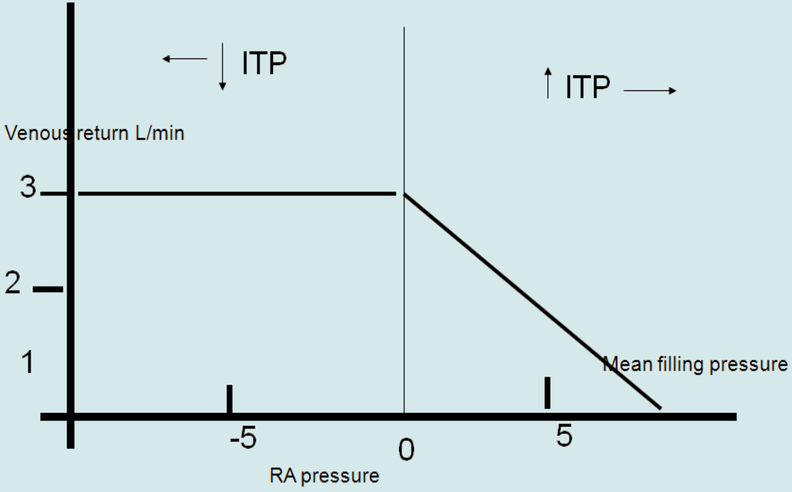
Venous return and RV preload

- **Spontaneous inspiration:**
  - P_{RA} falls, CVP constant, intra-abdominal pressure increases
  - increased pressure gradient increases VR
  - as P_{RA} approaches zero venous return maximized

- **Positive pressure ventilation:**
  - inhibits venous return to RA
  - ITP (+), decreases gradient between P_{RA} and mean CVP, RV filling falls
  - more pronounced in low output state

Effect of PEEP on RV preload

- **RV preload decreased:**
  - increases intrathoracic pressure (ITP)
  - increases intrathoracic volume
  - diaphragm descends
  - increases both CVP and P_{RA}
  - venous return shifts to right



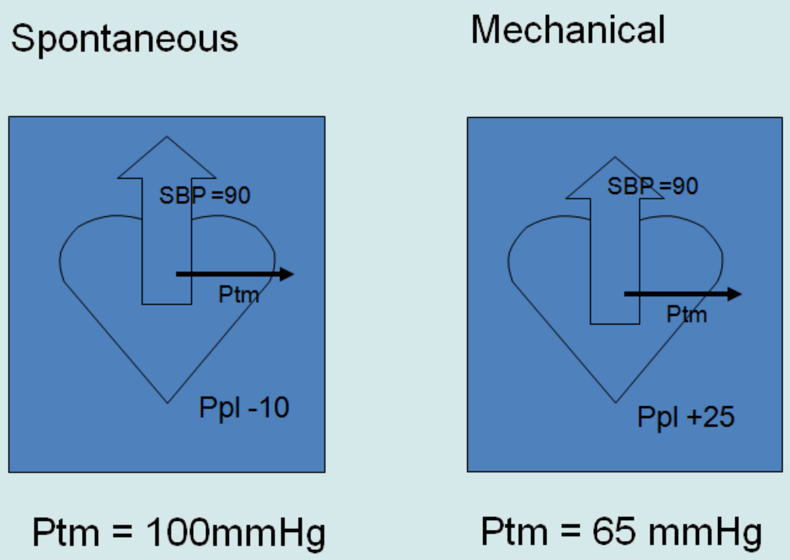
LV preload

- **Spontaneous Inspiration:**
  - RV volume increases
  - intraventricular septum shifts leftward
  - LV compliance and filling fall
  - “Ventricular interdependence”

- **Positive pressure ventilation:**
  - decreased VR--decreased LV filling
  - decreased RV volume--increased LV compliance
  - increased lung volume--restricted LV filling

LV afterload

- **Spontaneous inspiration:**
  – intrathoracic pressure falls and SBP unchanged and afterload increases
- **Positive pressure ventilation:**
  – intrathoracic pressure increases and afterload decreases



LV afterload

- **Spontaneous inspiration:**
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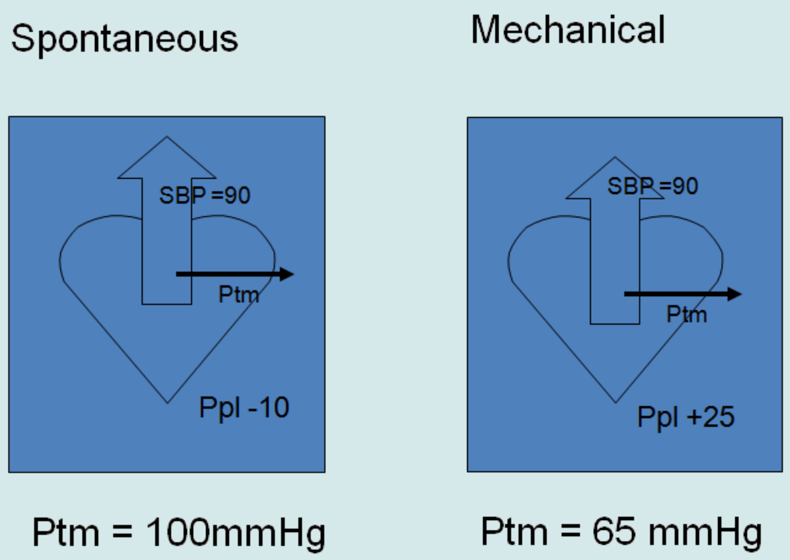
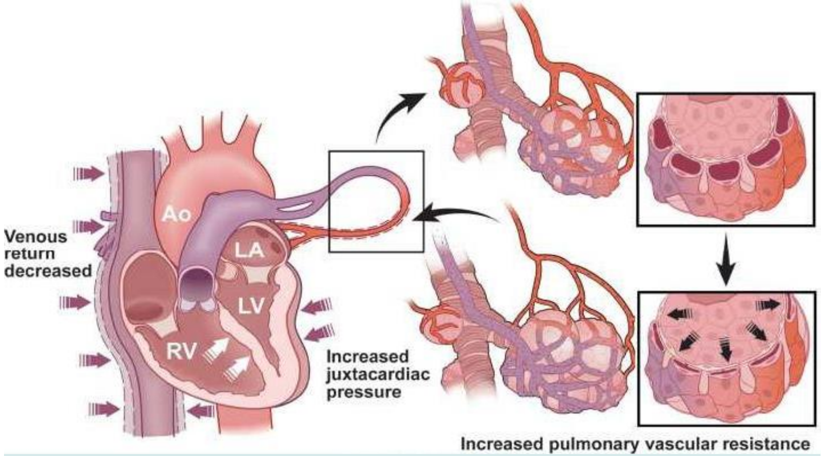


RV Afterload

- Determined by Pulmonary vascular resistance (PVR)
- PVR affected by lung volume via
  - passive compression of pulmonary vessels
  - hypoxic vasoconstriction

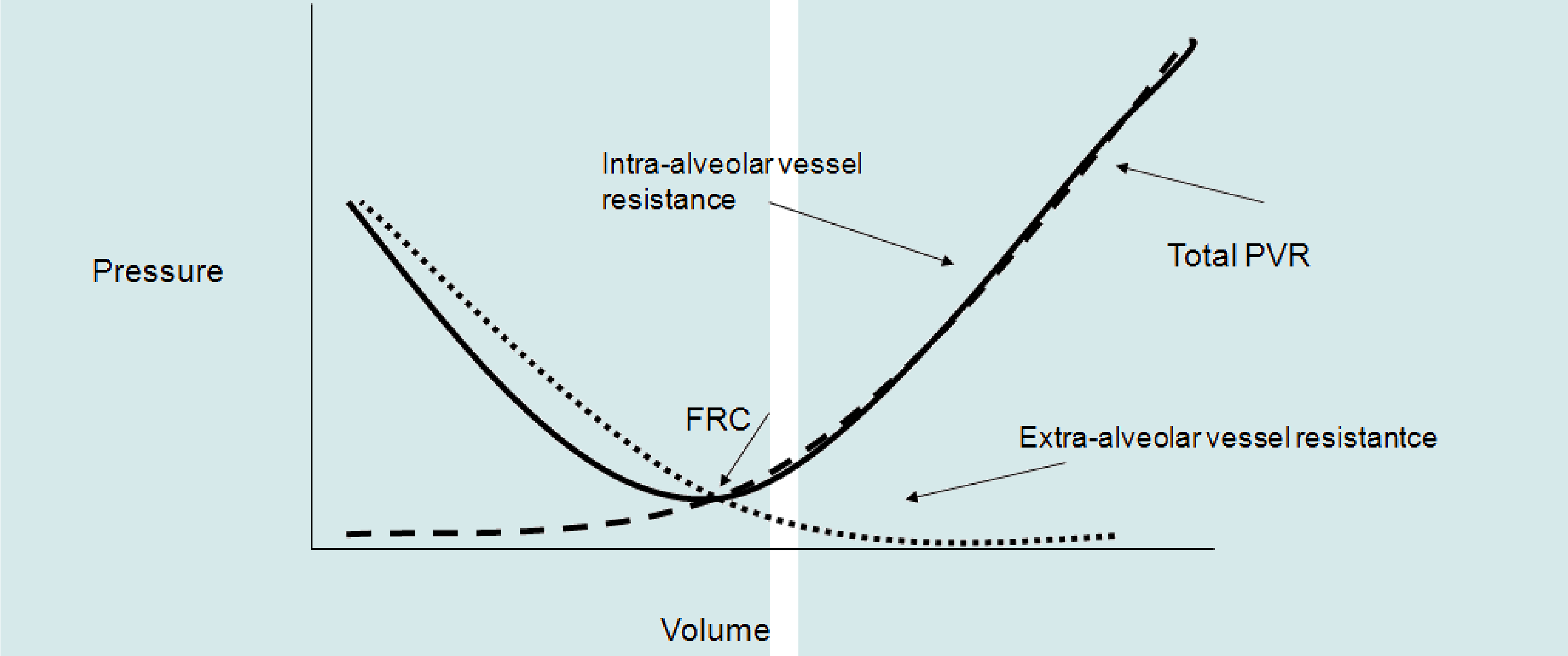


RV Afterload

- **Pulmonary vascular resistance + functional residual capacity:**

- PVR lowest at FRC
- **Below FRC** (atelectasis) = ↑ PVR
  – extra-alveolar vessels collapse
  – terminal airways close
  – alveoli collapse
  – hypoxia
- **Above FRC** (hyperinflation) = ↑ PVR
  – intralveolar vessels compressed

Lung volume and PVR



Mechanical ventilation and PVR

- **Increase PVR:**
  - Acidosis
  - Hypoventilation
  - Hyperinflation
  - overdistension

- **Decrease PVR:**
  - Hyperventilate
  - Alkalosis
  - PEEP
  - FIO_2

Overview

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- **Lesion specific approaches**
- Approach to extubation

Left - to - right shunts

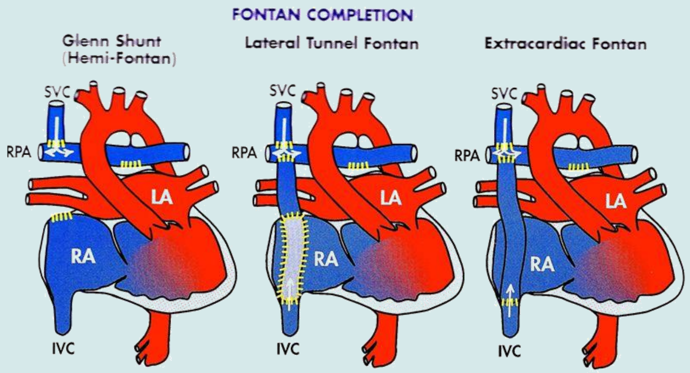
- Increasd PBF
- Compression of large airways can occur due to enlarged LA and Pas
- TOF / PA / MAPCAS:
  - compression of intrapulmonary bronchi by abnormal vessels
  - atelectasis, wheezing, poor gas exchange

- Bronchiolar narrowing from high flows and venous pressure
- Causes pulmonary edema
- Increased PBF associated with decreased $FEV_{25-75\%}$
- Prominent smooth muscle narrowing seen

Single ventricle: Fontan

- Venous return = PBF is “passive”
- Minimize positive pressure ventilation and PEEP
- Spontaneous ventilation ideal
- Early extubation
- Adequately volume load pt
- Slow rate, adequate tidal volume

Fontan procedure



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Extubation

- **Ideal setting for extubation:**
  - hemostasis
  - optimal cardiac output
  - stable hemodynamics
  - appropriate mental status
  - normothermia

1

Extubation

- $FIO2 < 0.4$ with satisfactory PaO_2
- PEEP < 5.0 cm H_2O
- pH > 7.35
- PCO_2 < 45 -50 mmHg
- Spontaneous TV 5 - 7ml/kg on PS < 8
- No significant grunting / flaring / retracting on CPAP or low rate
- Mechanical VT / spontaneous TV < 0.4
- Negative Inspiratory Force -20 - 30 cm H_20

Extubation

- Sinus rhythm or stably paced
- Optimal perfusion and filling pressures
- Adequate cardiac output
- Minimal inotropic support
- Conscious
- No residual paralysis
- Minimal sedative within 2-4 hrs
- Optimal hematocrit

3

Extubation

Reasons why patients fail

- **Pulmonary processes:**
  - effusions, pneumonia, atelectasis
  - diaphragm or vocal cord paralysis
  - airway obstruction, RAD
- **Cardiac reasons:**
  - residual anatomic leasion
  - poor function/low output
- **Neurologic reasons:**
  - oversedation, weakness
  - CNS event--hypoxic ischemic encephalopathy, intracranial hemorrhage

Thank you for your attention