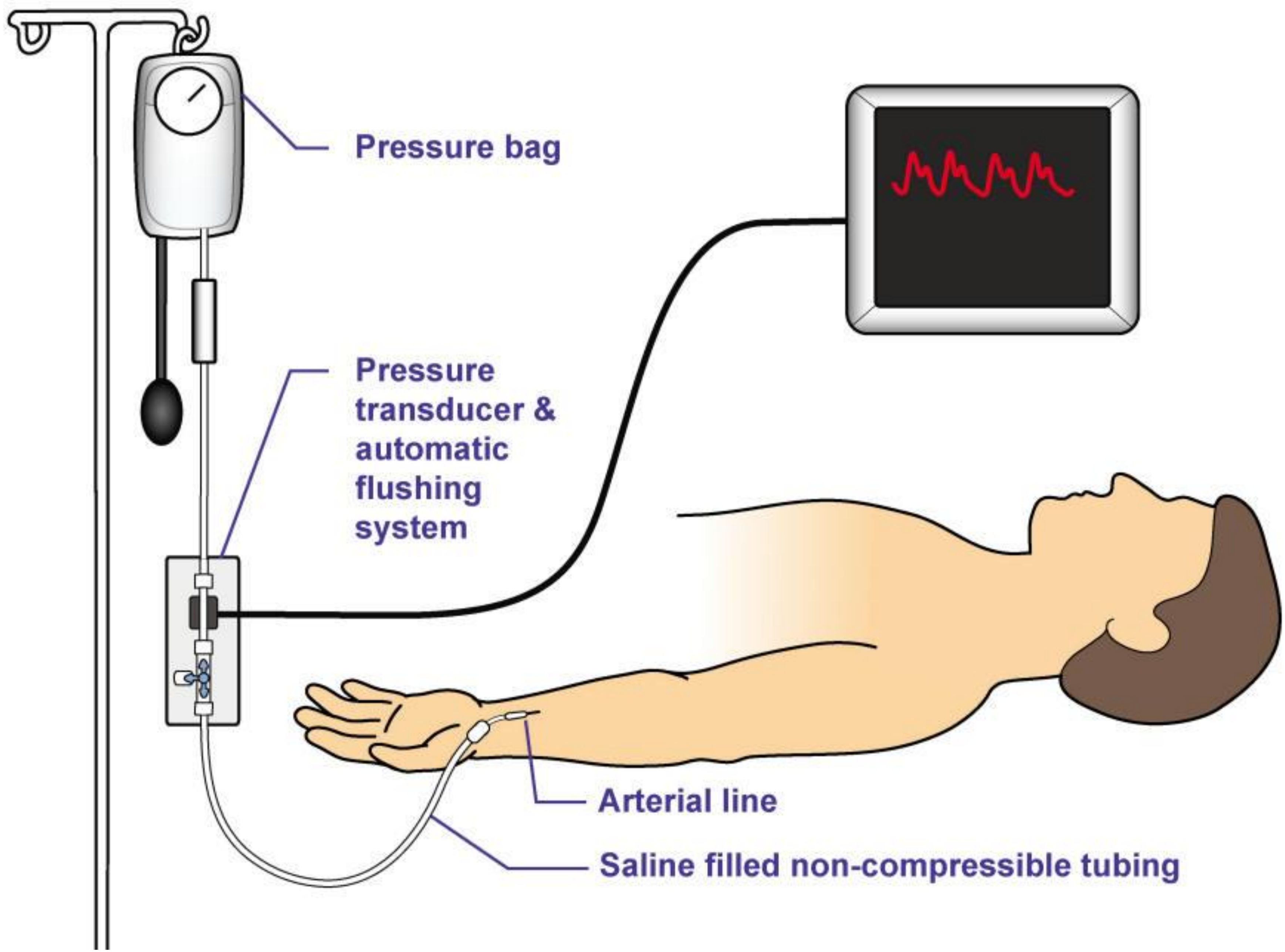
Arterial Lines

Helping to prevent a stab in the dark

Reasons for arterial line insertion

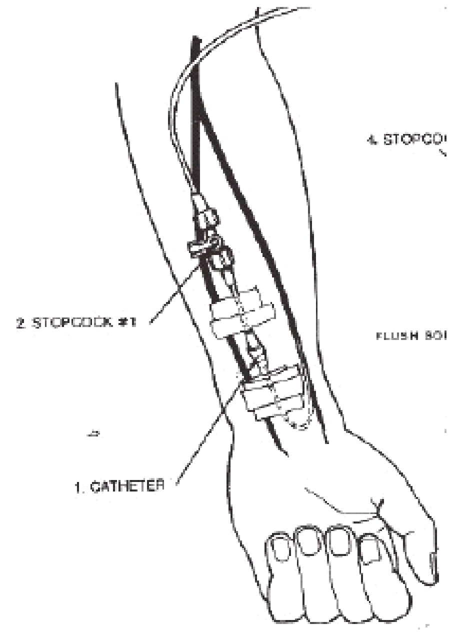
- Repeated blood gases required
  - Ventilated patients
  - Respiratory failure
  - Recognised acid base imbalance

- Continuous blood pressure monitoring
  - Sepsis
  - Ongoing fluid resuscitation
  - Cardiac instability

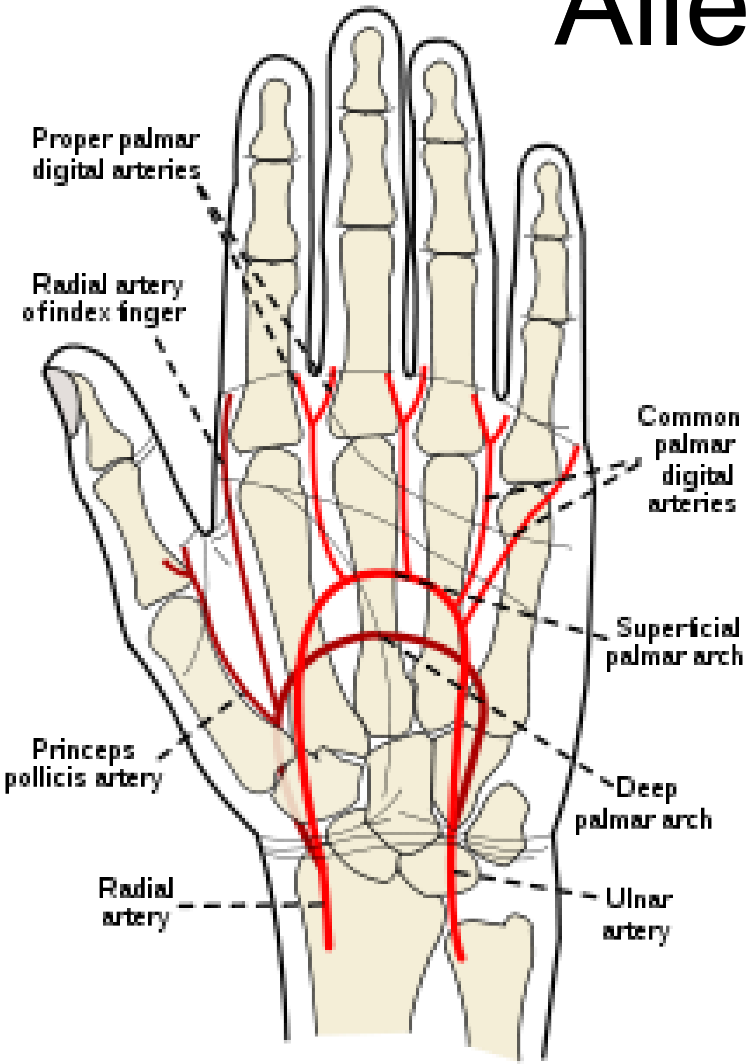

Pressure bag
Pressure transducer & automatic flushing system
Arterial line
Saline filled non-compressible tubing

Arterial Catheter Sites

- **Mostly Radial access**
  - **Advantages:**
    - **Easy access**
    - **Easy to identify**
    - **Easy to insert catheter**
    - **Minimal immobilisation of site**
    - **More comfortable than other sites**
  - **Disadvantages:**
    - **Thin catheter causing predisposition to overshoot artifact**
    - **Peripheral access ➔ Increase risk of Harmonic amplification**
    - **Vaso-reactive**
- **But also femoral, pedal, brachial, axillary access**



Allen' Test



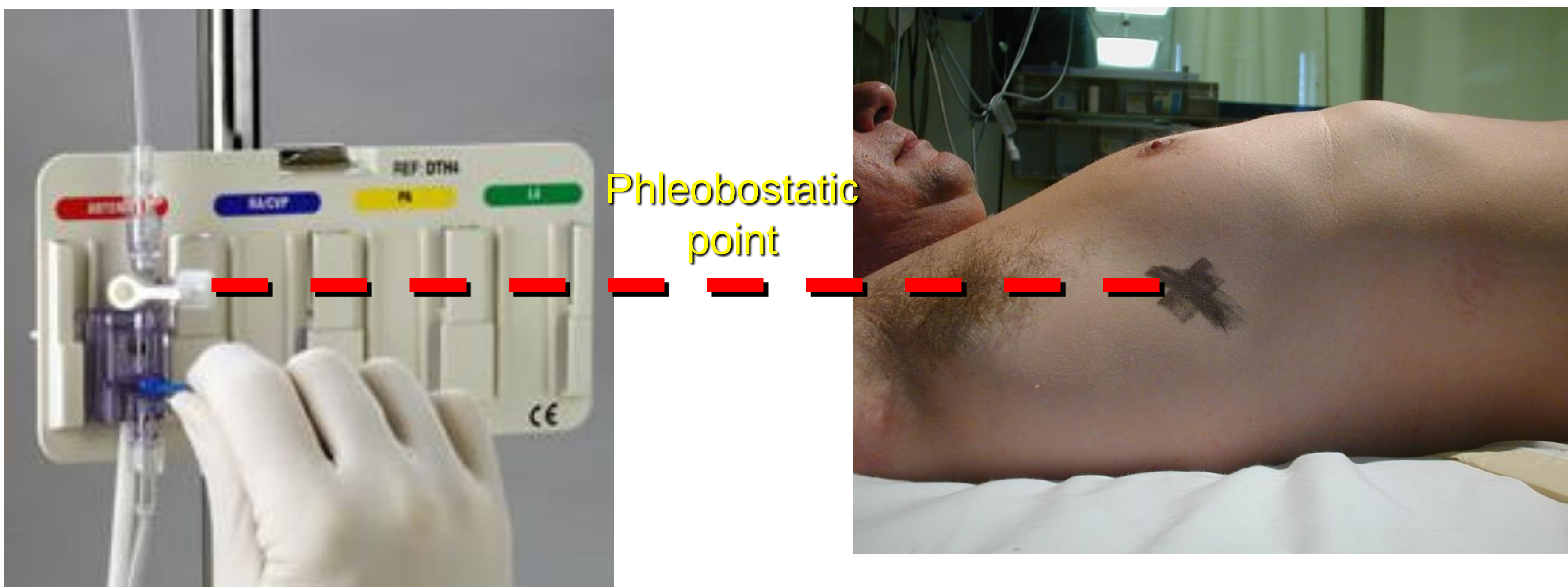
- The hand is elevated and the patient/person is asked to make a fist for about 30 seconds.
- Pressure is applied over the ulnar and the radial arteries so as to occlude both of them.
- Still elevated, the hand is then opened. It should appear blanched (pallor can be observed at the finger nails).
- Ulnar pressure is released and the colour should return in 7 seconds.
- If color returns as described above, the Allen's test is considered to be "positive." If color fails to return, the test is considered "negative" and the ulnar artery supply to the hand is not sufficient. The radial artery therefore cannot be safely pricked/cannulated.

Preparation

- Sterile dressing pack
- Sterile gloves
- Chlorhexidine swab
- Local anaesthetic
- Inco sheet
- Catheter set
- Pressure tubing
- Pressure bag
- IV solution

Zeroing & Leveling

1. Measure mid thorax
2. Identify 4^{th} ICS
3. Mark reference position
4. Measure pressures supine to 45^{o}



Safety issues

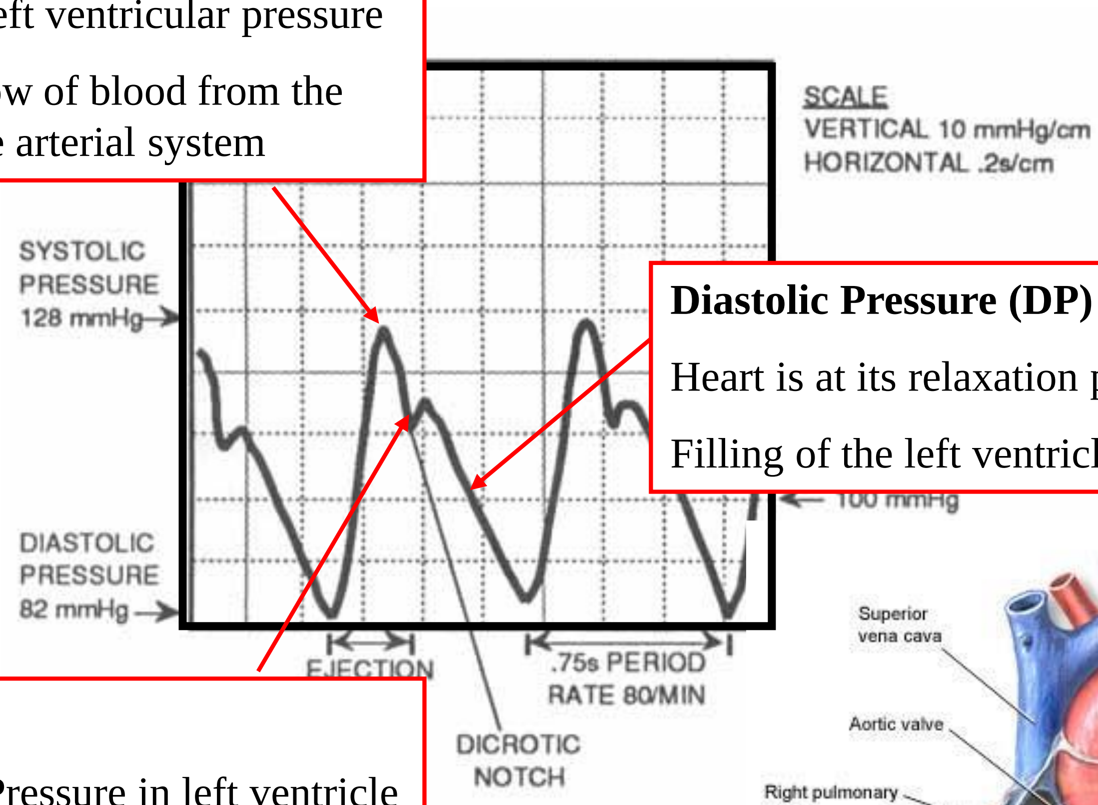
- Keep cannulated limb visible if possible
- Use non IV line caps on ports
- Label tubing and line

91
92
0
463
90/61 (68)
36.7
15
6
100
376
No local alarm recording available

Peak systolic pressure (PSP)

Reflects maximum left ventricular pressure

Sharp uprise = Ouflow of blood from the ventricle and into the arterial system



Diastolic Pressure (DP)

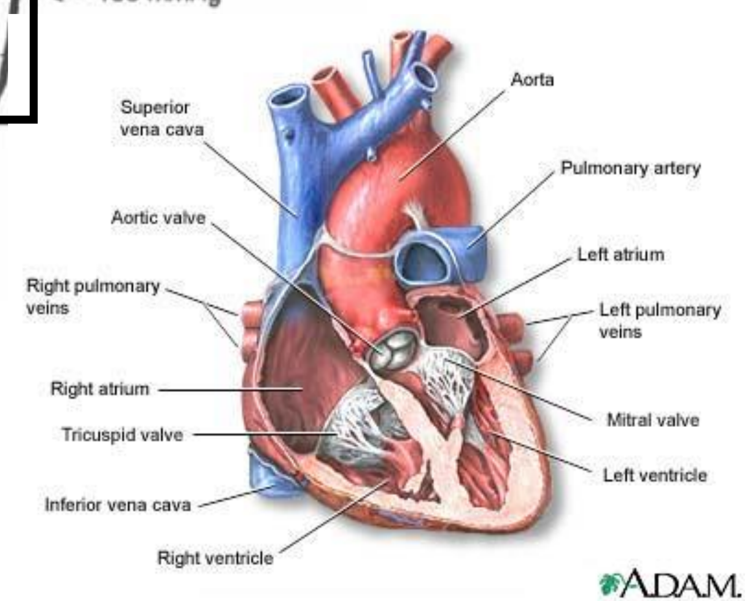
Heart is at its relaxation phase.

Filling of the left ventricle.

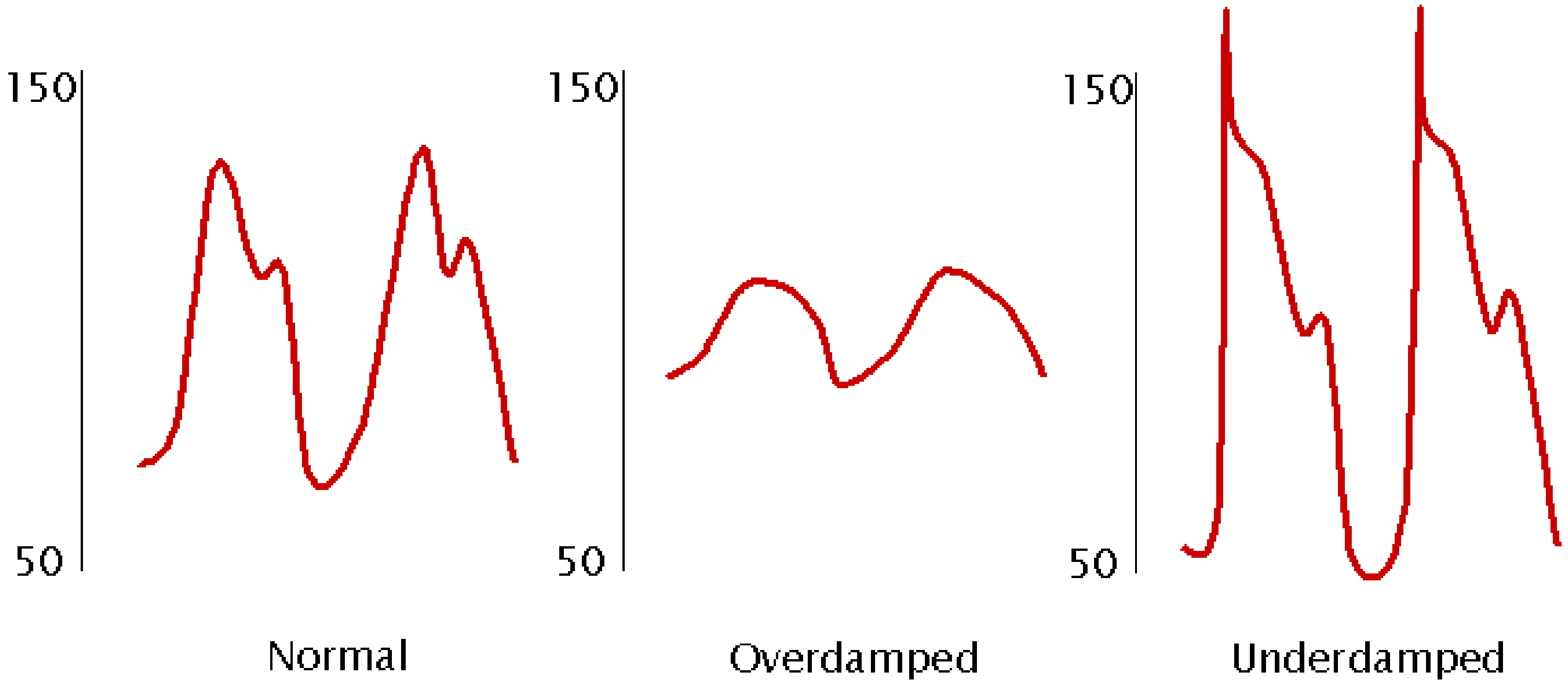
Dicrotic Notch

Pressure in Aorta > Pressure in left ventricle => Close of the aortic valve

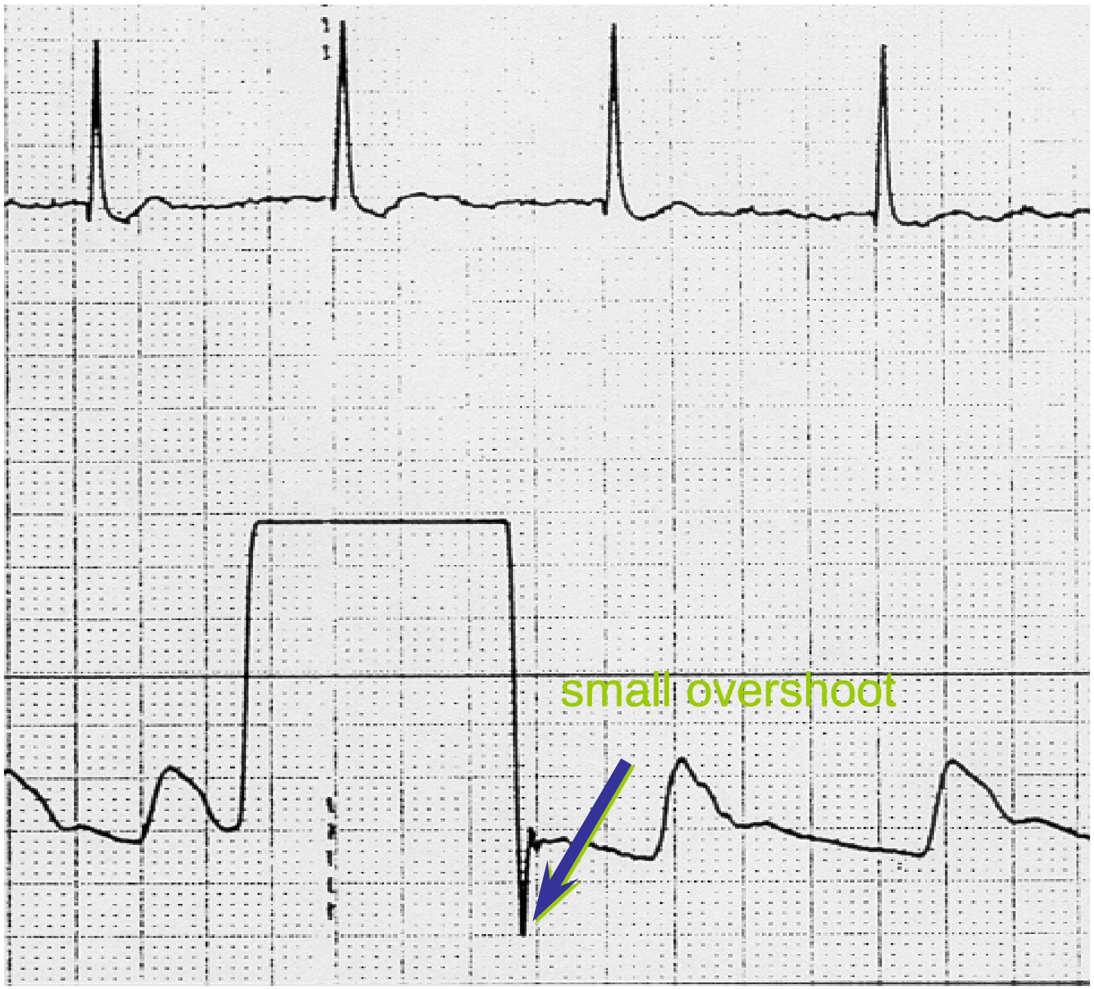
Marks the end of the systole and beginning of diastole



Dampening effects

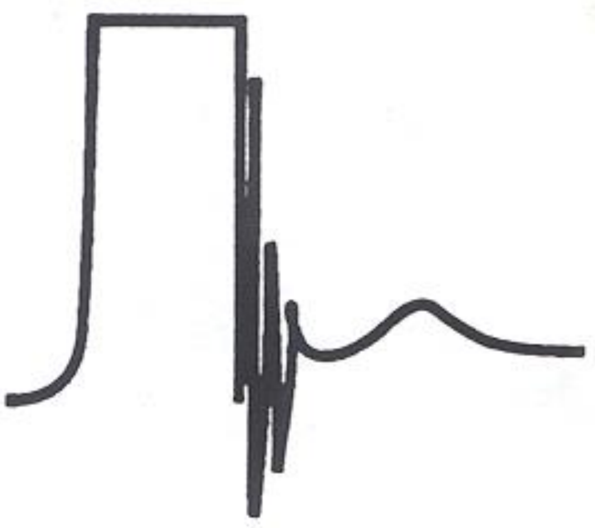


Normal Square Wave Test



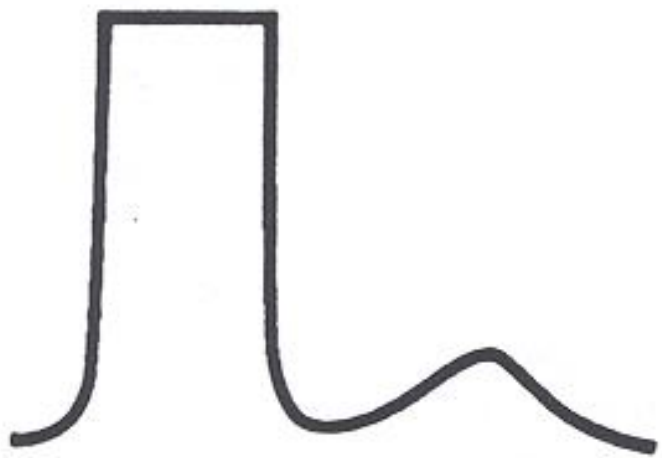
- Square wave
- quickly returns to baseline
- Followed by 1 or 2 bounces
- Bounce < 40 ms
- 2nd bounce $\leq$1/3 height of 1st

Critically Damped



- Pressure is accurate
- Number of oscillations should be 1-2 when returns to baseline

Overdamped

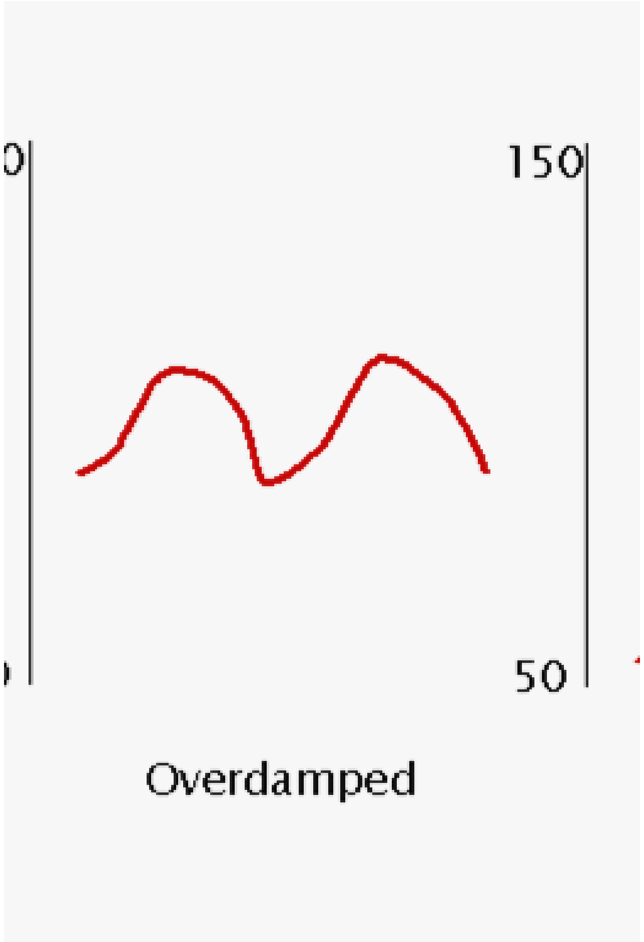


- No oscillations when waveform returns to baseline
- Pressure will be underestimated, i.e., lower than actual

Overdampened trace

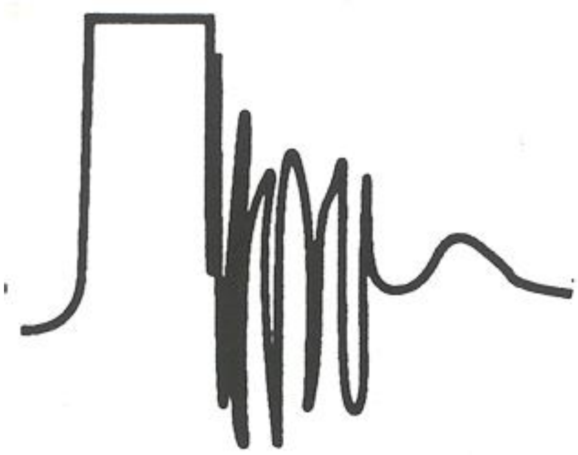
- Caused by
  - Occluded line
  - Low pressure in bag
  - Innappropriate scale on monitor
  - Air in transducer tubing

- Low cardiac output states
  - Hypovolaemia
  - Vasodilatation
  - Cardiogenic shock



Underdamped

- More than 2 oscillations when returns to baseline
- Pressure will be overestimated, i.e., higher than actual pressure



Avoiding Problems

- Always flush sample port and line after sampling
- Keep all connections tight and ensure stopcocks closes to air
- Don’t add extra stopcocks and tubing to lines
- Maintain flush bag at 300mm Hg
- Zero and maintain transducer at level of left atrium (4th intercostal space – mid axilla line

ARRHYTHMIA REVIEW

[redacted]
Unit: CCU
Bed: BED2
[redacted]

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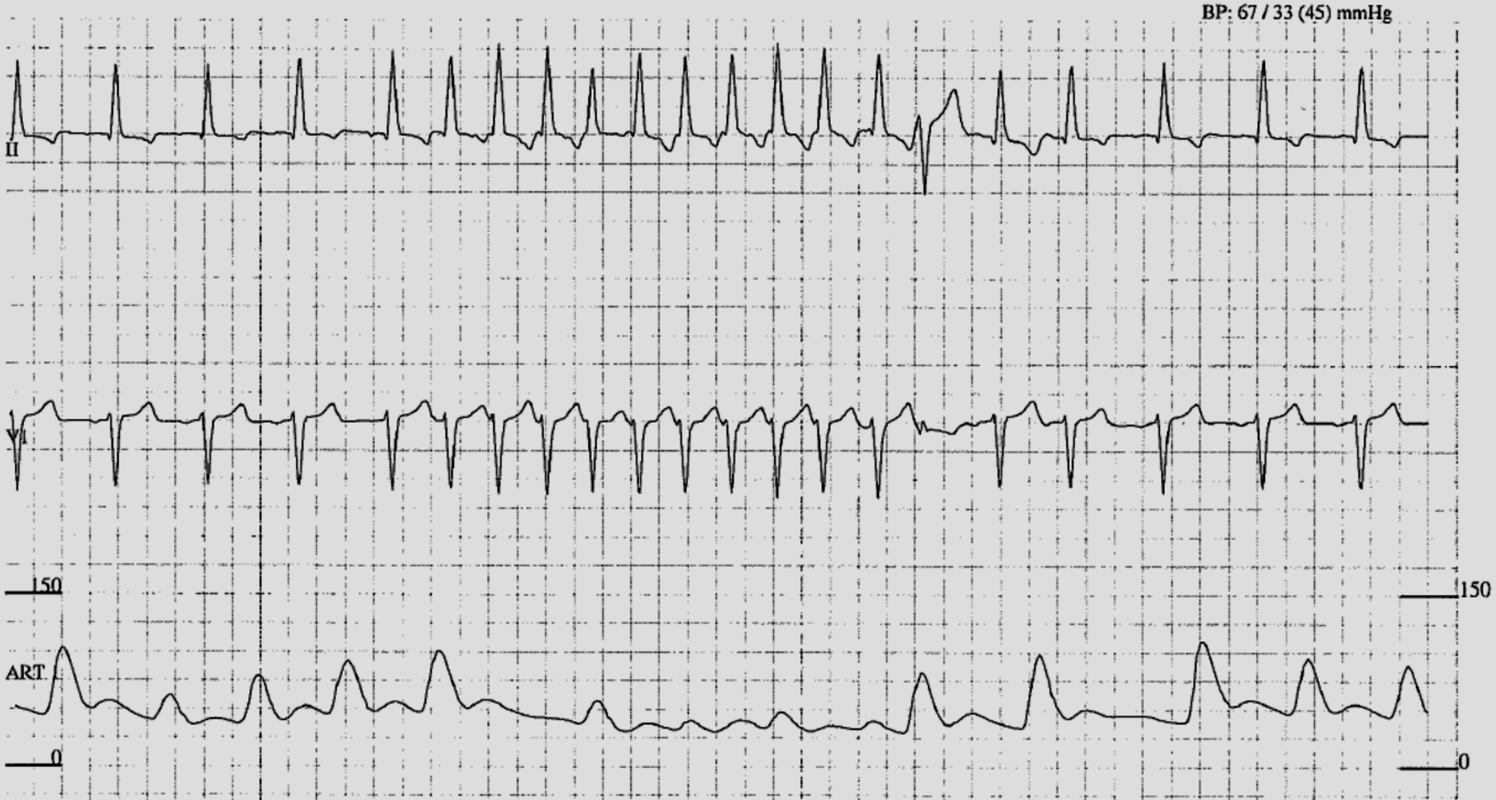
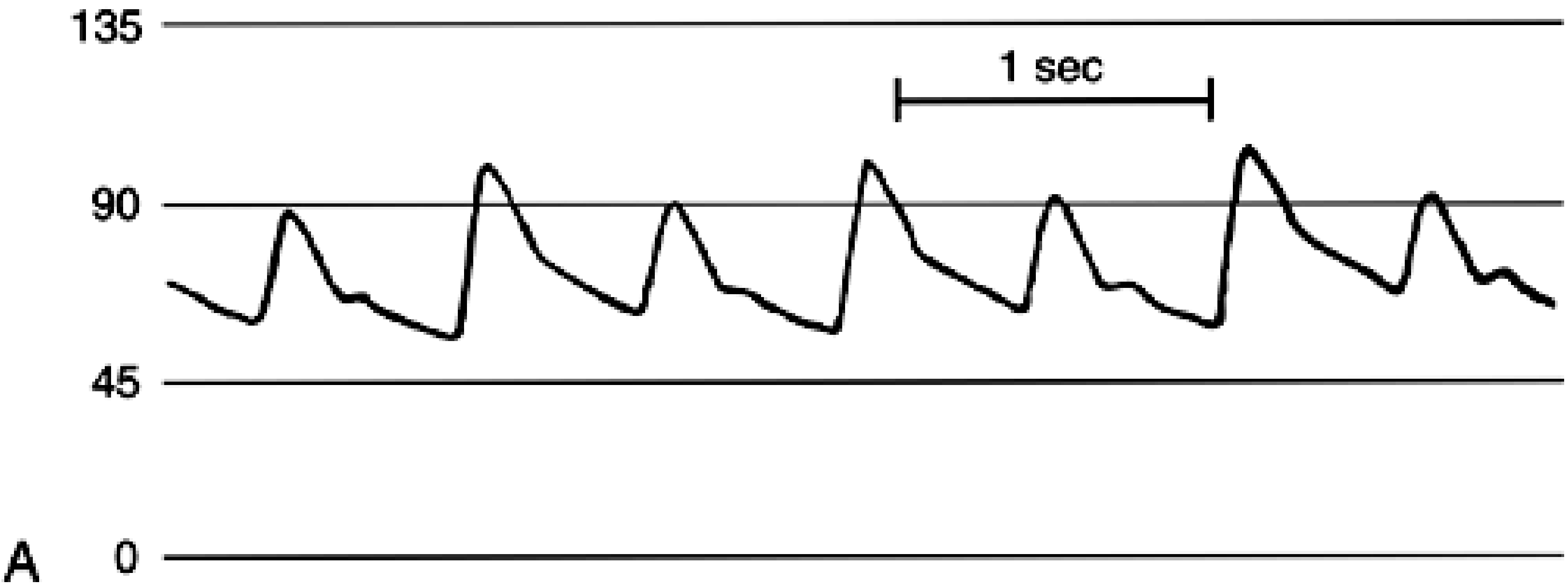
R ON T

HR: 181 bpm
BP: 67 / 33 (45) mmHg



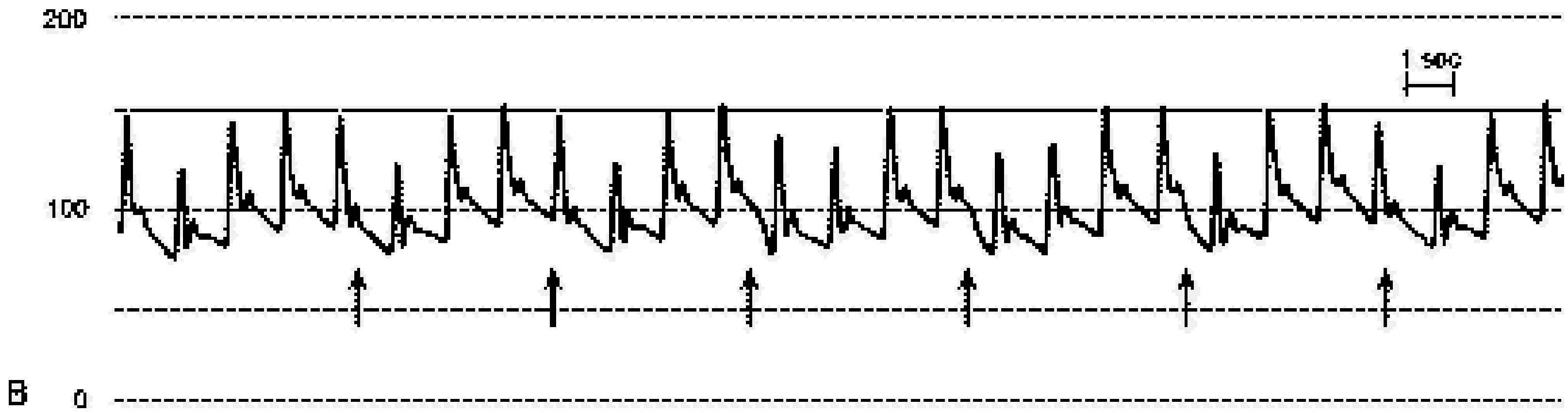
GE Medical Systems Information Technologies

Pulsus alternans



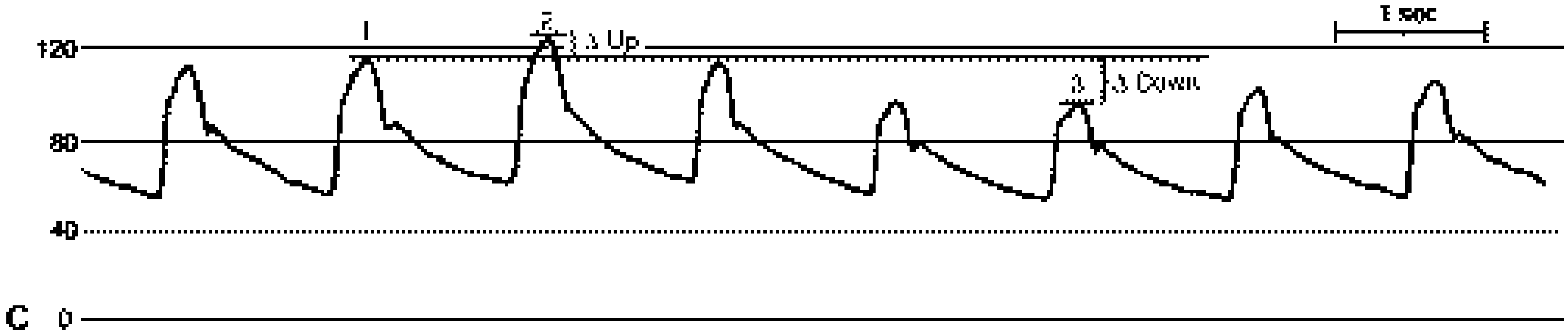
- Alternating beats of larger and smaller pressures
- Sign of severe left ventricular failure
- Can be exaggerated by anaesthesia when sympathetic tone and contractility is reduced

Pulsus Paradoxus



- Drops in systolic pressure during inspiration
- Often a sign of pericardial constriction (tamponade)
- Can also be seen with airway obstruction and bronchoconstriction.

Systolic Pressure Variation

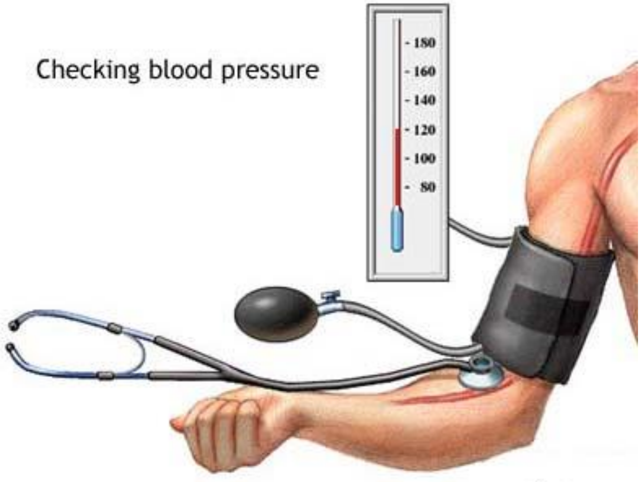


- Seen during positive pressure ventilation as evidence of hypovolaemia
- Stroke volume variation

What is Blood Pressure

- Defined as resistance (SVR) x flow (CO)

- Normal values:
  - Systolic (100-130mmHg)
  - Diastolic (60 – 90mmHg)



Cardiac Output (CO)

Determined by -

- Preload (cardiac filling or CVP)
- Myocardial contractility (force of contraction)
- Afterload (resistance to aortic ejection)
- Heart rate

MAP

Adequate organ perfusion depends on a continuous perfusion pressure.

Most organs require a mean blood pressure > 65 mmHg for normal function

$$\text{Mean BP} = \text{diastolic BP} + \frac{(\text{systolic} - \text{diastolic})}{3}$$

Not all hypotension is the same

- Mr Brown is hypotensive and tachycardic with warm extremities
- Mrs Clark is hypotensive and tachycardic with cold clammy extremities
- Mr Wall is hypotensive and bradycardia with cool extremities

Do we treat them all the same?

Shock Categories (P.R.O.V.V)

- Pump problems (cardiogenic)
- Rate and rhythm (cardiogenic)
- Obstructions (obstructive)
- Volume (hypovolaemic)
- Vasodilatation (distributive)

General response to shock

- Sympathetic response
  - Pump Increased cardiac contractility
  - Rate Tachycardia
  - Vasculature Vasoconstriction

- Increased respiratory rate
- Pupillary dilation
- Sweating

Limiters of shock response

- Bradycardia (heart blocks)
- Vasodilatory states (sepsis, anaphylaxis)
- Medication (beta blockers)

Dangers of shock response

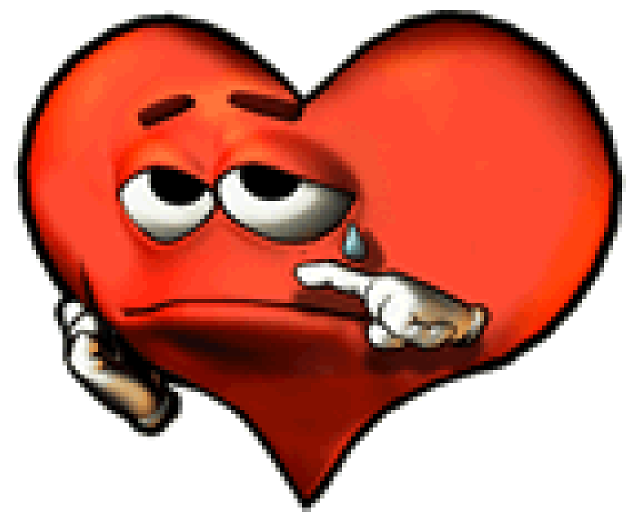
- Increased contractility and heart rate
  - – Increase myocardial oxygen demand
  - – May lead to myocardial ischaemia/infarction

- Increased vasoconstriction
  - – Reduction in tissue perfusion
  - – Decreased organ perfusion
  - – Acidosis

Untreated shock leads to

Release of cytotoxic and vasodilatory substances (lots of bad stuff that causes-)

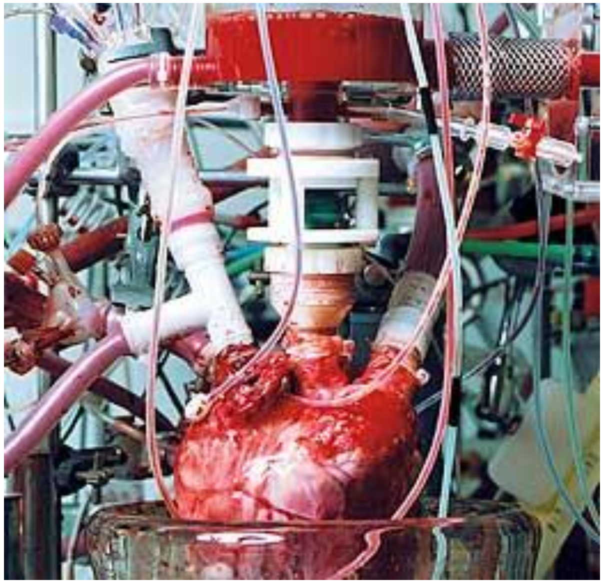
- Progressive vasodilatation
  - Overly wide pipes
- Increased capillary permeability
  - Leaky pipes
- Intravascular coagulation
  - Sticky blocked pipes
- Myocardial depression
  - A sad pump



Optimising perfusion

- Increase circulating volume

- Manipulate autonomic nervous system to affect



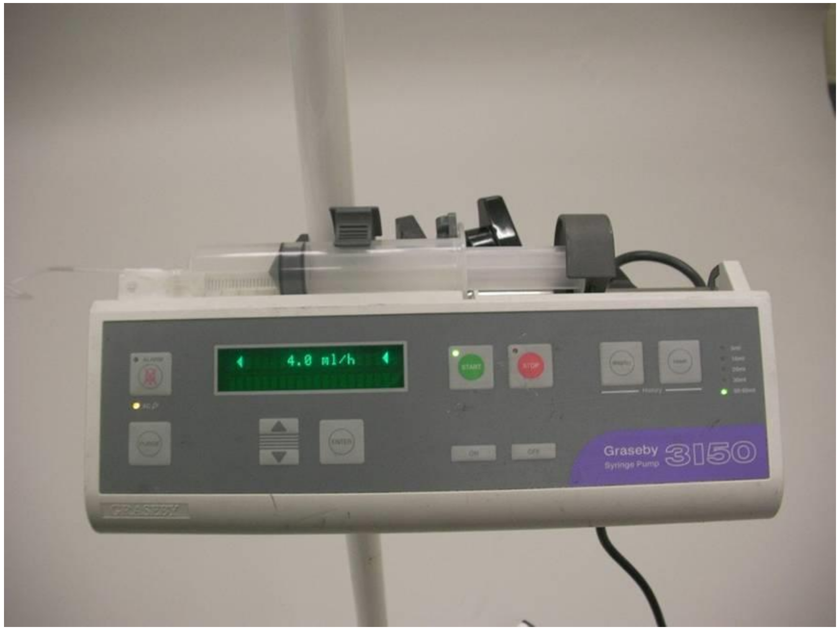
Alpha effects
– Vessel tone (vasoactive pressors)

Beta 1 effects
– Cardiac contractility (inotropic)
– Heart rate (chronotropic)

Beta 2 effects
– Broncho dilation
– Vasodilation

Perfusion drugs - Sympathomimetic

- Adrenaline
- Noradrenaline
- Phenylephrine
- Ephedrine
- Dobutamine
- Dopamine
- Isoprenaline



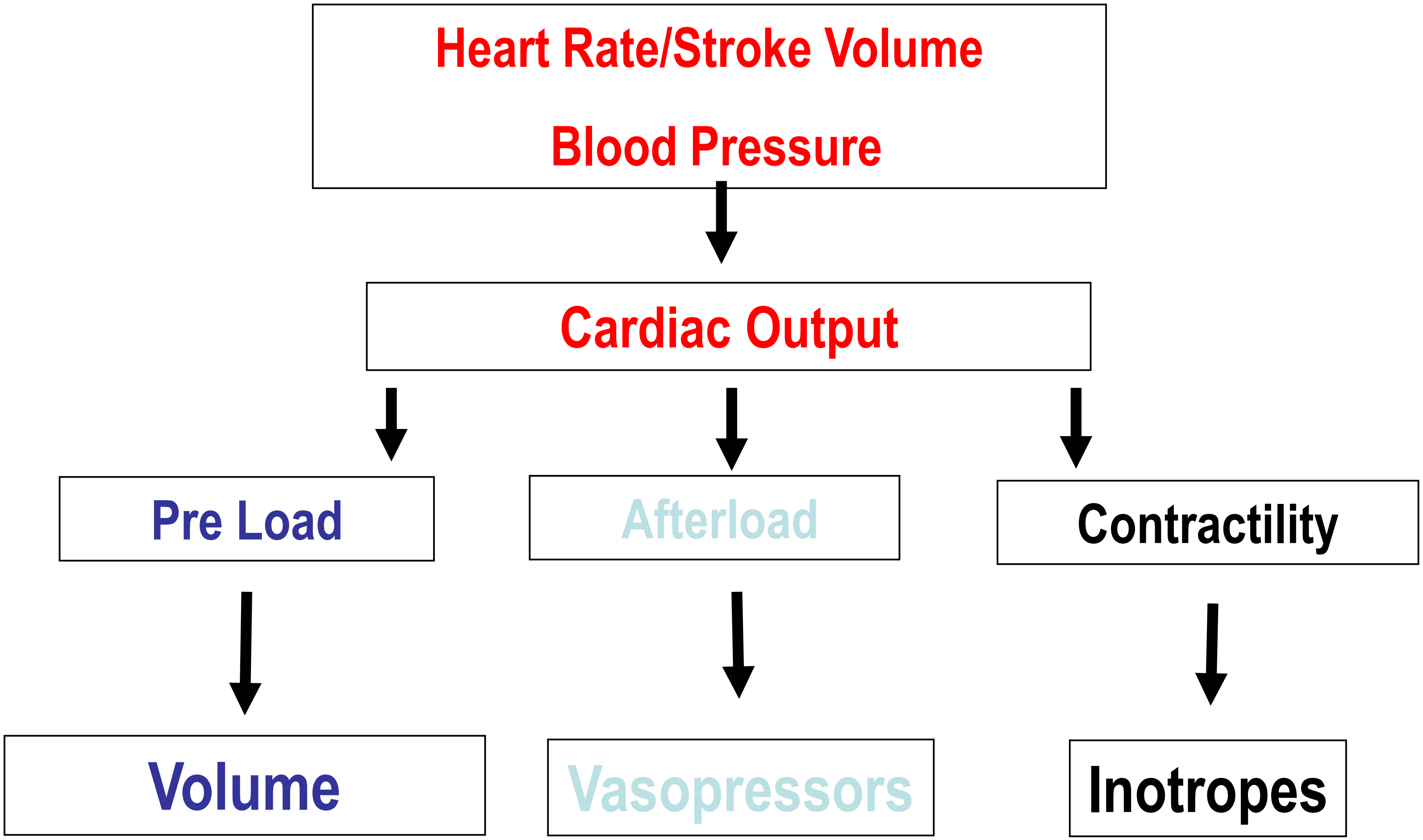
Choice determined by desired amount of alpha and beta effects required.

Table

Summary of Common Vasoactive Agents

Drug	Mean Arterial Pressure	Cardiac Output	Pulmonary Capillary Wedge Pressure	Systemic Vascular Resistance	Typical Dose	Receptors	Mechanism of Action
Dopamine					5-20 mcg/kg/min		
0.5-2 mcg/kg/min	↔	↑	↓/↔	↔		D_1, β_1, β_2	Vasodilation of renal, mesenteric, and coronary arteries
2-10 mcg/kg/min	↑	↑	↔	↑		α_1, D_1, β_1, β_2	Increases myocardial contractility, heart rate, and cardiac output; mild vasodilatory effects with preserved dopaminergic activity
10-20 mcg/kg/min	↑	↑	↔	↑		α_1, D_1, β_1, β_2	Peripheral vasoconstriction with preserved inotropic and chonotropic effects
Norepinephrine	↑	↑	↔	↑	2-30 mcg/min	α_1, β_1	Vasoconstriction and increased cardiac contractility
Phenylephrine	↑	↔	↔	↑	40-200 mcg/min	α_1	Arterial vasoconstriction
Dobutamine	↔	↑	↓	↓	5-15 mcg/kg/min	β_1, mild β_2	Increases cardiac contractility with mild vasodilation
Epinephrine	↑	↑	↔	↑	2-10 mcg/min	α_1, β_1, β_2	Peripheral vasoconstriction, increased cardiac contractility, smooth muscle relaxation
Vasopressin	↑	↓	↑/↔	↑	0.01-0.04 units/min	V_1, V_2, V_3	Vasoconstriction of the vascular smooth muscle

Circulatory Failure



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- Mr Wall is hypotensive and bradycardic with cool extremities

Do we treat them all the same?

Arterial Blood Gases

Matching the numbers to the clinical picture

Not all tachypnoea is the same

- Mr Blue is tachypnoeic and centrally cyanosed.
- Mr Scarlett is tachypnoeic and flushed
- Mrs White is tachypnoeic and pale
- Mr Edge is tachypnoeic and peripherally cyanosed

Are they all in respiratory failure?

Blood gases-Why the Big Deal?

They give us so much information about –

- Respiratory function
- Metabolic function
- Acid / Base balance in blood
- Bodies response to acid base imbalances
- Severity of illness
- Is the patient getting better or worse?

Blood Gas Values

pH	7.410		[7.350 - 7.450]
pCO_2	43.0	mmHg	[35.0 - 45.0]
pO_2	86.6	mmHg	[80.0 - 100]
$cHCO_3^-(P)_c$	26.7	mmol/L	
$ctCO_2(B)_c$	24.1	mmol/L	
$cBase(B)_c$	2.3	mmol/L	
sO_2	97.3	%	

Electrolyte Values

cNa^+	139	mmol/L
cK^+	3.9	mmol/L
cGlu	5.4	mmol/L
cLac	0.4	mmol/L

Oximetry Values

ctHb	122	g/L	[120 - 175]
Hct_c	37.4	%	
FO_2Hb_e	96.5	%	[96.0 -]
$p50_e$	26.41	mmHg	
$p50(st)_d$	26.84	mmHg	
$p50(T)_e$	26.41	mmHg	

Temperature Corrected Values

pH(T)	7.410	
$pCO_2(T)$	43.0	mmHg
$pO_2(T)$	86.6	mmHg

Notes

d	Default value(s)
c	Calculated value(s)
e	Estimated value(s)

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Production of acids

- Metabolic processes produce acids as byproducts
- Higher the metabolic rate the greater production of acids.

2 types of acids produced

- Volatile acids
  - CO_2 -able to cross alveolar capillary membrane and can be regulated primarily by respiration

- Fixed or non volatile acids
  - are regulated by the kidneys because they can only be excreted in solution

Respiratory & renal regulation

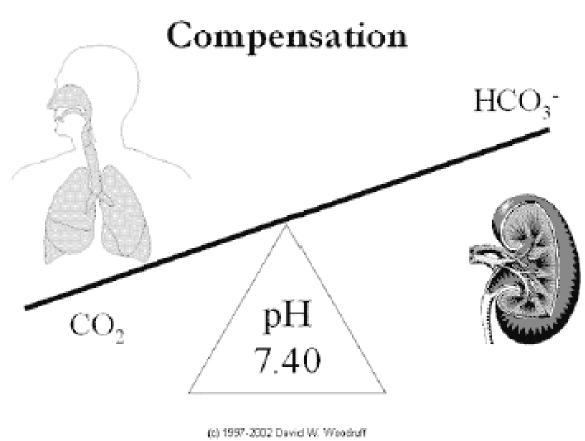
$$H_2O + \mathbf{CO_2} \Longleftrightarrow \mathbf{H_2CO_3} \Longleftrightarrow \mathbf{H^+} + HCO_3$$

Respiratory component

* Rapid response

Renal component

* Delayed response



Normal blood gas values

- pH 7.35 - 7.45
- PO2 80 - 100 mmHg
- PCO2 35 - 45 mmHg
- HCO3 22 - 26 m Eq/litre
- BE -2 - +2
- %O2 Sat 95 or greater

Blood gas analysis

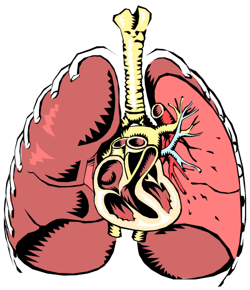
Acid versus **base** (alkaline)

pH

7.4

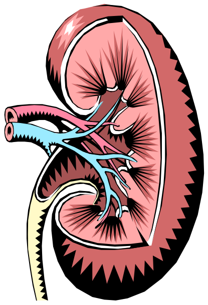
$PaCO_2$

35 – 45



HCO_3

22 – 26



ABG interpretation

Step 1

Consider each value independently

pH $PaCO_2$ HCO_3

Is the value normal

Is it an acid *or*

Is it a base

Eg	pH	7.2
	$PaCO_2$	50
	HCO_3	33

Reference Ranges:

pH	7.35-7.45
pCO2	35-45 mmHg
HCO3	22-26 mEq/L
pO2	80-100 mmHg
O2 Sat	95-100%

Step 1

pH	7.2	acid
$PaCO_2$	50	acid
HCO_3	33	base

Step 2

Look at pH to determine the nature of the imbalance

acidosis or alkalosis

pH 7.20 acid (acidosis)

eg pH 7.44

pH 7.18

Reference Ranges:

pH	7.35-7.45
pCO2	35-45 mmHg
HCO3	22-26 mEq/L
pO2	80-100 mmHg
O2 Sat	95-100%

Step 2

determine imbalance - acidosis or alkalosis

pH	7.2	acid (acidosis)
$PaCO_2$	50	acid
HCO_3	33	base

Reference Ranges:

pH	7.35-7.45
pCO2	35-45 mmHg
HCO3	22-26 mEq/L
pO2	80-100 mmHg
O2 Sat	95-100%

Step 3

now determine if the problem is respiratory or metabolic by looking at value with same status as pH

- If $PaCO_2$ → respiratory
- If HCO_3 → metabolic

Recapping

Step 1

Consider each value independently

- *is it acid or base*

Step 2

Look at pH to determine the nature of the imbalance

– *is it acidosis or alkalosis*

Step 3

Determine if the problem respiratory or metabolic

– *which value matches the pH*

examples

pH	7.18	pH	6.85	pH	7.56
$PaCO_2$	65	$PaCO_2$	26	$PaCO_2$	22
HCO_3	24	HCO_3	8	HCO_3	25

Compensation

Step 4

Determine level of compensation

is compensation absent**,** partial or complete?

pH	7.2	acid
$PaCO_2$	50	acid
HCO_3	33	base

Do this by looking at the value that does not match the pH

Is this within normal limits?

- if yes - *no compensation* has occurred
- if outside normal limits - *some compensation* has occurred

Compensation

Step 4 cont.

now determine if compensation is partial or complete

pH	7.2	base
$PaCO_2$	50	acid
HCO_3	33	base

Partial

- if the value that doesn't match the pH is outside normal limits *and* the pH is also outside normal limits then partial compensation has occurred

Complete

- if the value that doesn't match the pH is outside normal limits *and* the pH has returned to normal limits then complete compensation has occurred

Compensation

absent, partial or complete

examples

pH	7.18	acid
$PaCO_2$	65	acid
HCO_3	24	normal

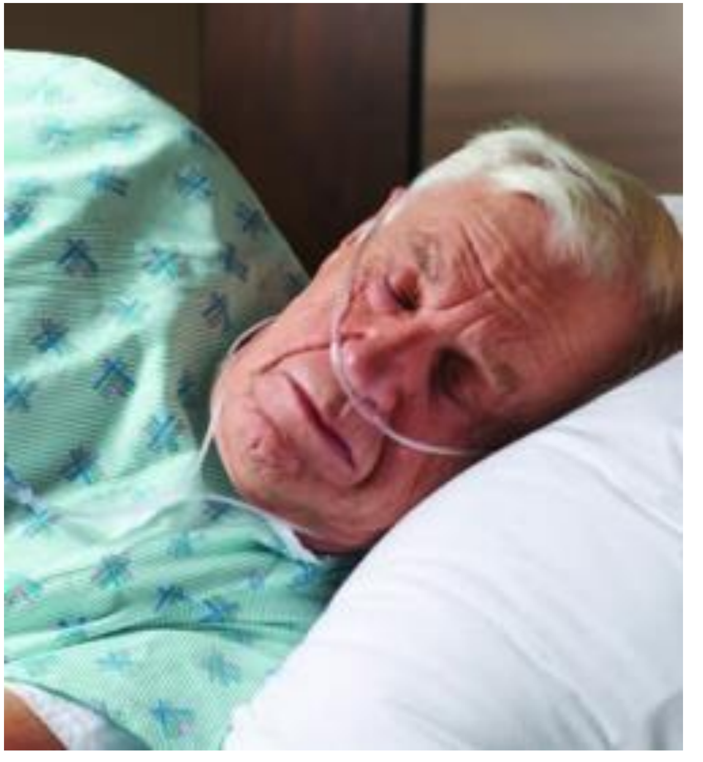
pH	6.85	acid
$PaCO_2$	29	base
HCO_3	8	acid

pH	7.45	base
$PaCO_2$	50	acid
HCO_3	33	base

Reference Ranges:

pH	7.35-7.45
pCO2	35-45 mmHg
HCO3	22-26 mEq/L
pO2	80-100 mmHg
O2 Sat	95-100%

Case A (anterior non-STEMI)



Mr Blue 84 yrs old

- Hypotensive BP 78/50
- Tachycardia 120
- Crackles in his bases
- Pulmonary oedema on X-ray
- Cool dusky peripheries

Case A

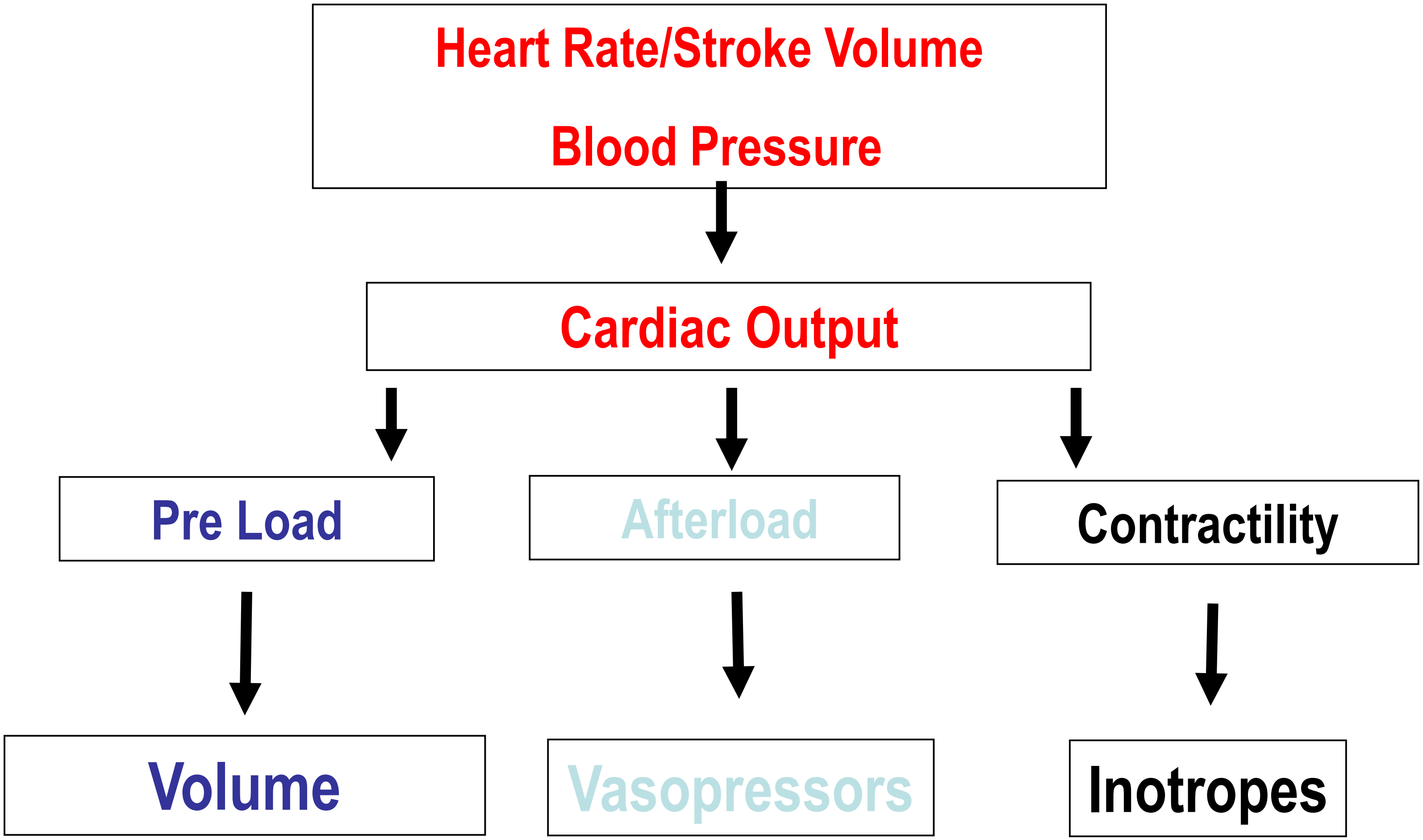
ABG result

pH	7.3
$PaCO_2$	26 mmHg
PaO_2	75 mmHg
HCO_3	18 mmol/L
SaO_2	92%

Lactate 2.4

Your interpretation -

Circulatory Failure



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2-10 mcg/kg/min	↑	↑	↔	↑		α_1, D_1, β_1, β_2	Increases myocardial contractility, heart rate, and cardiac output; mild vasodilatory effects with preserved dopaminergic activity
10-20 mcg/kg/min	↑	↑	↔	↑		α_1, D_1, β_1, β_2	Peripheral vasoconstriction with preserved inotropic and chonotropic effects
Norepinephrine	↑	↑	↔	↑	2-30 mcg/min	α_1, β_1	Vasoconstriction and increased cardiac contractility
Phenylephrine	↑	↔	↔	↑	40-200 mcg/min	α_1	Arterial vasoconstriction
Dobutamine	↔	↑	↓	↓	5-15 mcg/kg/min	β_1, mild β_2	Increases cardiac contractility with mild vasodilation
Epinephrine	↑	↑	↔	↑	2-10 mcg/min	α_1, β_1, β_2	Peripheral vasoconstriction, increased cardiac contractility, smooth muscle relaxation
Vasopressin	↑	↓	↑/↔	↑	0.01-0.04 units/min	V_1, V_2, V_3	Vasoconstriction of the vascular smooth muscle

Case B

Mrs Scarlett 57 yrs (cellulitis left leg)

- Hypotensive BP 78/50
- Tachycardia 120
- Warm peripheries
- Resp rate 28
- Chest clear on X-ray

Case B

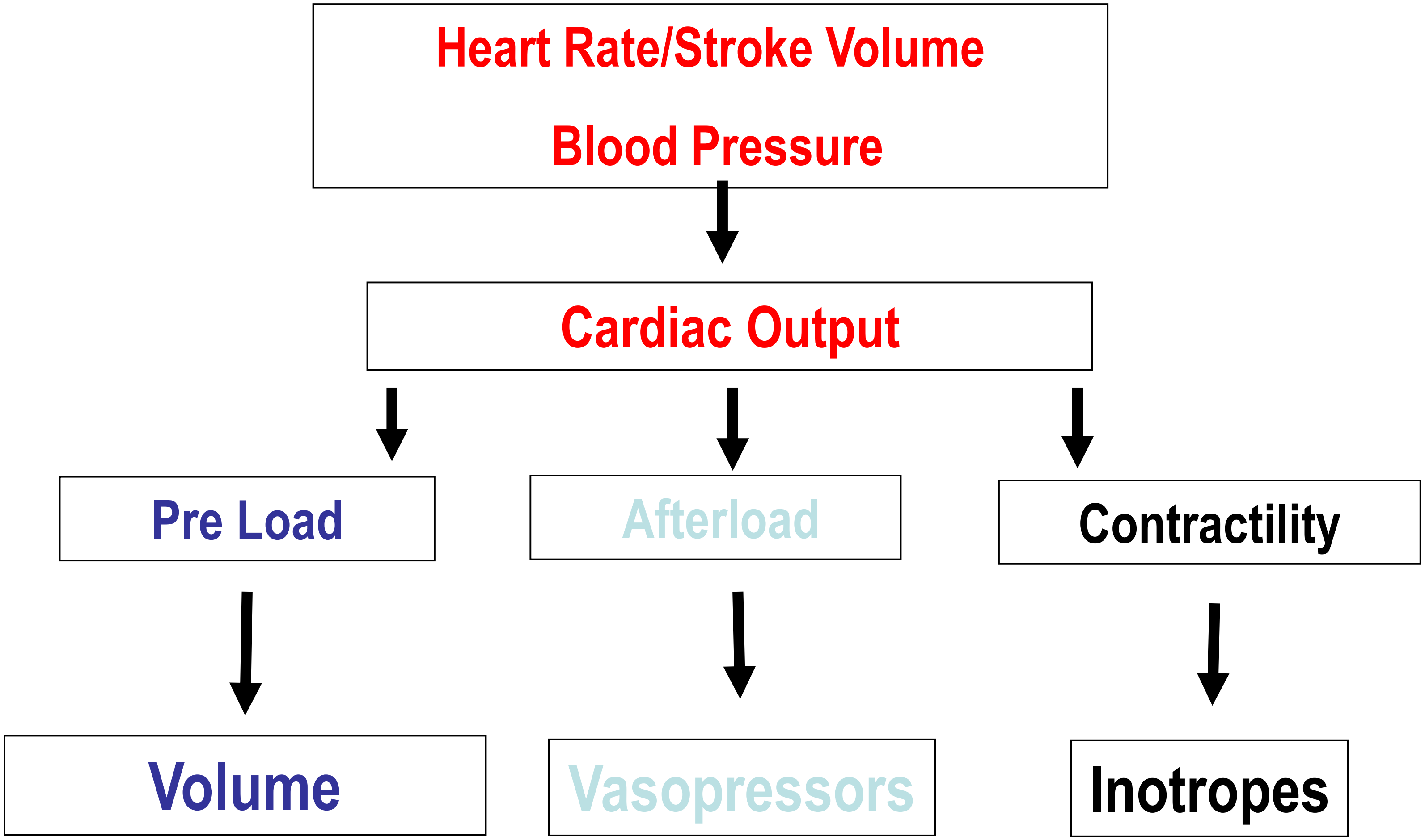
ABG result

pH	7.2
$PaCO_2$	26 mmHg
PaO_2	100 mmHg
HCO_3	16 mmol/L
SaO_2	97%

Lactate 4.3

Your interpretation -

Circulatory Failure



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2-10 mcg/kg/min	↑	↑	↔	↑		α_1, D_1, β_1, β_2	Increases myocardial contractility, heart rate, and cardiac output; mild vasodilatory effects with preserved dopaminergic activity
10-20 mcg/kg/min	↑	↑	↔	↑		α_1, D_1, β_1, β_2	Peripheral vasoconstriction with preserved inotropic and chonotropic effects
Norepinephrine	↑	↑	↔	↑	2-30 mcg/min	α_1, β_1	Vasoconstriction and increased cardiac contractility
Phenylephrine	↑	↔	↔	↑	40-200 mcg/min	α_1	Arterial vasoconstriction
Dobutamine	↔	↑	↓	↓	5-15 mcg/kg/min	β_1, mild β_2	Increases cardiac contractility with mild vasodilation
Epinephrine	↑	↑	↔	↑	2-10 mcg/min	α_1, β_1, β_2	Peripheral vasoconstriction, increased cardiac contractility, smooth muscle relaxation
Vasopressin	↑	↓	↑/↔	↑	0.01-0.04 units/min	V_1, V_2, V_3	Vasoconstriction of the vascular smooth muscle

Case C

Mrs Gasp 75 yrs old

- Hypotensive BP 88/50
- Tachycardia 130
- Temp 38.5
- Drowsy
- Resp rate 30
- Poor air entry to bases
- Basal consolidation on X-ray

Case C

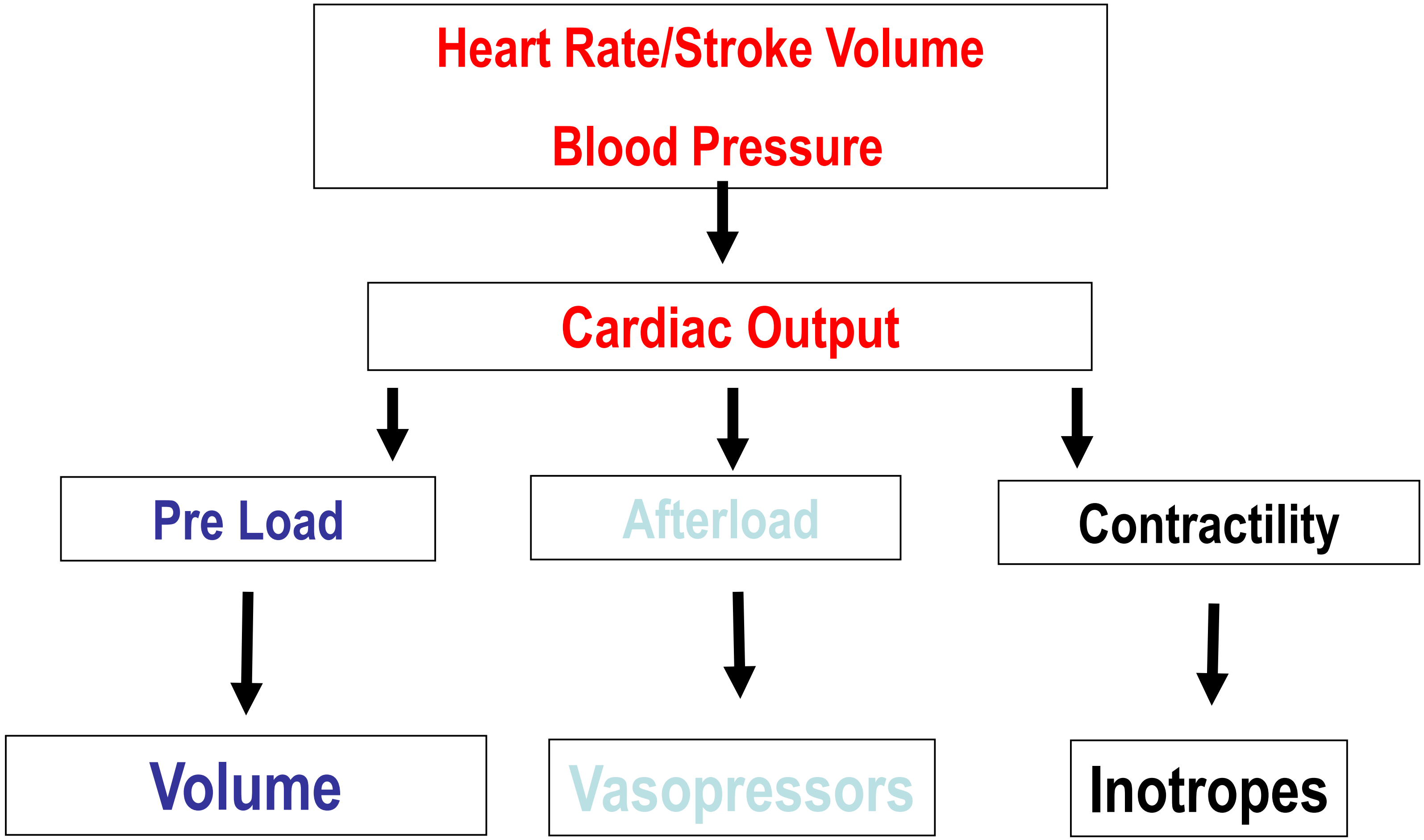
ABG result

pH	7.15
$PaCO_2$	80 mmHg
PaO_2	65 mmHg
HCO_3	30 mmol/L
SaO_2	86%

Your interpretation –

Ideal mangement

Circulatory Failure



Table

Summary of Common Vasoactive Agents

Drug	Mean Arterial Pressure	Cardiac Output	Pulmonary Capillary Wedge Pressure	Systemic Vascular Resistance	Typical Dose	Receptors	Mechanism of Action
Dopamine					5-20 mcg/kg/min		
0.5-2 mcg/kg/min	↔	↑	↓/↔	↔		D_1, β_1, β_2	Vasodilation of renal, mesenteric, and coronary arteries
2-10 mcg/kg/min	↑	↑	↔	↑		α_1, D_1, β_1, β_2	Increases myocardial contractility, heart rate, and cardiac output; mild vasodilatory effects with preserved dopaminergic activity
10-20 mcg/kg/min	↑	↑	↔	↑		α_1, D_1, β_1, β_2	Peripheral vasoconstriction with preserved inotropic and chonotropic effects
Norepinephrine	↑	↑	↔	↑	2-30 mcg/min	α_1, β_1	Vasoconstriction and increased cardiac contractility
Phenylephrine	↑	↔	↔	↑	40-200 mcg/min	α_1	Arterial vasoconstriction
Dobutamine	↔	↑	↓	↓	5-15 mcg/kg/min	β_1, mild β_2	Increases cardiac contractility with mild vasodilation
Epinephrine	↑	↑	↔	↑	2-10 mcg/min	α_1, β_1, β_2	Peripheral vasoconstriction, increased cardiac contractility, smooth muscle relaxation
Vasopressin	↑	↓	↑/↔	↑	0.01-0.04 units/min	V_1, V_2, V_3	Vasoconstriction of the vascular smooth muscle

Case scenarios

Case 1

A 10 year old boy arrives Hx increasing SOB over last week, now feverish and has moist unproductive cough

Vital signs

Temp	39^o C
HR	120
BP	90/60
RR	46
SpO_2	93% on 10 L oxygen via non-rebreather mask

Impression – possible pneumonia

Case 1

ABG result

pH	7.47
$PaCO_2$	26 mmHg
PaO_2	55 mmHg
HCO_3	20 mmol/L
SaO_2	90%

Your interpretation -

Case 2

A 12 year old girl is admitted with drowsiness and abdo pain

ABG on air

pH	6.9
$PaCO_2$	17 mmHg
PaO_2	92 mmHg
HCO_3	12 mmol/L
SaO_2	96%

Interpretation -

Case 3

An unconscious 30 year old with tricyclic OD responding only to pain is admitted, intubated & ventilated in ED

ABG 30 mins after intubation

pH	7.1
$PaCO_2$	47 mmHg
PaO_2	360 mmHg
HCO_3	18 mmol/L
SaO_2	100%

your interpretation -

Case 4

An unconscious 43 year old post head trauma and responding only to pain is admitted, intubated & ventilated in ED

ABG 30 mins after intubation

pH	7.50
$PaCO_2$	22 mmHg
PaO_2	560 mmHg
HCO_3	25 mmol/L
SaO_2	100%

your interpretation -

Case 5

A 6 year old girl involved in an MVA is brought in unconscious, she is intubated, her cardiac rhythm and absence of pulse indicates PEA and CPR is in progress.

Initial ABG

pH	7.1	
$PaCO_2$	10	mmHg
PaO_2	34	mmHg
HCO_3	18	mmol/L
SaO_2	67%	

Your interpretation -

Case 6

A 75 year old woman wpost op from repair of a # left tib and

fib requiring regular morphine has become increasingly

drowsy

ABG result

pH	7. 28
$PaCO_2$	70 mmHg
PaO_2	70 mmHg
HCO_3	30 mmol/L
SaO_2	92 %

Case 7

11 year old with paralytic ileus 3 days post appendectomy.
Nasogastric tube insitu with large outputs.

ABG

pH	7.48
$PaCO_2$	49 mmHg
PaO_2	95 mmHg
HCO_3	32 mmol/L
SaO_2	97%