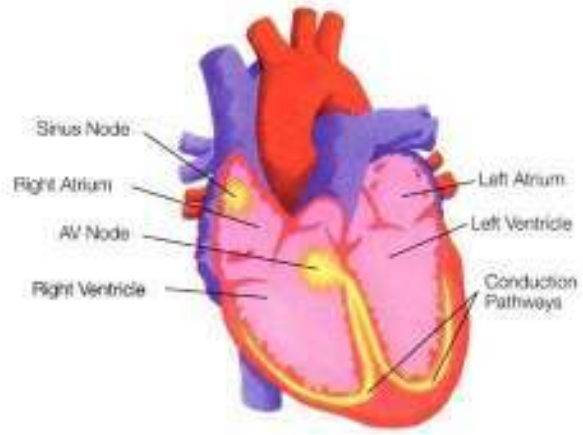
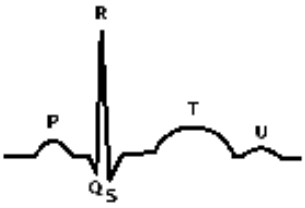
CARDIOVASCULAR SYSTEM
&
INOTROPES

To understand inotropes and their use, there must first be an understanding of the cardiovascular system.

Conduction





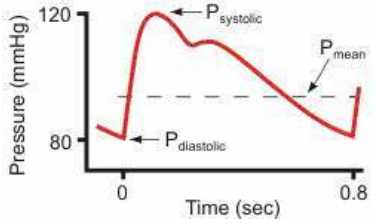
P wave: atrial depolarisation
QRS complex: depolarisation of ventricles
ST: transient period when no electronic current can pass through the myocardium
T wave: ventricles returned to resting phase

Blood pressure

When the heart contracts, blood is pumped out into the body. The maximum pressure measured in the arteries is called **systolic** pressure.
When blood fills the heart, the artery pressure relaxes and this is called **diastolic** pressure.
We measure blood pressure in mmHg, and monitor both the systolic, diastolic and mean pressure. It is determined by **cardiac output** (flow) and **systemic vascular resistance.**

Flow to the tissues is crucially dependent on mean blood pressure (MAP).

This is calculated by adding the diastolic pressure and 1/3 of the pulse pressure



Normal blood pressure

Age	Systolic	Diastolic
Birth (12 hours < 1000g)	39-59	16-36
Birth (12 hours, 3kg)	50-70	25-45
Neonate (96 hours)	60-90	20-60
Infant (6 months)	87-105	53-66
Toddler (2years)	95-105	53-66
School age	97-112	57-71
Adolescent (15 years)	112-128	66-80

Blood pressure (systolic) can be approximated over the age of 1 year by using the formula: 80 mmHg + (2 x age in years) [APLS]

Even when the blood pressure falls to 75% of normal, the tissues try to preserve metabolic functions. It does this by extracting more oxygen and nutrients from the blood supply still reaching them. Only at very low levels of tissue perfusion do the tissues give up and resort to anaerobic metabolism.

Cardiac output

Cardiac output is the amount of blood pumped by the ventricle per minute. It is determined by **stroke volume x heart rate**

what affects cardiac output?

systemic vascular resistance
circulating volume / cvp
contractility
sedation
pain

Heart rate

Heart rate varies with age and with activity

Age (years)	Heart rate (bpm)
<1	110-160
1-2	100-150
2-5	95-140
5-12	80-120
>12	60-100

An increase in temperature will raise a heart rate by 10bpm per degree of increase
Agitation, distress, pain all increase the heart rate
A variety of rhythm disturbances may compromise cardiac output.
Sinus tachycardia may give insufficient time for effective diastolic ventricular filling.

In **infants**, stroke volume is relatively fixed (1.5ml/kg at birth), so cardiac output is reliant on changes in heart rate only. Therefore the **heart rate** is the main cause of increase or decrease in cardiac output.

Stroke volume

Stroke volume is the amount of blood pumped out of the ventricle with each heartbeat. It is determined by preload, cardiac contractility & afterload.

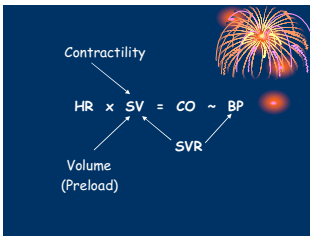
pre-load – the degree of myocardial stretch at the end of diastole. That is, the amount of blood available for expulsion by the myocardium
Preload is affected by:
any change to the circulating blood volume – dehydration ↓, haemorrhage ↓, hypovolaemia ↓
any change in the blood returning to the heart – vasoconstriction↑ / vasodilation ↓
any change to ventricular filling time – heart failure ↓, tamponade ↑, change in heart rate ↓ ↑

The greater the preload the greater the stroke volume and therefore the greater the cardiac output. However, it is possible to overstretch the myocardium, resulting in cardiac compromise.

cardiac contractility – the ability of the myocardium to contract effectively
May be reduced by myocardial oedema and decreased ventricular compliance that may follow damage such as in cardiomyopathy or post cardiac surgery. The myocardium can be further compromised by volume or pressure overload, which causes ventricular dilation, hypertrophy and ischaemia

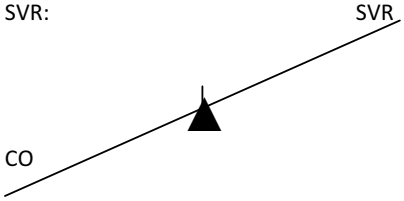
afterload

the resistance the ventricle must overcome in order to move blood in the pulmonary artery and aorta.
This can be divided into **systemic vascular resistance (SVR)** &
pulmonary vascular resistance (PVR)
SVR: resistance to blood flow through the arteries (left ventricle)
PVR: resistance to blood flow through pulmonary arteries (right ventricle)



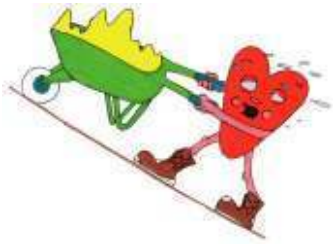
Increasing SVR improves the MAP but may reduce cardiac output to a level where tissue perfusion is reduced

SVR: SVR



CO

Increasing SVR may be needed in a vasodilated patient ie anaphylaxis or sepsis, in order to maintain MAP at a level able to perfuse, but if cardiac output is reduced, increasing SVR will have a negative effect by further reducing cardiac output while increasing BP (so making you feel better)



Remember: increasing blood pressure without improving cardiac output may lead to organ perfusion being compromised

The greater the preload, the greater the stroke volume and therefore the greater the cardiac output

↓ afterload = ↑ CO

↑preload → ↑ SV → ↑ CO

↑ ventricular function → ↑ CO

Cardiovascular support and inotropes

The body produces **catecholamines** which are a group of physiologically important substances including adrenaline, noradrenaline and dopamine.
They have different roles – mainly as **neurotransmitters** – in the functioning of the sympathetic and central nervous system. They occur naturally within the body.

Normally this is automatically controlled and can increase or decrease according to the body's need.
In stress the production increases to provide '**fight or flight**' response - see below
The body's coping mechanism can only compensate for so long before the body's equilibrium is challenged and becomes unable to maintain normostasis
Improvement in cardiac function / myocardial contractility at this time can be brought about by supplementing the body's own response with the use of catecholamine agents. These will stimulate the action of the sympathetic nervous system
For ease of understanding and clinical practice they can be simply classified as either **inotropes** or **vasopressors.** Some drugs however do both

An **inotrope** is an agent which increases or decreases the force of muscular contraction of the heart. It acts on α, β1 & β2 receptors
alpha (α): peripheral vasoconstriction
increased systemic vascular resistance
beta 1(β-1): increased heart rate
increased AV conduction velocity
increased ventricular contractility
beta 2 (β-2): peripheral vasodilation
bronchodilatation

Inotropes and their reactions

	ALPHA	BETA 1	BETA 2	COMMENTS
Dobutamine		++	+	Can be given peripherally
Dopamine	+	++	+	
Adrenaline	++	+++	++	Can be given peripherally if desperate
Noradrenaline	+++	+		

Positive inotropes are known as sympathiomimetic drugs. They stimulate contraction, directly affecting heart rate, peripheral perfusion, blood pressure and urinary output.
They include dopamine, dobutamine and adrenaline
Negative inotropes block β-adrenoreceptors in the heart, peripheral vessels, bronchi, pancreas and liver.
They include labetalol and propanolol
A **vasopressor** drug acts on α1-adrenergic receptors by increasing peripheral vascular tone and increasing systemic vascular resistance which leads to an elevation of blood pressure.
They include adrenaline & noradrenaline

In theory, inotropes treat impaired cardiac contractility and vasopressors treat peripheral vascular failure
Most drugs used for cardiovascular support within Critical Care display both inotropic and vasopressor activity.

Adrenaline and **noradrenaline** are **vasoconstrictive sympathomimetics**. They raise the blood pressure transiently by acting on the α-adrenergic receptors to constrict the peripheral vessels. But at the same time they reduce the perfusion of the kidneys and other vital organs.
Dopamine, dobutamine and **isoprenaline** are **inotropic sympathomimetics.** Dopamine & isoprenaline act on the β-receptors in the cardiac muscle and increase contractility with little effect on the heart rate, whereas dobutamine will increase the heart rate

Indications for use: **to maximise cardiac output and oxygen delivery to the tissues when other measures have failed to achieve the desired effect.**
They should be titrated to provide maximal therapeutic effects with minimal side or toxic effect

There is a very rapid onset and rapid offset
Except for milrinone, which has about a 20 minute onset and half-life of 4-6 hours

Down regulation
Critically ill patients can become resistant to effects of pressor agents
This is more prevalent in those with septic shock

aim:
to optimise distribution of cardiac output
use minimum effective dose to achieve desired outcome
to wean and discontinue to avoid undesirable side effects

‘fight & flight’ response

In response to stress or fear, our bodies automatically prepare themselves for fighting or fleeing. This is an autonomic response
The sympathetic impulses initiate an “emergency” system. The ongoing sympathetic signals increase greatly.

The body prepares to expend maximum energy by:

- ✓ Increasing heart rate: increases delivery of oxygen and glucose to the skeletal muscles
- ✓ Increasing contractility: increases rate of blood flow and increases delivery of oxygen and glucose to the skeletal muscles
- ✓ Dilation of coronary vessels of the heart: increases oxygen and nutrients to the cardiac muscles to sustain increased heart rate and contractility
- ✓ Constriction of blood vessels in the digestive and other organs: shunts blood to skeletal muscles to increase oxygen and glucose
- ✓ Contraction of spleen and other blood reserves: increasing general blood circulating the body which increases the oxygen and glucose to the skeletal muscles
- ✓ Dilation of respiratory airways: increases the oxygen load into the blood
- ✓ Increased repiratory depth and rate: increases the oxygen load into the blood
- ✓ Increased sweating: increased dissipation of heat generated by skeletal muscle
- ✓ Increased conversion of glycogen into glucose: increases the amount of glucose available to the skeletal muscle

In other words: faster, stronger heartbeat, dilated blood vessels in skeletal muscle, dilated bronchi, increased blood sugar levels from conversion of glycogen to glucose

Sympathetic effectors such as the heart, smooth muscle, glands have receptors for noradrenaline
Both adrenaline and noradrenaline can bind to the receptors of the sympathetic effectors to prolong and enhance the effects of the sympathetic stimulation by the autonomic nervous system

In simple terms, the body produces adrenaline, noradrenaline and dopamine. Normally this is controlled and can increase or decrease according to the body's need.
In stress, the production increases to provide the 'fight or flight' response. The effects on the body are detailed below, showing the sympathetic receptors as described above.

The body's coping mechanism can only compromise for so long before the body's equilibrium is challenged and becomes unable to maintain normostasis.
Improvement in cardiac function/myocardial contractility at this time can be brought about by the use of dopamine, dobutamine, and adrenaline. These will increase the heart rate, atrioventricular conduction velocity and ventricular contractility.

When a child presents as sick:

When children present as very sick, this fight or flight response is usually running at full tilt. They are compensating and running at maximum output.
Lower blood pressure is a very late sign of how sick they are. Fluid boluses may help the immediate situation, by increasing the circulating volume

When you give this child any sort of sedative or anaesthetic agent for intubation, you are immediately taking away their fight and flight response. It is this response that has been maintaining their heart rate and blood pressure and once you take away the drive that releases the increased release of catecholamines, then the child's cardiovascular function will plummet. Their heart rate may well drop, their BP will fall and you have an immediate emergency situation.

If considering intervention and ventilation in the sick child, and the blood pressure is lowered, it is wise to consider making up inotropes prior to intubation.
If not, then **have emergency drugs at hand**.
If time permits, gain access, give resuscitation fluids of 20mls/kg and reassess, prior to intubating and sedating.

If inotropes are being considered, then the child will need a lot of monitoring and constant review.
Draw up some emergency drugs and make up some inotropes – often adrenaline and dopamine to start with.
Once the situation is stabilised, then consider rationalising the inotropes, but initially the adrenaline will increase the child's heart rate and cardiac output, and the dopamine will increase the BP and improve CO.
Noradrenaline may also be used, but dobutamine should only be initiated once the circulating volume / CVP is adequate

What monitoring do they need?

Because these children are receiving drugs that have an immediate and constant effect on their cardiac output, they must be constantly monitored:

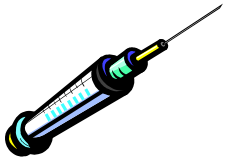
Heart rate
Blood pressure - by an arterial line
CVP
Temperature
Urinary output
Blood sugar

Why?

- effects of drugs continually act upon the heart and circulating volume. Changes in any parameter can indicate changes within the child's condition and must be reviewed and reassessed. Their requirement for the drug can change very quickly
- changing the syringe over can have extreme effects on the heart rate and blood pressure, so must be titrated and changed very carefully, observing HR & BP continually whilst changing over. NEVER let these syringes run out!

Who can make up inotropes?

All senior staff and those that have undertaken training and assessment can make up and check inotropes with any staff who have their IV certificate. Staff should not change over the syringes unless competent to do so or are being supervised



drug calculations:

drug – 3mgs x weight in 50 mls (dopamine, dobutamine, GTN, milrinone)
0.3mgs x weight in 50 mls (adrenaline, noradrenaline, isoprenaline)

eg **20 kg child to start an IVI of 5 mcg/kg/min of dopamine**
3 x 20 = 60 mgs in total of 50 mls of 5% dextrose

to calculate out: change mgs into mcgs	x 1000	= 60000 mcgs
mcgs/ml	/ volume i.e. 50	= 1200mcgs/ml
mcgs/ml/min	/ time i.e. 60	= 20mcgs/ml/min
mcgs/kg/ml/min	/ weight i.e. 20	= 1mcg/kg/ml/min
∴ **5mcgs**/kg/min	x rate i.e. 5	= 5mcgs/kg/min

eg **5 kg child to start an IVI of 0.2 mcgs/kg/min of adrenaline**
0.3 x 5 = 1.5 mgs in total of 50 mls of 5% dextrose

to calculate out: change mgs into mcgs	x 1000	= 1500 mcgs
mcgs/ml	/ volume i.e. 50	= 300mcgs/ml
mcgs/ml/min	/ time i.e. 60	= 5mcgs/ml/min
mcgs/kg/ml/min	/ weight i.e. 5	= 0.1mcg/kg/ml/min
∴ **0.2** mcgs/kg/min	x rate i.e. 2	= 0.2mcgs/kg/min

Adrenaline (Epinephrine)

Action:
Acts directly on alpha and beta receptors with levels changing according to the physiological status

- ✓ Imitates most actions of SNS except for the face& sweat glands
- ✓ Strengthens myocardial contraction, increasing cardiac output & rate
- ✓ Increases systemic vascular resistance
- ✓ Increases coronary artery perfusion pressure, increasing myocardial oxygenation
- ✓ Increases systolic but may decrease diastolic blood pressure
- ✓ Causes bronchial smooth muscle relaxation
- ✓ Constricts bronchial arterioles and inhibits histamine release – this reduces congestion and oedema, increasing tidal volume and vital capacity
- ✓ Constricts arterioles in the skin, mucous membranes and kidneys
- ✓ Dilates skeletal muscle blood vessels
- ✓ Raises blood sugar by promoting conversions of glycogen reserves in the liver to glucose and inhibits insulin release in the pancreas
- ✓ CNS stimulation believed to result from peripheral effects

Uses:

- ❖ **cardiac arrest**
- ❖ symptomatic bradycardia unresponsive to ventilation and oxygen
- ❖ poor systemic perfusion
- ❖ **hypotension with good CVP and stable rhythm**
- ❖ anaphylactic reactions
- ❖ acute asthmatic attack
- ❖ temporary relief of bronchospasm

Dose: arrest – 0.1ml/kg of 1:10 000 (0.01mg/kg)
Repeat as necessary

IVI: single strength made up as 0.3mg/kg in 50mls 5% glucose
= 0.1mcg/kg/min at 1ml/hr
adjust rate until ↑ HR / BP or systemic perfusion reaches acceptable level

nebuliser: 1ml of 1:1000 adrenaline diluted with 3mls saline

precautions / side effects

- ➢ metabolic acidosis – may decrease effect so should be corrected
- ➢ tachyarrythmias
- ➢ hypertension
- ➢ hyperglycaemia
- ➢ increased myocardial oxygen demand
- ➢ rates higher than 0.6mcg/kg/min cause profound vasoconstriction that can compromise extremities and skin perfusion
- ➢ lower doses may decrease renal and hepatic blood flow
- ➢ infiltration causes necrosis and ischaemia
- ➢ systemic reactions: nervousness, restlessness, anxiety, tremors, nausea, sweating, vomiting, dyspnoea, weakness, dizziness
- ➢ bronchial and pulmonary oedema
- ➢ urinary retention

Noradrenaline

Action:

- ✓ acts directly on alpha adrenergic receptors
- ✓ acts little on beta receptors except on the heart
- ✓ main therapeutic effect: vasoconstriction & cardiac stimulation
- ✓ reduces blood flow to the kidney, other vital organs, skin, skeletal muscle
- ✓ peripheral vasoconstriction
- ✓ inotropic stimulation of the heart which causes an
    - increase in systolic and diastolic pressure,
    - myocardial oxygenation,
    - coronary artery blood flow
    - myocardial output
- ✓ cardiac output varies with systemic blood pressure
- ✓ bradycardia
- ✓ causes less CNS stimulation and less effect on metabolism than adrenaline
- ✓ large doses can cause glycogenolysis and inhibits pancreatic release of insulin, so causing hyperglycaemia

uses:

- ❖ restore blood pressure in acute hypotensive states, **primarily used in shock**
- ❖ cardiac arrest
- ❖ useful in hypercyanotic episodes like Fallots, Tricuspid atresia, to overcome peripheral vasodilation
- ❖ useful in pulmonary vascular disease to overcome peripheral systemic vasodilation
- ❖ sepsis with significant vasodilation

dose: 0.3mg/kg in 5% glucose at 1ml/hr = 1mcg/kg/min
0.01 – 0.5mcgs/kg/hr

precaution / side effects

- ➢ significantly increases myocardial workload
- ➢ reducese peripheral tissue perfusion
- ➢ may cause hypoxia or hypercapnia
- ➢ hypertension
- ➢ bradycardia
- ➢ arrythmias
- ➢ respiratory difficulties
- ➢ headache
- ➢ tissue necrosis at infusion site
- ➢ CNS: tremors, dizzieness, restlessness, anxiety, weakness
- ➢ Hyperglycaemia
- ➢ Prolonged use: plasma volume depletion
    - Oedema
    - Haemorrhage
    - Intestinal hepatic and renal necrosis

Dopamine

Action

- ✓ 2-5mcg/kg/min: increases renal bloodflow (D receptors)
  - splanchic
  - coronary
  - cerebral blood flow
- ✓ 5-10mcg/kg/min: increases cardiac contractility without raising HR & BP
  - increases cardiac output
  - direct stimulation of cardiac B 1 adrenergic receptors
  - indirect cardiac stimulation thorough the release of noradrenaline stored in cardiac sympathetic nerves
  - may cause a recution in cardiac output in neonates
- ✓ 10-20mcg/kg/min: vasoconstriction
  - increases vascular resistance
  - tachycardia

uses:

- ❖ low cardiac output
- ❖ hypotension
- ❖ poor peripheral perfusion when adequate intravascular volume and stable rhythm

dosage: 3mg/kg in 50mls 5% glucose at 1ml/hr = 1mcg/kg/min single strength
2-10mcg/kg/min

precautions / side effects

- ➢ tachycardia
- ➢ increased oxygen demand from myocardium
- ➢ arrythmias especially SVT & VT
- ➢ hypertension
- ➢ 20mcgs/kg/min or more: vasoconstriction peripherally and ischaemia in the shocked child
- ➢ central infusion only if more than 5mcgs/kg/min
- ➢ local ischaemia and necrosis if extravasation of peripheral IVI
- ➢ inactivated in alkaline solutions

Dobutamine

Action

- ✓ Synthetic catecholamine
- ✓ Acts directly on B-adrenergic receptors, not dependent on noradrenaline stores
- ✓ Increases cardiac contractility
- ✓ Improves cardiac output
- ✓ Increased heart rate with mild peripheral dilation of vascular bed
- ✓ Increases cardiac output in cardiogenic shock
- ✓ Decreases pulmonary capillary pressure and systemic vascular resistance
- ✓ Less effective than adrenaline in septic shock as it may increase existing systemic vasodilation

Uses

- ❖ Hypoperfusion if associated with ↑systemic vascular resistance
- ❖ Severe congestive cardiac failure
- ❖ Cardiogenic shock – especially if caused by cardiomyopathy as it decreases peripheral vascular resistance

Dose: 3mg/kg in 50mls 5% glucose = 1mcg/kg/min at 1ml/hr
5-10mcg/kg/min
adjust rate as required to stabilise BP and perfusion

Precautions and side effects

- ➢ tachycardia
- ➢ tachyarrhythmias
- ➢ ectopic beats
- ➢ nausea / vomiting
- ➢ hypertension
- ➢ extravasation – tissue necrosis & ischaemia
- ➢ inactivated slowly if in alkaline solutions

Milrinone

A selective inhibitor of phosphodiesterase III – an enzyme responsible for catalysing the breakdown of cAMP
Found in high concentration in cardiac and vascular smooth muscle

Action:

- ✓ Inhibition of phosphodiesterase III leads to accumulation of cAMP (cyclic adenosine monophosphate) resulting in:
  - ❖ a positive inotrope reaction and
  - ❖ vasodilation
- ✓ an inotrope and afterload reducer (inodilator)
- ✓ Bronchodilatation
- ✓ Slightly increases heart rate
- ✓ Increases contractility
- ✓ **Vasodilation in skeletal muscle**

Uses:

- ❖ congestive cardiac failure
- ❖ decreased cardiac output and
- ❖ increased filling pressure
- ❖ post cardiac surgery

Milrinone has about a 20 minute onset and half-life of 4-6 hours
Can be given peripherally

Dose: **:** Loading 50mcg/kg over 10-20 mins
Then 0.3 – 0.75 mcg/kg/min
NB a monograph will be specifically calculated for each individual child, indicating the dose and volume to be made up. Pharmacy will prepare this at their earliest convenience.
However, should the child come in at night, then the following is recommended:
dilute a 10ml ampoule into 25mls of 5% glucose to give 400mcg/ml.
This is the most concentrated strength able to be given peripherally.
If the child has a central line, then dilute 20mlsof milrinone into 25mls of solution. This would give a strength of 800mcg/ml.
If the child is very fluid restricted then it can be given neat.

Precautions / side effects:

- ➢ hypotension – consider a fluid bolus when initiating treatment
- ➢ arrhythmias
- ➢ incompatible with frusemide:-> precipitation

Isoprenaline

Action:

- ✓ B-adrenergic agonist
- ✓ Increased heart rate
- ✓ Increased atrial ventricular conduction
- ✓ Increased cardiac contractility
- ✓ Increased myocardial oxygen consumption – causes peripheral vasodilation particularly in skeletal muscle
- ✓ Cardiac output increases if circulating blood volume adequate
- ✓ Pulse pressure increases because diastolic pressure falls with dilation of the blood vessels
- ✓ Bronchodilation

Uses:

- ❖ Generally now used as a short term emergency treatment of heart block or severe bradycardia
- ❖ bradycardia with poor perfusion adrenaline may be preferred as it is less likely to decrease diastolic pressure

dose: 0.3mg/kg in 50mls 5% glucose at 1ml/hr = 0.1mcg/kg/min
0.1-0.5mcg/kg/min
may require to go up to 1mcg/kg/min but results in tachycardia.

Precautions / side effects

- ➢ Isoprenaline may compromise coronary perfusion because it has no alpha-adrenergic effects and decreases diastolic pressure
- ➢ Increases myocardial oxygen consumption by increasing myocardial contractility and heart rate
- ➢ Increased heart rate may further decrease coronary perfusion by decreasing diastolic filling time
- ➢ NOT given in arrest
- ➢ Isoprenaline cardiotoxicity following IVI with asthmatic child
- ➢ arrhythmias,
- ➢ tachycardia,
- ➢ hypotension,
- ➢ sweating
- ➢ headache

Glyceryl Trinitrate

Action:

- ✓ Systemic venous and arterial vasodilator
- ✓ Pulmonary vasodilation
- ✓ Relaxes stiff ventricles
- ✓ Acts on veins and coronary vessels
- ✓ Potent coronary vasodilator
- ✓ Decreases venous return and therefore decreases left ventricular work

Uses:

- ❖ Left ventricular failure

Dose: 3mcg/kg in 50mls 5% glucose at 1ml/hr = 1mg/kg/min
0.5-5mcg/kg/min

precautions / side effects

- ➢ maximum concentration 1mg/ml
- ➢ tachycardia – care with administration
- ➢ non PVC giving set – reactive to light
- ➢ hypotension
- ➢ headache
- ➢ flushing
- ➢ dizziness
- ➢ hypothermia

Propanolol

action

- ✓ non-selective beta blocker of cardiac and respiratory adrenoreceptors.
- ✓ competes with adrenaline and nonadrenaline for available beta-receptor sites
- ✓ blocks cardiac effects of beta-adrenergic stimulation causing decreased heart rate and decreased myocardial irritability
- ✓ decreased force of contraction
- ✓ depresses automaticity of sinus node and ectopic pacemaker
- ✓ higher doses depress cardiac function
- ✓ also blocks the bronchodilator effect of catecholamines and decreases plasma levels of free fatty acids
- ✓ promotes sodium retention – often need a diuretic
- ✓ decreased platelet aggregability
- ✓ reninactivity suppressed, along with beta blocker effect causes decreased cardiac output causing hypotension

Uses

- ❖ hypertension
- ❖ arrythmias and thyrotoxic crisis
- ❖ obstructive cardiomyopathy
- ❖ myocardial infarction
- ❖ tachycardia's associated with digoxin toxicity
- ❖ anaesthesia
- ❖ hypertrophic subaortic stenosis

dose: 0.02mg's/kg test dose
then 0.1mg/kg (max 5mg) over 10 mins
repeat x 1-3 prn
then 0.1-0.3mg/kg/dose, 3 hourly

precautions / side effects

- ➢ pruritis, rash, allergy
- ➢ fever
- ➢ respiratory distress
- ➢ CNS: psychosis, sleep disturbances, depression, confusion, agitation, fatigue, syncope, weak, drowsy, hallucinations
- ➢ CVS: palpitations, profound bradycardia, AV heart block, hypotension, tachyarrythmia, acute CCF,
- ➢ Ear, eye: visual disturbances, conjunctivitis, tinnitus
- ➢ GI: nausea, vomiting, diarrhoea, constipation, abdominal pains, hypoglycaemia,
- ➢ Haematology: non-thrombocytopenic purpura, anaemia's, hypocalcaemia
- ➢ Resp: dyspnoea, laryngospasm, bronchospasm
- ➢ Skeletal muscle vasoconstricition