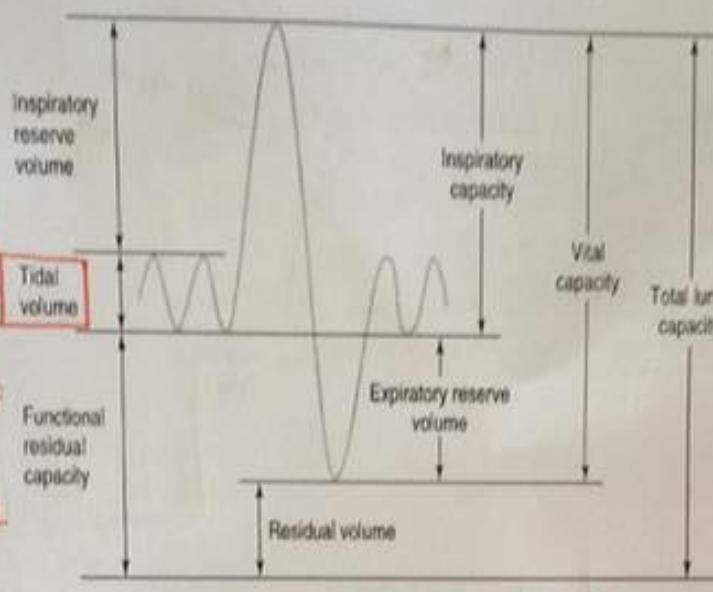


Pulmonary function test

the volume of air displaced between normal inhalation and exhalation when extra effort is not applied.

normally = 500 mL
or 7 mL/kg



$$\text{Vital capacity} = 70 \text{ mL/kg}$$

$$\text{Tidal volume} = 7 \text{ mL/kg}$$

$$\text{FRC} = 30-40 \text{ mL/kg}$$

average in adults =

$$2500 \text{ mL}$$

21% of them is oxygen

$$500 \text{ mL O}_2$$

we consume 250 mL/min

$$\text{O}_2 \rightarrow 50-500 \text{ mL O}_2$$

will be enough for only 2 minutes

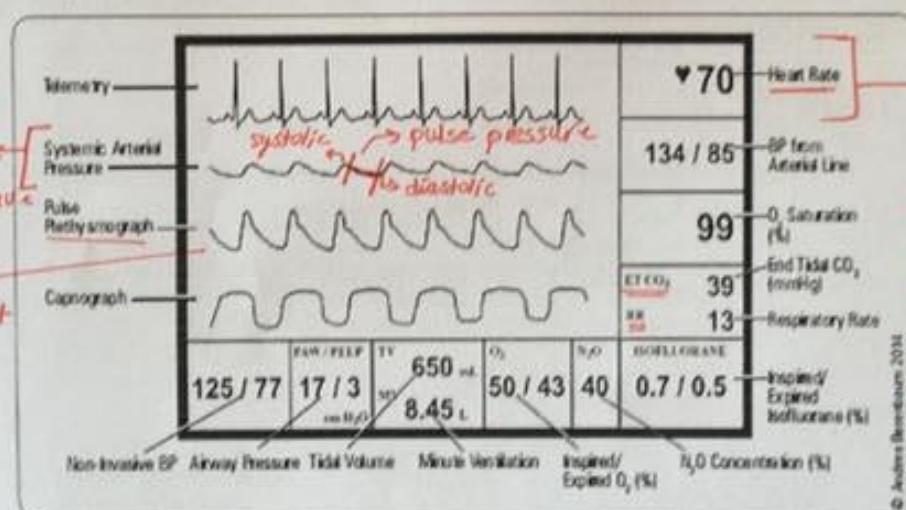
That's why we make pre-oxygenation or de-nitrogenation to substitute the N_2O with O_2 , so all the 2500 mL will be O_2 → enough for 10 minutes.

- invasive BP measurement is more accurate than the non-invasive -

- invasive BP.
The waves: invasive BP waves

lower limit of normal O_2 sat = 92%.

$$\text{PaO}_2 = 60$$

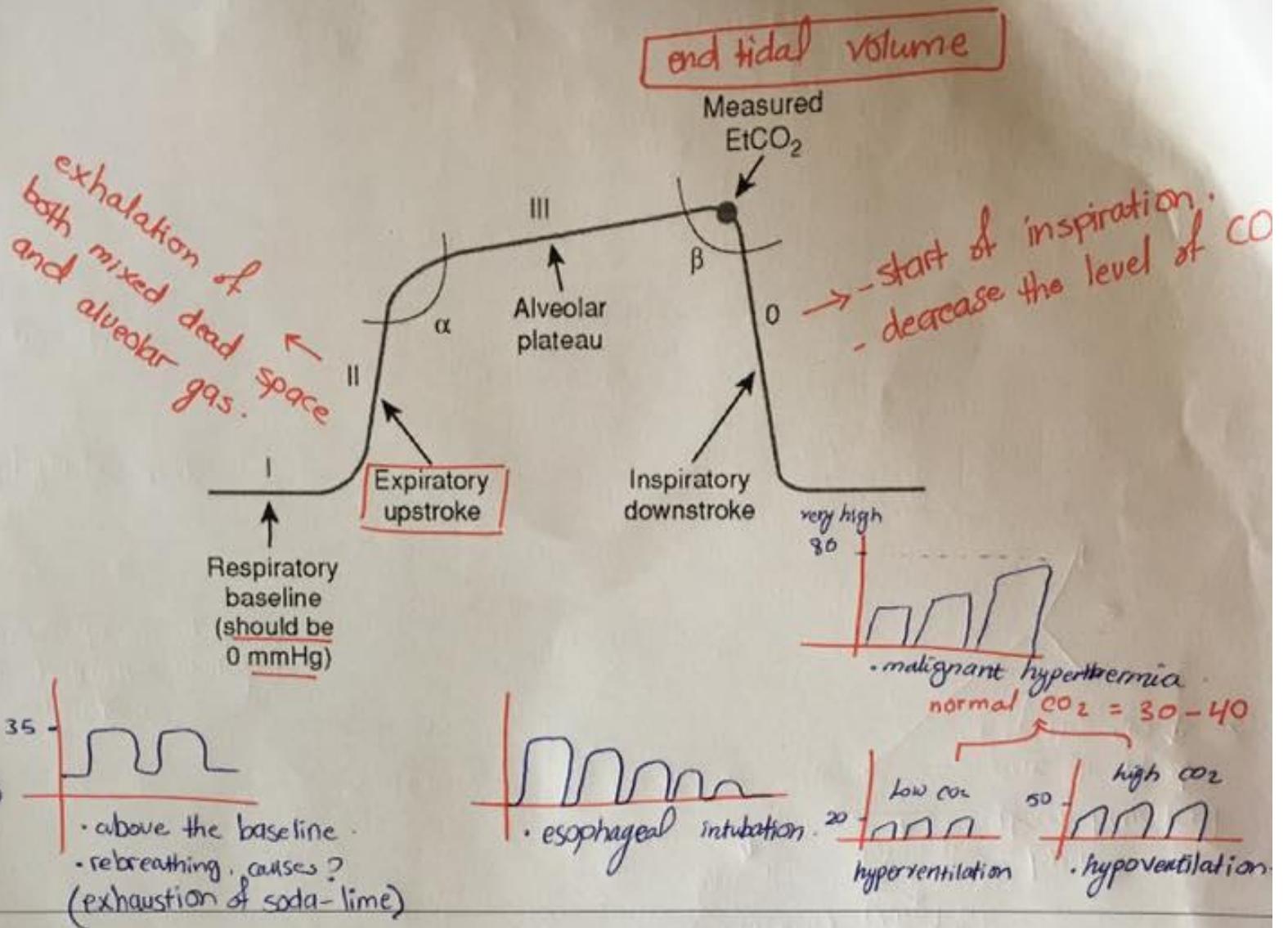


- standard ECG → Lead II or V_5
- The ECG and HR reflects the electricity of the heart, not the mechanical function that's why there's electromechanical dissociation or pulseless electrical activity.

Figure 2. Typical anesthesia monitor

capnograph

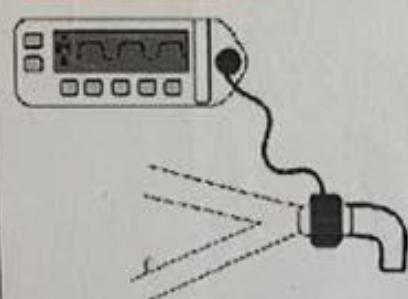
- what it reflects? (ventilation)
- what does it measure?
 - End tidal CO_2
 - respiratory rate



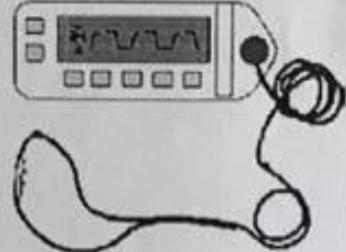
These are capnometers

attached to ←
the breathing
system.

Mainstream



Sidestream



- * The shock has 3 phases :
 1. compensatory phase.
 2. decompensatory phase.
 3. multi-organ dysfunction phase.

• Shock symptoms after :

1 pneumonia \Rightarrow
septic shock

2 Penicillin \Rightarrow
anaphylactic shock

3 Central line \Rightarrow

Tension pneumothorax

\Rightarrow obstructive cardiogenic shock.

Shock: Classification

- Hypovolemic shock - due to decreased circulating blood volume in relation to the total vascular capacity and characterized by a reduction of diastolic filling pressures
- Cardiogenic shock - due to cardiac pump failure related to loss of myocardial contractility/functional myocardium or structural/mechanical failure of the cardiac anatomy and characterized by elevations of diastolic filling pressures and volumes
- Extra-cardiac obstructive shock - due to obstruction to flow in the cardiovascular circuit and characterized by either impairment of diastolic filling or excessive afterload
- Distributive shock - caused by loss of vasomotor control resulting in arteriolar/venular dilatation and characterized (after fluid resuscitation) by increased cardiac output and decreased SVR



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* Shock \Rightarrow MAP < 40 \Rightarrow MAP = $\frac{CO}{SVR}$

\downarrow
when this decreases (cardiogenic shock)

\downarrow
when this decreases (vasogenic shock)

* if the CO $\downarrow \rightarrow$
SVR should \uparrow
 \Rightarrow compensatory phase.

$$CO = HR * \frac{SV}{preload(LVEDV)} * \frac{contractility}{after load (resistance)} * \frac{ejection fraction}{}$$

example :-

patient with
normal BP

HR = 105

urine output =

25 mL/hr



Class II

Clinical Correlates of Hemorrhage

compensatory decompensatory multi-organ dysfunction

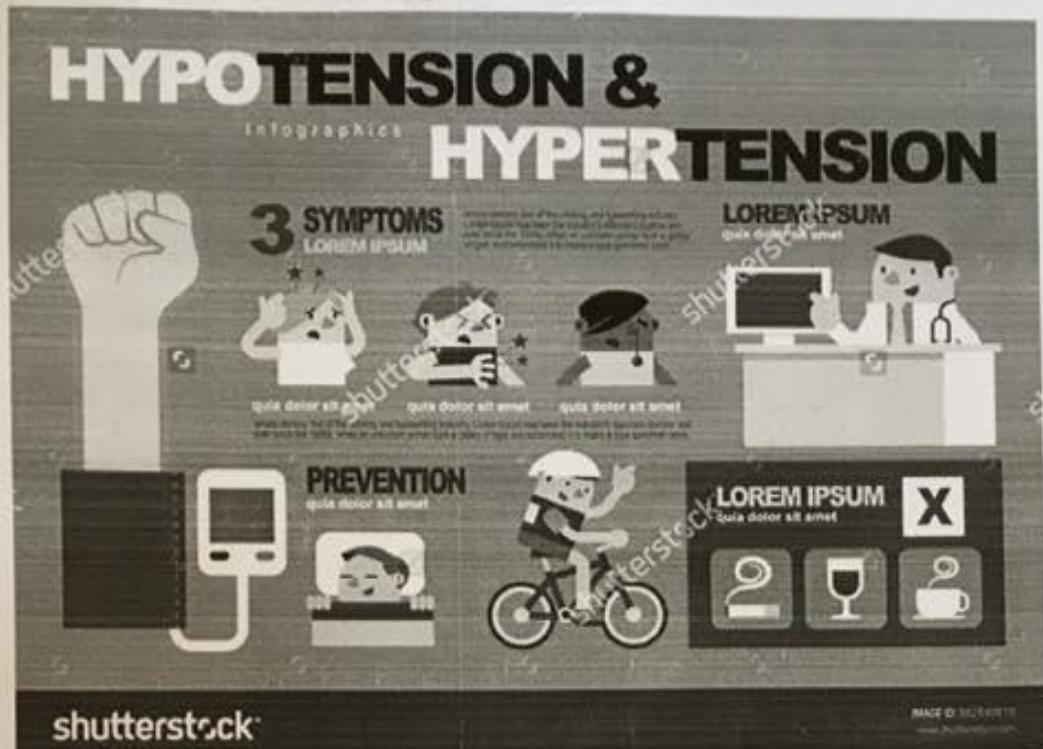
	Class I	+	Class II	Class III	Class IV
* Blood loss (mL)	> 750		750 - 1500	1500 - 2000	> 2000
Blood loss (% total)	> 15%		15 - 30%	30 - 40%	> 40%
* Pulse rate	< 100		> 100	> 120	> 140
* Blood pressure	Normal		Normal	↓	↓
Pulse pressure	Normal or ↓		↓	↓	↓
Orthostasis	Absent		Minimal	Marked	Marked
Capillary refill	Normal		Delayed	Delayed	Delayed
Resp rate	14 - 20		20 + 30	30 - 40	> 34
* UO (mL/hr)	> 30		20 - 30	5 - 15	< 5
CNS mental status	Slight anxiety		Mild anxiety	Anxious/confused	Confused/lethargic
CI (L/min)	↓ 0-10%		↓ 20-50%	↓ 50-75%	↓ > 75%



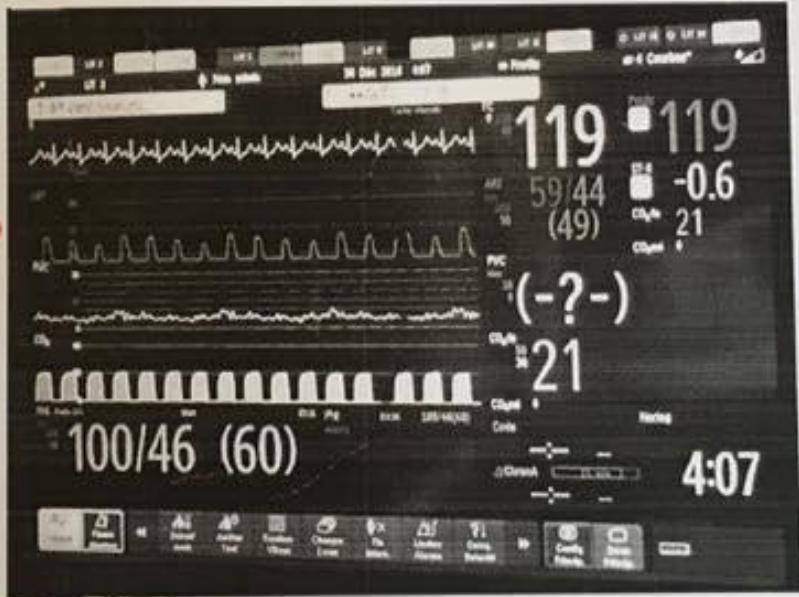
American College of Surgeons, 1999

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- if the preload decreases \Rightarrow hypovolemic shock (most common)
- if the contractility decreases \Rightarrow cardiogenic shock (non-obstructive)
- if the preload \downarrow and after load $\uparrow \Rightarrow$ obstructive cardiogenic shock (aortic stenosis, HTN, cardiac tamponade, tension pneumothorax)



* Shocked patient,
what is the
management if
he needs surgery?



Road
Traffic
accident

- RTA,, need GA,,, YOUR management,,,
- THREE MAJOR CONCERN FOR ANESTHESIA THIS PT,,,

- 1 rapid sequence induction (The patient ~~has~~ full stomach)
- 2 we give Ketamine (to increase the circulation) → don't give propofol
- 3 maintenance ⇒ opioid or inhalational agents or Thiopental

ABCDE
large venula (?)
colloid or blood
arterial line
central line



* what is this maneuver?

1) in-line stabilization, the patient is suspected to have neck injury after RTA

2) Laryngoscopy.

(air-Fluid Level)



* where you can see it?
in intestinal obstruction.

* what are the cardinal
features of intestinal obstruction?

- 1) abdominal distension.
- 2) constipation
- 3) vomiting
- 4) abdominal pain.

* what's the type of
acid-base disturbance in
this patient?

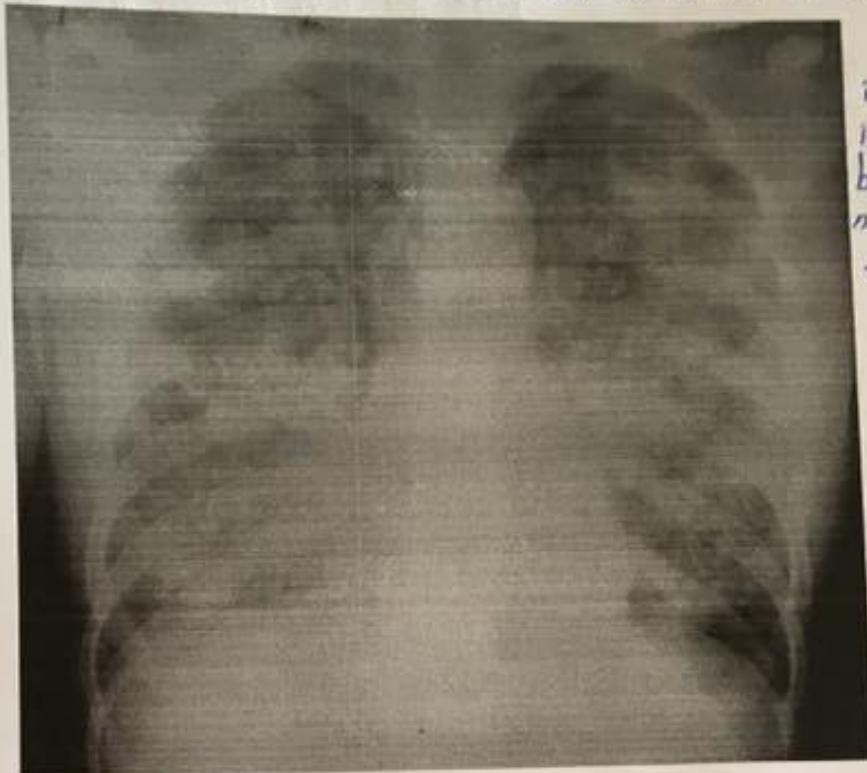
metabolic alkalosis
(patient is losing acids
by vomiting)

* what's the anesthesia concern in this patient?

1. patient is dehydrated → rehydrate!
2. patient has electrolyte disturbances → correct it
3. risk of aspiration → rapid sequence induction
4. ICU admission and good approach for pain management.

* What's the cause of this patient x-ray?

This is pulmonary edema which can be cardiac or non-cardiac



in this patient
it's non-cardiac
because of the
normal size of
the heart

↓
This is
ARDS.

non-cardiac (cardiogenic pulmonary edema)

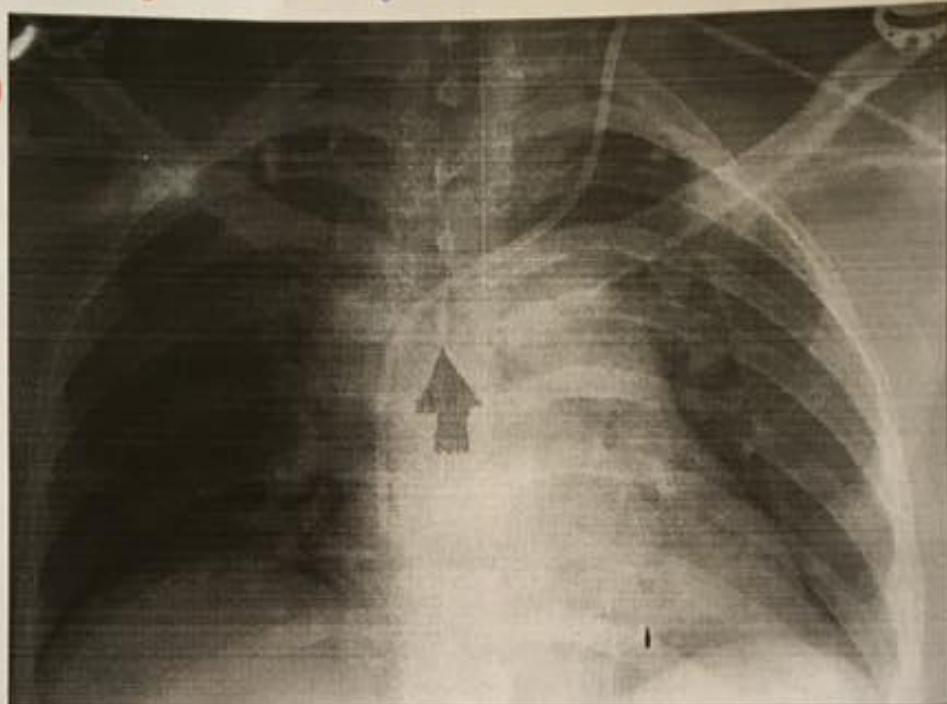


← cardiomegaly

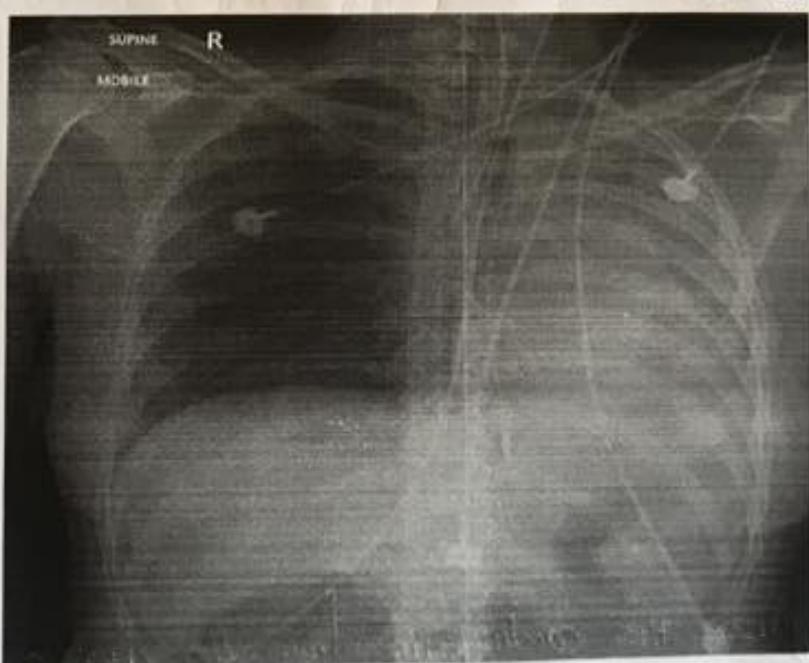
* We can recognize the cardiogenic from the non-cardiogenic pulmonary edema by:

- ① the size of the heart → cardiomegaly in cardiogenic pulmonary edema
- ② wedge pressure → more than 18 mmHg in cardiogenic.

- * What's the arrowed structure? Central line.
- * Where does it go? at the junction between the SVC and right atrium.
- * Through which vein we introduced it? (in this pic)
Left internal jugular vein



- * What is the cause of this patient's x-ray? Lung collapse. (Left)
- * mention the causes of Lung collapse?
 1. bronchial obstruction (may be foreign body, Tumor, blood)
 2. external obstruction (Lymphoma)
 3. tube introduced to one major bronchi, not to the other.

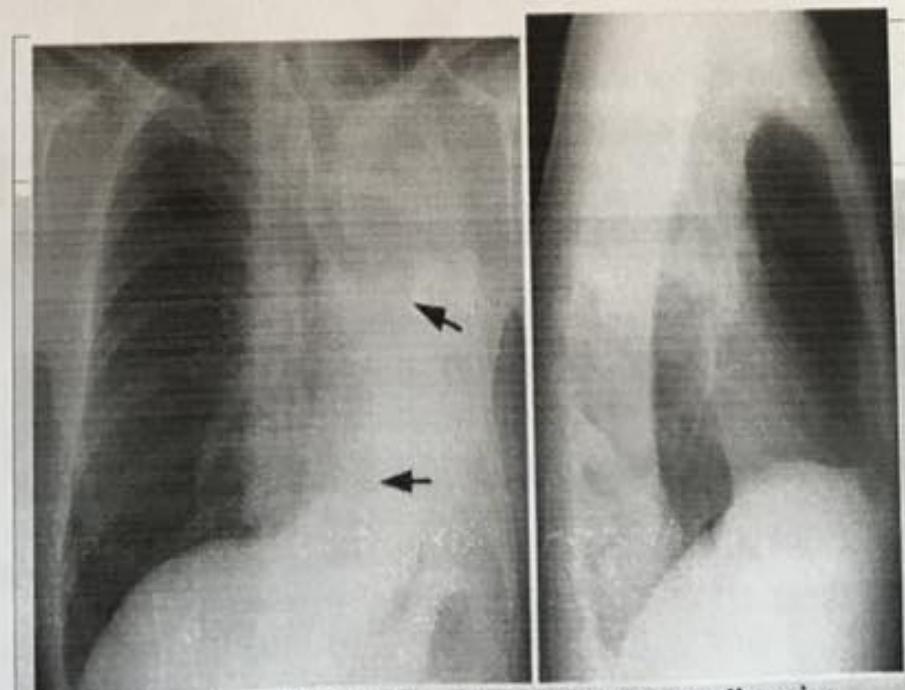




- * This is Lung collapse with tracheal deviation.
- * Possible cause? bronchogenic carcinoma (mostly) or tension pneumothorax.

Lung collapse, causes

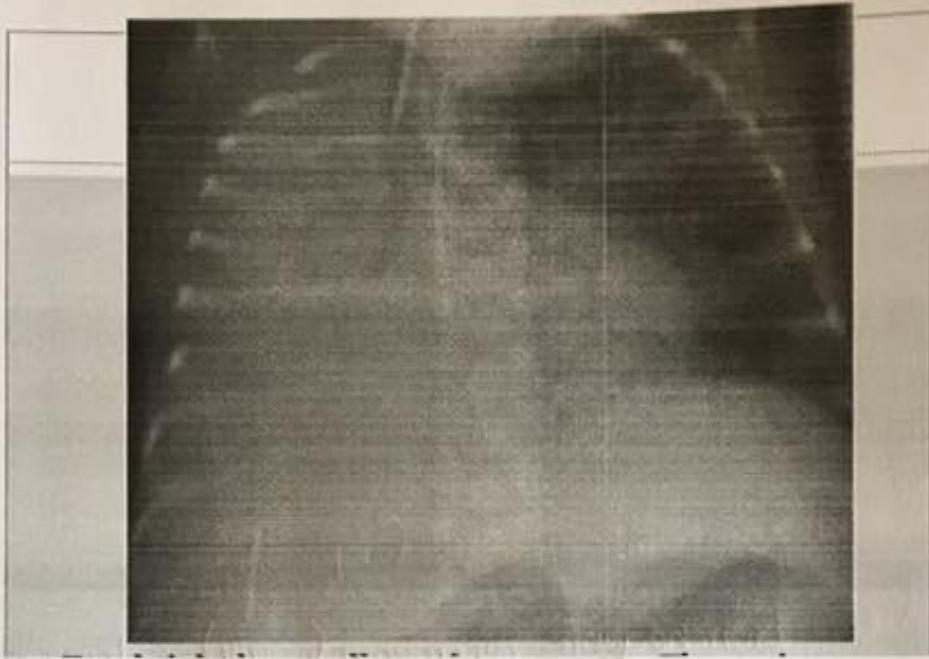
1. endobronchial intubation
2. bronchial obstruction (foreign body)



* Frontal and lateral chest radiographs. The cause of the collapse is a

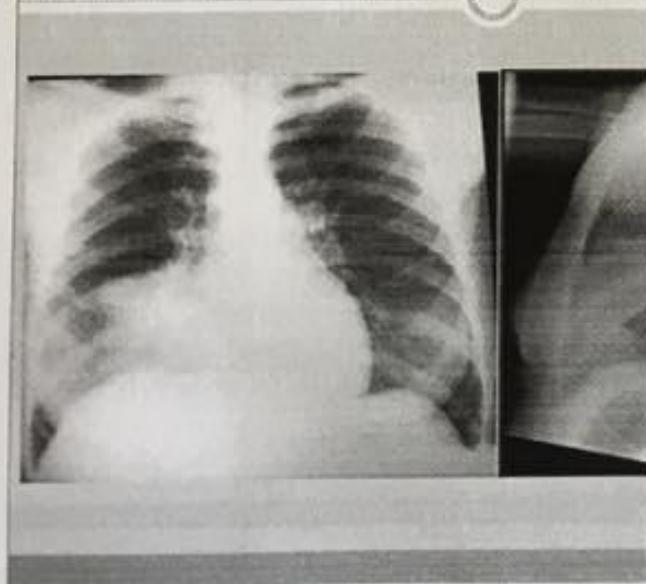
This is a neonate with Lung collapse.

The cause? endobronchial intubation.

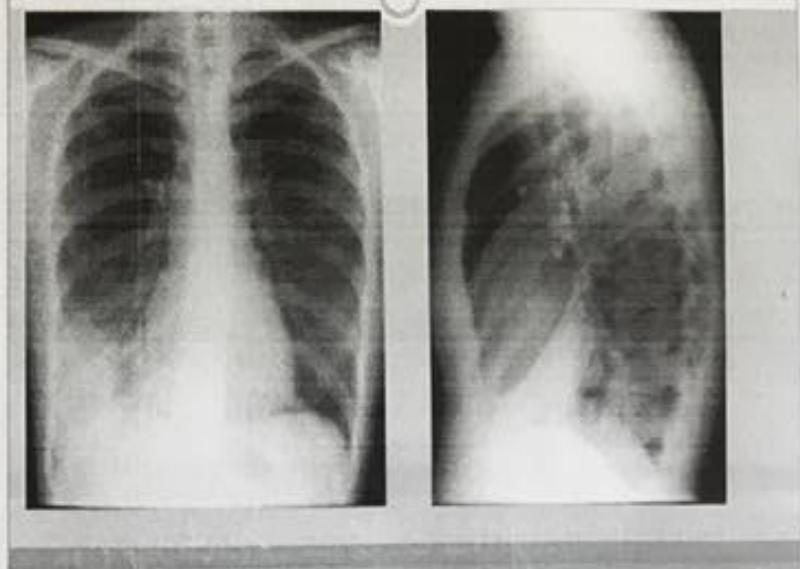


- * This patient presented to the hospital with cough and fever
What's the possible cause of these symptoms, with this chest x-ray?
 - pneumonia (lower lobe)

Right middle lobe conso



Right lower lobe consolidation



- * Underwater Seal System, attaches the patient's chest tube.
- * indications:- 1. pneumothorax | 3. haemothorax | 5. post-op
2. pleural effusion | 4. chylothorax. ↓

CABEG

MORE ABOUT DRAINAGE CANISTERS

ONE-BOTTLE SYSTEM

- SIMPLEST SET UP
- FIRST TUBE SUBMERGED IN 2 CM WATER CREATES A WATER SEAL
- SECOND TUBE CONNECTED TO WALL SUCTION
- EXCESSIVE ACCUMULATION OF FLUID CAN CAUSE DECREASED FUNCTION OF THE UNIT

One-bottle System

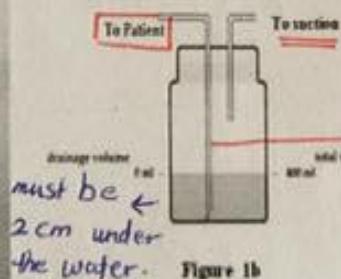


Figure 1b

complication:

→ if this tube goes out from the water, the air will enter the pleura (pneumothorax)

because of the pressure difference.

CLASSIFICATION

HYPOTENSIVE TYPE I

ABG CHANGES:

- $\text{PaO}_2 \downarrow$
($< 60 \text{ mmHg} / 8.0 \text{ kPa}$)
- PaCO_2 normal or \downarrow
($< 50 \text{ mmHg} / 6.7 \text{ kPa}$)
- $\text{PA-aO}_2 \uparrow$

VENTILATORY TYPE II

ABG CHANGES:

- $\text{PaO}_2 \downarrow$
($< 60 \text{ mmHg} / 8.0 \text{ kPa}$)
- $\text{PaCO}_2 \uparrow$
($> 50 \text{ mmHg} / 6.7 \text{ kPa}$)
- PA-aO_2 normal
- $\text{pH} \downarrow$ (Acidosis)

- uncompensated respiratory acidosis, Resp. failure Type II
 - pH 7.02, CO₂ 65, HCO₃ 23 and PaO₂ 55
 acidosis hypercapnia normal low.
 - compensated metabolic acidosis, possible cause
 - pH 7.25, CO₂ 22, HCO₃ 19, pO₂ 60
 acidosis low low (acidosis)
 - pH 7.52, CO₂ 21, HCO₃ 23, pO₂ 45
 Alkalosis low normal hypoxia.
 - uncompensated resp alkalosis, Resp. failure Type I.
- acute or chronic renal injury
 aspirin intoxication
 alcohol intoxication

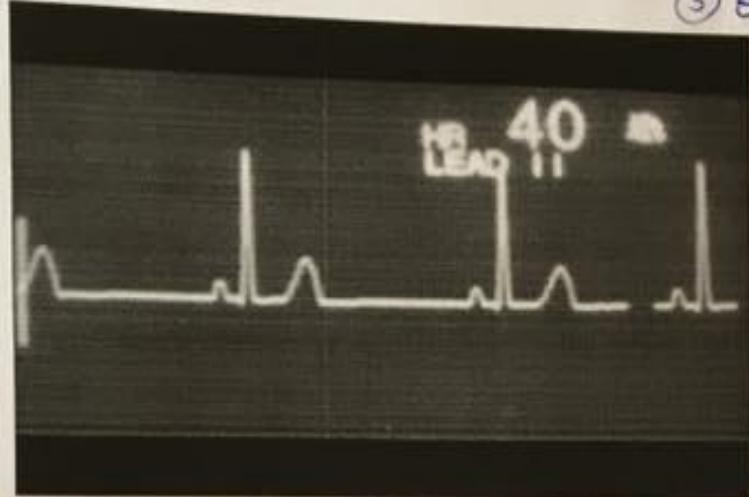
Glasgow Coma Scale						
	Points Scored					
	①	②	③	④	⑤	⑥
Best motor response	OS	Extends to pain (decerebrate)	Flaxes to pain (decorticate)	Withdraw from pain	Locally to pain	Obeys commands
Best verbal response	No verbal response	Incomprehensible sounds	Inappropriate words	Confused	Oriented	
Eye opening	No opening	Pain	Open your eyes	Spontaneous		

sketchymedicine.com

Remember: even a toddler has a GCS of 3!

You must think about anesthesia
related causes =>

- * This is bradycardia, possible causes ① propofol ② morphine
③ scopolamine.



* what's the
management?
Atropine.

* Diagnosis?

Sinus Tachycardia.

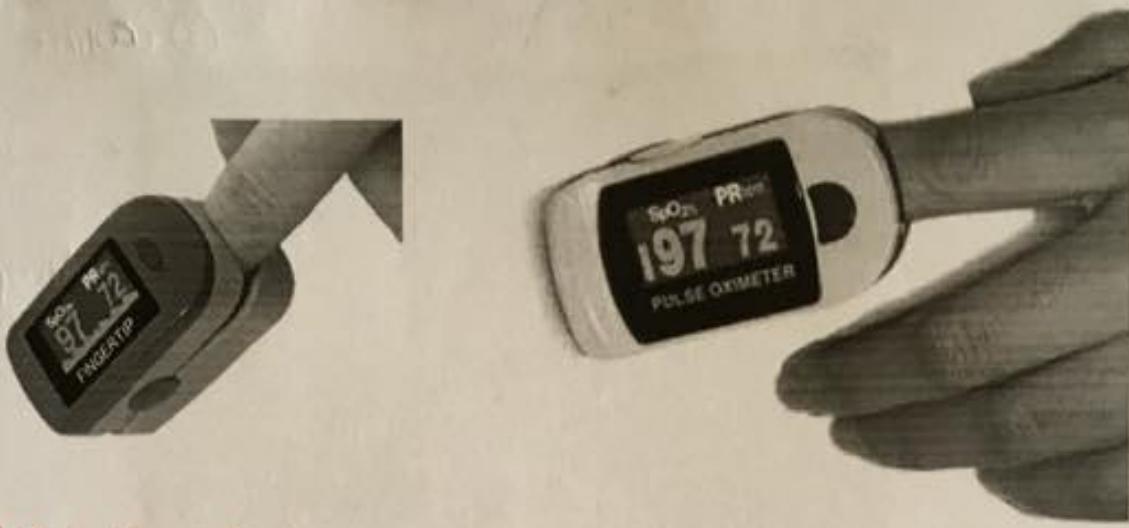
* causes?

1. pain
2. fever
3. hypovolemic shock
4. Thiopental
5. Ketamine

* management? 1. Treat the underlying cause 2. β -Blocker (1st choice)

3. calcium channel blocker (2nd choice)





* What is this device ? Pulse oximeter

* What does it reflect ?

1. pulse wave.

2. pulse → reflects the circulation.

3. SpO_2 → reflects the oxygenation.

Estimating FiO_2

every one

Litre increases ←
the FiO_2
by 0.04
↓

we can give
maximum 6L

→ after that's
there will be irritation.

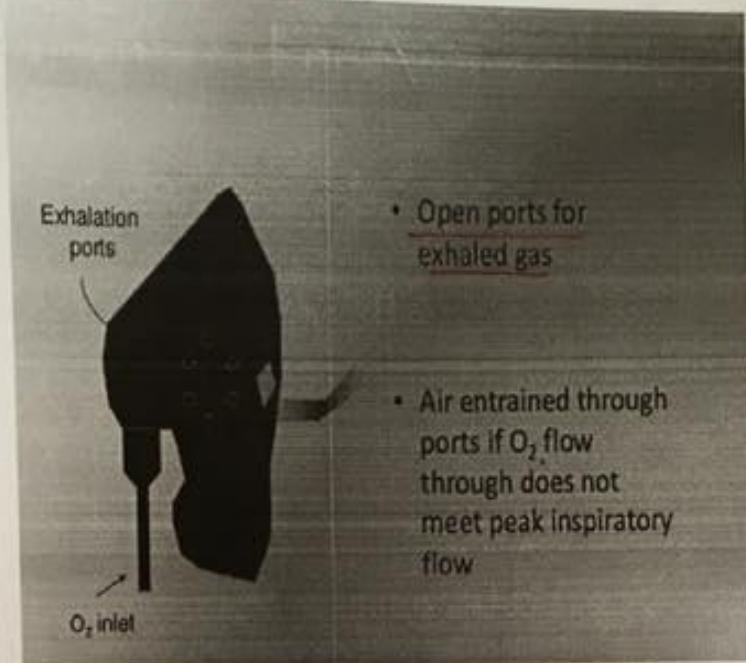
O ₂ Flow rate	FiO ₂
Nasal cannula	
1	0.24
2	0.28
3	0.32
4	0.36
5	0.4
6	0.44

O ₂ Flow rate	FiO ₂
Oxygen mask	
5-6	0.4
6-7	0.5
7-8	0.6

O ₂ Flow rate	FiO ₂
Mask with reservoir	
6	0.6
7	0.7
8	0.8
9	0.80+
10	0.80+

→ here we can make titration, so we can prevent hypoxia.

Simple face mask.

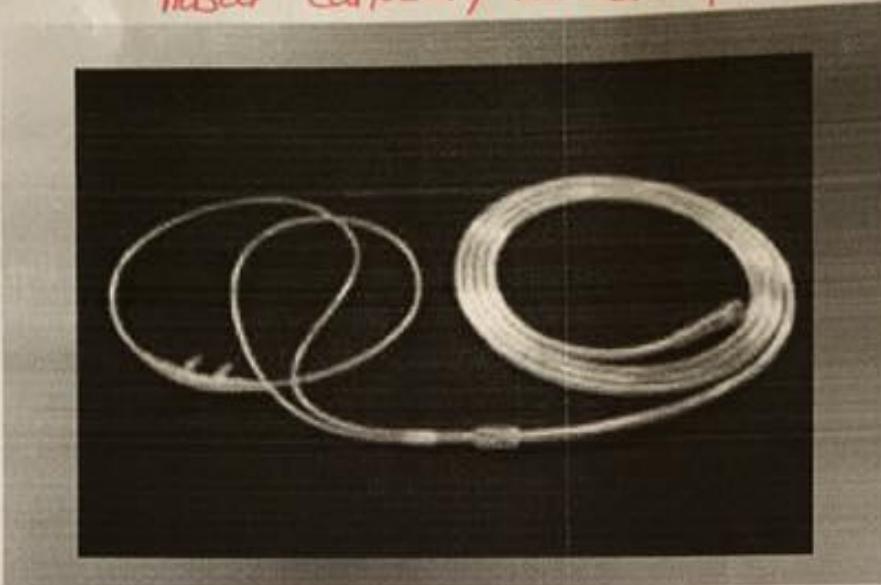


* There's leak,
that's why the
O₂ saturation
does not reach
100 %

Simple face mask :
Variable performance
system , small capacity
Low flow (6 L/min)
↓
(40% or 0.4)
FO₂.

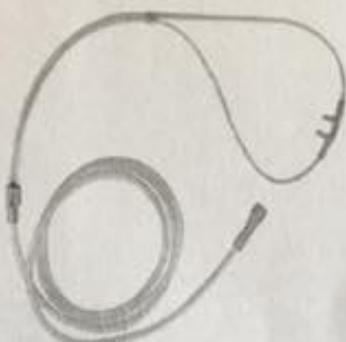


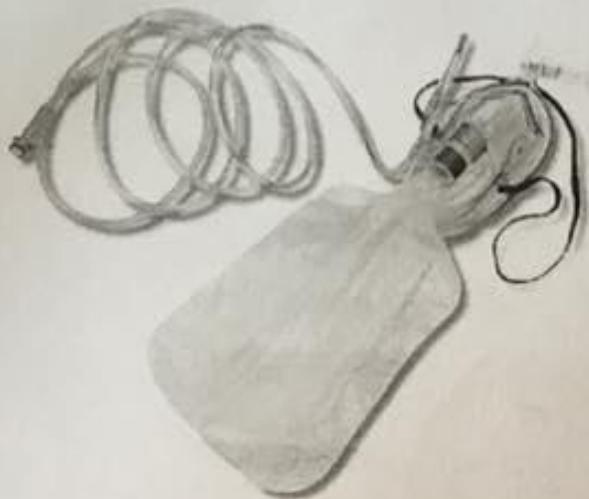
nasal cannula / variable performance.



Nasal cannula :
Variable performance system

* what's the minimum F_iO_2 does it give? 0.24





- There are exhalation ports but there's unidirectional valve
- the air can go out, but can't go in, so there's no leak, and the O₂ can reach high value (around 80%)

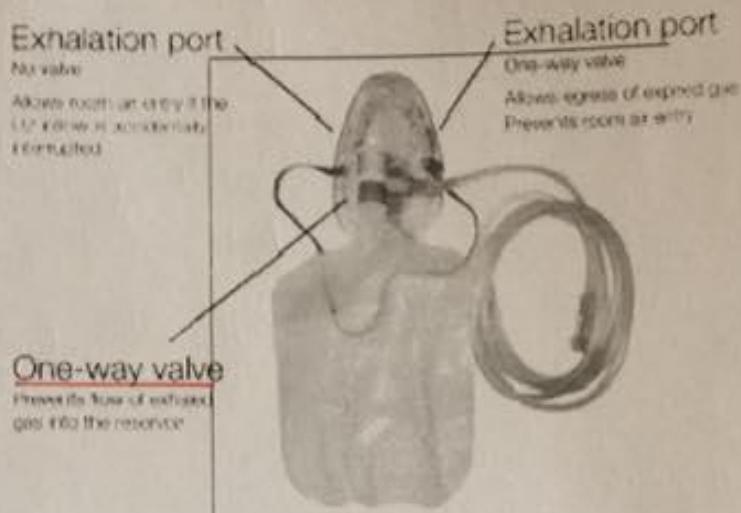
Non-rebreathing mask :
Variable performance system



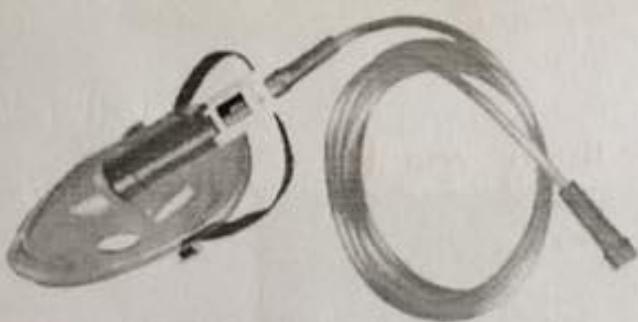
non-rebreathing mask .



reservoir bag .

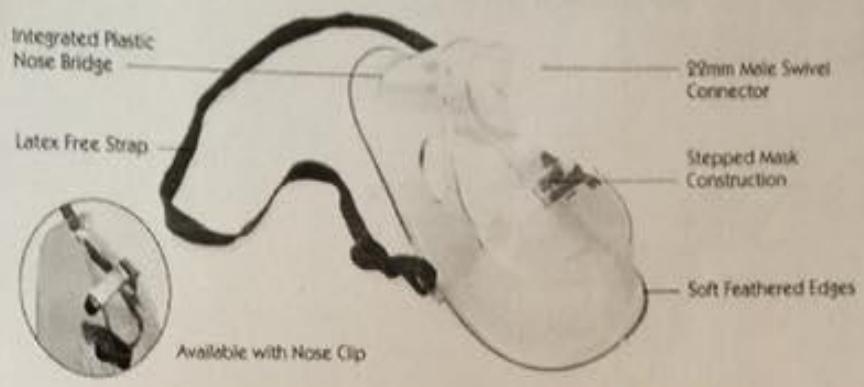


- * if it presents in both sides → the mask is non-rebreathing completely.
- * if just in one side (non-rebreathing mask) → partial non-rebreathing

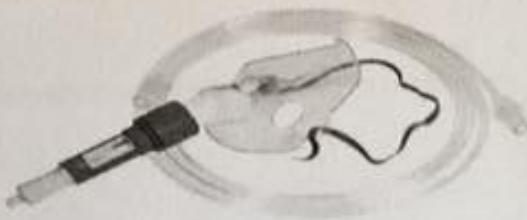


Venturi mask : → it can maximum give 60% O₂.
Fixed performance system
Bernoulli principle

Venticaire®



Q14)- based on what principle does this mask work? Bernoulli's
2- what type of mask is this? Fixed performance system



A) What is mask 1 & what is the maximum FiO₂ that can be delivered by it?

non-rebreathing mask, maximum 80%.



B) what is FiO₂ of mask 2 when the flow is 5 L?

simple face mask, maximum 60%,
when the flow is 5L \Rightarrow FiO₂ = 40%.

* absolute:

- Q15) 1- what is this **Laryngeal mask**.
2- give a contraindication for its use
3- name two conditions where it is used? ① short procedure.
② difficult air way management

* relative:

1. poor oral opening.
2. poor lung compliance.
3. Long procedure (> 2 hrs)
4. head and neck surgery.





* what is this ?

pulmonary function test device.

* what does it measure ?

1. volume (forced vital capacity)
2. capacity

* what is its use ?

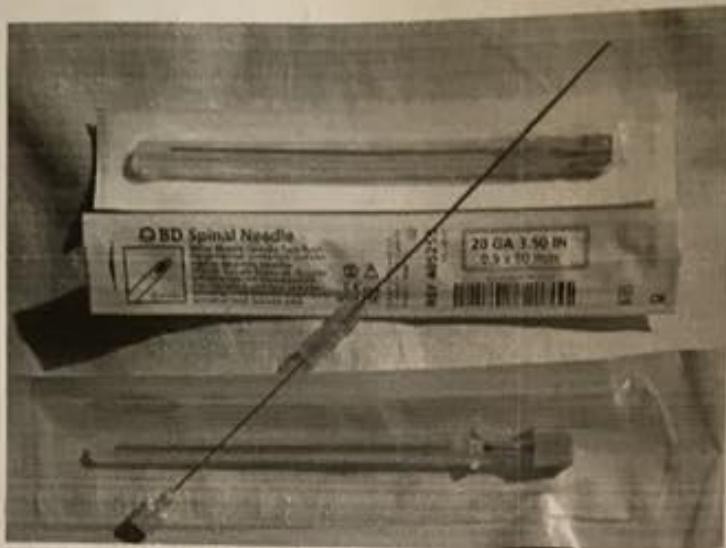
to prevent atelectasis in post-operative patients.

* This is Tuohy needle (epidural needle)

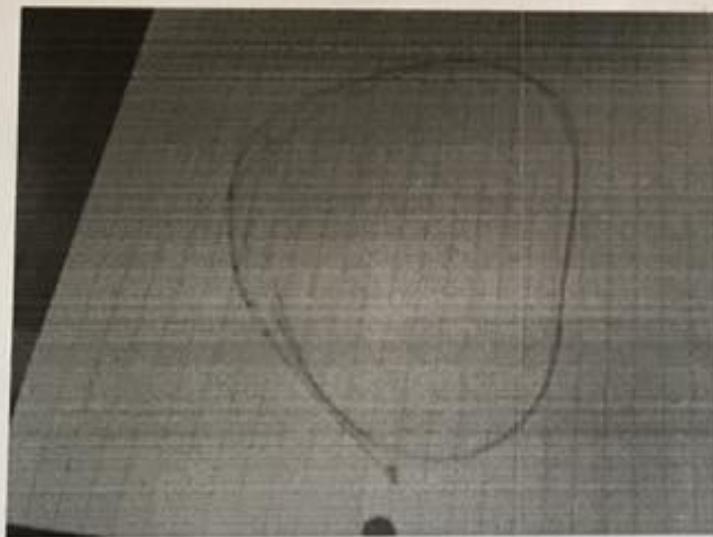
* gauge = 18

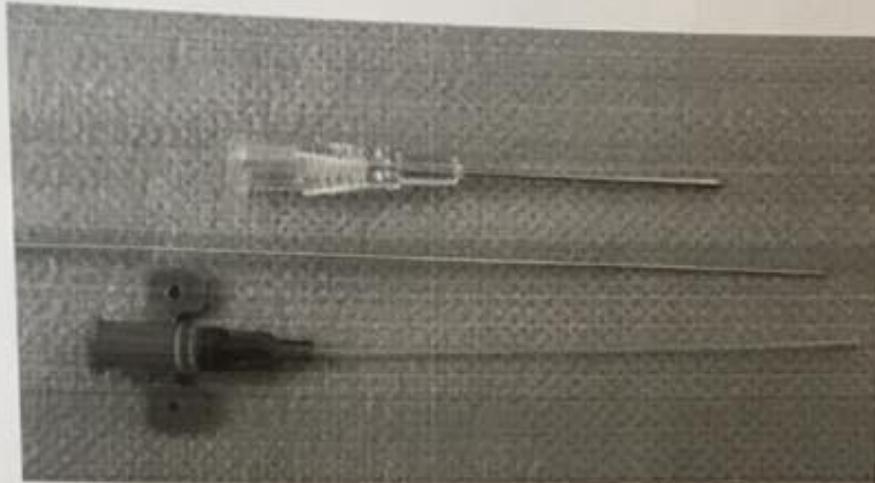


Spinal needle \Rightarrow Quincke (cutting)



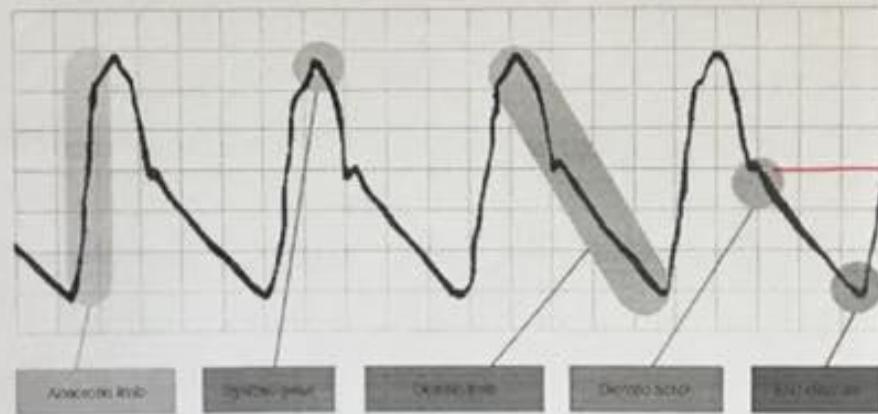
Epidural catheter \rightarrow between ligamentum flavum and dura matter.





* equipment of arterial line .

Normal arterial waveform



→ dicrotic notch
↓
closure of aortic valve.

The aortic root marks the waveform's initial upstroke, which occurs as blood is rapidly ejected from the ventricle through the open aortic valve into the aorta.

Atrial pressure then falls sharply, resulting in the systolic peak – the waveform's highest point.

As blood continues into the peripheral vessels, arterial pressure falls and the waveform begins a downward trend, called the diastolic rise. Arterial pressure usually falls until pressure in the ventricle is less than pressure in the aortic root.

When ventricular pressure is lower than aortic root pressure, the aortic valve closes. This event appears as a small notch on the waveform's downward slope, called the dicrotic notch.

When the aortic valve closes, diastole begins, progressing until systolic root pressure gradually falls to its lowest point. On the waveform, this is known as end diastole.

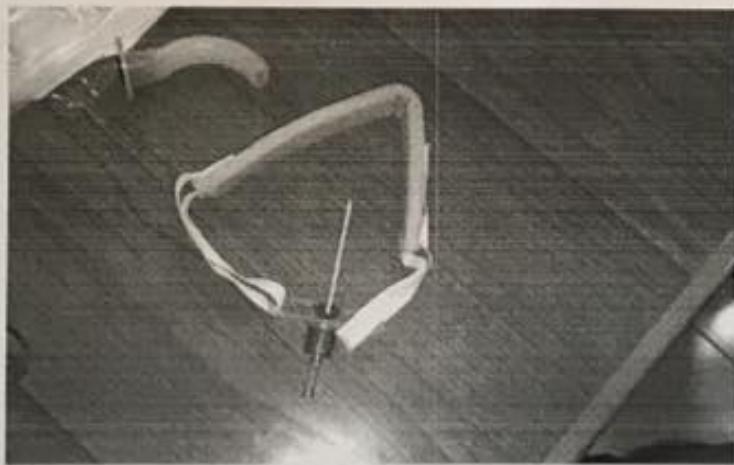
Cricoid pressure (rapid sequence induction)

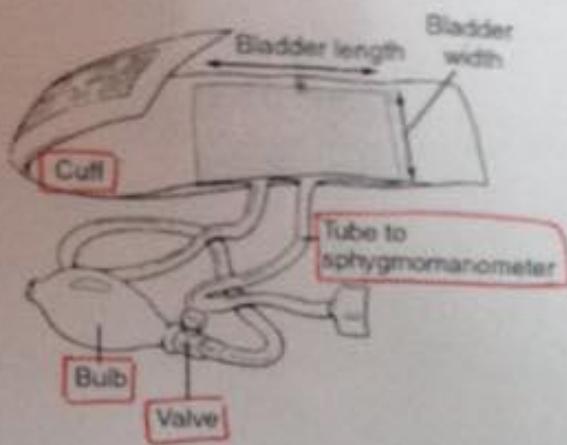


→ head tilt, chin lift,
jaw thrust.

Cricothyrotomy set when we use it ? when we

can't intubate,
can't ventilate .





what is this? device used for non-invasive BP monitoring.

Table 3. Causes of Abnormal CO₂ Levels

Hypocapnia (Decreased CO ₂)	Hypercapnia (Increased CO ₂)
Hyperventilation	Hypoventilation
Hypothermia (decreased metabolic rate)	Hypothermia
Decreased pulmonary blood flow (decreased cardiac output)	Improved pulmonary blood flow after resuscitation or hypotension
Technical issues:	Technical issues:
Incorrect placement of sampling catheter	Water in capnography device
Inadequate sampling volume	Anesthetic breathing circuit error
V/Q mismatch	<ul style="list-style-type: none"> • Inadequate fresh gas flow • Rebreathing • Exhausted soda lime • Faulty circuit absorber valves
<ul style="list-style-type: none"> • Pulmonary thromboembolism • Acute pulmonary edema • Air embolism 	Low bicarbonate

Ventilation	metabolism	circulation	device
hypercapnia	hypoventilation	hyperdynamic	dissociation of valve the device + unidirectional
hypocapnia	hyperventilation	↓	exhausted soda-lime

→ semi-open circuit

MAPLESON SYSTEM

The best one for spontaneous ventilation.

The best for controlled ventilation
it is called (Bain circuit)

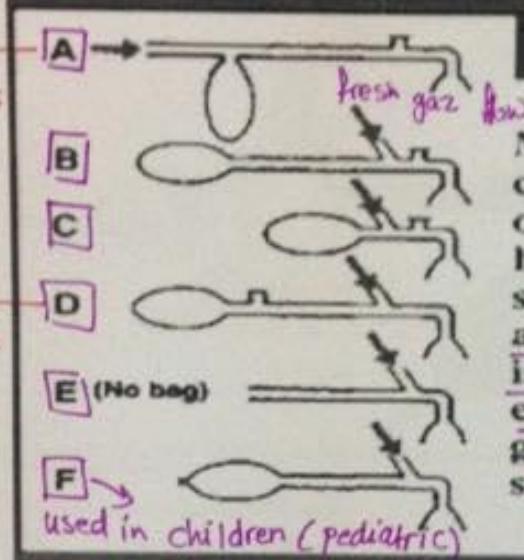
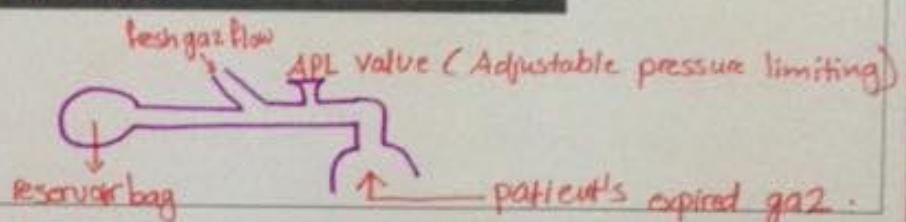


Figure 1

Mapleson classification of anaesthetic breathing systems. The arrow indicates entry of fresh gas to the system.



Mapleson D

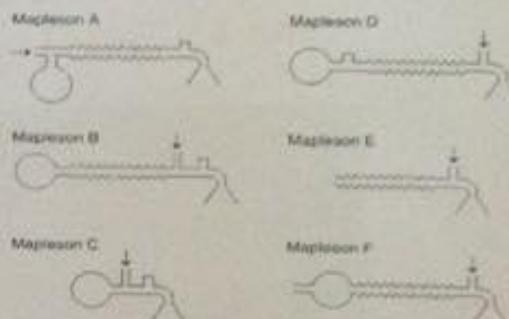


FIGURE 4-3. Mapleson circuits.

Mapleson F (Jackson Rees Modification)

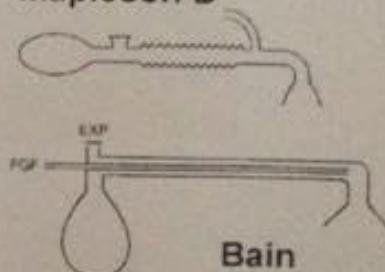
Ayres - 1927

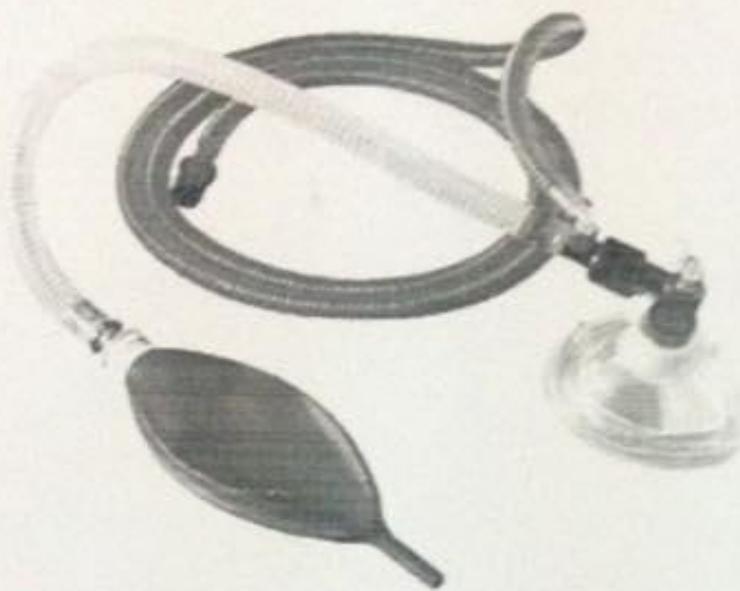
JR - 1950



- The Mapleson F or Jackson Rees modification of the Ayres T-Piece is a basic system for use with very small patients.
- It is a big disadvantage that you cannot remove waste gases safely.

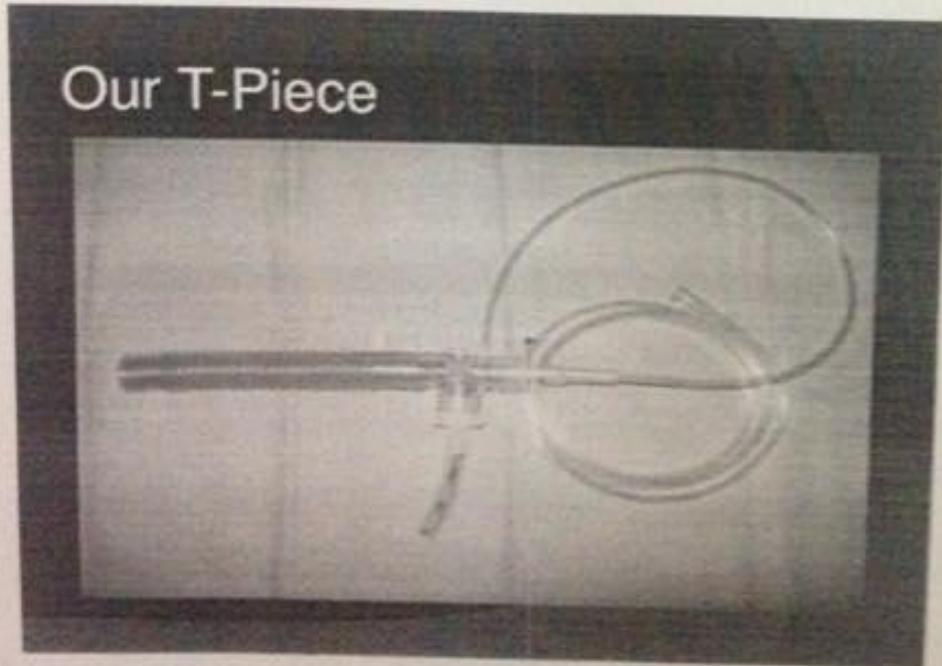
Mapleson D

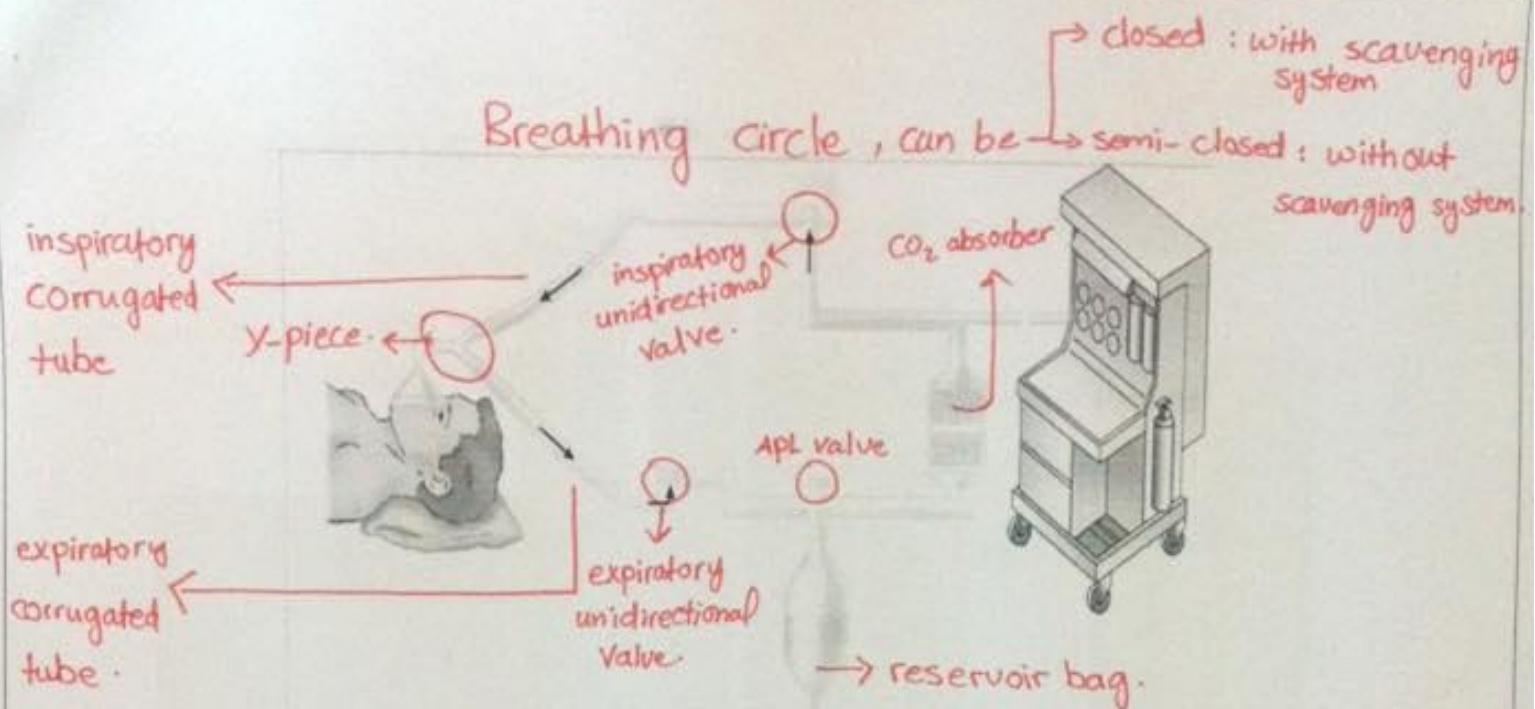




- * Mapelson F (Jackson Rees)
- * Semi - open circuit

* This is Tracheostomy tool attached to Mapelson E and it's called T-Piece which is a semi- open circuit



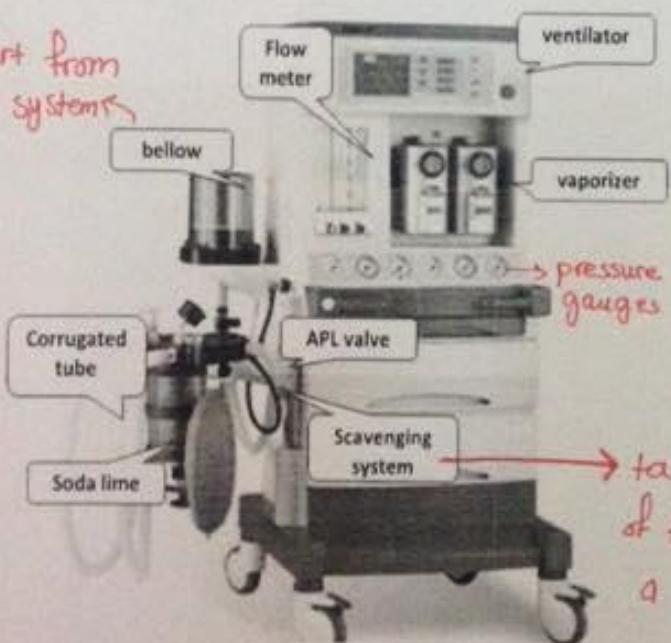


Source: Butterworth JF, Mackey DC, Wasnick JD: Morgan & Mikhail's Clinical Anesthesiology.
20th Edition: www.accessmedicine.com

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- The APL valve is used only during spontaneous breathing (manual).
- The function of the APL valve is to control the pressure inside the circle,
⇒ when the APL valve is closed, the pressure inside the reservoir bag will increase and create pressure gradient with the lung.

It is a part from the circle system.



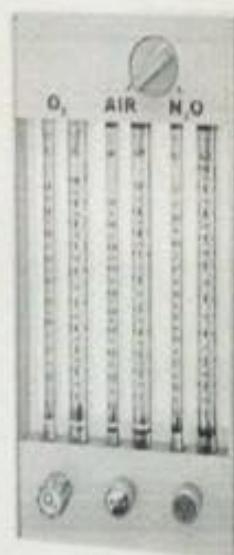
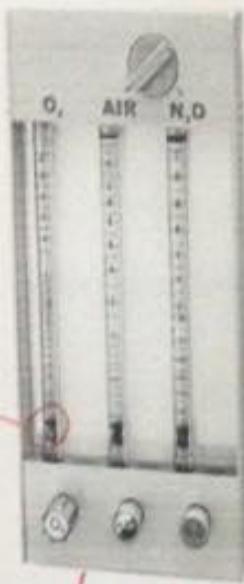
→ converts the anesthetic drug from liquid to vapor.

Yellow → sevoflurane (MAC = 2)
Purple → isoflurane (MAC = 1.2)

→ take the waste gas out of the operating theater, has a negative pressure

Q5) picture shows flowmeter and the Q was what is the function of it and there is an arrow on CO₂ absorbent and the Q was identify the structure?

composed of : sodium hydroxide, potassium hydroxide, calcium chloride, water, silica.



[The] function of the flow meter is measurement of the flow.
* when we open the N₂O flow, the O₂ will be opened (3:1)

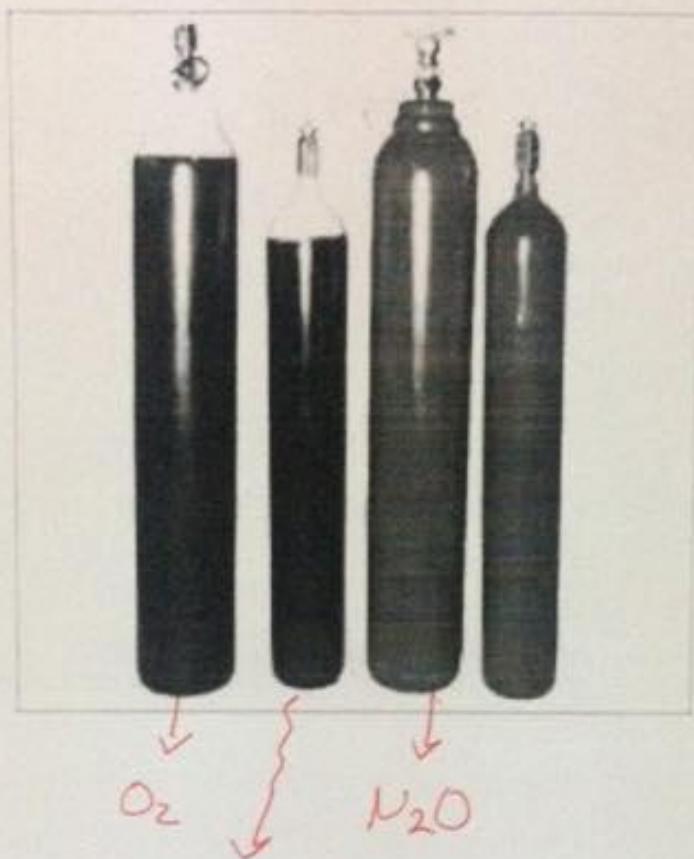
N₂O O₂
proportioning
System

vaborizer

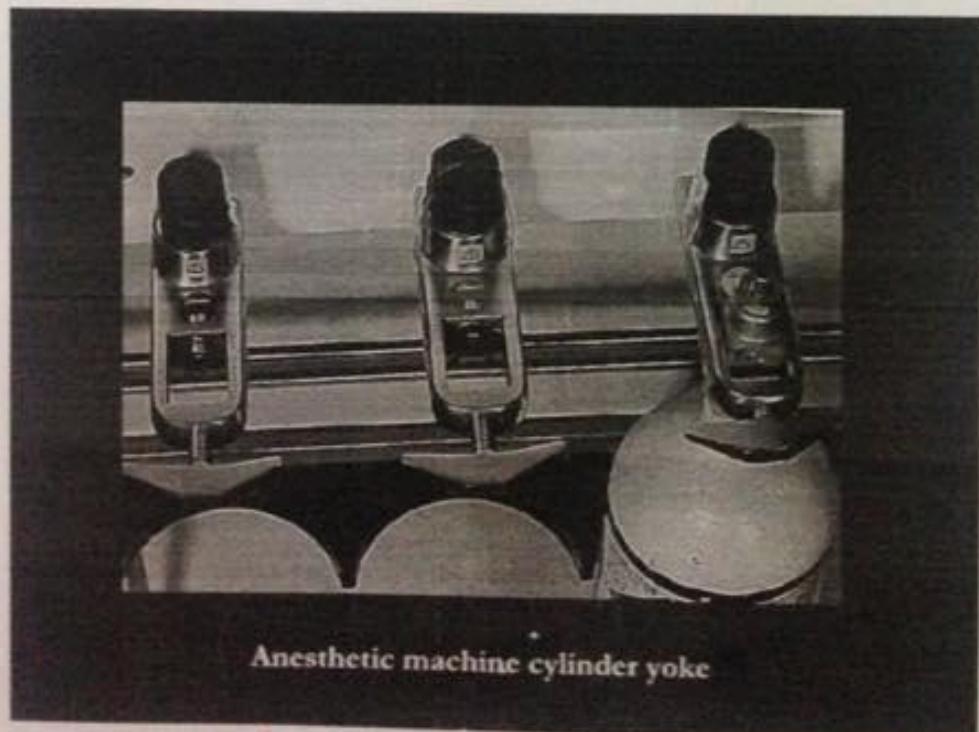


- * sevoflurane is better for induction \Rightarrow non-irritant.
- * Sevo is more expensive than iso. (pleasant)
- * Sevo is used for pediatrics
- * iso is more potent (less MAC value)
- * Sevo has more risk for emergence awareness because it has higher MAC value and less potency
- * iso has faster offset

* go and see
the colored picture



The small one is E size (behind the anesthesia machine)



Anesthetic machine cylinder yoke

* it has safety system → every cylinder has its special yoke

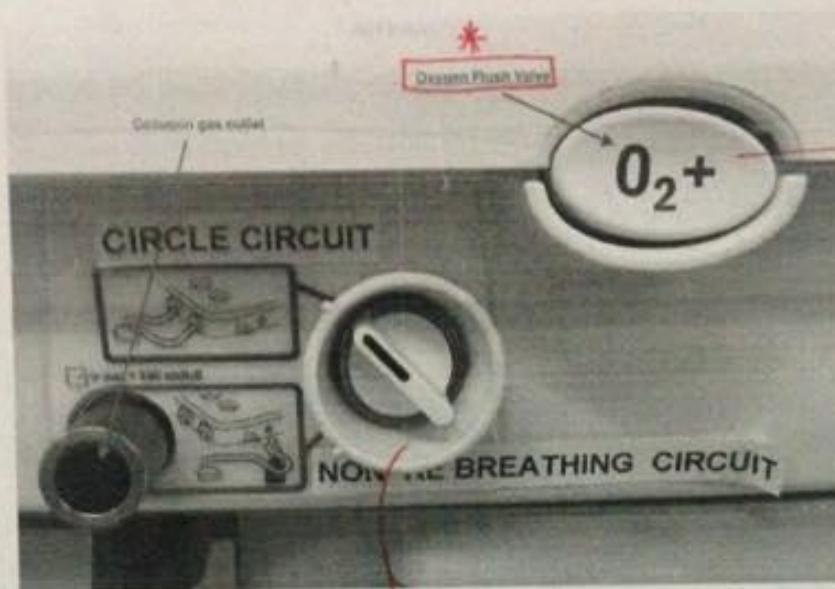
Hanger Yolk

- Hanger Yolk: orients and supports the cylinder, providing a gas-tight seal and ensuring a unidirectional gas flow into the machine
- Index pins; Pin Index Safety System (PISS) is gas specific → prevents accidental rearrangement of cylinders (e.g., switching O₂ and N₂O)



Diameter index safety system (DISS)

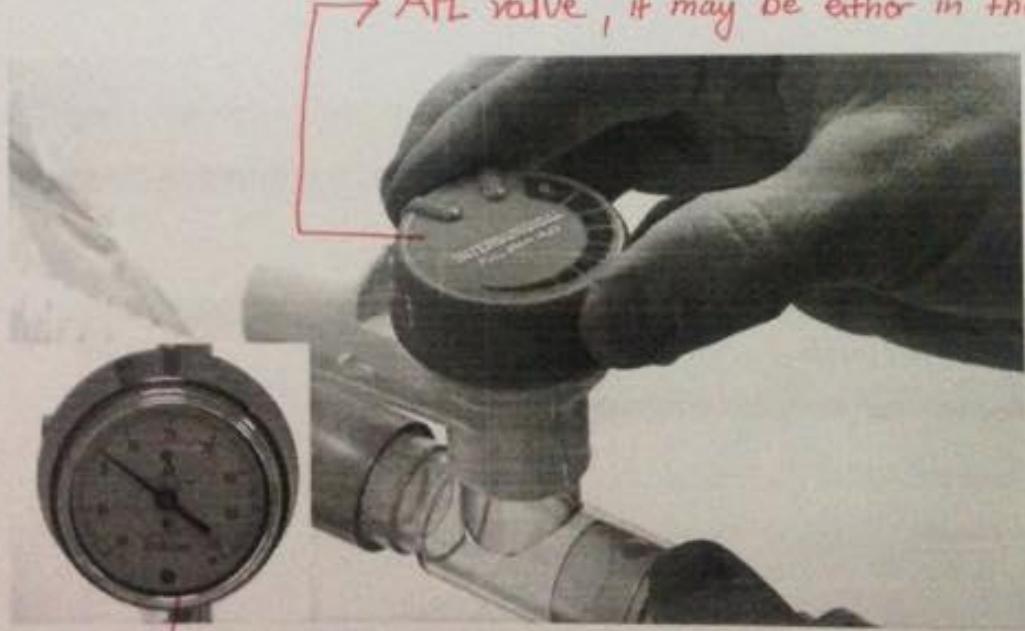




→ it give O_2 at high pressure around (3 bar) in emergency cases
e.g.: Laryngospasm

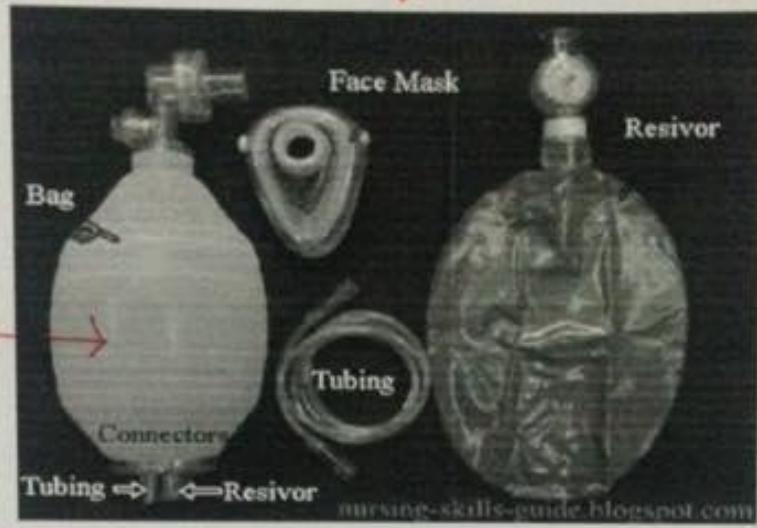
note : 1 bar = 760 mmHg

Switch to transfer the system
from circle → circuit
(closed) → (opened)



gauge pressure, it may be to
the cylinder
the pipeline
the airway pressure.

① Face mask is rigid unlike the simple face mask which is rubber
 ② Face mask gives maximum FIO_2 around 100% but the simple face mask around 60%,



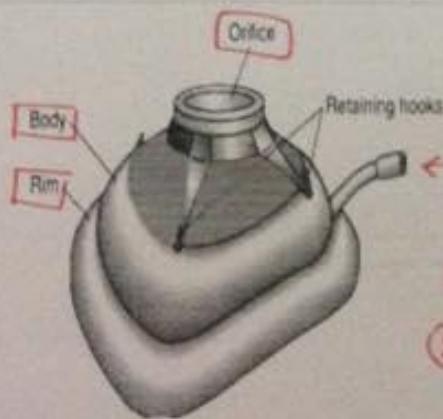
ambu bag
 ↓
 self-inflating
 unlike the resivor
 bag.

③ Face mask doesn't have ports but simple face mask does.

PARTS OF FACEMASK

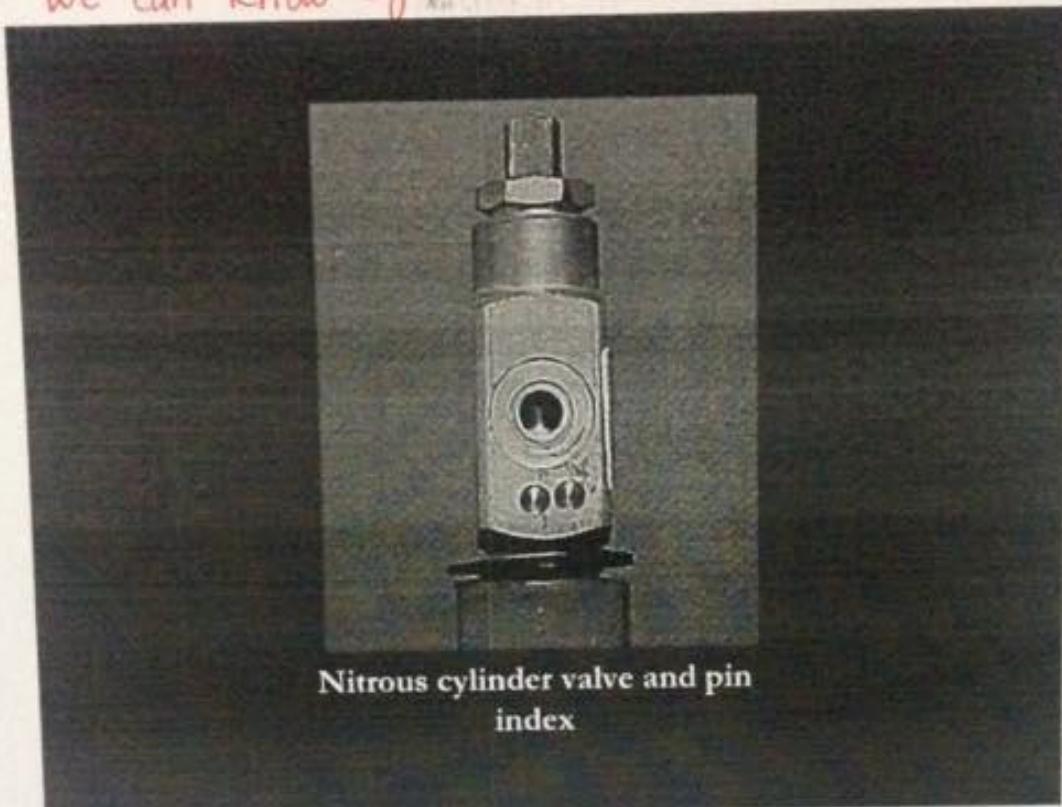
Body (Shell or Dome)

- Constitutes the main part of the mask
- Transparent facemask allows observation for vomitus, secretions, blood, lip color, and exhaled moisture and is better accepted by a conscious patient.



← important for inflating the cuff to:
 ① fit the patient's face
 ② prevent any leak.

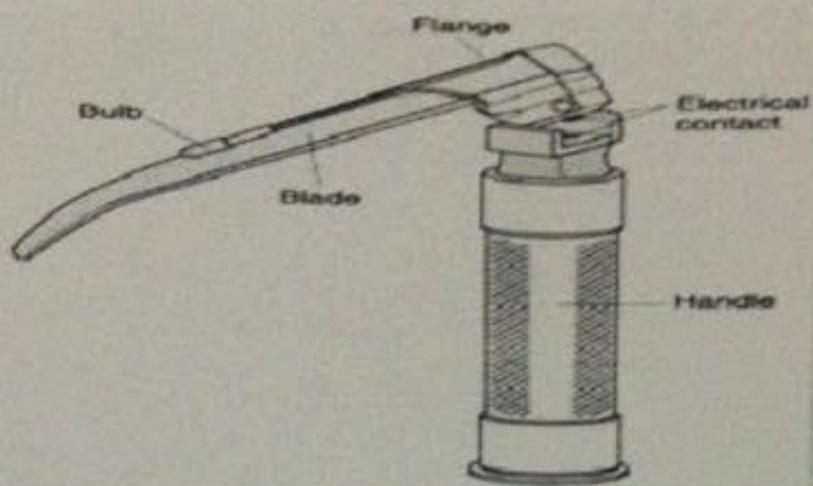
we can know by its color (blue) what does it contain (N_2O)



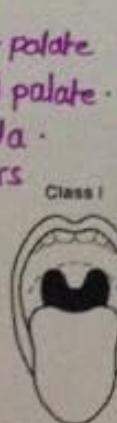
**Nitrous cylinder valve and pin
index**

PRESSURE GAUGE

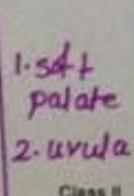




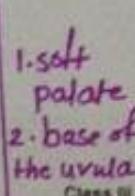
1. soft palate
2. hard palate
3. uvula
4. pillars



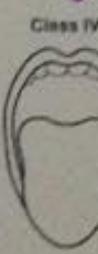
1. soft palate
2. uvula



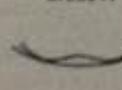
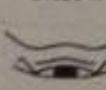
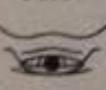
1. soft palate
2. base of the uvula



hard palate only.



Grade I Grade II Grade III Grade IV



grade I : full view of glottis and epiglottis

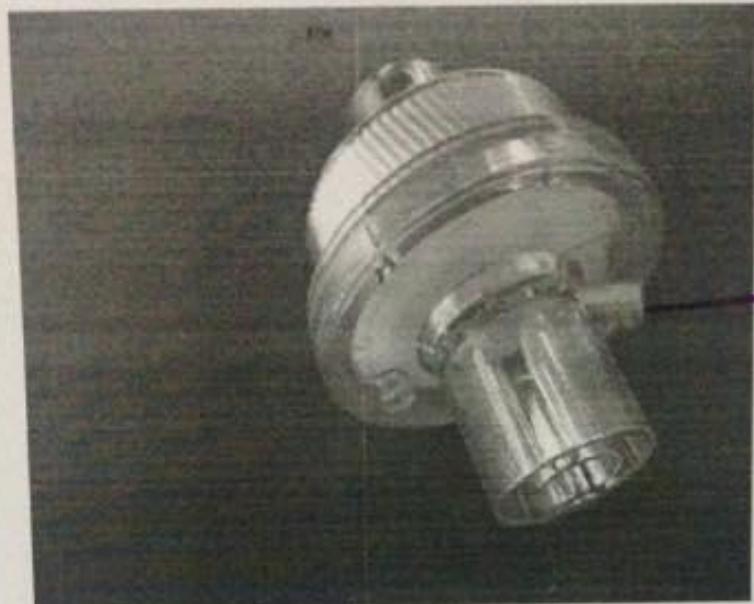
⇒ grade II : epiglottis and posterior extremity of glottis

grade III : epiglottis only

grade IV : neither glottis nor epiglottis

1. (Mallampati classification)

2. Cormack - Lehane classification



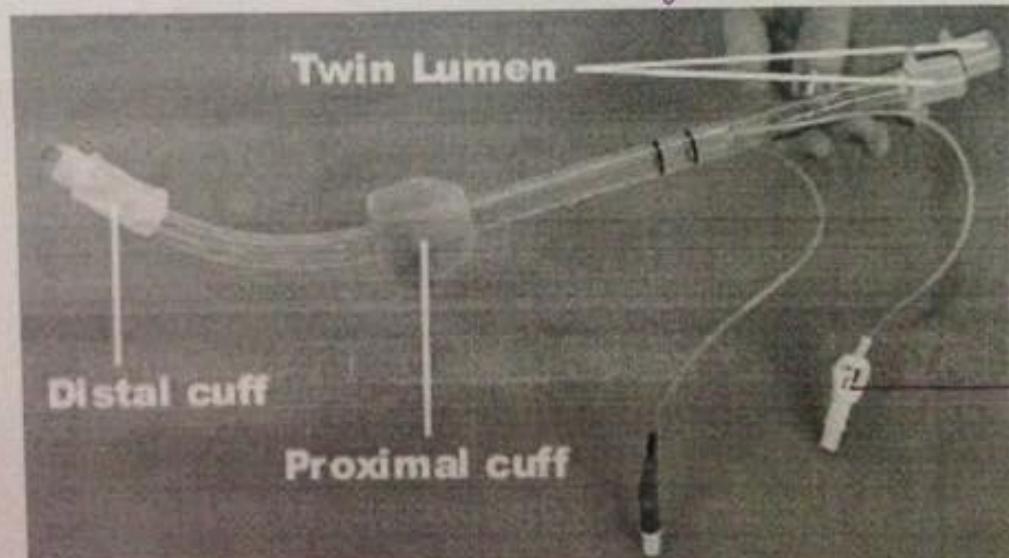
→ This attaches to the mainstream analyzer (slide #4) to take CO₂ sample to the capnograph.

* This is HME (heat moisture exchanger)

* Function ? ① humidification : storage of the warm exhaled gas from the patient and mixing it with the cold inhaled gas from the machine .

② filtering (antibacterial)

Combi-tube airway



- blind induction airway device .
- The distal cuff is smaller in size than the proximal one .



• nasal airway

⇒ it is flexible , unlike the oral airway .

gag reflex ←

Oral airway



⇒ To hold the tongue away from the posterior pharynx .

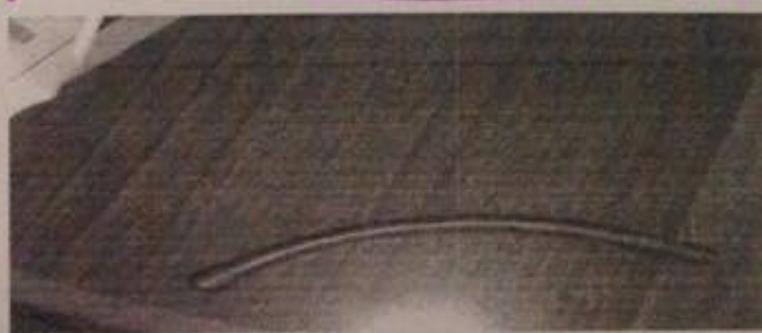


no gag reflex

←

Nasal airway

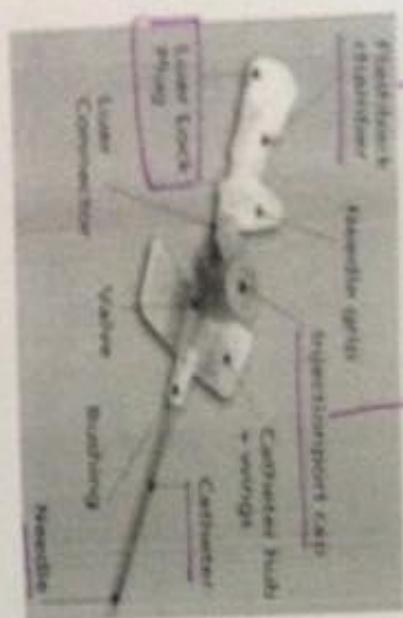
→ contraindications :-



- ① nasal polyp
- ② basal skull fracture .
- ③ bleeding tendency

(in and out flush)

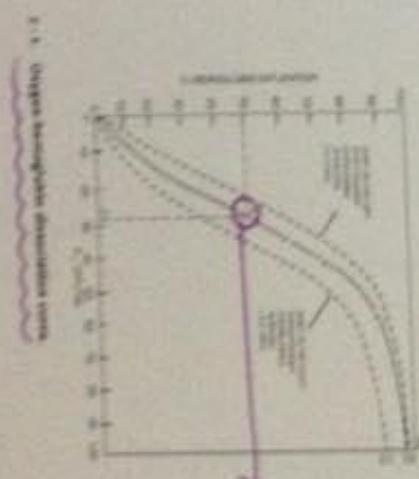
In flush only.



Tooth needle
(10 cm)



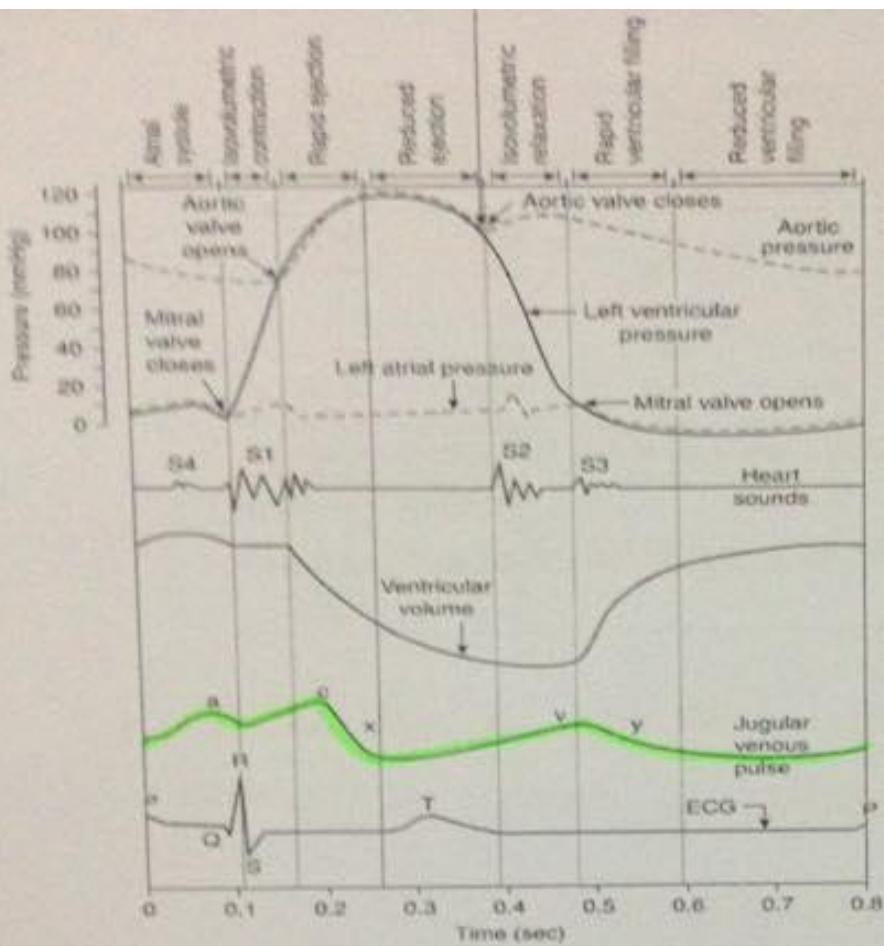
Spiral needle.



This point is
P₅₀: the O₂
tension at which
haemoglobin is
50% saturated

* The importance of P₅₀ is to
specify the position of O₂
dissociation curve.

→ normally 26.4 mmHg



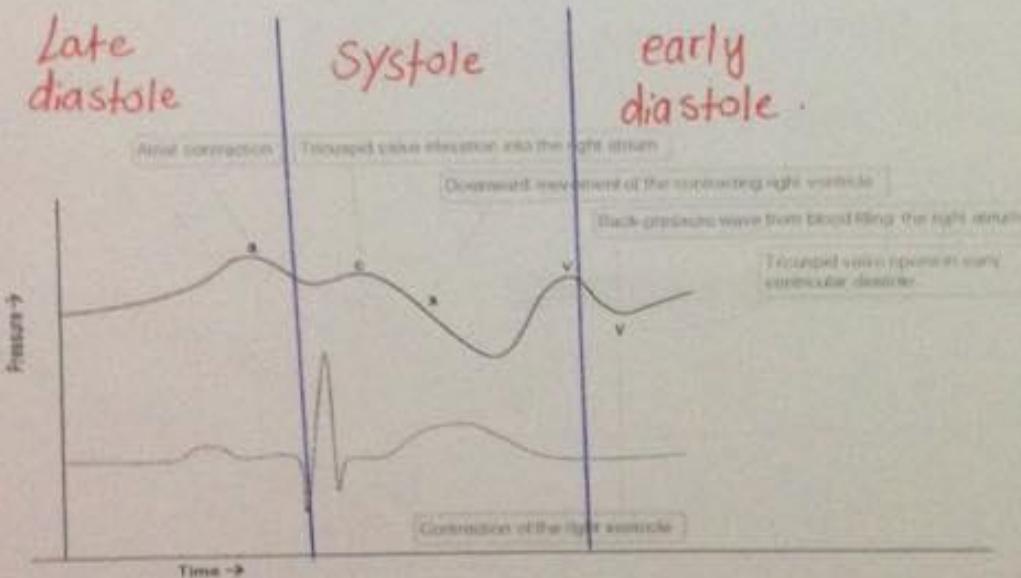
e cardiac cycle.

a: atrial contraction.
c: ventricular contraction and closure of the tricuspid valve (early systole)

x: continue ventricular contraction and atrial relaxation.

v: atrial filling.

y: opening of tricuspid valve (ventricular filling)



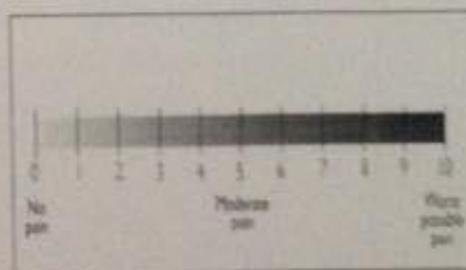
* wave a → The atrial contraction is active filling to the ventricle.

* wave y → passive filling, because of the pressure difference.

Table 4. Assessment of Hemorrhagic Shock

	Class I	Class II	Class III	Class IV
Percentage Blood Loss	0-15%	15-25%	30-40%	>40%
Percentage TBW loss	0-2%	2-4%	5-7%	>7%
Heart Rate	<100	>100	>120	>140
Blood Pressure	Normal	Normal	Decreased	Decreased
Treatment	Rapid infusion of 1/2L of crystalloid (e.g. Ringer's lactate, maintenance fluids)	Rapid infusion of 2 L of crystalloid, unless losses with crystalloid (1:3 or 1:600), colloid (1:1)	Rapid infusion of 2 L of crystalloid, unless losses with crystalloid (1:3 or 1:600), colloid (1:1)	Rapid infusion of 2 L of crystalloid, unless losses with crystalloid (1:3 or 1:600), colloid (1:1)
Note		Goal is to increase SBP to 90 mm Hg or more >25 mm Hg above baseline	Goal is to increase SBP to 90 mm Hg or more >25 mm Hg above baseline	

(pain scoring system)



مخطط الالم / pain score

نقطة الالم / point

1-3 → mild pain → give (NSAID)

4-7 → moderate pain → give weak opioid (tramadol, codeine)

8-10 → severe pain → give strong opioid (morphine).

CO₂ ABSORBERS

- Soda lime composition: 80% calcium chloride, 15% water, 4% sodium hydroxide, and 1% potassium hydroxide.
 - Small amounts of silica added for hardness (and minimize alkaline dust).
- * CO₂ absorber is whitish in color because most of its content is calcium chloride (80%).

exhaustion of soda lime

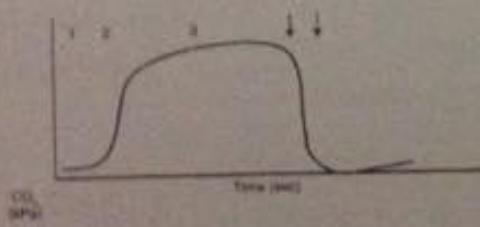


FIGURE 3-1. Capnography tracings.

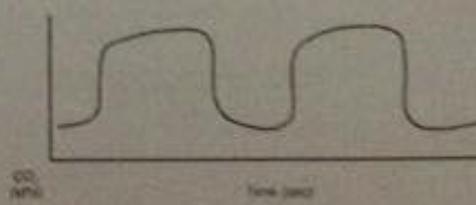


FIGURE 3-2. Capnograph (rebreathing).

* patient with brain edema \rightarrow giving him glucose will increase his edema.

Q7) picture shows D5W (dextran Swater) and the q was what is the content of dextran mg/ml in this solution and if this solution hypo/iso/hyper tonic?

Table 6. IV Fluid Solutions

		ECF	Nerger's Lactate	0.9% NS	0.45% NS in D5W	D5W	3/2 D5W + 1/2 NS	Plasmalyte
mg/L	Na ⁺	142	138	154	11	-	9	140
	K ⁺	4	4	-	-	-	-	5
	Ca ²⁺	4	3	-	-	-	-	-
	Mg ²⁺	3	-	-	-	-	-	3
	Cl ⁻	103	109	154	11	-	91	98
	HCO ₃ ⁻	27	28 [*]	-	-	-	-	27
mOsm/L		290-310	273	308	154	252	281	294
pH		7.4	6.5	5.0	4.5	4.0	4.3	7.4

Concentrations

* any percentage when you multiply it by 10, the unit will be converted to mg/mL.

- ① D5W contains 5% glucose, which equals to 50 mg/mL.
 ② The solution is hypotonic (252 mOsm/L)

note: isotonic solution osmolarity is 290 ± 10 mOsm/L

* what are the layers you go through when you introduce the needle in epidural anesthesia?

skin



subcutaneous fat



supraspinatus ligament



infraspinatus



ligamentum flavum



epidural space

Epidural and Spinal Anesthesia

- most useful for surgeries performed below level of umbilicus

Anatomy of Spinal/Epidural Area

- spinal cord extends to L2, dural sac to S2 in adults
- nerve roots (cauda equina) from L2 to S2
- needle inserted below L2 should not encounter cord, thus L3-L4, L4-L5 interspace commonly used
- structures penetrated
 - skin
 - subcutaneous fat
 - supraspinous ligament
 - interspinous ligament
 - ligamentum flavum (last layer before epidural space)
 - dura + arachnoid for spinal anesthesia

Table 8. Epidural vs. Spinal Anesthesia

	Epidural	Spinal
Deposition Site	LA injected in epidural space (space between ligamentum flavum and dura)	LA injected into subarachnoid space in the dural sac surrounding the spinal cord and nerve roots
Onset	Spinal blockade requires 10-15 min Slower onset of epidural effects	Spinal blockade occurs in 2-5 min
Effectiveness	Effectiveness of blockade can be variable	Very effective blockade
Difficulty	Technically more difficult, greater failure rate	Lesser risk due to visual confirmation of CSF flow
Patient Positioning	Position of patient not as important (can lie gravity neutral)	Requires LA solution + position of patient upright

Spinal anesthesia

- ① rapid and more effective than the epidural.

- ② more risk of epidural puncture.

* epidural anesthesia

→ needs more titration.

Table 9. Epidural vs. Spinal Anesthesia (continued)

	Epidural	Spinal
Specific Gravity/Spread	Epidural epinephrine spreads throughout the potential space; specific gravity of solution does not affect spread.	LA solution may be much hypertonic (higher specific gravity than the cerebrospinal fluid) by mixing with 10% dextrose, thus increasing spread of LA to the dependent (low) areas of the subarachnoid space.
Dosage	Large volumes of LA (usually > toxic IV dose).	Smaller dose of LA required (usually < toxic IV dose).
Continuous Infusion	Use of catheter allows for continuous infusion or repeat injections.	None.
Complications	Failure of technique Hypotension Bradycardia if cardiac sympathetics blocked (only L1-T2-4 block), e.g. "high speed" Epidural or subarachnoid hemorrhage Accidental subarachnoid injection can produce spinal anesthesia (and any of the above complications) Systemic toxicity of LA (acetaminophen induced) Catheter complications (leaking, kinking, dislodged or subarachnoid placement) Infection Dural puncture	Failure of technique Hypotension Bradycardia if cardiac sympathetics blocked (only L1-T2-4 block), e.g. "high speed" Epidural or subarachnoid hemorrhage Post-spinal headache (CSF leak) Transient paresthesias Spinal cord trauma, infection
Combined Spinal-Epidural	Combines the benefits of rapid, reliable, intense blockade of spinal anesthesia together with the flexibility of an epidural catheter.	

Contraindications to Spinal/Epidural Anesthesia

- absolute contraindications
 - lack of trained personnel
 - lack of resuscitative drugs/equipment
 - patient refusal
 - allergy to local anesthetic
 - infection at puncture site or underlying tissues
 - coagulopathies/bleeding diathesis
 - raised ICP
 - sepsis/bacteremia
 - severe hypovolemia
 - cardiac lesion with fixed output states (e.g. severe mitral/aortic stenosis)
 - lack of IV access
- relative contraindications
 - pre-existing neurological disease (demyelinating lesions)
 - previous spinal surgery, severe spinal deformity
 - prolonged surgery
 - major blood loss or maneuvers that can compromise reaction

Pharmacological family

Phenol

Barbiturate

phenocyclidine

Benzodiazepines

Table 4. Intravenous Inductive Agents

	Propofol (Diprivan®)	Thiopental (Sodium Thiopental)	Ketamine (Ketalar®, Ketaset®)	Benzodiazepines (Midazolam [Versed®], Diazepam [Valium®], Nitrazepam [Maze®])
Class	• Alkylphenol - hypnotic	• Ultra short acting barbiturate - hypnotic	• Phenocyclidine (PCP) derivative - dissociative	• Benzodiazepine - anesthetic
Action	• Inhibition at GABA synapse • Decreased cerebral metabolism, rate and blood flow, decreased ICP; decreased SVR, decreased BP, and decreased HR	• Decreased tone Cr channels open, blocking GABA and suppressing glutamatergic • Decreased cerebral metabolism and blood flow, decreased CBF, increased CO ₂ , decreased HR, decreases capillary tension, decreased respiratory rate	• May act on NMDA, sigma, and other receptors • Increased HR, increased BP; increased SVR, increased coronary flow, increased myocardial O ₂ uptake • CNS and respiratory depression, bronch smooth muscle relaxation	• Causes increased glycogen efflux • Facilitates GABA • Produces sedation and skeletal muscle relaxant effects • Minimal cardiac depression
Indications	• Induction • Maintenance • Total intravenous anesthesia (TIVA)	• Induction • Control of convulsive status	• Major trauma, hypoxemia, severe sepsis because sympathomimetic	• Used for sedation, anxiolytic, and anaesthesia
Caution	• Allergy (eg, soy) • Patients who convert ketones to ketone bodies decreased BP (e.g. heart failure output overload)	• Allergy to barbiturates • Uncontrolled hypertension, shock, cardiac failure • Patients over 60 years, status asthmaticus, myasthenia	• Anesthetic allergy • TCA medication (antidepressants cause HTN and dysrhythmia) • History of psychosis • Patients who cannot tolerate HTN (e.g. CHF, increased ICP, septic shock)	• Myxedema respiratory depression
Dosing	• N induction: 2.5-3.0 mg/kg (less with opioids) • Unconsciousness <1 min • Loss 4-6 sec • t _{1/2} =95 min • Decreased post-operative sedation, recovery time, N/V	• N induction: 3.5 mg/kg • Unconsciousness about 2 s • Loss 5 min • Accumulation with repeat dosing - not for maintenance • t _{1/2} = 5-10 h • Post-operative sedation lasts hours	• N induction 1.5 mg/kg • Unconsciousness about 15 s, unconscious, amnesia, and unconsciousness in 45-60 s • Unconsciousness for 10-15 min, unconscious for 40 min, unconscious for 1-2 h • t _{1/2} = ~2 h	• Overdose less than 5 min if patient R • Duration of action long but variable (unpredictable)
Additional Notes	• 0-30% decreased BP due to vasodilation • Reduce burning of N velocity mixing with ketones	• Combining with rocuronium causes precipitation to form	• High incidence of emergence reactions (visual changes, auditory sensations, illusions) • Precise with glycocyclidine to decrease sedation	• Antagonist flumazenil (Benzospin®) competitive antagonist 0.2 mg IV over 15 s, repeat with 0.1 mg after (max of 2 mg), full of 60 min. • Midazolam also has anterograde amnesia effect and increased risk of thromboembolism

- * The uses of these drugs →
 - ① induction of anesthesia
 - ② maintenance of anesthesia
 - ③ anxiolytic (diazepam)

④ sedation
⑤ anti-epileptic.

- * The doctor mentioned these SEs ...

* propofol :

1. hypotension.
2. bradycardia.
3. pain on injection

* Thiopental :

- 1.hypotension.
- 2.Tachycardia
3. bronchospasm

	CV	PULMONARY	CEPHALIC	Others
Barbiturates	Depression of central vasomotor center in the brain stem; vasoconstriction and T-HR.	V _A and MV _A : normal response to Propofol and Hypoxia is blunted.	Vasodilation, ↓ CBF and ICP, CMRO ₂ , ↓ production to global ischemia.	Ketamine to induce anorexia, analgesia, and synesthesia; may precipitate acute intermittent porphyria.
Benzodiazepines	Minimal change in BP, HR, and CO. However, with combination of opioids, SVR and BP can ↓.	Nominal response to CO ₂ is minimally affected.	CMRO ₂ is mildly depressed along with CBF and ICP much less than barbiturates. Not known to be significantly neuroprotective.	
Opioids	Do not tend to be myocardial depressants. Histamine release associated with morphine and fentanyl.	Depress ventilation. Nominal response to CO ₂ is blunted. T resting pCO ₂ . Associated with chest wall rigidity.	Cause ↓ in CMRO ₂ , CBF and ICP activation of chemoreceptor trigger zone induces nausea and vomiting.	Gastrointestinal: Delayed gastric emptying and peristalsis. Head and neuropathy.
Ketamine	BP, HR, and CO are all ↑ due to CNS sympathetic stimulation.	Normal ventilation and where reflexes are maintained. Facial hemichemosis.	CMRO ₂ , CBF and ICP are all ↑, unlike other nonbarbiturate agents.	Shares similar structure as phenothiazine, therefore similar psychomotor effects including ↑ in ICP and hallucinations.
Exsufflation	No significant effects on respiratory compliance, BP, and CO.	Ventilation is maintained. Induction typically does not cause apnea.	Like barbiturates, CO ₂ HCO ₃ and CMRO ₂ are all ↑.	Inhalation of β -1-1 hydroxyethyl and alpha-1 adrenergic receptor antagonists are useful.
Propofol	Direct myocardial depression, vasodilation.	Nominal response to Propofol and Hypoxia are blunted.	CBF and ICP are ↑.	Anxiolytic and sedative properties. Effective in terminating status epilepticus.

BP: blood pressure; CO: cardiac output; CMRO₂: cerebral metabolic rate of O₂; CO₂: partial pressure of CO₂; HR: heart rate; ICP: intra-cranial pressure; MV_A: minute ventilation; pCO₂: partial pressure of carbon dioxide; V_A: total volume.

* Ketamine :

1. hypertension.
2. Tachycardia

* Benzodiazepines

respiratory depression .

* All of them cause nausea and vomiting except Propofol (anti-emetic)

* Ketamine is the most one to cause bronchodilation

Table 12. Opioids

Agent	Relative Dose to 10 mg Morphine IV	Moderate Dose	Dose*	Duration	Special Considerations
Codine	200 mg PO	15-30 mg PO	Slow (30-60 min)	<u>Moderate</u> (4-6 h)	Promotes post-operative use, not for N use
Morphaïne (Deseret®)	75 mg IV	1-3 mg/kg IV	Moderate (10 min)	<u>Moderate</u> (4-6 h)	Anxiolytic, relaxant, less pupillary constriction than morphine, metabolic build up may cause seizures
Morphine	10 mg IV 20 mg PO	0.2-0.2 mg/kg IV 0.4-0.6 mg/kg PO	Moderate (5-10 min)	<u>Moderate</u> (4-6 h)	Histamine release leading to decrease in BP
Oxycodone Controlled Release	15 mg PO	15-20 mg PO (no N)	Slow (30-45 min)	<u>Long</u> (8-12 h)	Do not split, crush, or chew tablet
Oxycodone Regular Tablet	15 mg PO (no N)	5-15 mg PO	Moderate (15 min)	<u>Moderate</u> (4-6 h)	Percocet® = oxycodone 5 mg + acetaminophen 325 mg
Hydrocodone (Dihydro) [†]	2 mg N 10 mg PO	40-60 µg/kg N 2-4 mg PO	Moderate (15 min)	<u>Moderate</u> (4-6 h)	
Fentanyl	100 µg N	0.3 µg/kg N	Rapid (<5 min)	<u>Short</u> (0.5-1 h)	Transient muscle rigidity at very high doses
Sufentanil	100 µg N	0.5-1.5 µg/kg N	Rapid (1-3 min)	<u>Ultra-short</u> (<10 min)	Only use during induction and maintenance of anesthesia

* In general, pain control rate is 2.2× more potent than analgesic.

★ opioid's side effects --

- ① respiratory depression
- ② addiction, euphoria
- ③ ileus (constipation)
- ④ miosis

Table 14. Volatile Inhalational Agents

	Sevoflurane	Desflurane	Isoflurane	Ethrane	Halothane	Nitrous oxide (N ₂ O)*
MAC (% gas in O ₂)	<u>1.0</u>	<u>5.0</u>	<u>1.2</u>	<u>1.7</u>	<u>0.8</u>	<u>104</u>
CNS	Increased ICP	Increased ICP	Decreased cerebral metabolic rate Increased ICP	ECG sawtooth-like activity Increased ICP	Increased ICP and cerebral blood flow	—
Resp	Respiratory depression (severely decreased TV, increased RR), decreased response to respiratory CO ₂ reflexes, bronchospasm	—	—	—	—	—
CVS	Less decrease of contractility, stable HR	Tachycardia with rapid increase in concentration	Decreased BP and CO, increased HR, theoretical chance of coronary steal**	Stable HR, decreased contractility	Decreased BP, CO, HR, and conduction Bradycardia, myocardial ischaemia, hypotension-induced arrhythmias	Can cause decreased HR in patients with those with existing heart disease
MSK	Muscle relaxation, prevention of other muscle relaxants, uterine relaxation	—	—	—	—	—

Properties and Adverse Effects of N₂O:

Due to its high MAC, nitrous oxide is combined with other anesthetic gases in certain surgical anesthesia. A MAC of 10% is possible in a preoxygenated situation only.

Second Gas Effect: use of desflurane instead of isoflurene allows a lower MAC.

Expansion of blood vessels in clinical scenarios such as atherosclerosis, the middle ear, bowel lumen and ETT cuff will rapidly change MAC if administered.

Offices have noted during anesthesia, the sound of N₂O from body mass can start to blow the eardrum (O₂ creating a hyperbaric situation if the oxygen is 50%) is low.

** Coronary steal: uterine cramps and vessel dilation which may compromise blood flow to areas of the heart with fixed pathology (e.g. stenosis, atherosclerosis).

Table 14. Distinguishing Muscle Relaxants (Mayo Clinic) vs. Succinylcholine (SCCh)	
Mechanism of Action	SCCh vs. Non-depolarizing muscle relaxants
Anesthetizing Drugs	Non-depolarizing muscle relaxants bind to ACh receptors causing prolonged depolarization, which is reversed by neostigmine, atropine, followed by a tertiary phosphodiesterase inhibitor to block ACh receptors by SCCh.
Onset	SCCh < 1 minute vs. 10-20 minutes of all muscle relaxants.
Dose/Effect	SCCh is rapidly offset by plasma cholinesterase (succinylcholinesterase), usually an hour and half on the field.
Side Effects	1. Rapid onset 2. Reversal of neuromuscular blockade, cholinergic autonomic responses (in addition to neuromuscular blockade) - Myo-ocular block (dysphoria, mydriasis, tachycardia, sinus tachycardia, increased sweating, salivation, ptosis especially in children) 3. Hypertension - Description of motor nerve activity increase post-treatment of succinylcholine (succinylcholine-induced hypertension) - Description of an increased incidence of malignant hyperthermia by SCCh may lead to muscle stiffness, tachycardia, hypertension, and tachypnea. - Myopathy and rhabdomyolysis - Bradycardia, tachycardia, arrhythmias, other drugs - Tachycardia, post-operative respiratory depression (e.g., muscular spasm) - Decreased arterial blood pressure - Decreased cerebral blood flow - Upper airway obstruction 4. Eye irritation/IRIS 5. Increased risk of malignant hyperthermia post-treatment of susceptible patients or those who lack the capacity to dissipate heat 6. Pseudocholinesterase, pseudocholinesterase inhibitor, may be increased if small doses of non-depolarizing agents cause SCCh antagonism
Contraindications	Known hypersensitivity to SCCh, known history of malignant hyperthermia, myopathy, arrhythmias, etc., hypothyroidism, pseudocholinesterase deficiency, drug and food hypersensitivity.
Relative	Known history of plasma cholinesterase deficiency, cholinesterase pseudocholinesterase deficiency, known pseudocholinesterase, open eye injury.

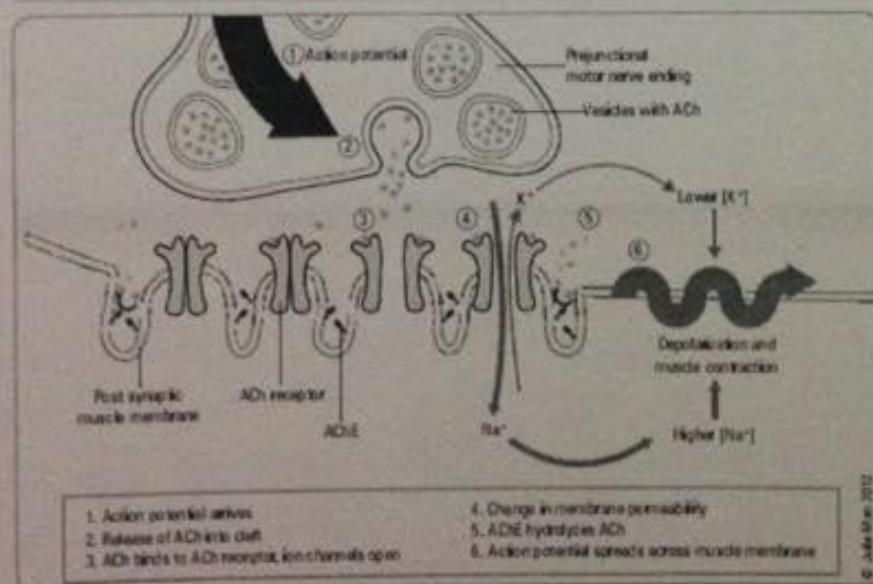
Table 15. Main Distinguishing Muscle Relaxants (Dongen et al.)

Mechanism of Action	Comparative半數抑制劑 (IC ₅₀) of nondepolarizing ACh receptors producing depolarization			
	筒箭毒碱	肉桂箭毒碱	胆碱酯酶抑制剂	琥珀酰胆碱
Anesthetizing Drugs (single)	0.1	0.8-1.0	0.1	0.2
Onset (min)	0.3	1.5	2.5	3.5
Duration (min)	15-25	30-45	45-60	90-120
Mechanism	Plasma cholinesterase	Ligand receptor	Cholinesterase	Muscarinic cholinesterase
Antagonism	A agent antagonists, direct muscarinic receptor blockers, selective benzodiazepines, and direct acetylcholinesterase inhibitors to SCCh patients, indirect benzodiazepines and direct acetylcholinesterase inhibitors to non-SCCh patients.	Direct antagonists, M receptor antagonists, and M receptor blockers.	Cholinesterase inhibitors, M receptor antagonists, and M receptor blockers.	Direct antagonists, M receptor antagonists, and M receptor blockers.
Side Effects	None	None	None	None
Metabolism/Excretion	None	None	None	None
Offices	None	None	None	None
Contraindications	Increased serum level of pseudocholinesterase, liver failure.	Cholinesterase inhibitors, M receptor antagonists, and M receptor blockers.	Pseudocholinesterase, plasma cholinesterase, and M receptor blockers.	None

* mention diseases affect this region :- (neuromuscular junction)

- ① myasthenia gravis
- ② Tetany

- ③ Lambert - eaton .



* note that gullian barre disease does not affect the junction, it affects the nerve it self

- * side effects of these drugs: (increase the parasympathetic effect)
1. bradycardia
 2. salivation
 3. lacrimation
 4. urination
 5. diarrhea
 6. miosis

Table 17 Reversal Agents for Non-Depolarizing Relaxants

Cholinesterase Inhibitor	Neostigmine	Pyridostigmine	Edrophonium
Onset and Duration	Intermediate	Longest	Shortest
Mechanism of Action	Blocks enzymatic degradation of ACh, increases ACh at nicotinic and muscarinic receptors, displaces non-depolarizing muscle relaxants.	Muscarinic effects of reversing agents include unwanted bradycardia, salivation, and increased bowel peristalsis.	
Dose	0.04-0.08 mg/kg	0.1-0.4 mg/kg	0.5-1 mg/kg
Recommended Anticholinergic	Glycopyrrolate	Glycopyrrolate	Atropine
Dose of Anticholinergic per mg	0.2 mg	0.05 mg	0.014 mg

* Atropine and glycopyrrolate are anticholinergic agents administered during the administration of reversal agents to minimize muscarinic effects.

Table 18. Local Anesthetic Agents

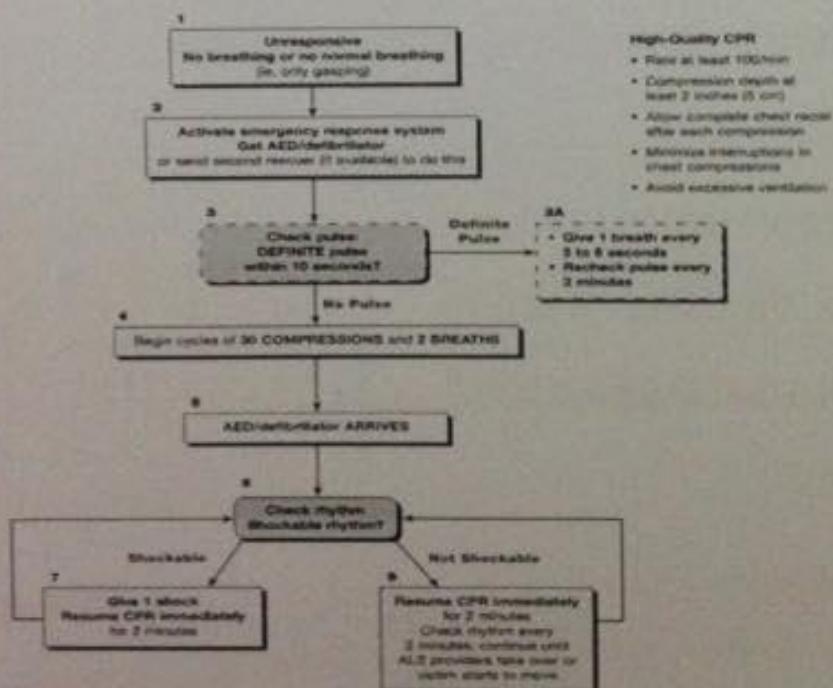
	Maximum Dose	Maximum Dose with Epinephrine	Potency	Duration
chloroprocaine	11 mg/kg	14 mg/kg	Low	15-30 min
lidocaine	5 mg/kg	7 mg/kg	Medium	1-2 h
bupivacaine	2.5 mg/kg	3 mg/kg	High	3-8 h
ropivacaine	2.5 mg/kg	3 mg/kg	High	2-8 h

* do not give IV bupivacaine → highly cardiotoxic

* Glycopyrrolate is recommended with neostigmine.
pyridostigmine.

* Atropine is recommended with Edrophonium.

Adult BLS Healthcare Providers



Note: The boxes bordered with dashed lines are performed by healthcare providers and not by lay rescuers.

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Advanced Cardiac Life Support (ACLS) Guidelines

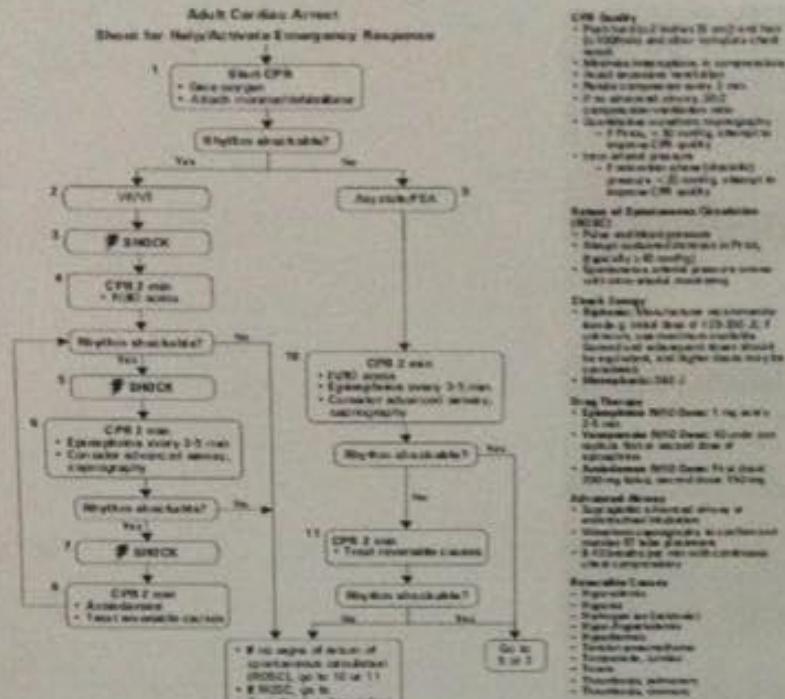
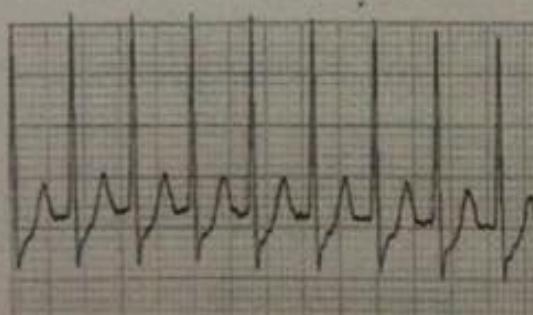


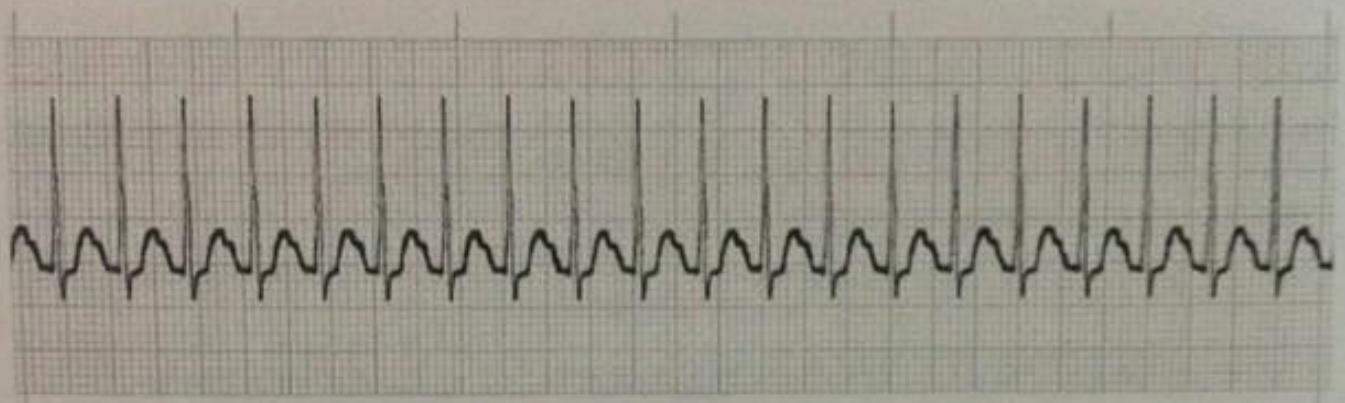
Figure 18. Adult *Acyrthosiphon pisum* viviparae.

Figure 16. Adult cardiac arrest algorithm.
Reprinted with permission: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 8: Adult Advanced Cardiac Life Support. Circulation. 2010;122:S767-827. © 2010 American Heart Association.

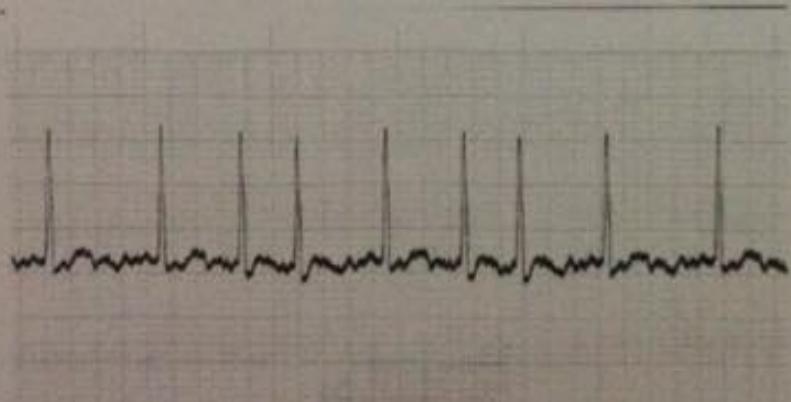
3- a patient presented to you with this ECG, the patient was fully conscious, not in pain, normal vital signs.



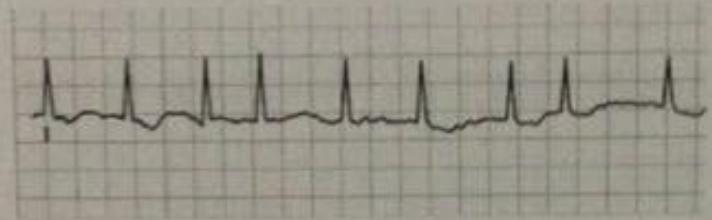
- This represents SVT
 - patient is stable , the management will be ^{vagal maneuvers}
^{adenosine}.
 - if he is unstable , the management will be (DC shock) .



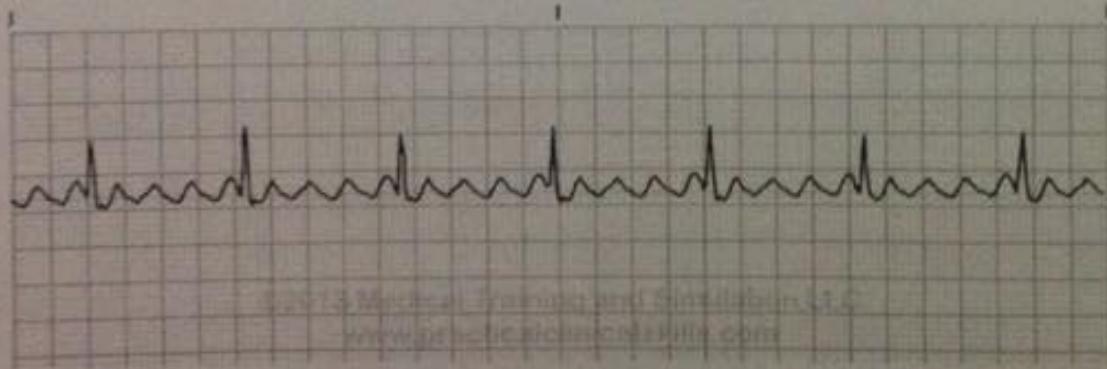
(SVT)



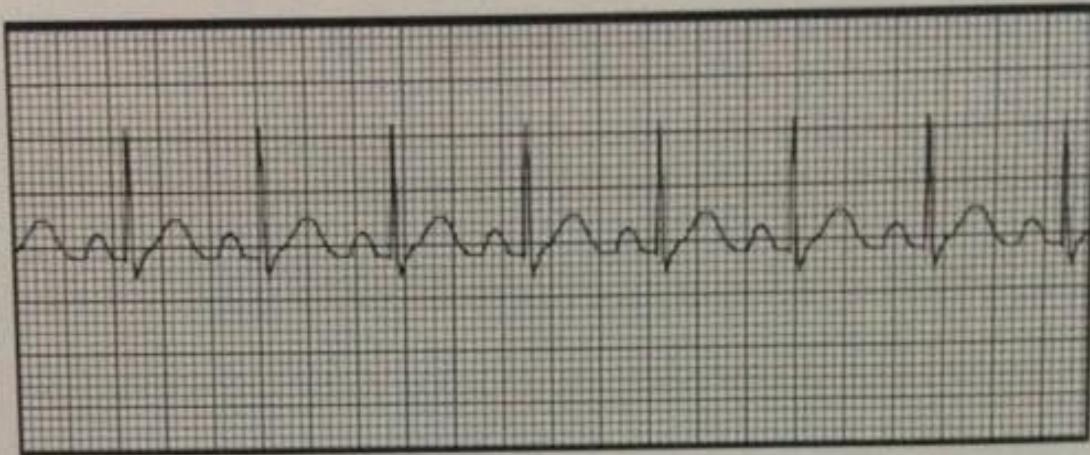
(AFib)



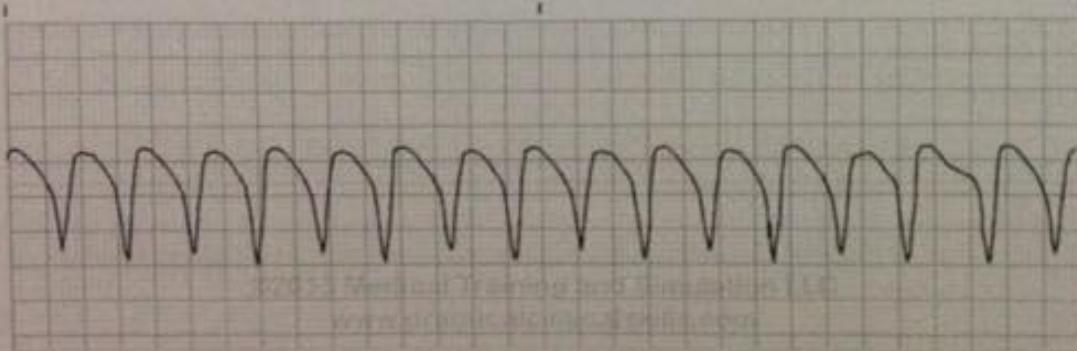
(AFib)



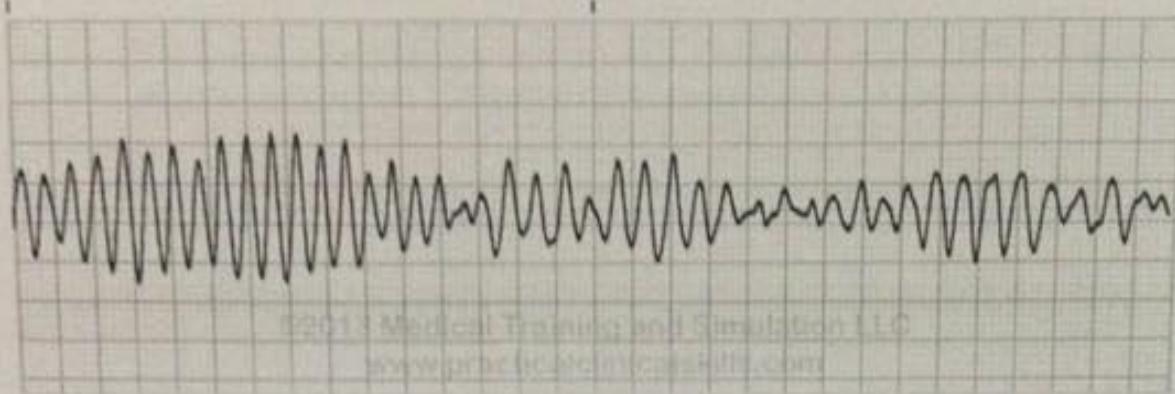
(Atrial Flutter)



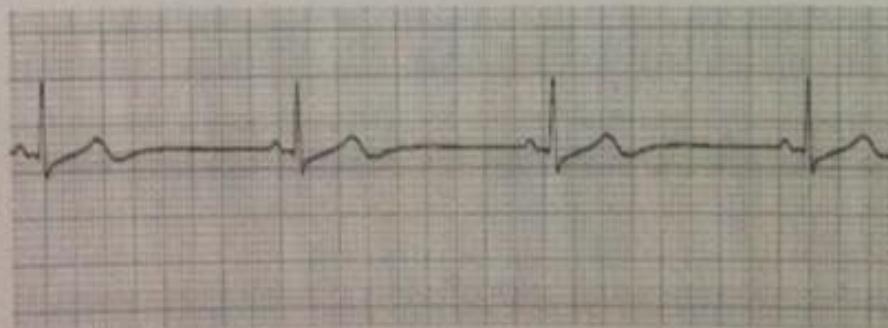
sinus tachycardia



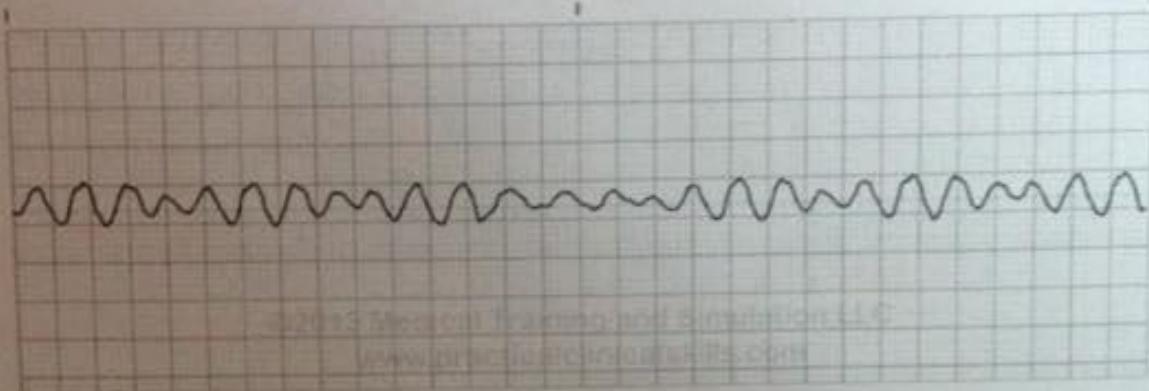
ventricular tachycardia
(monomorphic)



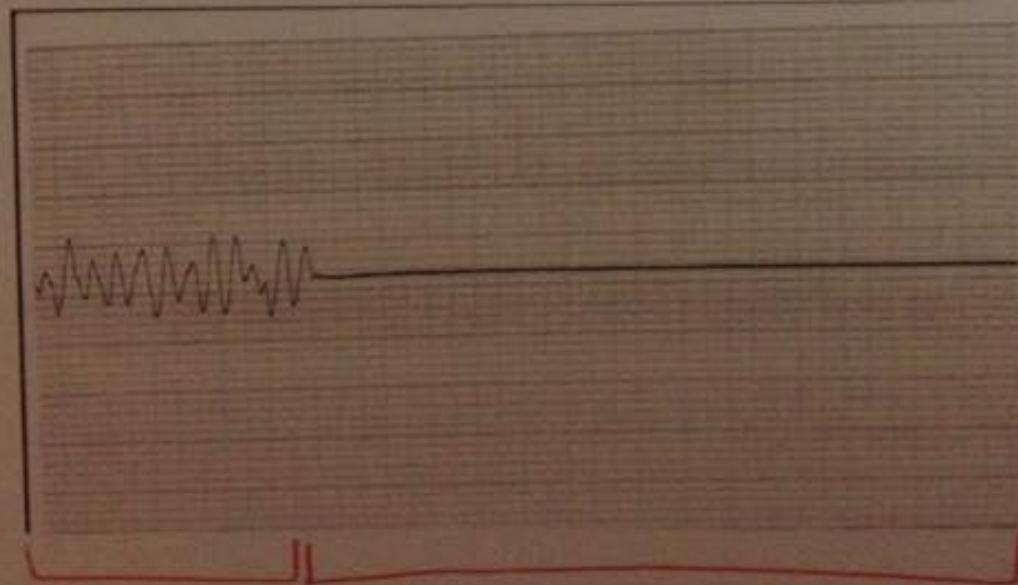
polymorphic ventricular tachycardia
(Torsade de pointes)



- sinus brady cardia .
- Drug of choice : Atropine



- Ventricular fibrillation . (coarse)
- management: synchronized Dc shock .



VFib asystole
(shockable) → (nonshockable)

- * where should you put the paddles of the defibrillator?
 - one is apical → left anterior axillary line between the 4th and 5th rib.
 - the other one is sternal → right lateral border of the sternum, 2nd intercostal space.

Q10) mention two types of this device and two indications for its usage

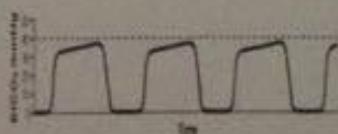


- * Types : ① monophasic
② biphasic →

الخطير مجاور هنا
الحادي ، ليس بيتوح
حلق لطعن
- * indications : ① VFib
② pulsless VTac .

Q12) give two measurements that can be taken from this graph:

- ① end tidal CO₂
- ② Resp. rate .





* pulse oxymeter

98 represent O₂ sat.

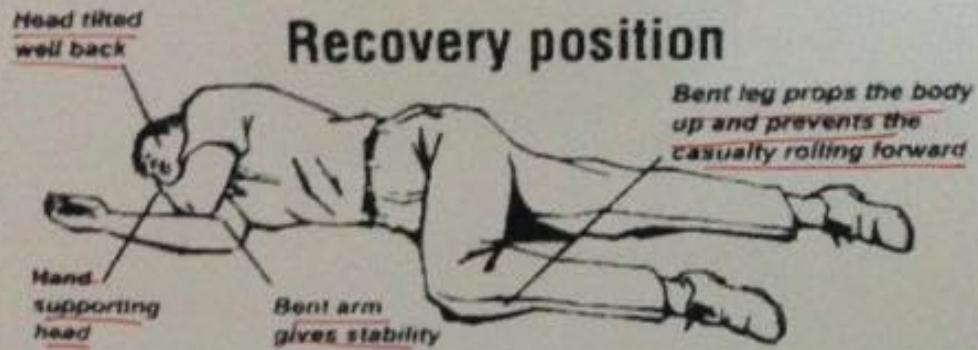
64 represent the pulse .

Q9) identify four events in this picture?

head tilt, chin lift, look, listen, feel

Breathing monitoring.





* The importance of this position e-

- ① Keep the airway opened.
- ② prevent the gastric content aspiration.



* She's checking for responsiveness of patient, which is part from the BLS



* Chest compression , part of both BLS and ALS

11-A) what is

B) mention two ways to counteract it?

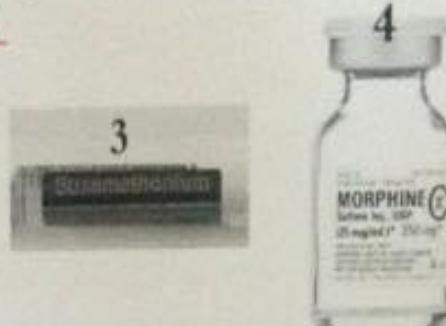
- ① neostigmine
- ② pyridostigmine



- 12- match:
- -Long acting analgesic. 4



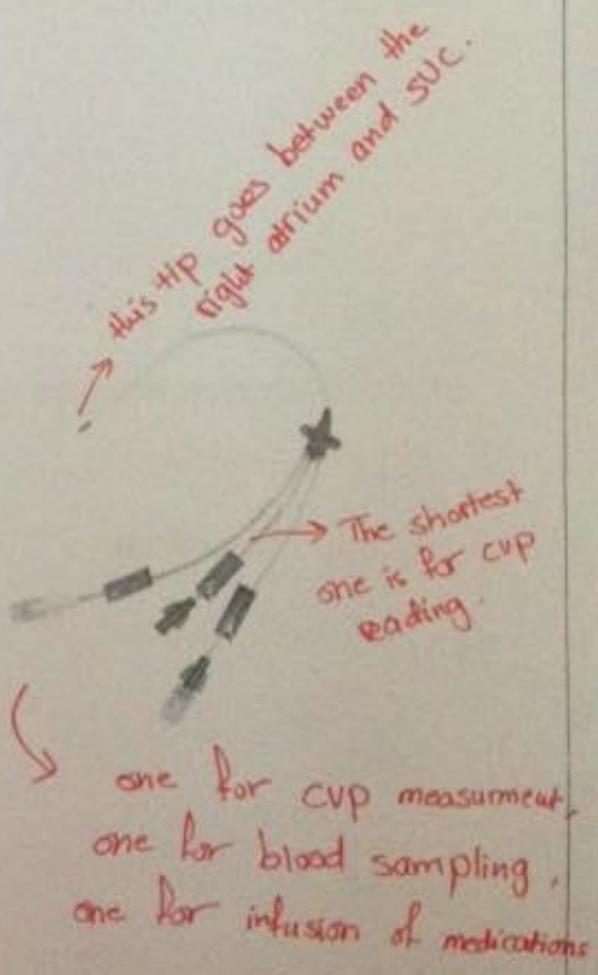
- -most potent analgesic. 2
- -depolarizing muscle relaxant. 3
- -short acting muscle relaxant 3



13- A) What is this? central line
used for CVP measurement

B) Mention two complications of its use

- ① pneumothorax
- ② Bleeding and hematoma
- ③ arterial puncture
- ④ infection
- ⑤ arrhythmias



This is IV set.

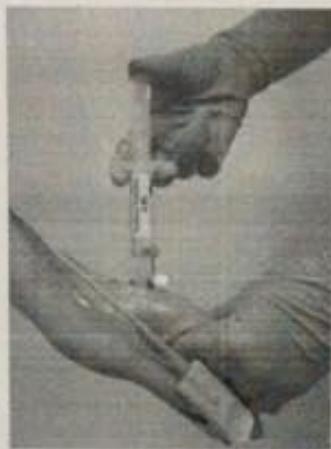


14- In ALS, if the rhythm is shockable, explain the doses and administration of the drugs.

Adrenaline 1 mg
amiodarone 300 mg → IV or interosseous

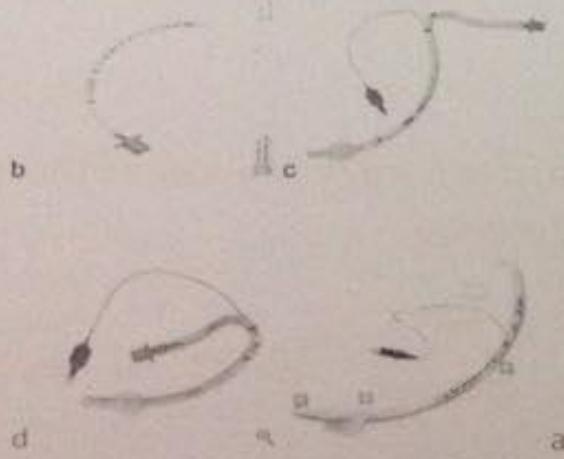
* he is doing flushing.

why? to insure no bulging, no resistance.



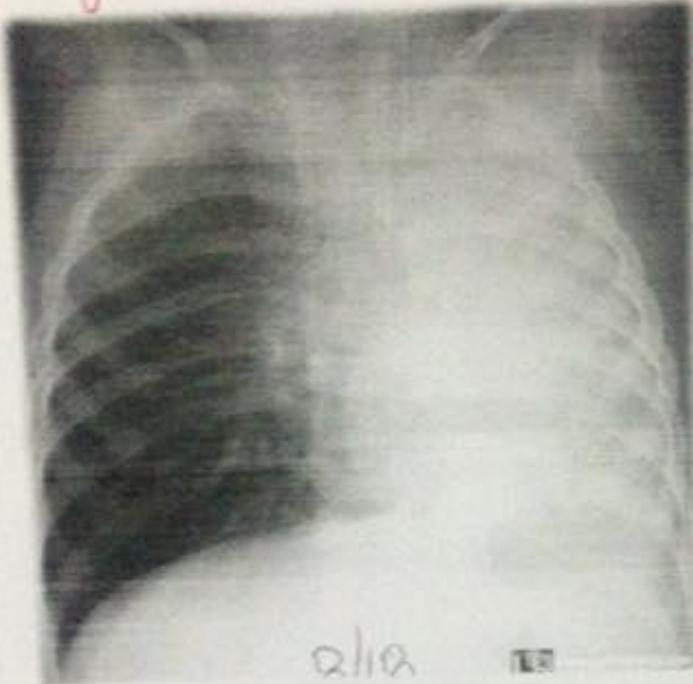
3) pictures about endotracheal tubes and the is match between images and it indicates

- a) abdominal hernia surgery cuffed endotracheal tube **a**
- b) child abdominal surgery non cuffed endotracheal tube **b**
- c) mandibular surgery preformed endotracheal tube **c**
- d) maxillary sinus surgery preformed endotracheal tube **d**.



Lung collapse (left), causes

right endobronchial intubation
blug in the left
endobronchial carcinoma
in the left.



Which drug is more potent? **propofol**

- Which one is contraindicated in porphyria? **Thiopental**

* The most stable one is Etomidate.

* The most one to cause hyperdynamic circulation is Ketamine

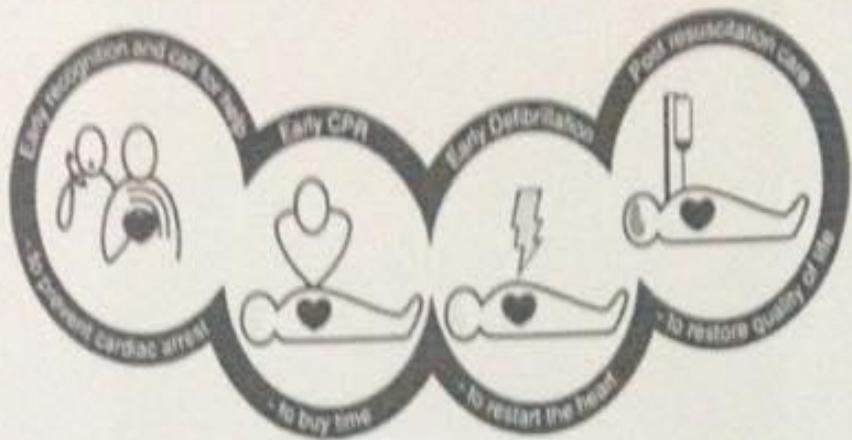
* The most one to cause instability is propofol.

Thiopental ←



propofol. ←





Collapsed lung

Pneumothorax

Displaced diaphragm

→ The heart is shifted

Mediastinal shift