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UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE "CAROL DAVILA" BUCUREŞTI

AD-COR Program inovativ de formare in domeniul cardiologiei pediatrice
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MODUL TEORETIC

PAEDIATRIC CARDIAC CRITICAL CARE

Imputernicit: Prof. Dr. Tammam Youssef

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Acest material a fost documentat/ validat/ prezentat la sesiunile de formare în cadrul proiectului *„AD-COR Program inovativ de formare în domeniul cardiologiei pediatrice”* - POSDRU/179/3.2/S/152012, proiect cofinanțat din Fondul Social Operațional Sectorial Dezvoltarea Resurselor Umane 2007-2013.

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

PAEDIATRIC CARDIAC CRITICAL CARE

Bucharest, October 15, 2015

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RobinAid Foundation
Hamburg / Germany

Declaration of Disclosure

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

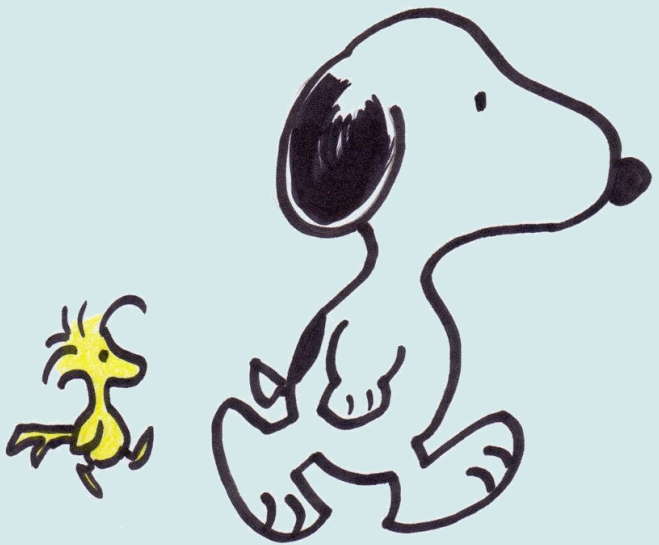
Let‘s continue our walk

Important topics in Paediatric Cardiac Critical Care

Today‘s agenda

- Welcome and short flashback
- Three parts à 60 min
- 15 minutes break after each part

14 important topics



1. *The importance of cardiac output and cardiovascular medications √*
2. Human factors in paediatric cardiac critical care
3. Receiving patient from OT: assessment, preparation and handing-over process
4. Invasive and non-invasive methods of monitoring in the PCICU
5. Respiratory assessment and management; respiratory complications
6. Importance of pain management in paediatric cardiac critical care
7. Fluid management and treatment of metabolic disorders

14 important topics

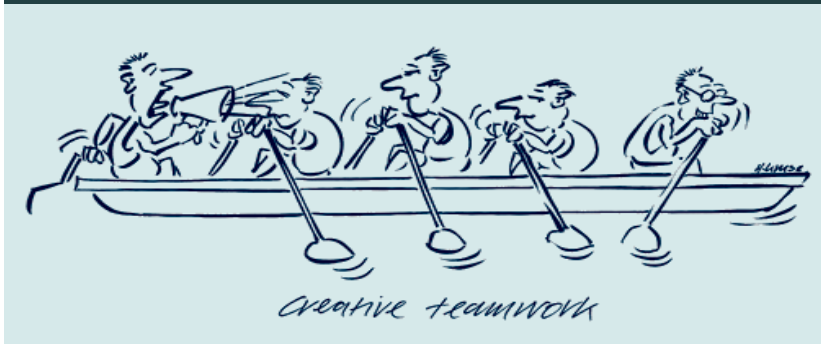
8. Fast-track extubation in pediatric cardiac surgery
9. Resuscitation: management of cardiac arrest
10. Medistinal bleeding and cardiac tamponade blood transfusion, coagulation disorders, and postoperative anticoagulation
11. Infections in the cardiac intensive care unit
12. Cardiac arrhythmias; care of patients with temporary pacemaker;
13. Renal failure: prevention, identification, management, and replacement therapy
14. Neurological complications, gastrointestinal complications, and nutrition

First important message

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.

Paediatric Cardiac TEAM

- Intensivist
- Cardiologist
- Neonatologist
- Anaesthetist
- Surgeon
- Nurses
- Physiotherapist
- Technicians
- Perfusionist
- Radiologist



Second important message

The care of critically ill children after cardiac surgery is challenging and best performed with the emphasis on an **anticipatory** (rather than reactive) **approach**.

In other words

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.

Now let‘s start with the todays‘s topics

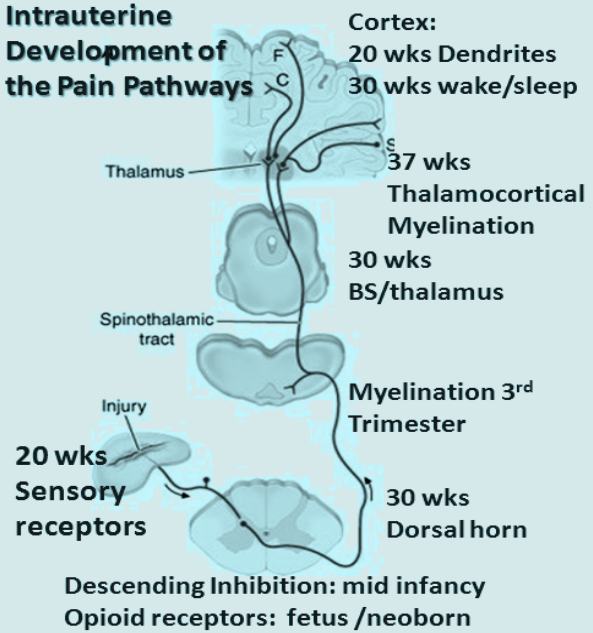
Importance of pain management in paediatric cardiac critical care

Pain management myths

- Neonates do not feel pain
- Infants are less sensitive to pain than adults
- Neonates have no memory of pain
- Children will tell you when they are having pain
- If a child can be distracted, he is not in pain
- Neonates are not able to tolerate the effects of analgesics
- Narcotics can lead to addiction in children
- Infants become accustomed to pain

What are the facts?

- Newborn infants have functional nervous systems which are capable of perceiving pain
- Physiologic means of assessing pain (VS) can be an unreliable predictor of pain
- Infants often develop an increase in signs of discomfort with repeated painful procedures
- Premature infants can have unpredictable responses to painful stimuli
- Unmanaged pain in the neonatal period can cause long term developmental complications

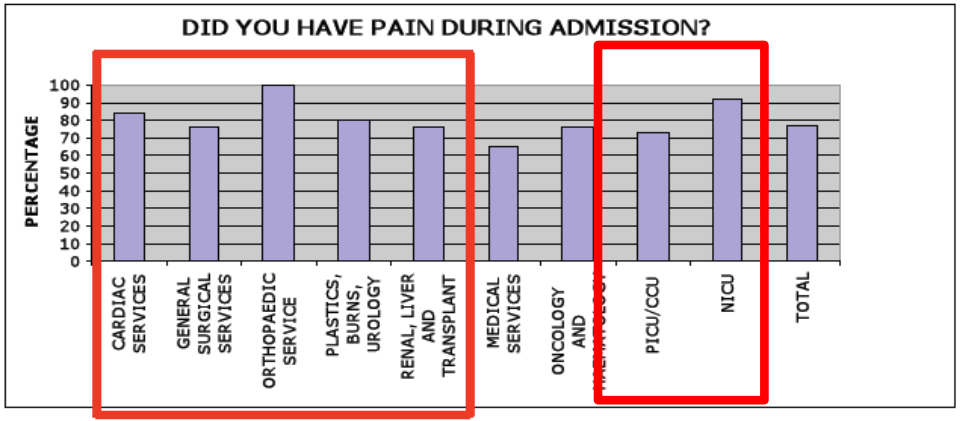


Facing the reality

SickKids Inpatient Pain Audit

Taylor EM, Boyer K, Campbell FA. Pain Res Manag. 2008 Jan-Feb;13(1):25-32.

77% of inpatients have pain during admission
44% - moderate-severe in previous 24h



Facing the reality

SickKids Inpatient Pain Audit

Taylor EM, Boyer K, Campbell FA. Pain Res Manag. 2008 Jan-Feb;13(1):25-32.

Hospitals are always **painful zones** for the kids especially cardiac and orthopaedic departments as well as intensive care units!

What is pain?

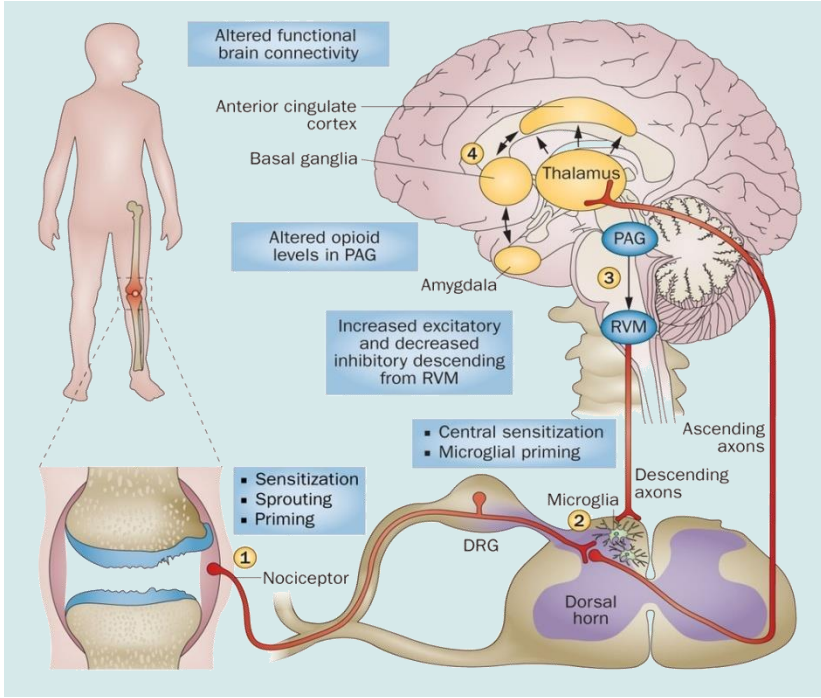
The experience of pain is complex, multifaceted, and

“an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage”

as defined in 2008 by the International Association for the Study of Pain.

Process of nociception

- **Transduction** conversion of pain stimulus into a nerve impulse by sensory receptors
- **Transmission** of nerve impulses from the periphery to the brain and spinal cord
- **Perception** the recognition of these impulses or signals as pain
- **Modulation** whereby descending neuronal tracts from the brain modify the nociceptive transmission in the spinal cord



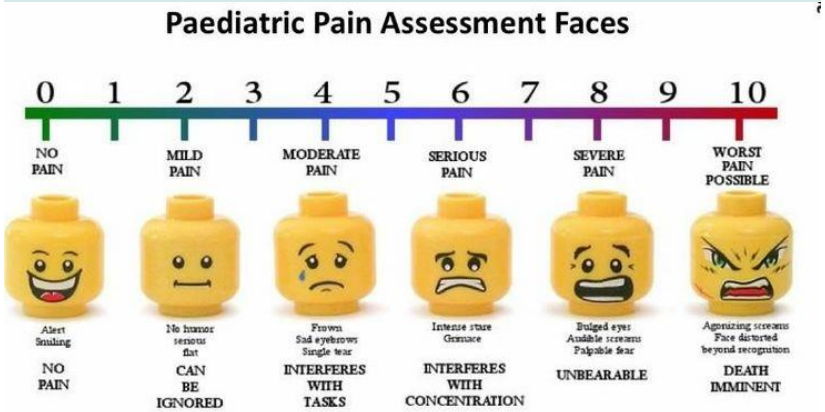
Negative effects of pain

- Physiological effects
- Behavioral effects
- Hormonal / metabolic responses

Consider pain as a vital sign

Physiological effects

- **Variations in HR**
  - from tachycardia down to bradycardia
- **variations in BP**
  - from hypertension down to hypotension
- **increased ICP**
  - up to more than two times higher as normal
- **increase in oxygen consumption**
  - oxygen extraction can be increased to ~70%
- **change in color**
  - from hyperperfusion down to hypoperfusion
- **increased or decreased muscle tone**
  - from hypertonus down to hypotonus



Behavioral effects

- **Crying**
  - can vary from high pitched, tense to soft moaning or whining
- **Facial expressions**
  - grimacing, quivering of chin
  - squeezing eyes shut, furrowed brow
- **Difficult to soothe, comfort or calm**

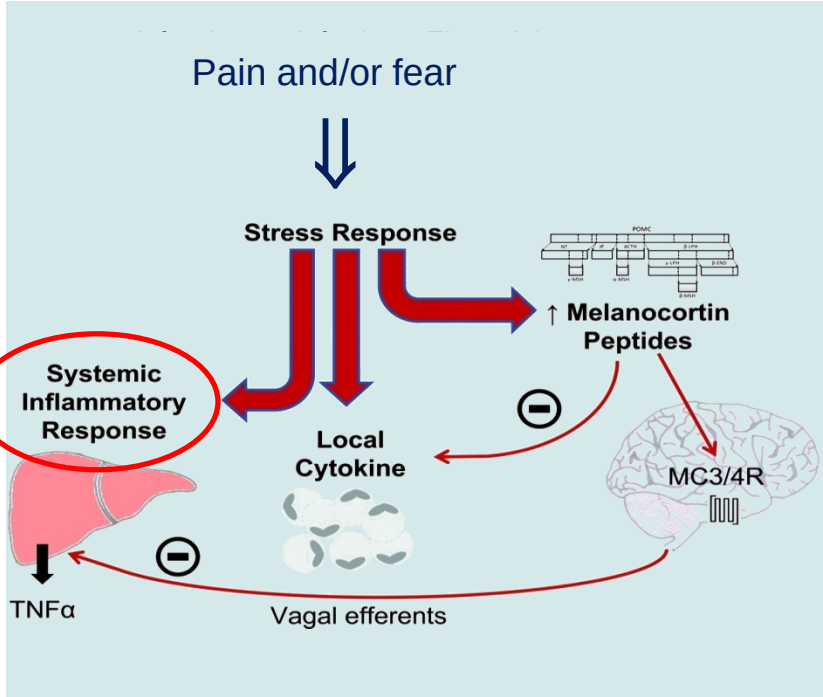
- **Body movements**
  - limb withdrawal, fist clenching
- hypertonicity or hypotonicity
- **State changes**
  - changes in sleep-wake cycles
  - changes in activity levels
  - increased fussiness or irritability

Hormonal / metabolic responses

- **Increase** in (endogen) **epinephrine** and **norepinephrine**
- Increase in **growth hormone** and **endorphins**
- **Decrease** in **insulin secretion**
- **Increased s**ecretion of **cortisol, glucagon,** and **aldosterone**
- Increased **serum glucose, lactate** and **ketones**

⇓

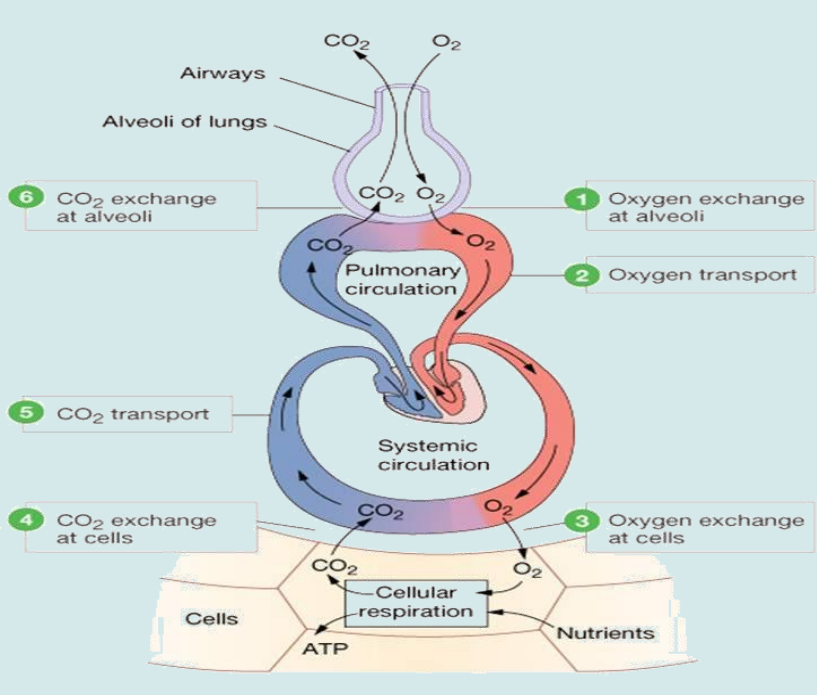
- Can lead to **lactic acidosis**



Important systems

Important systems in postoperative paediatric cardiac management:

- Cardiovascular
- Pulmonary
- Renal
- CNS
- Nutrition
- **Pain management**



O_2 Supply / Demand Imbalance

Four reasons for increased O_2 consumption:

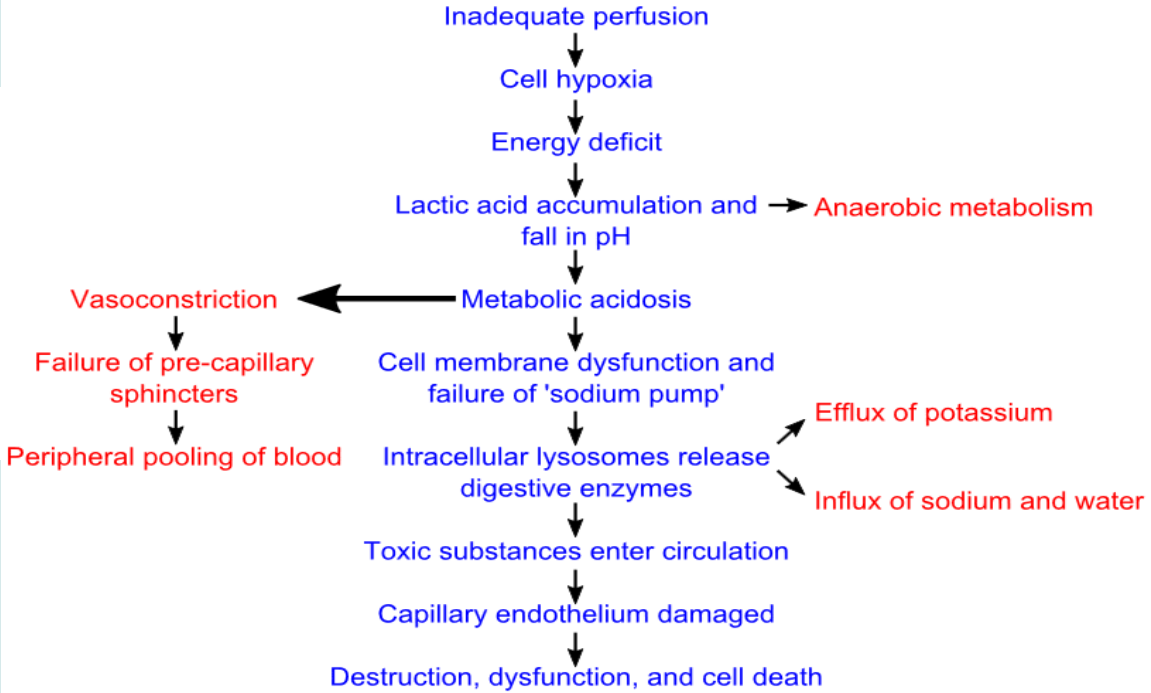
- **Stress / Pain**
- Hyperthermia
- Shivering
- Infection / Sepsis

Table 2 Clinical conditions and their effects on O_2 delivery and O_2 consumption and on venous oximetry

Decrease in $ScvO_2/SvO_2$
O_2 consumption ↑
Stress
Pain
Hyperthermia
Shivering
O_2 delivery ↓
CaO_2 ↓ (anemia, hypoxia)
Cardiac output ↓
Increase in $ScvO_2/SvO_2$
O_2 delivery ↑
CaO_2 ↑
Cardiac output ↑
O_2 consumption ↓
Analgesia
Sedation
Mechanical ventilation
Hypothermia

O_2 Supply / Demand Imbalance

An **imbalance** between tissue oxygen consumption and delivery appears with the development of **cellular hypoxia** and leads lastly to **cell death**.

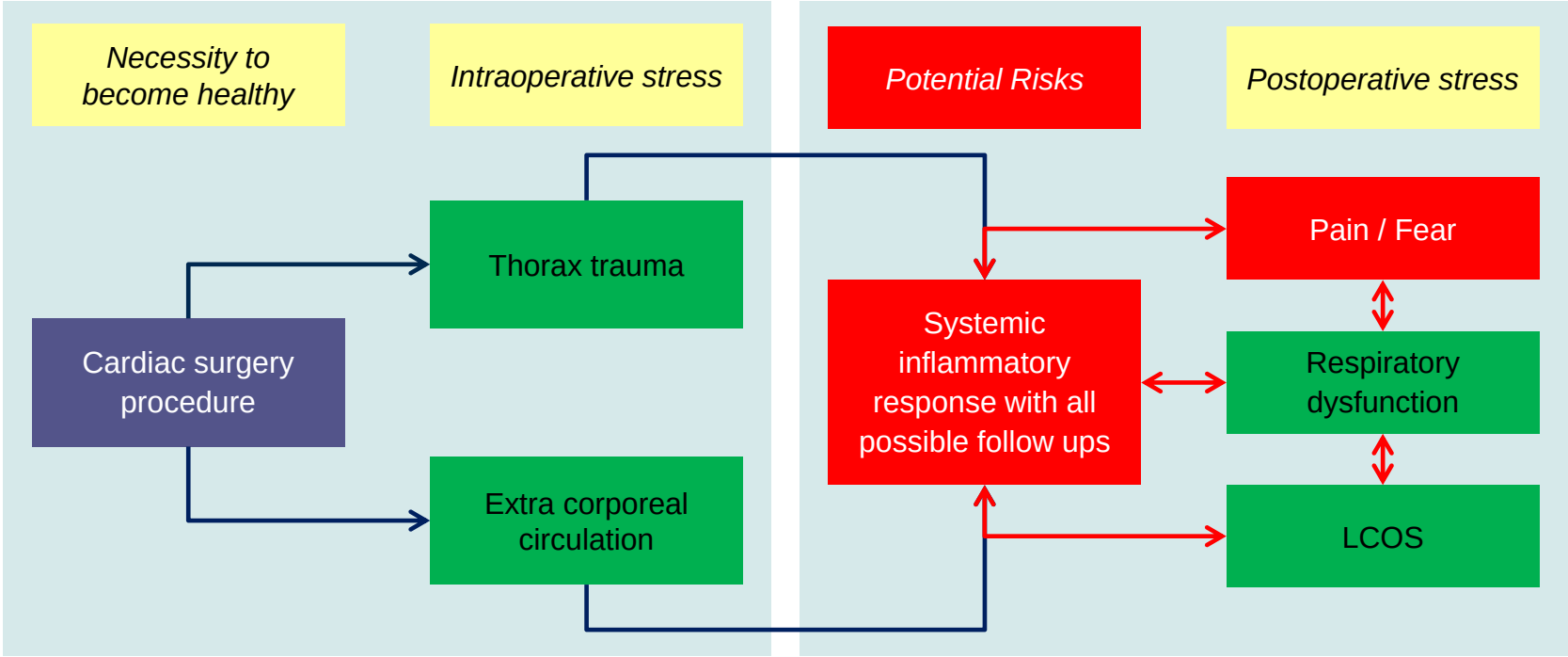


LCOS in paediatric cardiac surgery

Etiology of Low Cardiac Output Syndrome following congenital heart surgery

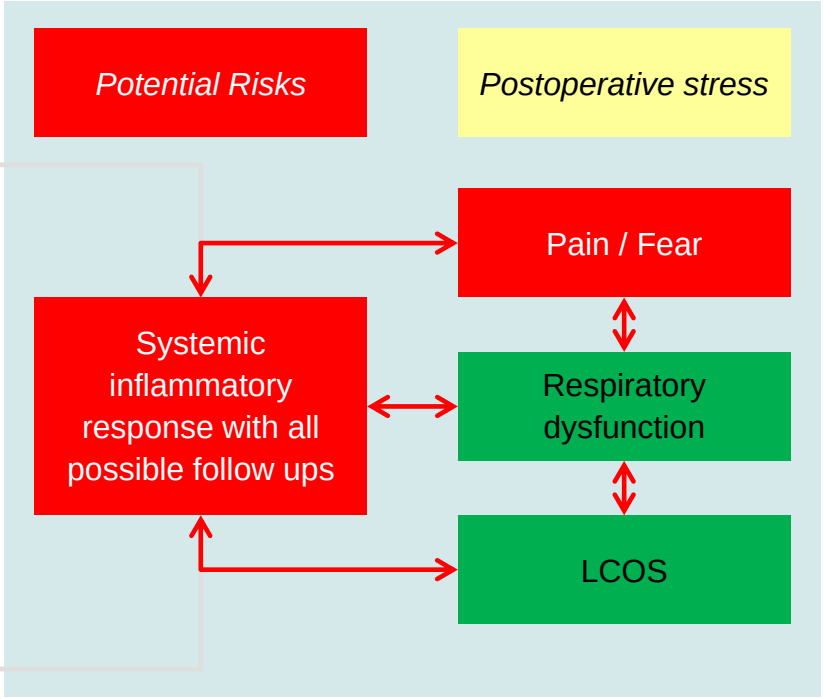
- Loss of AV synchrony
- Residual cardiac lesion
- Post-op. bleeding
- Pulmonary hypertension
- **Stress by pain and / or fear**
- Endocrine derangement (Cortisol, Thyroid, Vasopressin)

Situation by undergoing cardiac surgery



Never forget

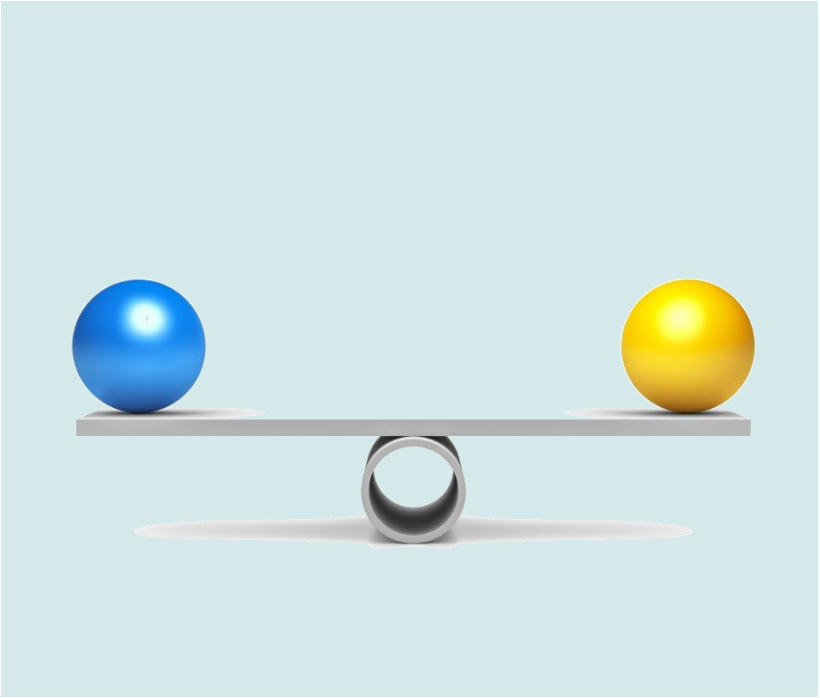
Inadequate pain management in postoperative care of children undergoing cardiac surgery can lead to a very big disaster!



General principles in pain management

- **Anticipate** and **prevent** pain
- Assess pain **adequately**
- Use a **multimodal approach**
- Use non-noxious and **non-painful route** of administration

General principles in pain management



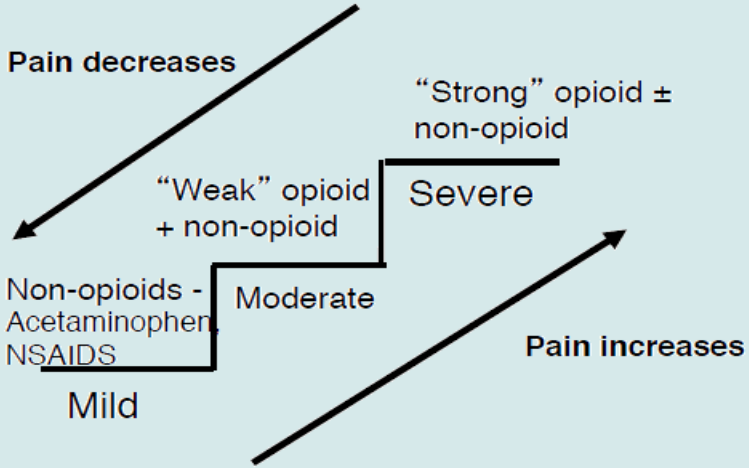
- **Balanced or multimodal analgesia**:
  - more than one class of analgesic or adjuvant each working in a different way

⇓

  - synergism
  - opioid sparing
  - reduced risk of tolerance and morphine sensitisation
  - reduced side effects and complications

General principles in pain management

- **Medications should be taken**
  - by the ladder
  - schedulded



Analgesia Ladder

Important

- **Pain management is not** the same as **sedation**
- **Anxiolysis** is also an **impotant need** in postoperative pain control

Pain-related psychological factors important in postoperative pain

Childrens anxiety is associated with increased postoperative pain and analgesic use

- Kain et al., Pediatrics. 2006 Aug;118(2):651-8.

Pain anxiety significantly associated with pain intensity and functional disability 2 weeks after discharge

Pain catastrophizing - associated with pain unpleasantness

Girls - higher levels of acute postoperative anxiety & pain unpleasantness.

- Pagé MG, Stinson J, Campbell F, Isaac L, Katz J. Pain Res. 2012;5:547-58

Effects of analgesia

- Reduces anxiety and stress response
- Reduces respiratory complications
- Reduces cardiovascular complications
- Reduces autonomic effects

The perfect analgesic

- **Effective** and **quick onset**
- **Safe** / no side-effects:
  - no CNS or cardiorespiratory depression
  - no constipation
  - no nausea
- No drug interactions
- **Cheap**
- No withdrawal, dependence, tolerance, addiction
- Useful in **all patient populations**
- Reversible effect
- Acceptable duration of effect (long, short)

The perfect analgesic

- Unfortunately this perfect analgesic doesn't exists!

Pharmacological substances

- Paracetamol
- NSAIDs
  (*Nonsteroidal Anti-Inflammatory Drugs*)
- Opiods
- NMDA antagonists (f.e. Ketamine)
  (N-Methyl-D-aspartate antagonists)
- Alpha-2 agonists (f.e. Clonidine)

Non-opioid analgesics	Paracetamol
	NSAIDs (including COX-2 inhibitors)
Weak opioids	Codeine
	Tramadol
Strong opioids	Morphine
	Pethidine
	Oxycodone
Adjuvant	Ketamine
	Clonidine, Dexmedetomidine

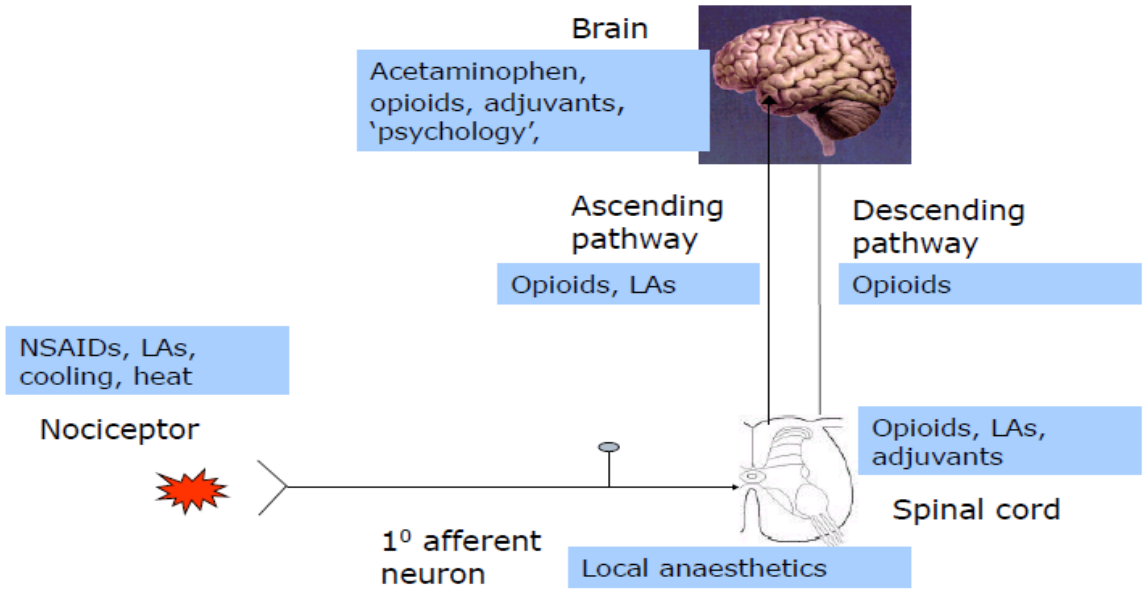
Used painkiller in children

- **Mild to moderate pain relief**
  - Acetaminophen (Paracetamol)
  - NSAIDs
- **Moderate pain relief**
  - Tramadol
  - Codeine
- **Severe pain relief**
  - Morphine
  - Nalbuphine
  - Ketamine

4 most used painkiller in children undergoing cardiac surgery

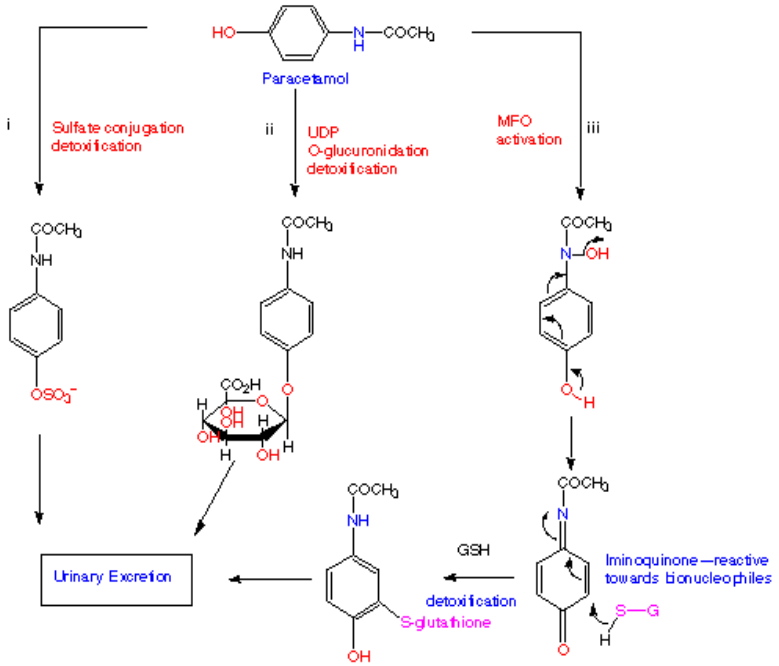
- Mild to moderate pain relief
  - **Acetaminophen (Paracetamol)**
  - **NSAIDs**
- Moderate pain relief
  - Tramadol
  - Codeine
- Severe pain relief
  - **Morphine**
  - Nalbuphine (part. antagonist)
  - **Ketamine**

Mechanism of action



Acetaminophen (Paracetamol)

- Inhibits COX-3 (cyclooxygenase 3) in the hypothalamus, very low activity on COX-1, possible 5-HT (5-hydroxytryptamine) activity
- No anti-inflammatory properties
- mild **analgesic**, good **antipyretic**
- First-line treatment if no contraindication
- **Morphine sparing effect** in pediatric patients
- Good **safety profile**
- **Contraindicated** by server **liver impairement**
- Metabolized in liver; half life is ~ 2-3 hours



Acetaminophen (Paracetamol)

- **Dose per weight:**

- IV application
- 10 kg or more:
  - 15 mg/kg in 30 min Q 6 H
- Less than 10 kg
  - 7.5 mg/kg in 30 min Q 6 H
- Peak analgesic effect of IV paracetamol occurs in 1 hour, duration 4-6 hrs
- Also oral or rectal application possible

Acetaminophen (Paracetamol)

- **NAPQI** (N-acetyl-p-benzoquinone imine) is a **toxic byproduct** produced during the metabolism of Acetaminophen
- It is normally produced only in small amounts, and then almost immediately detoxified in the liver
- The dosage at which Acetaminophen causes toxicity usually is **10 g in the average adult**
- The **dosage is much lower in children**, adapted to the weight
- The antidote **N-acetylcysteine** acts as a precursor for glutathione

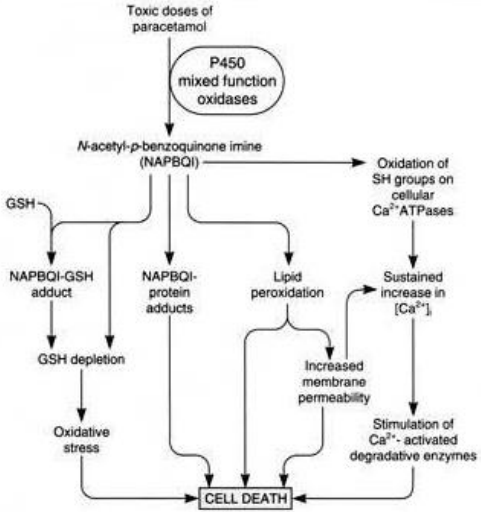


Fig. 49.1 Potential mechanisms of liver cell death resulting from the metabolism of paracetamol to *N*-acetyl-*p*-benzoquinone imine (NAPBQI). (GSH = glutathione) (Based on data from: Boobis A R et al. 1989, and Nelson S D, Pearson P G 1990 Annu Rev Pharmacol Toxicol 30: 169) See also Figure 13.1.

NSAIDs

- Analgesic, Antipyretic, Anti-inflammatory
- Block cyclooxygenase (COX) enzyme → ↓ prostaglandin synthesis
- COX-2 → Prostaglandins → pain, inflammation, fever
- COX-1 → Prostaglandins → gastric protection, hemostasis

- Avoided in:
  - infants less than 6 months (?)
  - children with aspirin or NSAID allergy
  - renal or hepatic failure
  - coagulation disorders
  - peptic ulcer disease
  - significant risk of haemorrhage (?)

NSAIDs

NSAIDs

Nonselective COX inhibitors

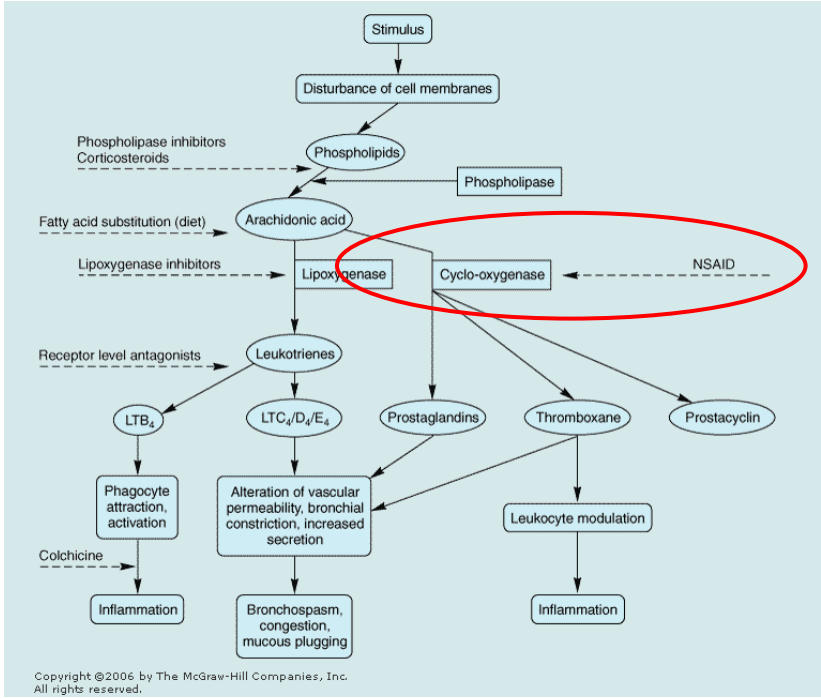
- *Salicylates:* Aspirin.
- *Propionic acid derivatives:* Ibuprofen, Ketoprofen, Flurbiprofen.
- *Fenamate:* Mephenamic acid.
- *Enolic acid derivatives:* piroxicam, Tenoxicam.
- *Acetic acid derivatives:* Ketorolac, Indomethacin.
- *Pyrazolone derivatives:* Phenylbutazone, Oxyphenbutazone.

Preferential COX-2 inhibitors:

- Nimesulide, Diclofenac, Aceclofenac, Meloxicam, Etodolac.

Selective COX-2 inhibitors:

- Celecoxib, Etoricoxib, Parecoxib.



NSAIDs

- **Ketorolac**
  - IV NSAID (available p.o.)
  - dose 0.5 mg/kg/dose Q 6 H
  - onset after 10 minutes
  - maximum i.v. dose 30 mg Q 6 hours
  - do not use more than 5 days
  - significant increase in side effects after 5 days
  - monitor renal function

- **Ibuprofen**
  - Oral NSAID (available rectal)
  - dose 5 to 10 mg/kg Q 6 to 8 hours
  - the recommended maximum daily dose is 40 mg/kg
  - potent antipyeretic, analgesic
  - Ibuprofen should be avoided in patients with severe hepatic disease

Opioids

- Used for thousands of years to produce:
  - euphoria
  - analgesia
  - sedation
  - relief from diarrhea
  - cough suppression

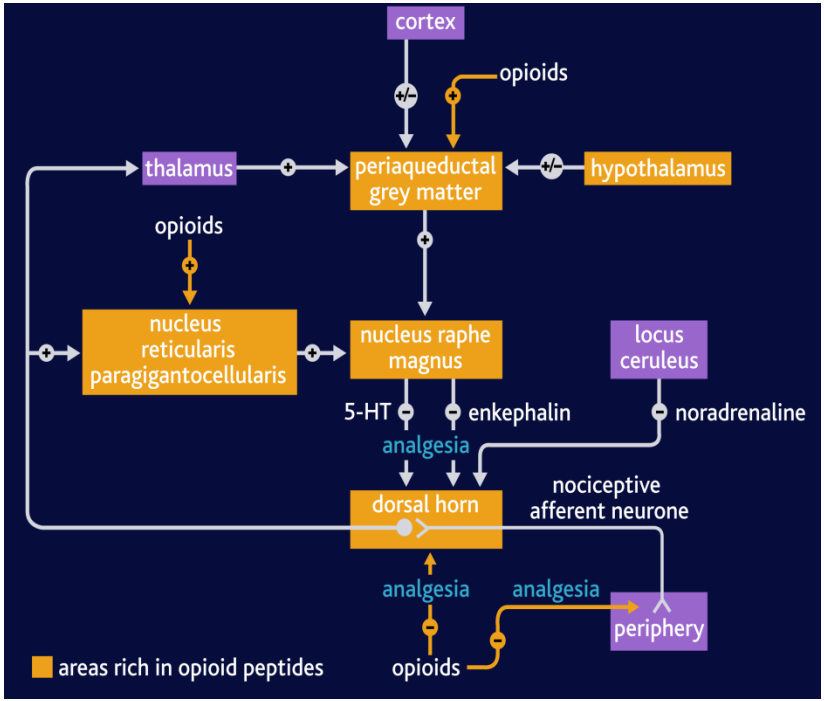
- Used medicinally and recreationally from early Greek and Roman times
- Opium and laudanum (opium combined with alcohol) were used to treat almost all known diseases
- Morphine was isolated from opium in the early 1800's and since then has been the most effective treatment for severe pain

Opioids

- Natural opioids occur in 2 places:
  - in the juice of the opium poppy (morphine and codeine)
  - as endogenous endorphins
- All other opioids are prepared from either morphine (semisynthetic opioids such as heroin) or they are synthesized from precursor compounds (synthetic opioids such as fentanyl)

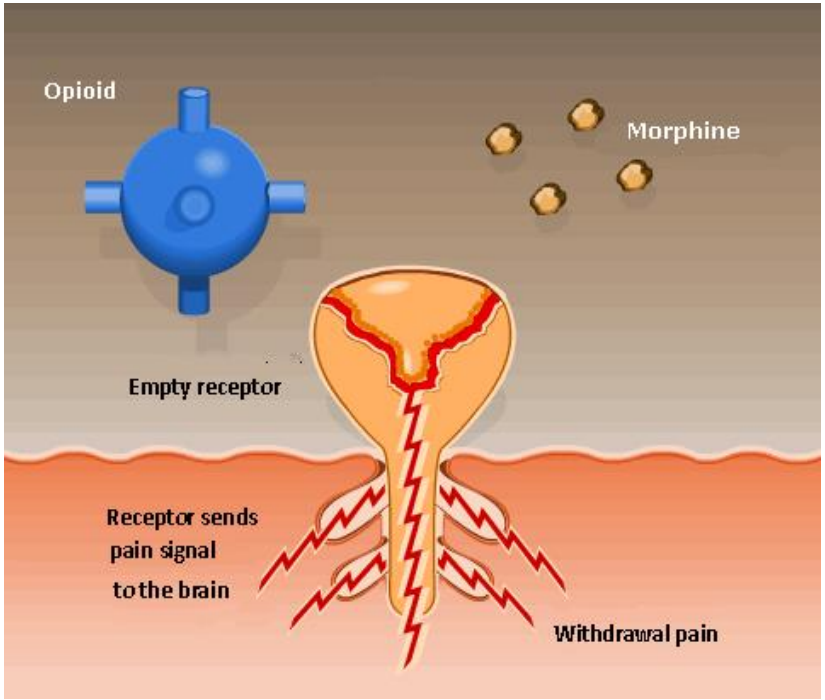
Mechanism of action

- Reduction or inhibition of neurotransmission, due largely to opioid-induced presynaptic inhibition of neurotransmitter release:
  - involves changes in transmembrane ion conductance
  - increase potassium conductance (hyperpolarization)
  - inactivation of calcium channels



Mechanism of action

- Mechanism of action via opioid receptors
  - μ - receptor
  - κ - receptor
  - δ - receptor



Mechanism of action

- **Delta receptor:**
  - it is unclear what delta's responsible for
- **Two types of Mu receptors:**
  - **Mu-1:** located outside spinal cord, responsible for central interpretation of pain
  - **MU-2**: located throughout CNS, responsible for respiratory depression, spinal analgesia, physical dependence, and euphoria
  - **Kappa receptor**: modest analgesia, dysphoric effects

Response	Mu-1	Mu-2	Kappa
Analgesia	★	★	★
Respiratory Depression		★	
Euphoria		★	
Dysphoria			★
Decrease GI motility		★	
Physical Dependence		★	

Pharmacological effects

- **Sedation and anxiolysis**
  - drowsiness and lethargy
  - apathy
  - cognitive impairment
  - sense of tranquility
- **Depression of respiration**
  - main cause of death from opioid overdose
- **Cough suppression**
  - opioids suppress the “cough center” in the brain
- **Pupillary constriction**

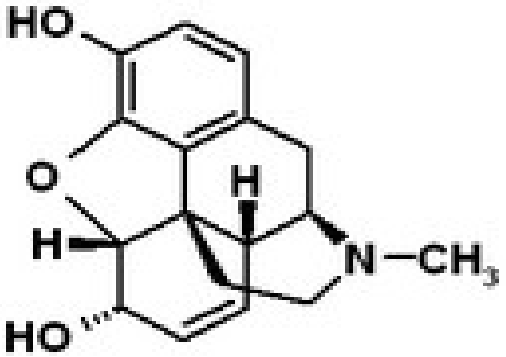
Pharmacological effects

- **Nausea and vomiting:**
  - stimulation of receptors in an area of the medulla called the chemoreceptor trigger zone causes nausea and vomiting
  - unpleasant side effect, but not life threatening
- **Gastrointestinal symptoms:**
  - opioids relieve diarrhea as a result of their direct actions on the intestines

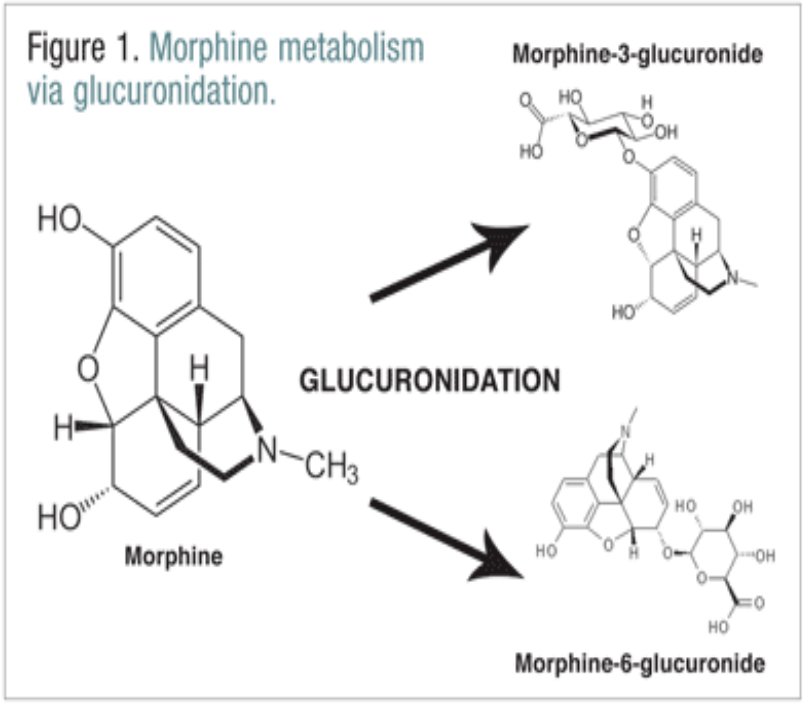
- **Other effects:**
  - opioids can release histamines causing itching or more severe allergic reactions including bronchoconstriction
  - opioids can affect white blood cell function and immune function
- **Important:**
  - Development of tolerance and addiction

Morphine

- **Most widely used** and studied opioid in children
- **Safe and effective in all ages**
- **Drug of first choice by server pain**
- Can be given by the subcutaneous, intramuscular, intravenous, epidural, intraspinal, and rectal routes
- Continuous or intermittent infusion of the dose is adjusted according to individual analgesic requirements



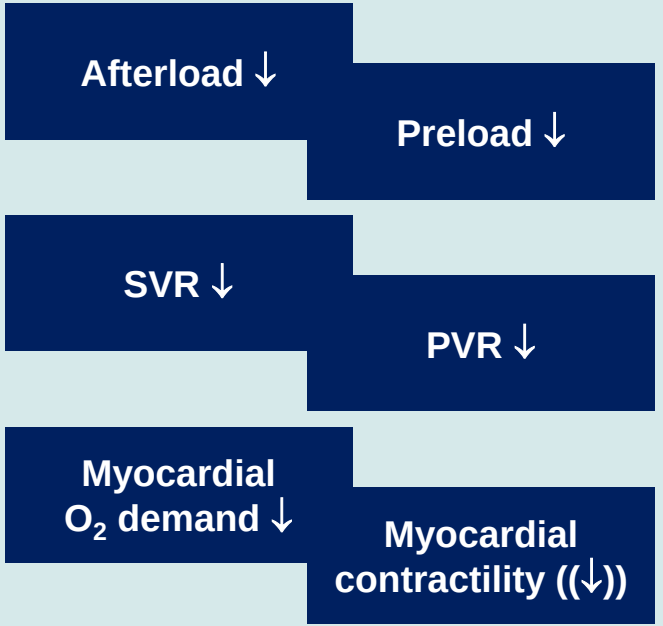
Morphine



- **Dose: 0.05 - 0.1 mg/kg IV dose Q 4 H**
- **Continous application: 10 – 60 mcg/kg/h**
- There is no maximum dose per day
- Metabolism:
  - hepatic glucoronidation: morphine-3-glucuronide (M3G) and -6-glucuronide (M6G)

Morphine cardiovascular effects

- Cardiovascular effects of Morphine lead to **vasodilatation**, thus a **decrease** in **systemic and pulmonary arterial pressure**
- Morphine causes the release of histamine
- **Suppression of central adrenergic tone**
- Suppression of reflex vasoconstriction



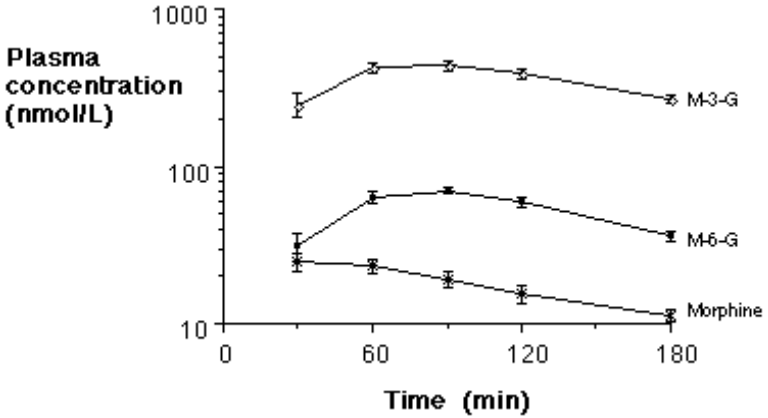
Morphine pharmacokinetics

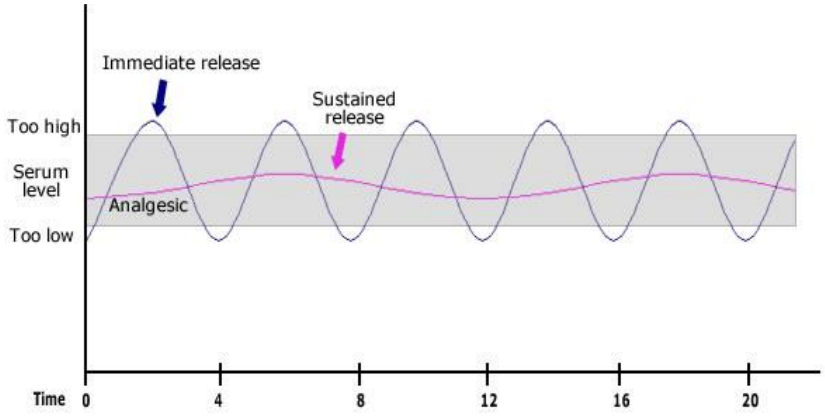
- Elimination halftimes for morphine following bolus i.v. administration is about 1.7-4.5 hours
- Oral application route is defeated by the 1st pass metabolism
- Intramuscular and subcutaneous application routes are not recommended in postoperative cardiac critical care because of unstable absorption by disorders in peripheral circulation

- Following bolus administration onset time is relatively slow (15-30 minutes) because:
  - morphine exhibits relatively low lipid solubility
  - at physiological pH, morphine, a weak base, is primarily ionized
  - the ionized form does not favor passage through the lipid membrane
  - accordingly, only about 10% - 20% of molecules are un-ionized

Morphine pharmacokinetics

- Best routes of application are i.v. bolus doses or continuous infusion by syringe pump!





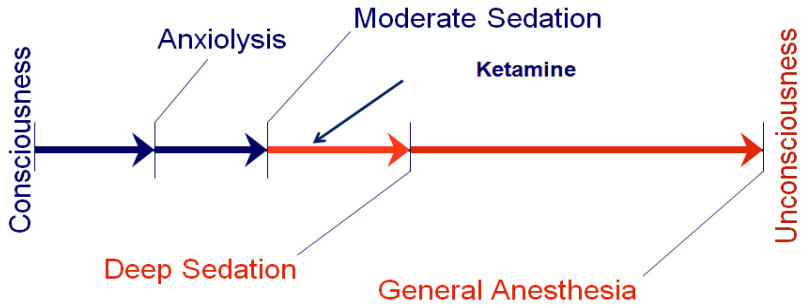
Agonists and Antagonists

- **Opioid Agonists:**
  - Morphine, Codeine, Meperidine, Fentanyl, Sufentanil, Methadone, Tramadol, etc.
- **Opioid Agonist / Antagonist + partial Agonist:**
  - Pentazocine, Nalbuphine, Butorphanol, Buprenorphine
- **Opioid Antagonists:**
- Nalorphine, Naloxone, Naltrexone, Naltrindole, Nalmefene

DRUGS	MU	KAPPA
Pure Agonists	Agonist	Agonist
Agonist-Antagonist	Antagonist	Agonist
Pure Antagonists	Antagonist	Antagonist

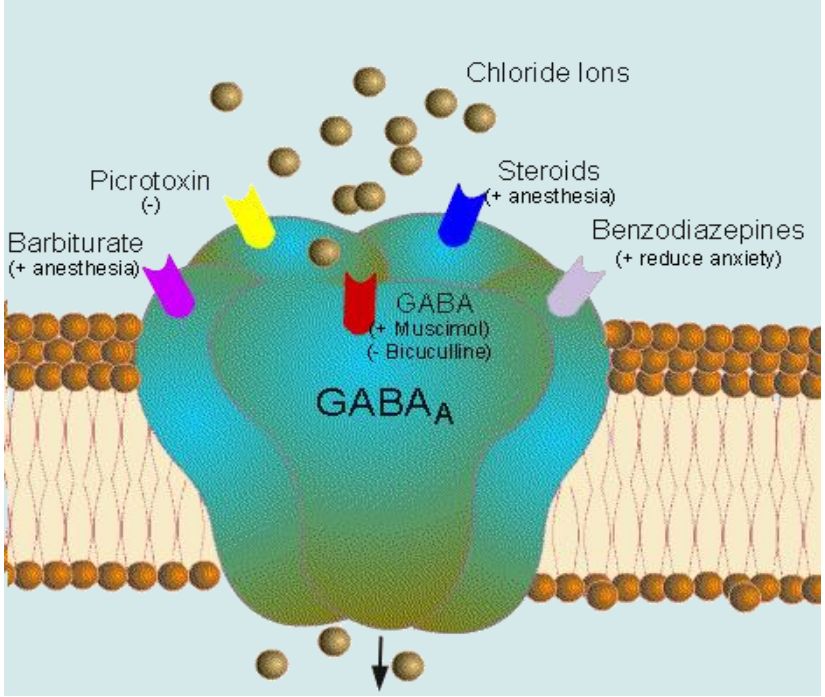
Ketamine

- N-Methyl-D-aspartate (NMDA) receptor antagonists
- Parenteral anaesthetic agent with amnestic and analgesic properties
- rapid onset
- useful for short, painful procedures
- Dissociative anesthesia may not produce reliable immobility
- May produce transient hypertension and increased HR
- Contraindicated with head trauma, increased ICP



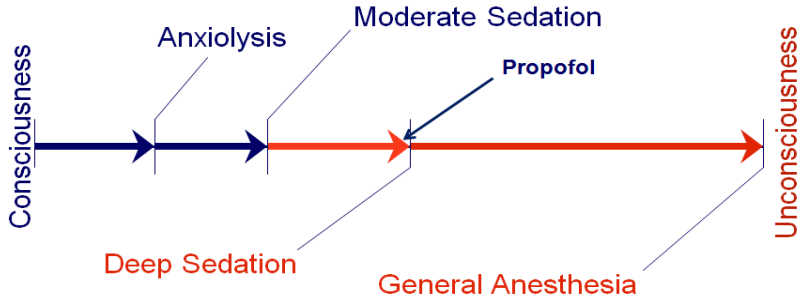
Propofol

- Propofol works by increasing GABA-mediated inhibatory tone in the CNS
- Propofol decreases the rate of dissociation of the GABA from the receptor, thereby increasing the duration of the GABA-activated opening of the chloride channel with resulting hyperpolarization of cell membranes



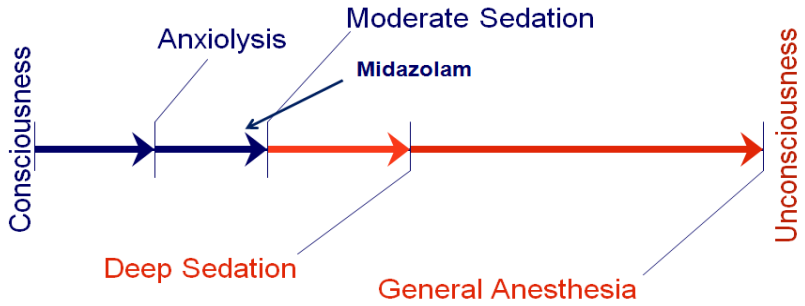
Propofol

- Sedative, anesthetic, amnestic, anticonvulsant
- Respiratory and cardiovascular depression
- Use only in mechanically ventilated patient
- Hypotension occasionally limits its use
- Rapid onset, short duration
- Onset <1 min, peak 2 min, duration 4-8 min
- Clearance not changed in liver or kidney disease
- Highly lipid soluble
- Metabolised by conjugation in the liver
- Increases triglycerides



Midazolam

- Belongs to the Benzodiazepines
- Rapid onset (1 - 3 min) and relatively rapid offset
- Expected duration: 60 - 90 minutes
- Anxiolysis plus amnesia, no analgesia
- Anti-convulsant
- Hemodynamic stability
- Reversible with Flumazenil (specific antagonist)
- Produces "true" moderate sedation in children
- Versatile + painless routes of administration



Our protocol

- Propofol
- Ketamine
- Morphine
- Ibuprofen / Ketorolac
- Midazolam
- Paracetamol

Our protocol

- Sedation with Propofol by ventilated patients coming out from OR when extubation is expected within the first 4 hours

Propofol

Our protocol

- Sedation with Propofol by ventilated patients coming out from OR when extubation is expected within the first 4 hours
- By expected extubation time more than 4 hours but less than 24 hours we are giving Propofol and Morphine additional as pain killer

Propofol

Morphine

Our protocol

- Sedation with Propofol by ventilated patients coming out from OR when extubation is expected within the first 4 hours
- By expected extubation time more than 4 hours but less than 24 hours we are giving Propofol and additional Morphine as pain killer
- By need of long term ventilation (> 24 hours) we change to Midazolam and Morphine

Midazolam

Morphine

Our protocol

- After extubation regularly application of Morphine i.v. max. 0.1 mg/kg/ Q 4 H or continuously with 10 – 60 mcg/kg/h
- Additional application of Paracetamol i.v. Q 6 H

Morphine

Paracetamol

Our protocol

- After extubation regularly application of Morphine IV max. 0.1 mg/kg/ Q 4 H or continuously with 10 – 60 mcg/kg/h
- Additional application.of Paracetamol i.v. Q 6 H
- To prevent the risk of vomiting we give Ondansetron regularly 4 Q H

Ondansetron

Morphine

Paracetamol

Our protocol

- All painful postoperative procedures (f.e. removing drains) are done under Ketamine anaesthesia

Ketamine

Our protocol

- Regularly on the second postoperative day we are shifting our patients to normal ward under pain managament with Paracetamol and Ibuprofen or Ketorolac

Paracetamol

Ibuprofen / Ketorolac

Our protocol

- Regularly on the second postoperative day we are shifting our patients to normal ward under pain managament with Paracetamol and Ibuprofen or Ketorolac
- At the 6th day we stop Paracetamol and also Ketorolac (if it was used) and continue with Ibuprofen orally as long as it is needed

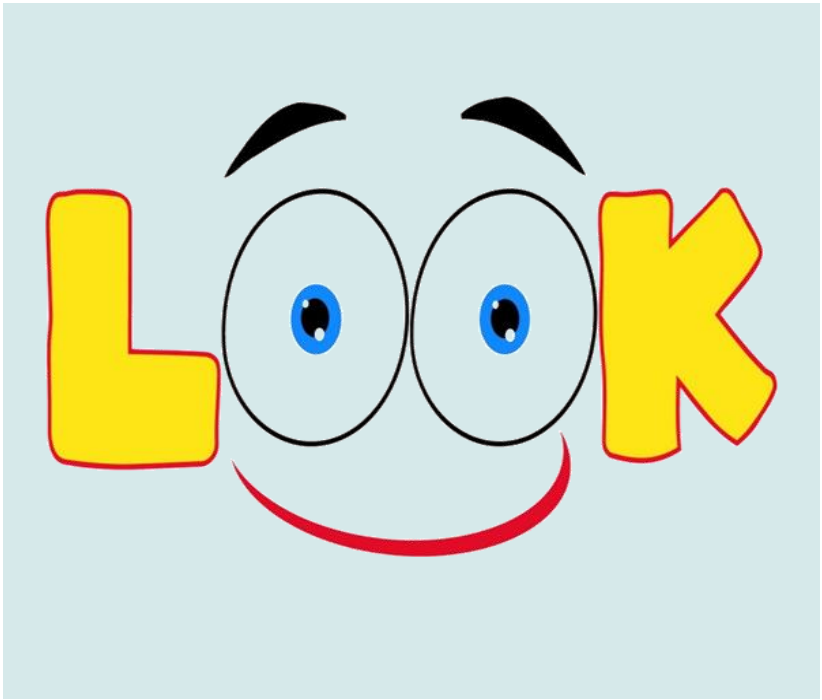
Ibuprofen

Take home messages

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.

1.

Take home messages



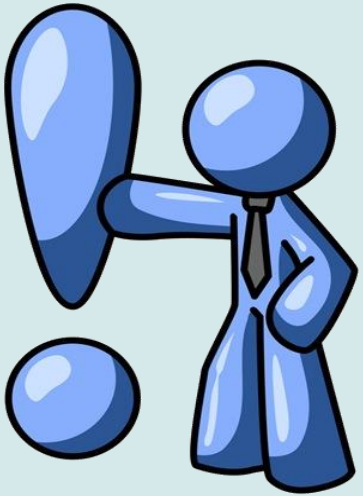
Anticipation and prevention are the most important **keys for recognition, diagnosis, and successfull management** of all potential risks and complications in the postoperative critical care of paediatric cardiac patients.

2.

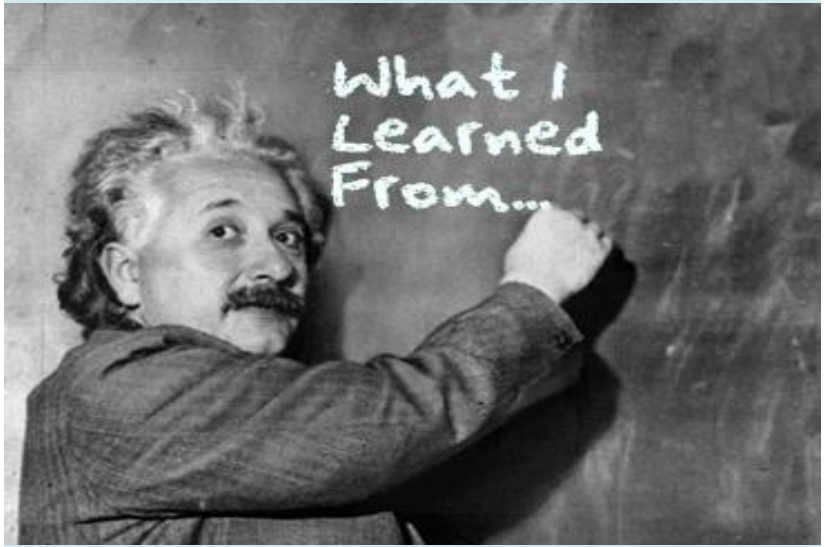
Take home messages

Hospitals are always **painful zones** for the kids especially cardiac and orthopaedic departments as well as intensive care units.

3.



Take home messages



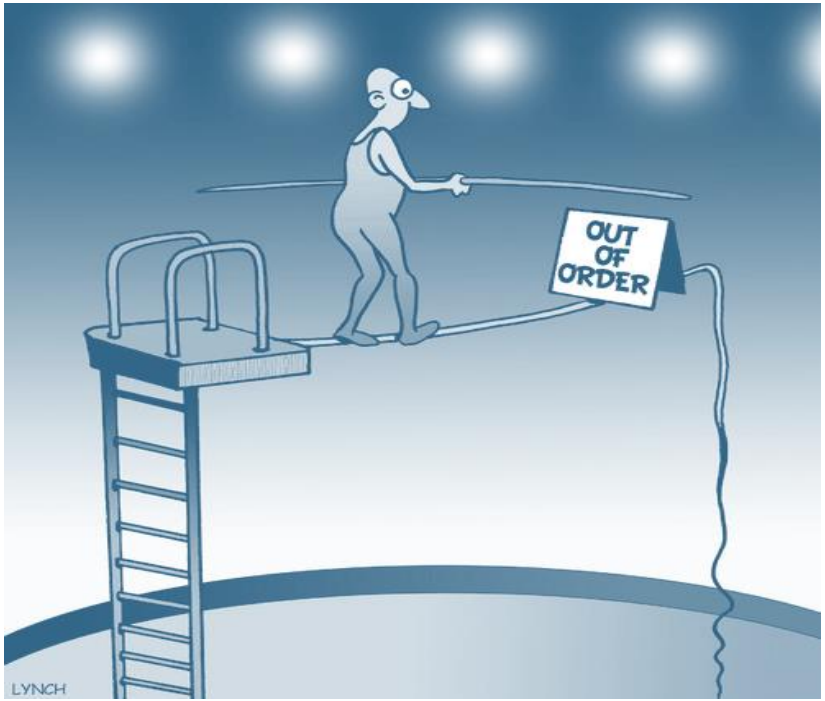
Consider pain as a **vital sign**.

4.

Take home messages

Inadequate pain management in postoperative care of children undergoing cardiac surgery can **lead to a very big disaster!**

5.



Take home messages

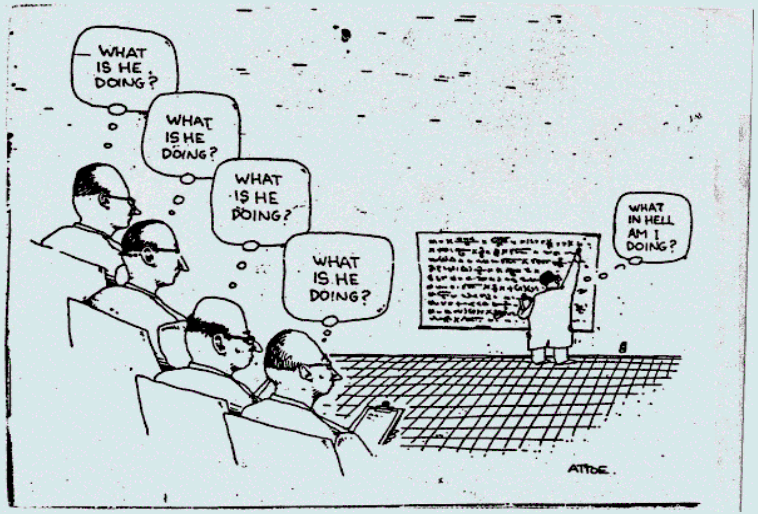
- **Anticipate** and **prevent** pain
- Assess pain **adequately**
- Use a **multimodal approach**
- Use non-noxious and **non-painful route** of administration

6.

Take home messages

- **Pain management is not** the same as **sedation**
- **Anxiolysis** is also an **impotant need** in postoperative pain control

7.



Take home messages

- Effects of **successful analgesia**:
  - **Reduces anxiety and stress response**
  - Reduces respiratory complications
  - Reduces cardiovascular complications
  - **Reduces autonomic effects**

8.

Take home messages

- Unfortunately there doesn’t exists a perfect analgesic
- In ICU we are able to manage all possible side effects

9.

Take home messages

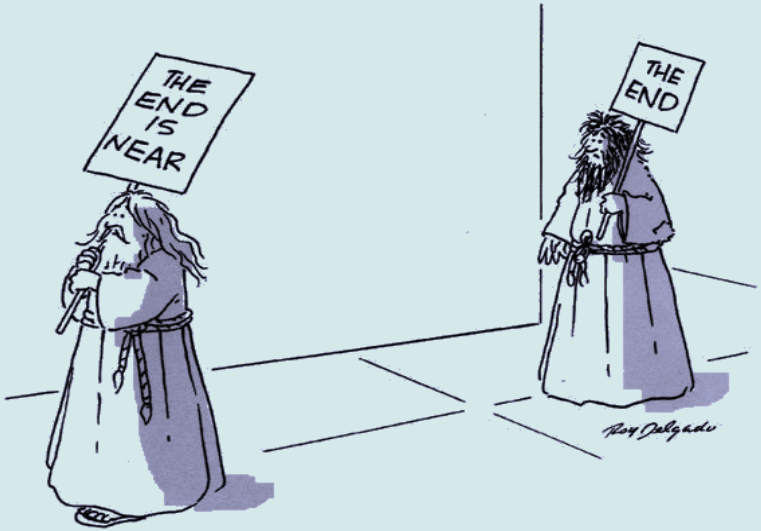
- **Most used painkiller** in children undergoing cardiac surgery:
  - **Acetaminophen (Paracetamol)**
  - **NSAIDs**
  - **Morphine**
  - **Ketamine**

10.

Take home messages

- **Morphine is**
  - **the most widely used** and studied opioid in children
  - **safe and effective in all ages**
  - has important positive effects we want in paediatric cardiac critical care

11.



Take home messages

Propofol

Midazolam

Ketamine

Morphine

Paracetamol

Ibuprofen / Ketorolac

- Use a structured and succesful protocol for postoperative pain management in paediatric cardiac critical care

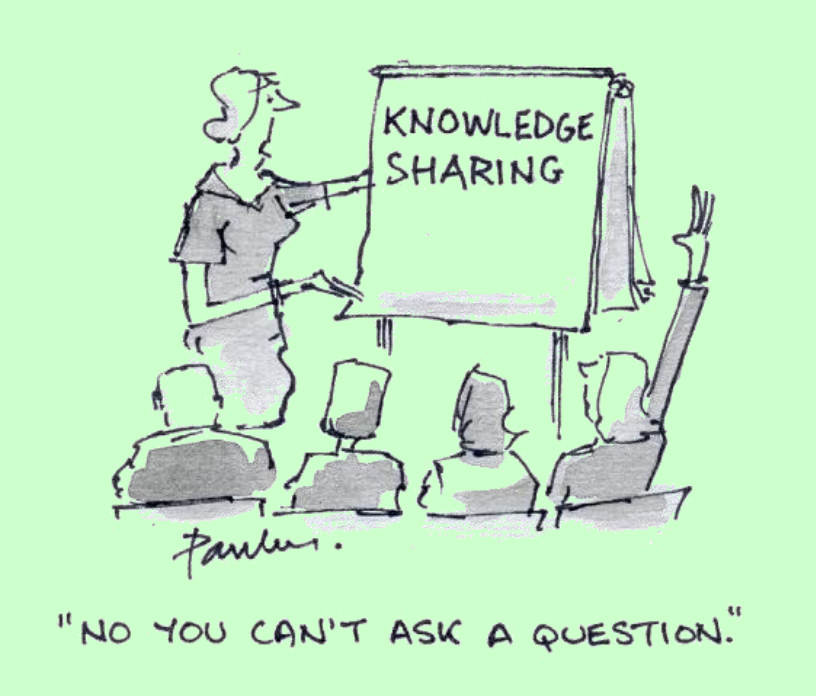
12.

Take home messages

Never let a child stay in pain!

13.

Thank you for your attention



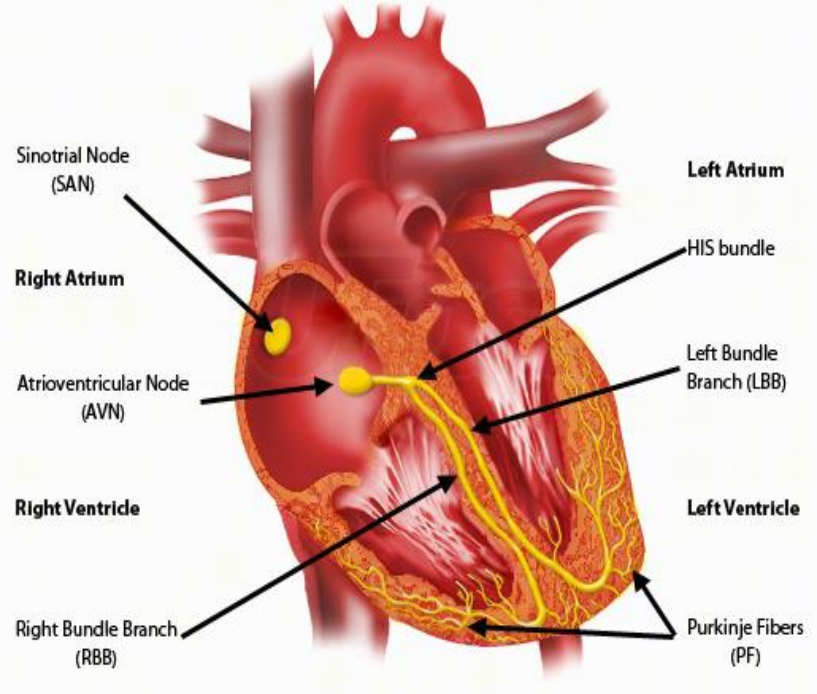
Yes, you can!

Let‘s continue with the next topic

Cardiac arrhythmias; care of patients with temporary pacemaker

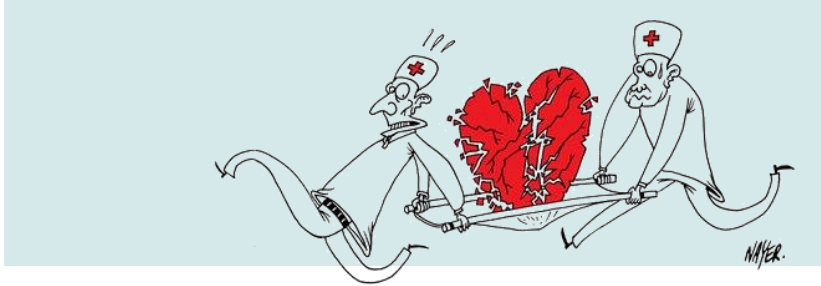
The cardiac conduction system

- 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

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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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JET: Junctional ectopic tachycardia, SVT: Supraventricular tachycardia

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

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

Junctional ectopic tachycardia

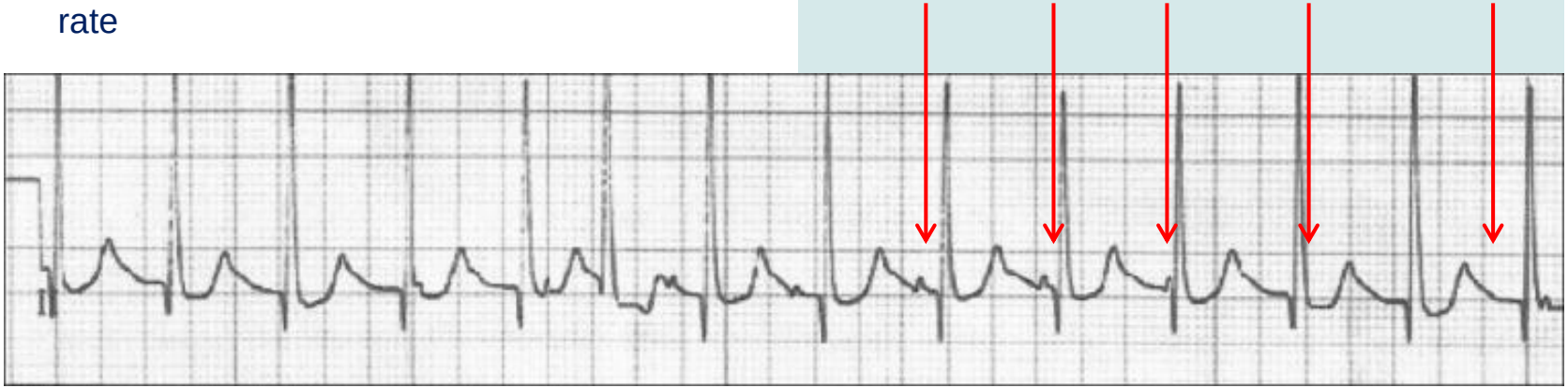
- A **narrow complex tachycardia** with atrio-ventricular dissociation

- 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

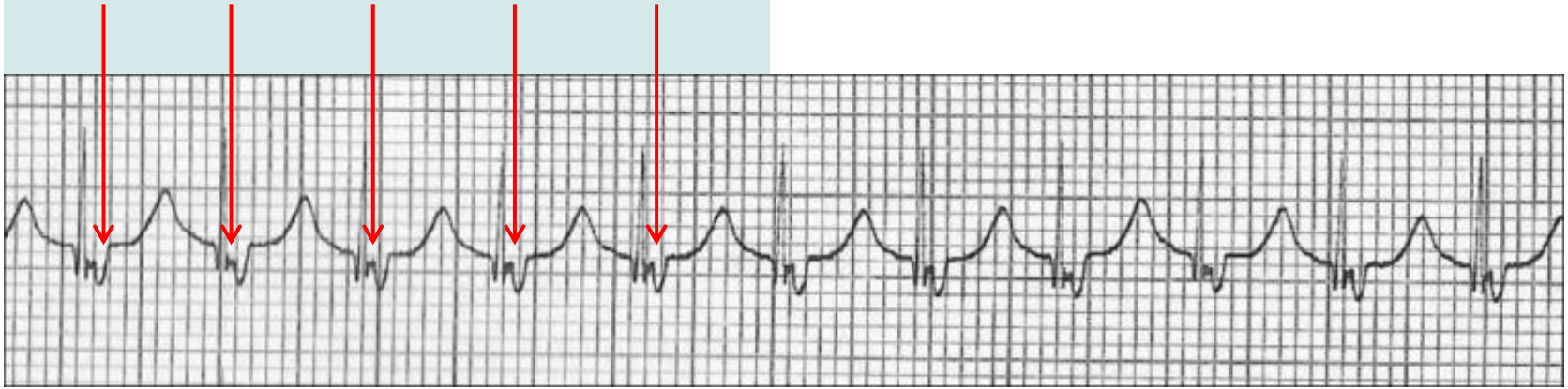
P waves



Junctional ectopic tachycardia

P waves

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



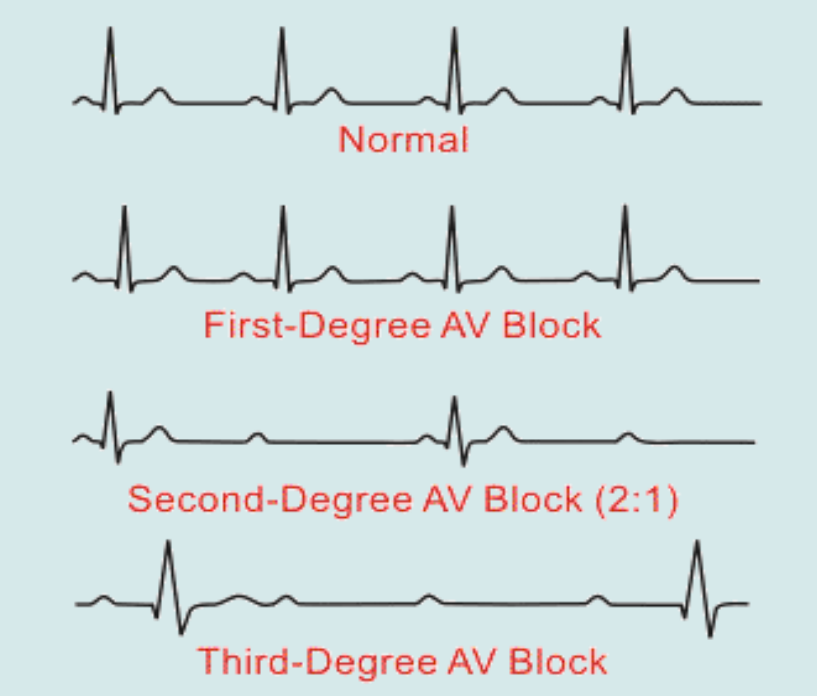
Therapeutic interventions

- **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

Principles of pacing

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

Principles of pacing

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

Principles of pacing

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

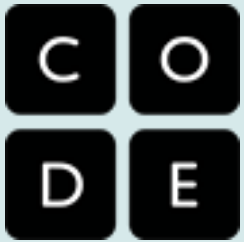
Principles of pacing

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

Principles of pacing

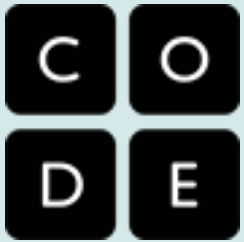
3 - letter NBG Pacemaker Code (for temporary pacer):



- **First letter: Chamber Paced**
  - V - Ventricle
  - A - Atrium
  - D - Dual (A & V)
  - O - None

Principles of pacing

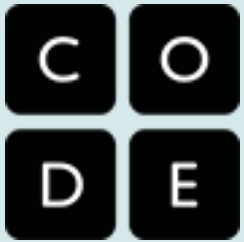
3 - letter NBG Pacemaker Code (for temporary pacer):



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

Principles of pacing

3 - letter NBG Pacemaker Code (for temporary pacer):



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

Principles of pacing

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

Principles of pacing

- **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

Principles of pacing

- **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

Principles of pacing

- **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

Pacemaker

- **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

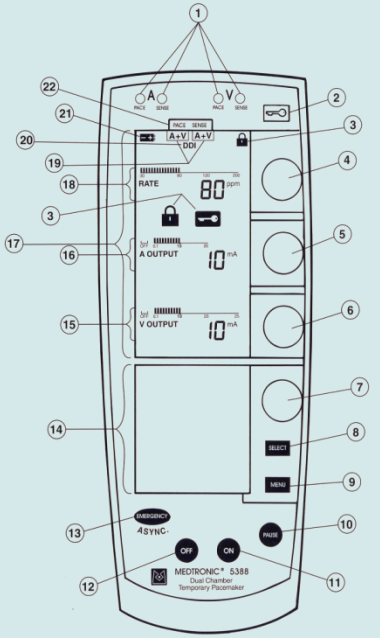


Figure 3-1. *Controls and Indicators of the Model 5388.*

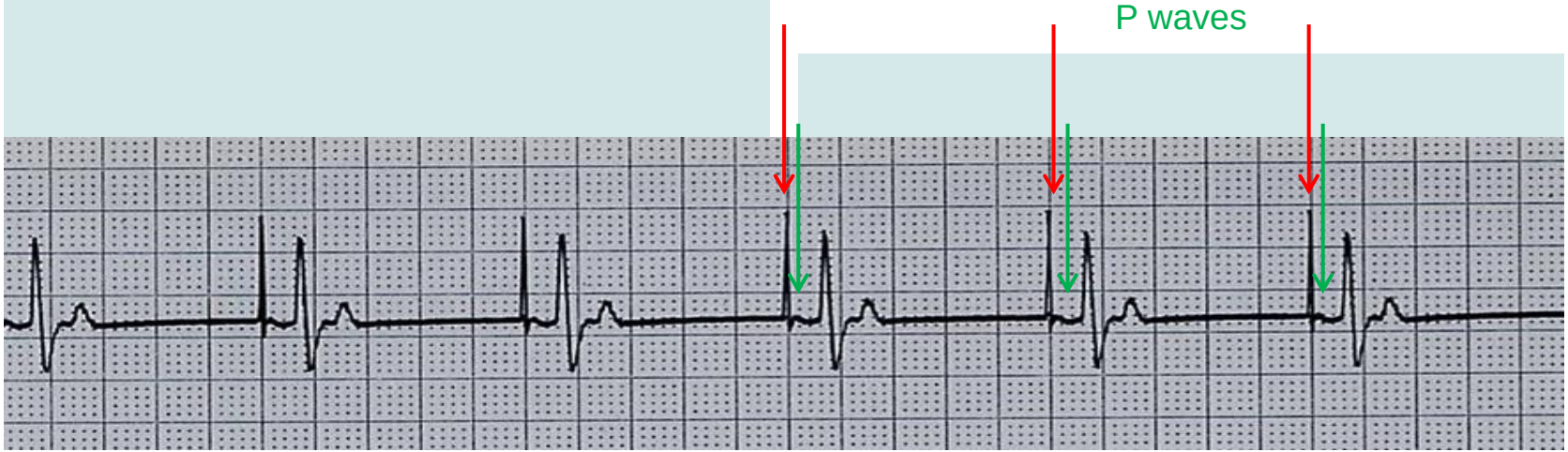
Pacemaker ECG

- 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

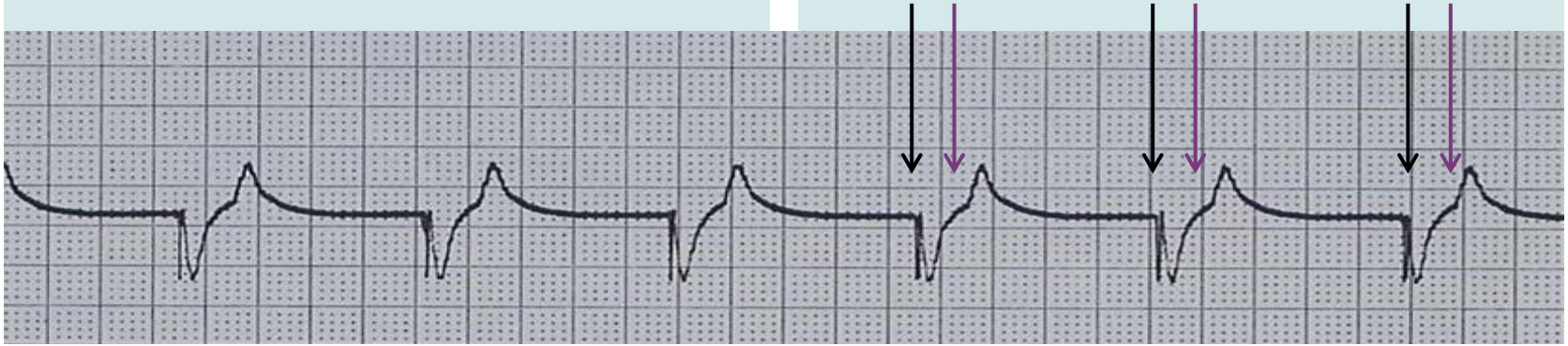


Normal pacing

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

Ventricular pacing spikes

QRS complexes

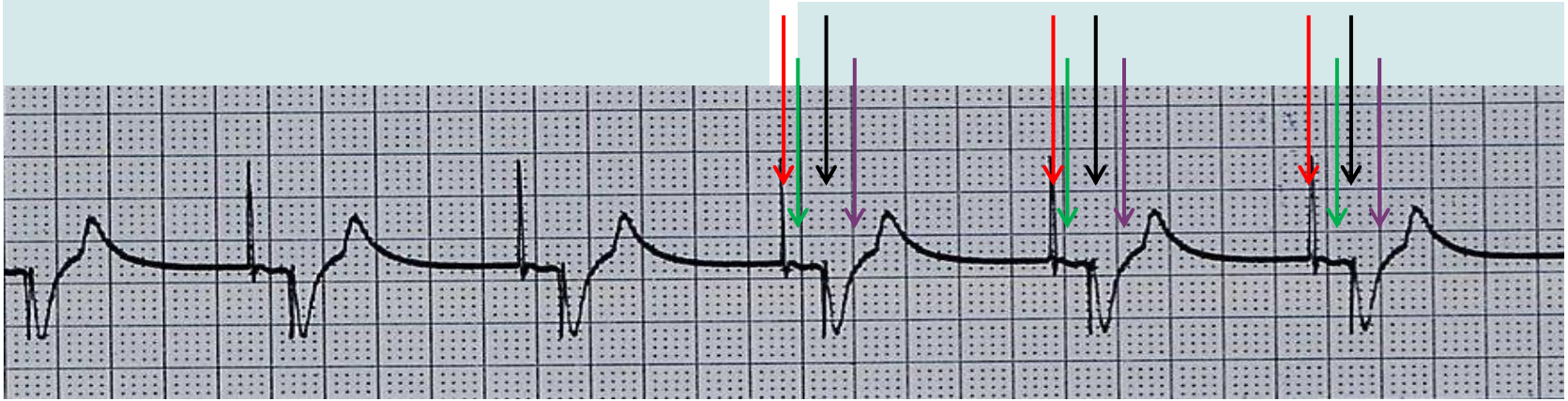


Normal pacing

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

Atrial + Ventricular pacing spikes

Atrial + QRS complexes

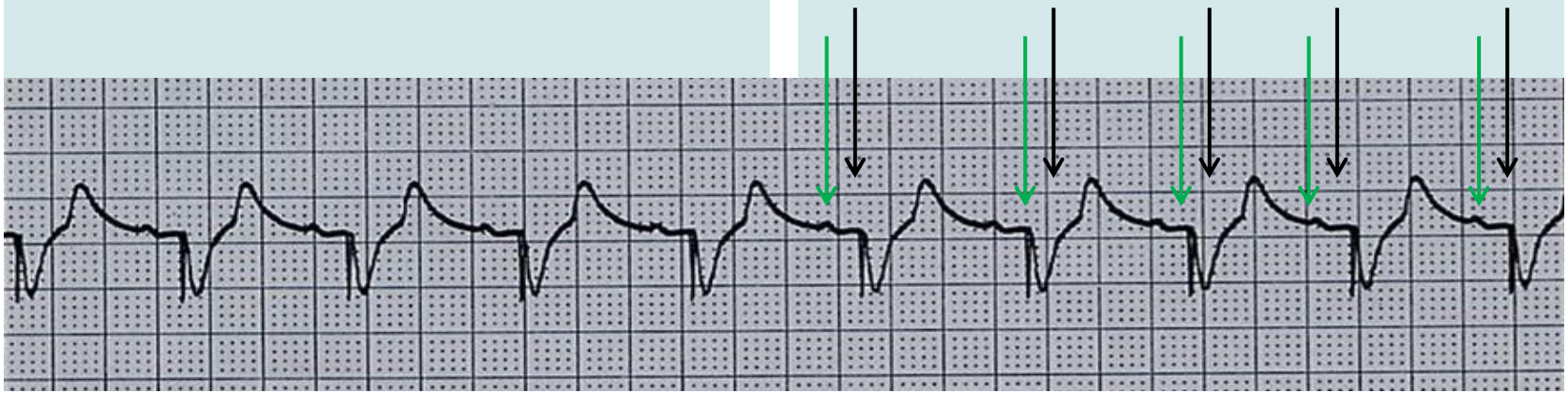


Normal pacing

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

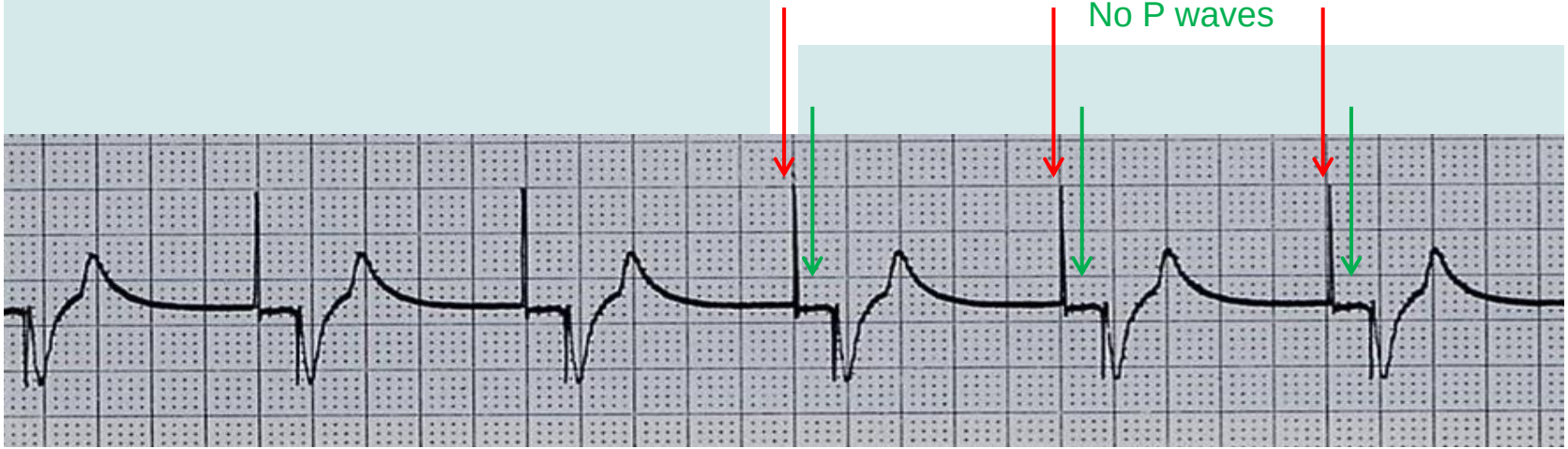
Ventricular pacing spikes

P waves



Abnormal pacing

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

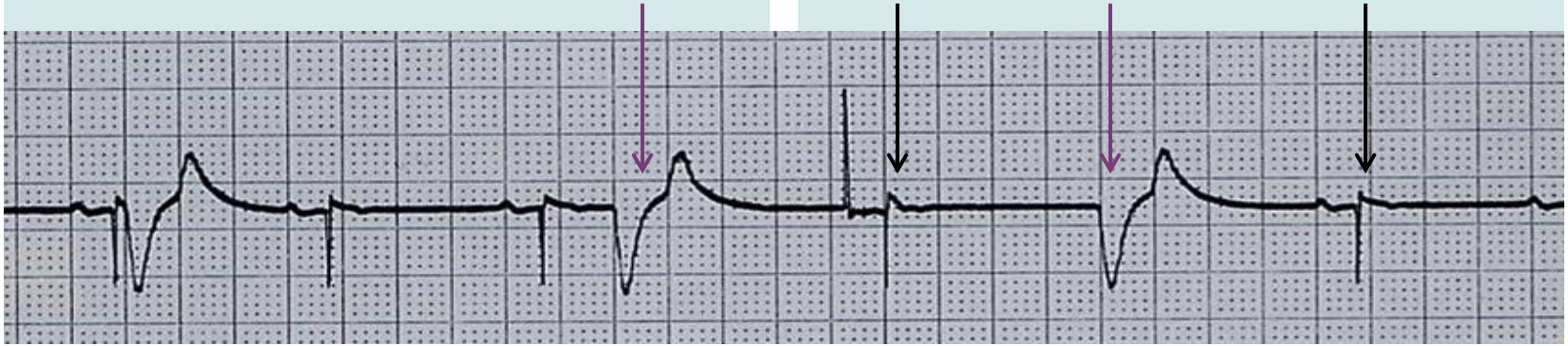


Abnormal pacing

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

Ventricular pacing spikes

QRS complexes



Failure to capture

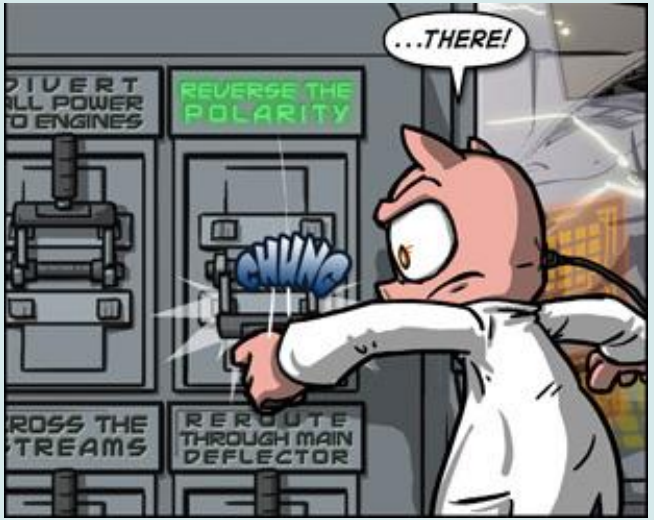
- **Causes:**
  - insufficient energy delivered by pacer
  - low pacemaker battery
  - dislodged, loose, fibrotic, or fractured electrode
  - electrolyte abnormalities
    - acidosis
    - hypoxemia
    - hypokalemia
- **Danger - poor cardiac output**

Failure to capture

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

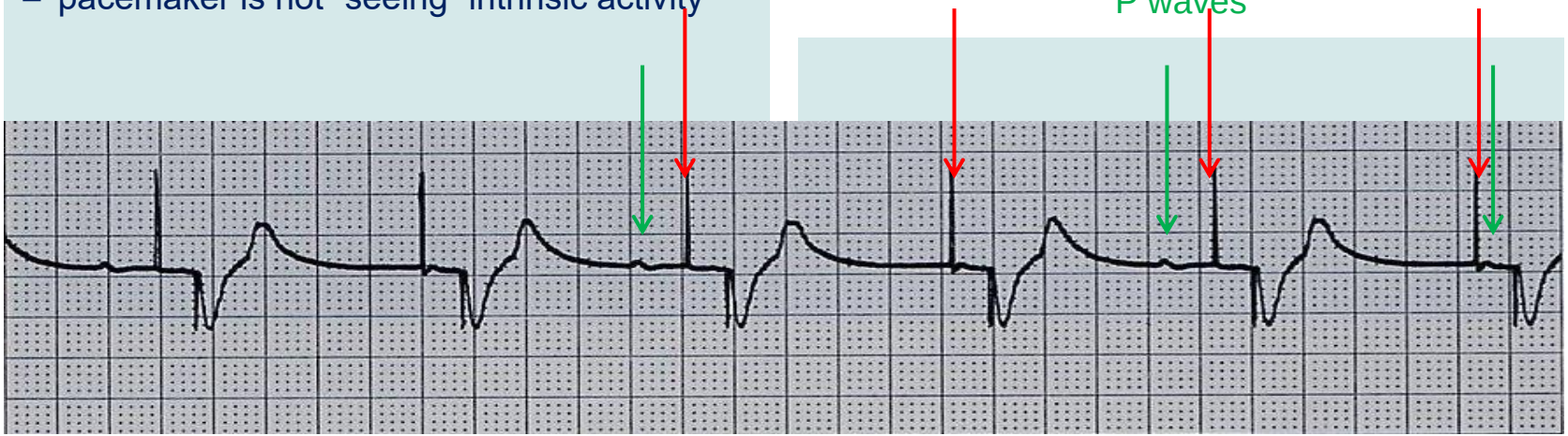
Reversing polarity

- **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

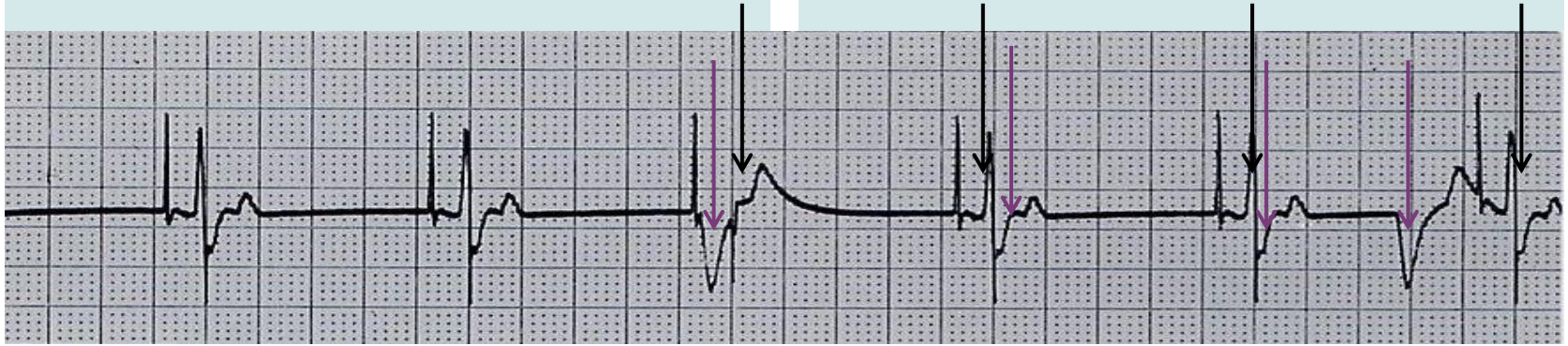


Abnormal pacing

- **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

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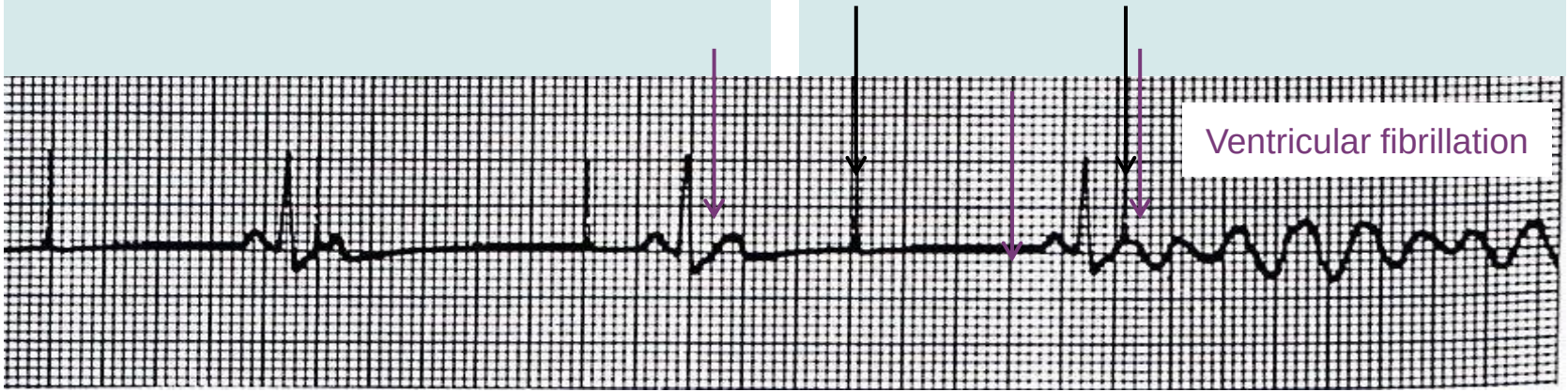
  R- on -T phenomenon

Ventricular pacing spikes

QRS complexes

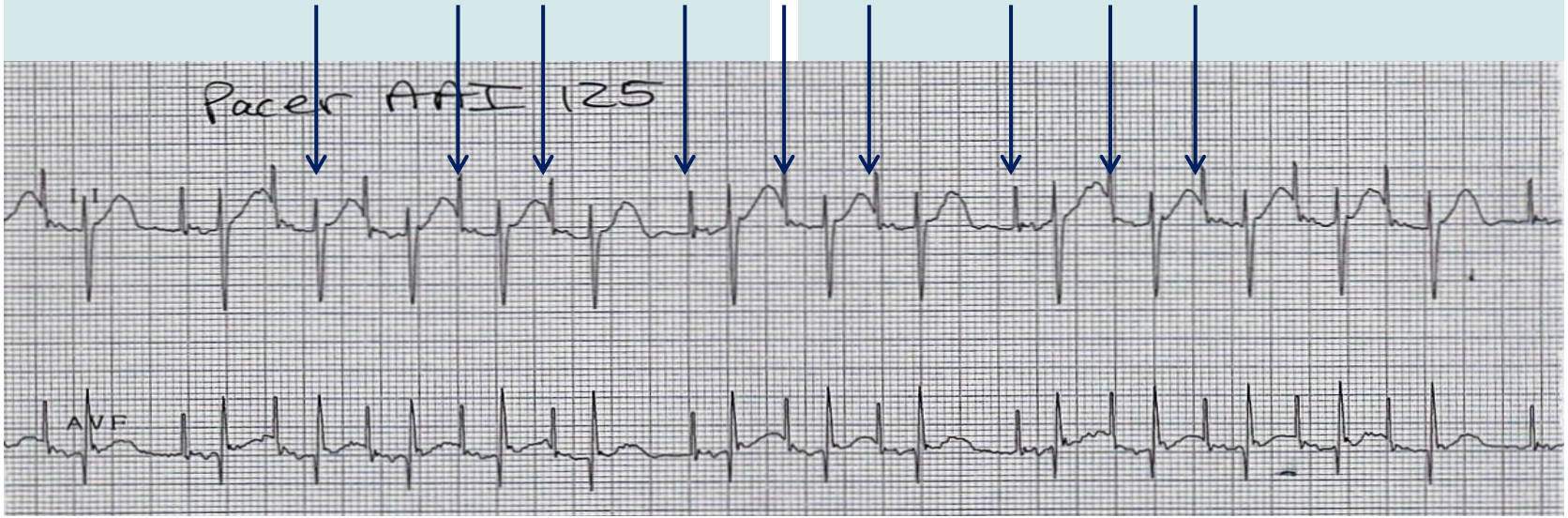


Failure to sense

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

Oversensing

- Pacing does not occur when intrinsic rhythm is inadequate



Oversensing

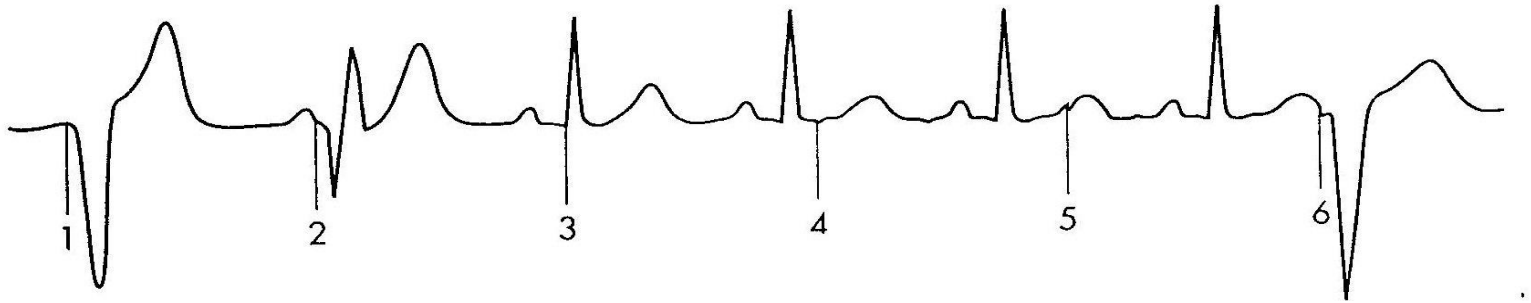
- **Causes:**
  - pacemaker inhibited due to sensing of “P” waves + “QRS” complexes that do not exist
  - pacemaker too sensitive
  - possible wire fracture, loose contact
  - pacemaker failure
- **Danger:**
  - heart block, asystole

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

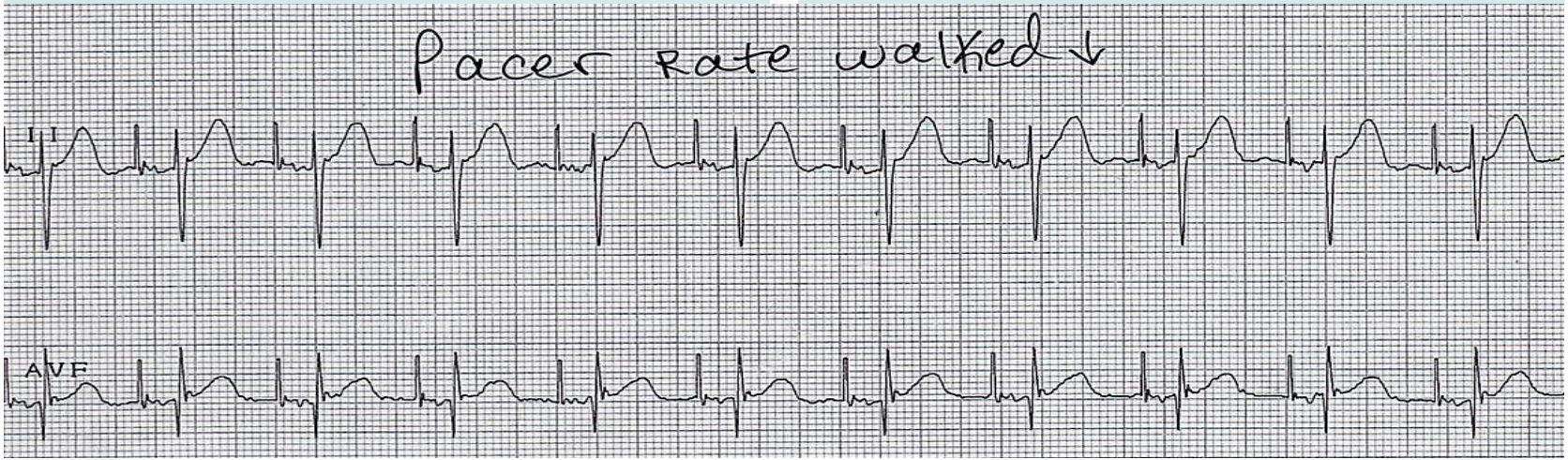
- **Causes:**
  - asynchronous pacing
  - failure to sense
  - mechanical failure: wires, bridging cables, pacemaker
  - loose connections
- **Danger**
  - impaired cardiac output
  - potential (low) for paced ventricular beat to land on T wave

Competition

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

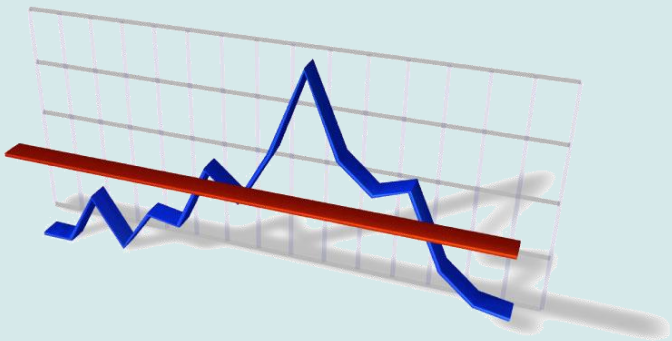
Assessing Underlying Rhythm

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



Threshold testing

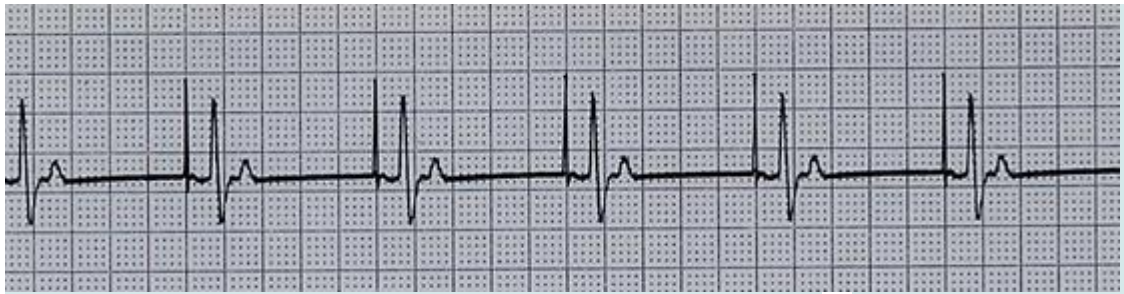
- **Stimulation threshold:**
  - Definition: Minimum current necessary to capture + stimulate the heart
- **Testing:**
  - set pacer rate 10 ppm faster than patient's HR
  - decrease mA until capture is lost
  - increase output until capture is regained (threshold capture)
  - output setting to be 2x's threshold capture



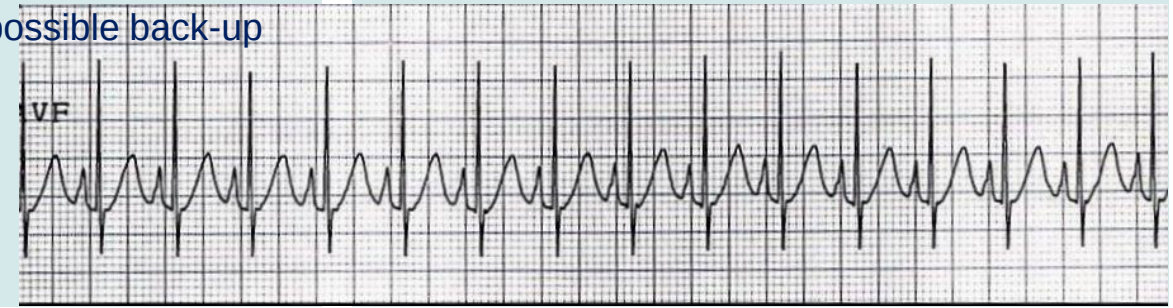
Sensitivity threshold testing

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

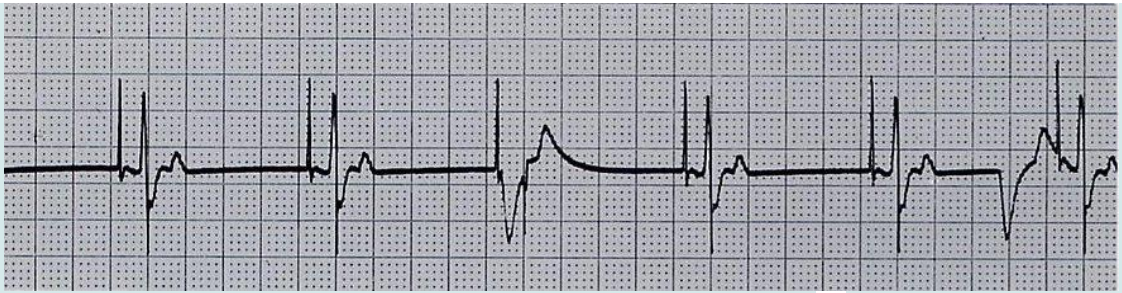
Mode of pacing, rhythm/problem, solution



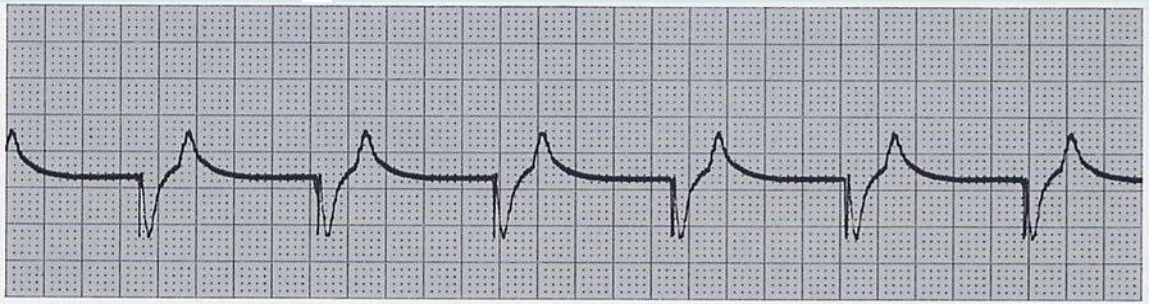
- AAI: normal atrial pacing
- Sinus rhythm: no pacing; possible back-up setting AAI, VVI, DDD



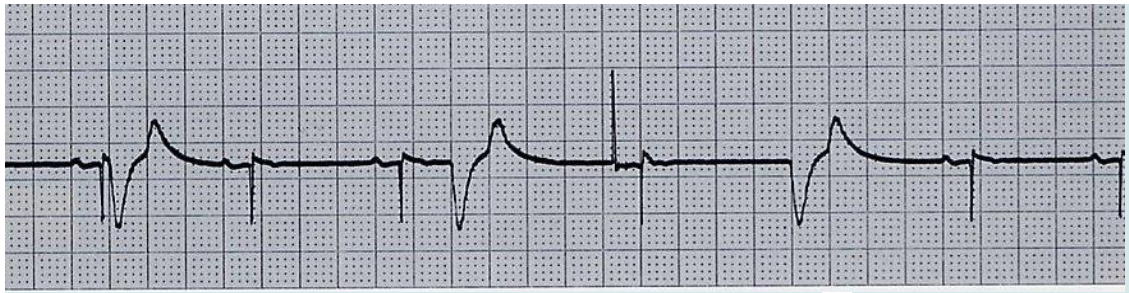
Mode of pacing, rhythm/problem, solution



- DDD: failure to sense ventricle; increase ventricular mA
- VVI: ventricular pacing

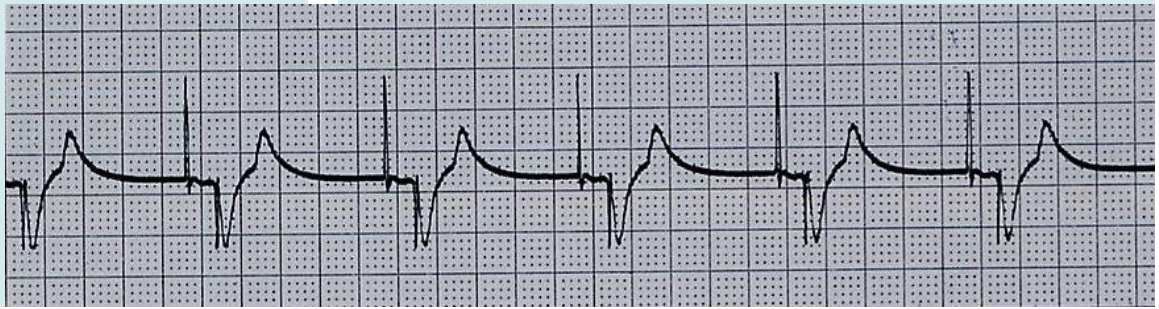


Mode of pacing, rhythm/problem, solution

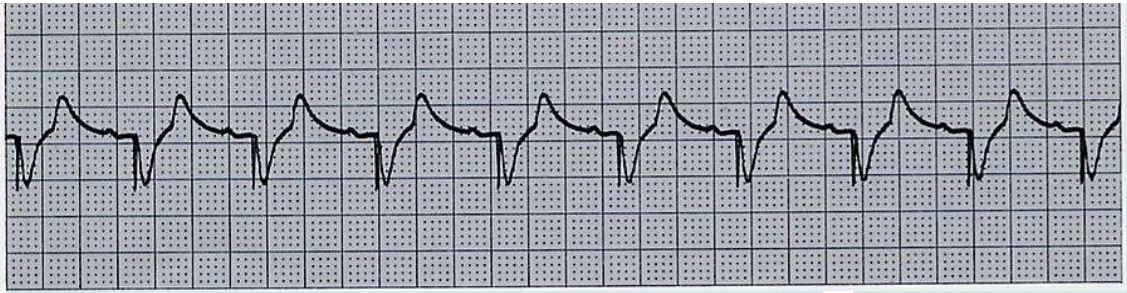


- DDD: failure to capture atria or ventricle; increase atrial & ventricular mA

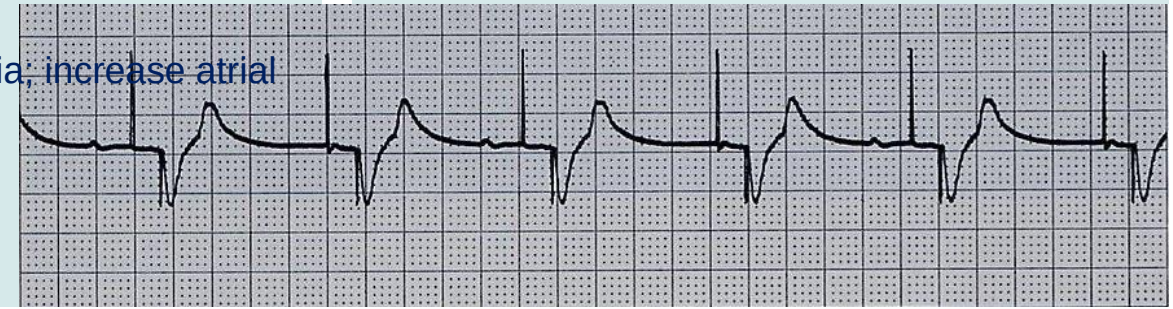
- DDD: normal atrial & ventricular pacing



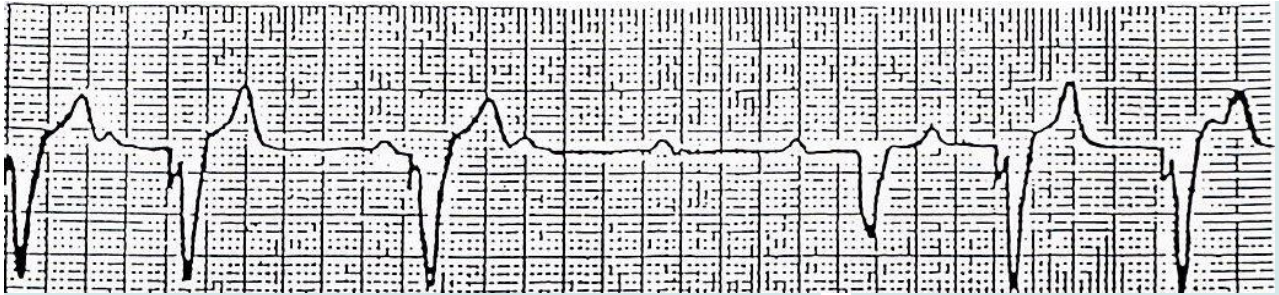
Mode of pacing, rhythm/problem, solution



- DDD: normal atrial sensing, ventricular pacing
- DDD: failure to capture atria; increase atrial mA



Mode of pacing, rhythm/problem, solution



- DDD: oversensing; decrease ventricular sensitivity

Thank you for your attention