Anaesthesia

2018

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Airway Management

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Concept

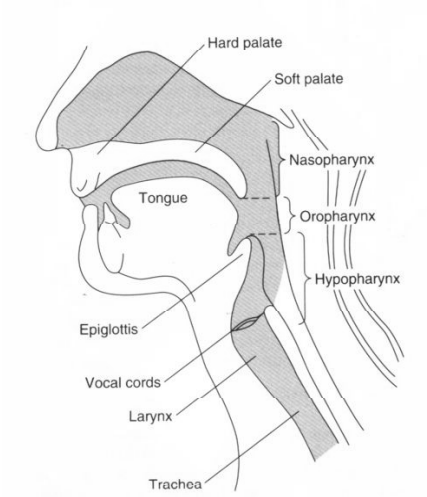
- In the ABCs of Life, A stands for Airway Patency
- Apart from Cardiopulmonary by-pass (with extracorporeal oxygenatation), oxygen cannot reach the blood when the flow of Oxygen to the Gas exchange membrane is obstructed, and even CPB is initiated under controlled airway conditions.

Oxygen Reserve in the Body

Is provided by:

- Expired Air Oxygen tension of ~ 120 mmHg **VS.** Alveolar Oxygen tension of around 100mmHg.
- Oxygen flux of ~1000 ml/min **VS.** Oxygen Consumption of ~ 250 ml/min
- Intracellular Oxygen tension 5-40 mmHg ***VS.*** Mitochondrial critical oxygen tension 1-3 mmHg

Airway anatomy



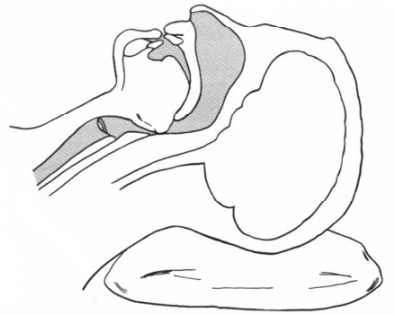
General Diagnostic Rules

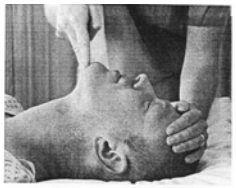
- Obstruction can occur due to many causes
- Always think of and exclude common things first (tumors come at the end of the list)
- In unconscious patients, the tongue falling back against the posterior pharyngeal wall is the commonest airway obstructing cause.

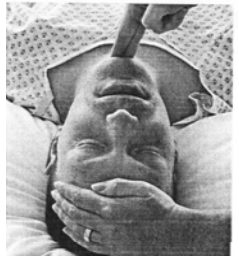
General management Rules

- Always start by shouting at and shaking the patient (remember the Pickwickian syndrome!?)
- Look into the patient's mouth and if applicable Swab the mouth and pharynx with your finger to remove any foreign bodies (e.g. a loose denture, vomitus, etc.)
- Patient still not breathing proceed ➔

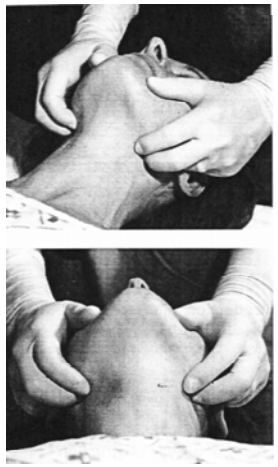
Head tilt-Chin Lift Maneuver







Jaw thrust Maneuver



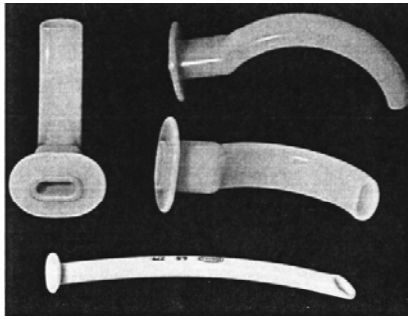
Applying face mask with jaw thrust

- USING SINGLE HAND TECHNIQUE

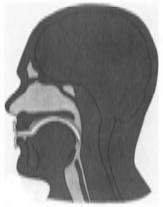
- USING TWO HAND TECHNIQUE

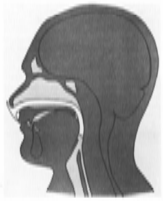
Maintaining patency of airway using airway assistant devices

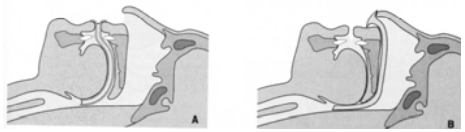
- Oral airways
- Nasal airways

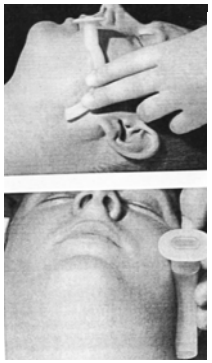


Maintaining patency of airway using airway assistant devices

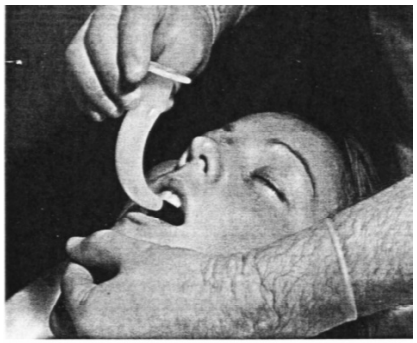


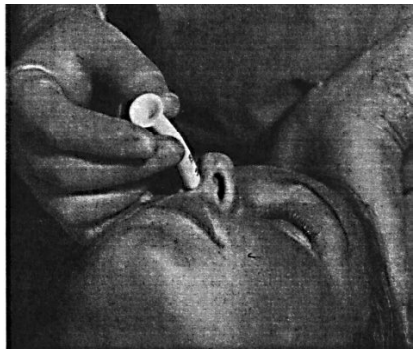






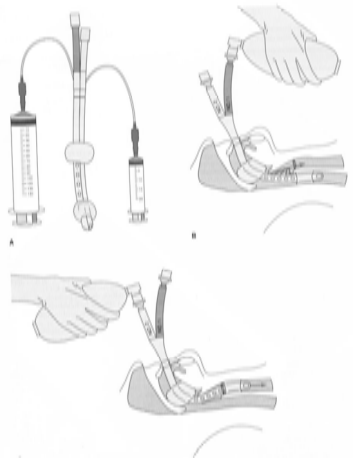
Insertion technique of the Oral and Nasal airways



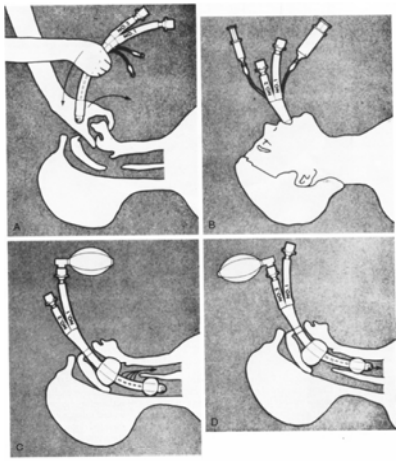


Maintaining patency of airway using the Combitube

- An Esophageal-tracheal combined tube with two lumens and two cuffs
- Blindly inserted
- Ventilation lumen depends on wheatear the distal end goes into the esophagus or the trachea



Combitube
(Insertion Technique)

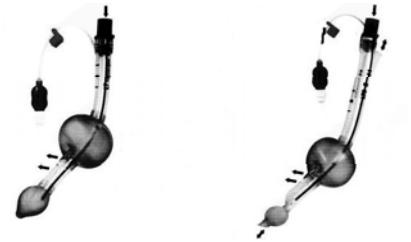


Disadvantages and contra-indications of Combitube

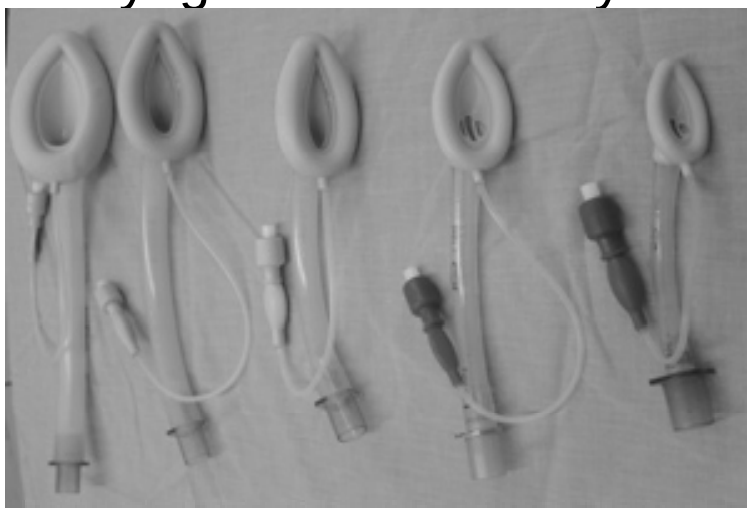
- ***Disadvantages:***
  - Available only in one size, proper for patients >15 years
  - Expensive
  - Can't be used to guide fiber-optic intubation
- ***Contra-indications:***
  - Not practical for pediatric patients
  - Patients with intact gag reflexes
  - Patients with esophageal pathology
  - Patients with caustic substance ingestion

Laryngeal tube

- A single lumen tube with both an esophageal and pharyngeal cuff
- A single pilot balloon inflates both cuffs
- Available in a variety of sizes
- Successful insertion by non-anesthetists
- New versions have an open esophageal end allowing for drainage and suctioning

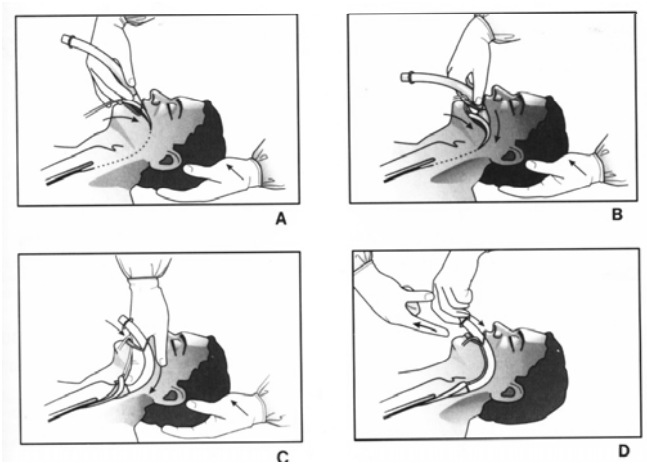


Laryngeal Mask Airway



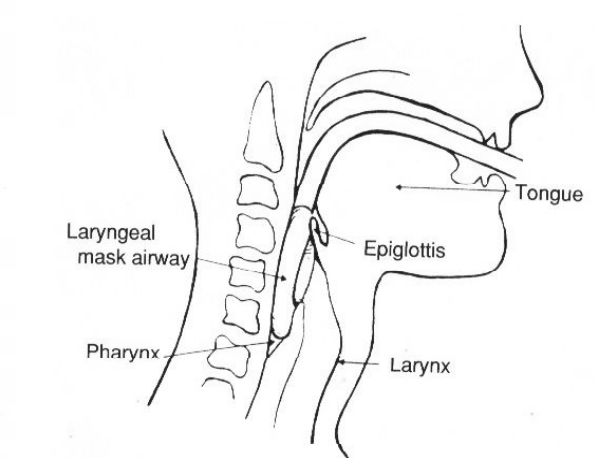
Laryngeal Mask Airway

(Technique of insertion)



Laryngeal Mask Airway

(In Position)



Choice of LMA size

Mask Size	Patient Size	Weight (kg)	Cuff Volume (mL)
1	Infant	<6.5	2–4
2	Child	6.5–20	Up to 10
2½	Child	20–30	Up to 15
3	Small adult	>30	Up to 20
4–5	Normal and large adult		Up to 30

Advantages of LMA compared with Endo-tracheal tube

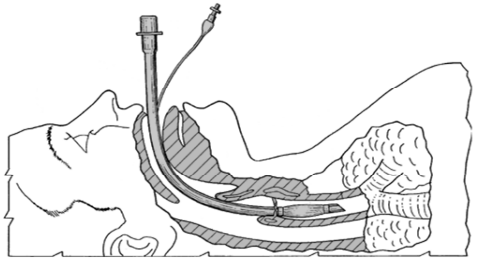
- Less invasive
- Less anesthetic depth required
- Useful in difficult airway management
- Less tooth and laryngeal trauma
- Less laryngospasm and Bronchospasm
- Does not require muscle relaxation
- Does not require neck mobility
- Less effect on B/P,H/R,ICP,IOP
- Less risk of Esophageal, or endo-broncheal intubation

Disadvantages of LMA compared with Endo-tracheal tube

- Increased Risk of GI content aspiration
- Not practical in prone or jackknife positions
- Unsafe in Morbidly obese
- Limits maximum PPV
- Less secure airway
- Greater Risk of gas leak and pollution
- Can Cause gastric distention

End of Part One

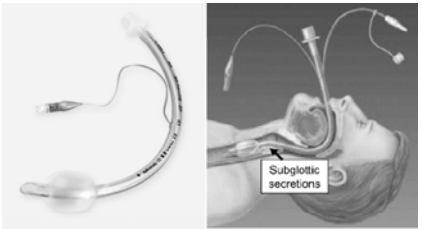
Endotracheal Intubation

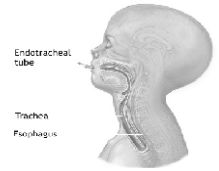


Types of Endotracheal Tubes

1- Sealing of trachea

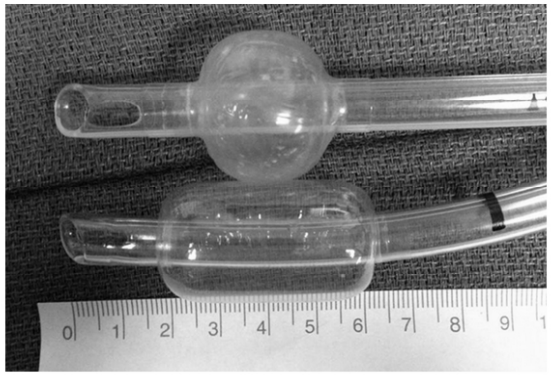
- Cuffed vs. Non-cuffed





2- Type of Cuff:

- High pressure-low volume Vs. Low pressure-high volume.

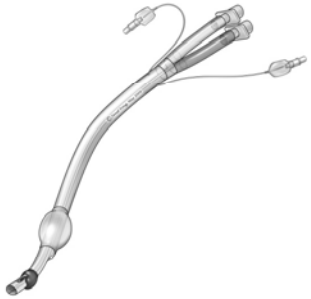


3- Shape of tube

- Regular, pre-formed or armored (Reinforced or non-kinkable)

3- Lumen

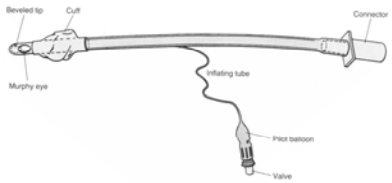
Single lumen vs. Double lumen

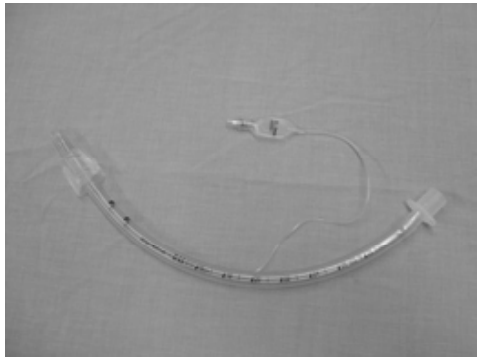


4- Usage times

- Disposable vs. Disposable

Endotracheal Tube
(Design)



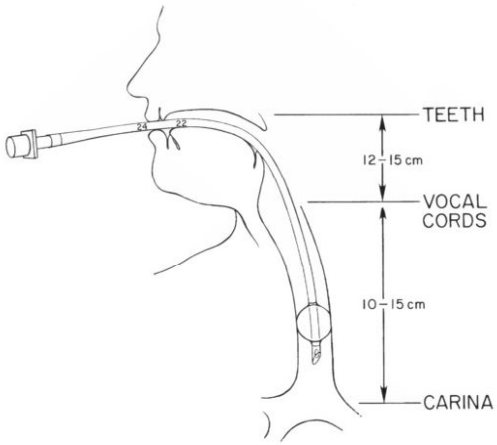


Endotracheal Tube
(Choice)

Oral endotracheal tube size guidelines.

Age	Internal Diameter (mm)	Cut Length (cm)
Full-term infant	3.5	12
Child	$4+\frac{\text{Age}}{4}$	$14+\frac{\text{Age}}{2}$
Adult		
Female	7.0–7.5	24
Male	7.5–9.0	24

Endotracheal Tube (in Position)

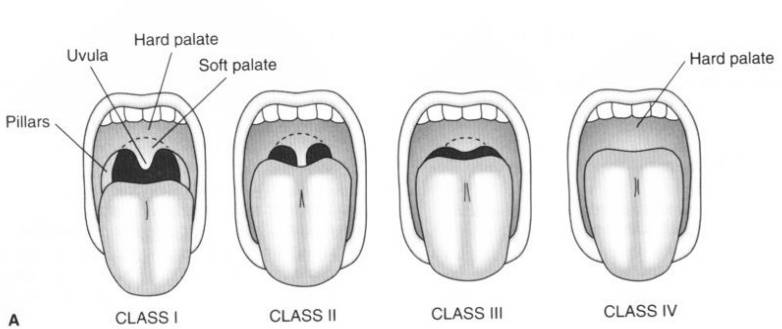


Endotracheal Intubation

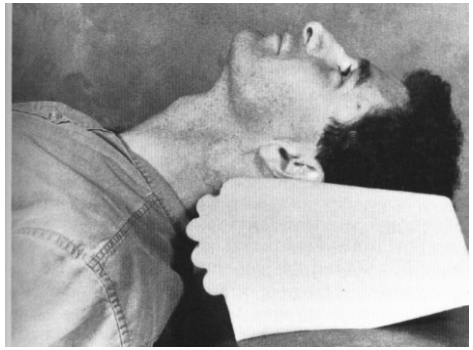
- Airway assessment:

Feature	***Visualization of Larynx during laryngoscopy***	
	Likely Easy	***Likely Difficult***
History	free	Previous difficulty/snoring/ Neck pathology/radiation
Facial Features:	symmetry	Asymmetry/Jaw recession
Pharyngeal view	Non-crowded	Crowded
Dental condition	Good	Protruding teeth/ mobile teeth
Head extension:	> 35 °	< 35 °
Neck length	Normal	Short
Mouth opening: > 2 fingers width	> 2 finger breadths (3 cm)	< 2 finger breadths
Thyro-mental distance: < 6 cm >	> 6 cm	< 6 cm
Jaw protrusion ability.	able	Not able

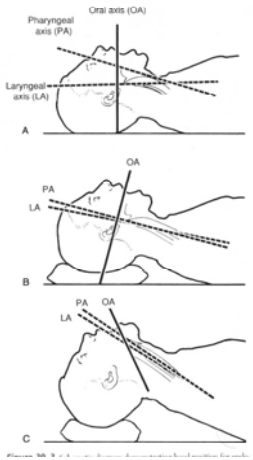
Mallampati classification



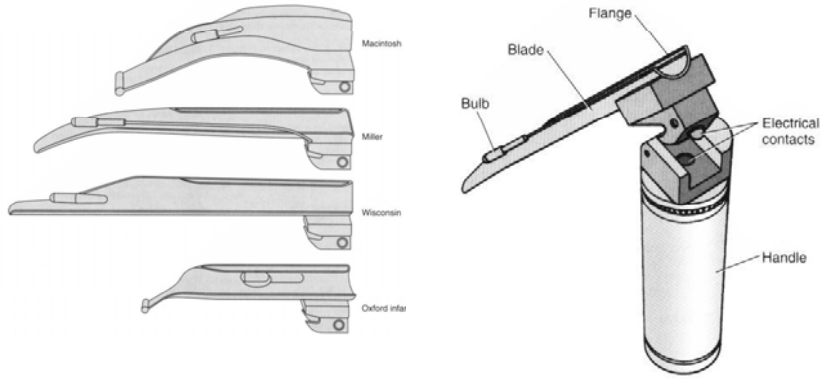
Patient Head Positioning Sniffing Position



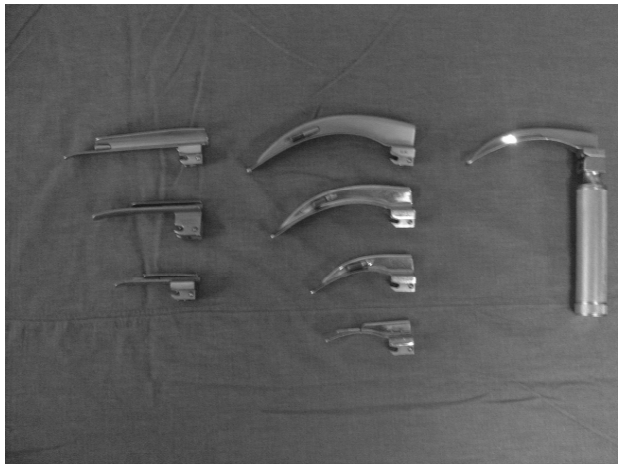
Sniffing Position-Alignment of Airway Axes



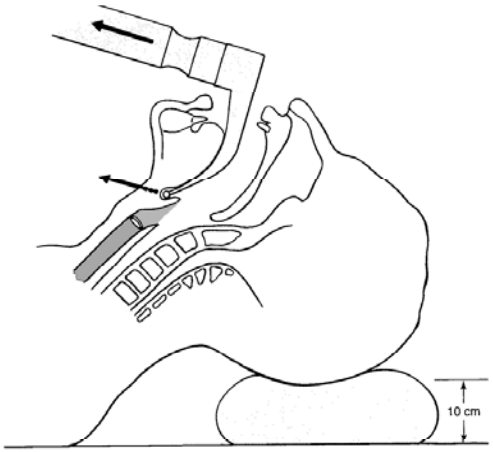
Direct Laryngoscopy
"The Laryngoscope"



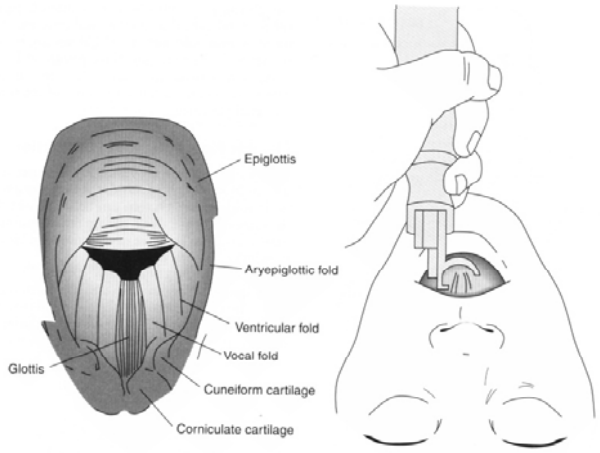
"The Laryngoscope"



Laryngoscopy



Laryngoscopy



Principles of Direct Laryngoscopy

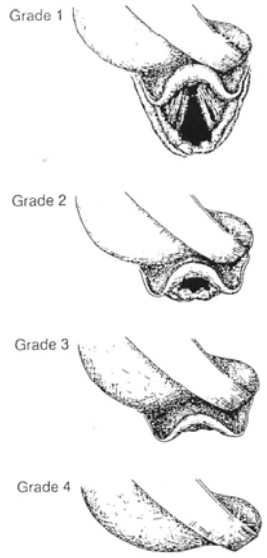
- Ideal Patient Position
- Ideal table Height: Head of patient at the Level of the operator's Xiphi-sternum.
- Proper size of Laryngoscope Blade
- Check adequacy of laryngoscope light
- Proper Endotracheal tube size

- Hold the Laryngoscope handle firmly with your left hand
- Introduce the laryngoscope blade at the right side of the tongue, with the right tongue border lying on the laryngoscope's blade flange.
- Slide the blade gently inward along the tongue, with gentle elevation of the tongue in the upward and forward axis direction until you see the epiglottis
- Advance the tip of the laryngoscope blade anterior to the Epiglottis ((i.e. in the vellucula against the Hypoid bone)
- Moving the tip of the laryngoscope blade anteriorly *(upward for you since the patient is supine),* will move the epiglottis away from the laryngeal inlet

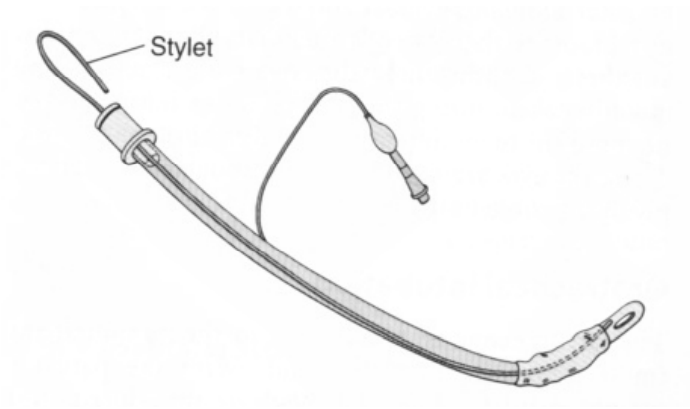
Principles of Intubation

- Hold the curved endotracheal tube with your right hand with its distal end directed forward.
- Introduce the tube through the Vocal cords into the trachea for an adequate distance *(until the horizontal black line mark on the tube is at the vocal cord level)*
- Avoid undue force when introducing the tube *(try slight rotation of the tube of it did not bypass the laryngeal inlet, otherwise, check the tube size)*
- Gently withdraw the laryngoscope blade out with your right hand firmly holding the tube in place
- Inflate the tube Cuff with proper volume(adequate to provide the seal, and not exceeding 30 cmH_2O)
- Ventilate through the tube and check that both lungs are equally ventilated by auscultating both lungs
- The tube distal end should ideally be 1 to 2 cm above the Carina, otherwise right bronchial intubation is likely with only the right lung being ventilated
- Adapt the depth of the tube up or down according to you check and record the required depth
- Firmly secure the tube by plaster taping it to the face or by the use proper tying tape

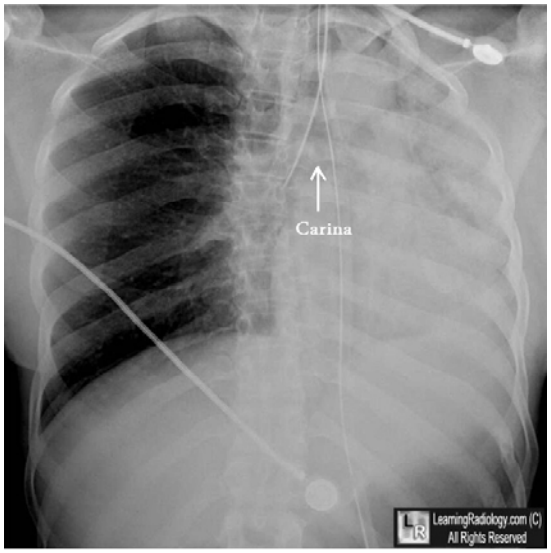
Laryngeal View Classification



Endotracheal tube with Stylet



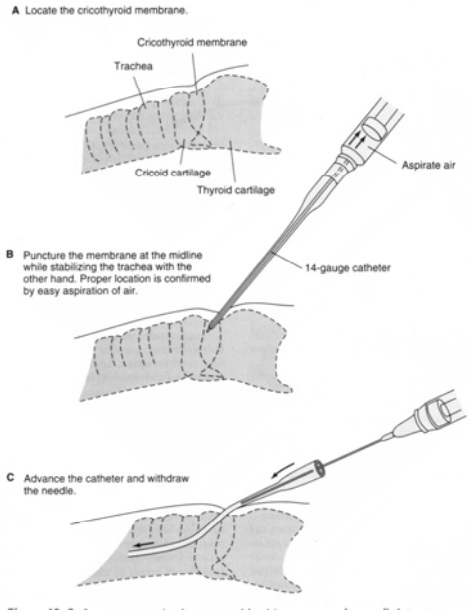
Right endo-bronchial Intubation



Cricothyrotomy

- In Can’t intubate-can’t ventilate Scenario
- A temporary life saving measure awaiting Fiber-optic Intubation / Tracheostomy
- Transtracheal Jet Ventilation adopted

Cricothyrotomy- Procedure



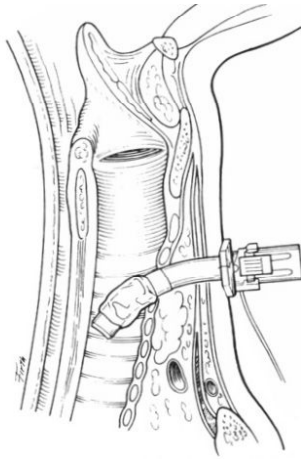
Cricothyrotomy

Tracheostomy

- Classic Tracheostomy is done by specially trained people
- Percutaneous Tracheostomy is becoming popular and similar in principle to Cricothyrotomy except for the site (2nd and 3rd tracheal rings) and Tracheostomy Tube

Tracheostomy Procedure

Tracheostomy tube in Position



Thank You

Insertion of Peripheral Intravenous Canula

Prepared by Dr Khaled AL-Zaben
Associate prof., Dept. of Anesthesia, Faculty of Medicine – Jordan University

1- Preparation

Prepare the Following:

- **Pressure cuff / tourniquet**
- **Disinfectant and swab (alcohol swab)**
- **Intra-venous Canula (2 -3 sizes)**
- **Fixation plaster**
- **A normal saline/other crystalloid filled syringe**
- **Gloves (if required)**

2- Explain the procedure to the patient

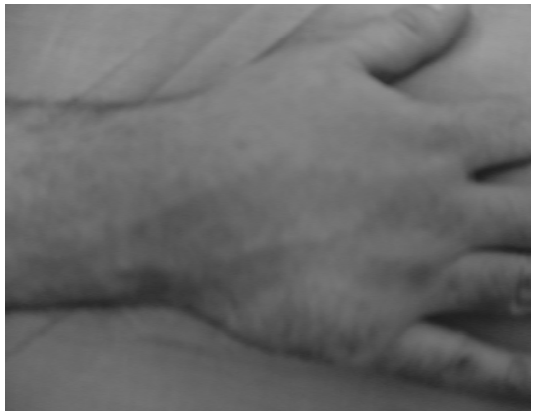
- Be empathic and reassuring as much as you can
- Explain reason and importance of what you are intending to do

3- Position:

- **the patient is lying down or sitting, with his/her hand outstretched and supported from below.**
- **You should sit next to the patient on the insertion side in a comfortable position**

4- Choose the target vein

- **It should be straight for enough distance to accommodate the Canula's length**
- **Its size should be large enough for your canula's gauge and match the intended aim of the I.V access (e.g. Blood Transfusion needs larger bore canulas)**
- **If applicable, choose sites where the veins and arteries are far apart and/or veins are less mobile (e.g. dorsum of hand)**

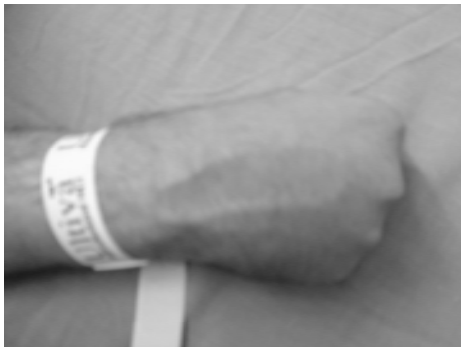


5- Compress (fill) the vein:

** **by applying the tourniquet proximal to the puncture site**

- **Broad thin tourniquets are best used**
- **The pressure applied by the tourniquet should not be very high (only compressing the veins but not the arteries)**
- **Ask the patient to make a fist (to pump blood back into the vein) and wait**
- **Tap with your fingers over the vein to make further dilatation.**

***** **<u>This step may precede step 4 if you don't see veins</u>**

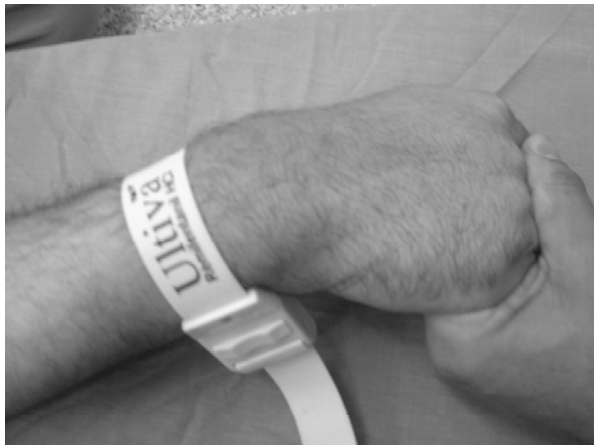


6- Disinfect

Disinfect the puncture site as quickly as possible using an alcohol swab and allow drying for 15 seconds

7- Tighten the vein

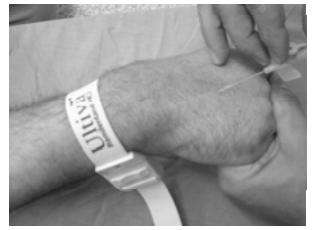
- **by tightening the skin above it using your other hand**
- **This should continue until the canula is fully inserted (remember that you can't easily penetrate a mobile object)**

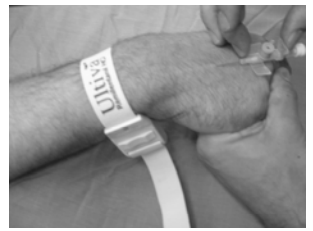


***** You can practice the above steps on yourself, colleagues or at home, to gain practice*****

7- Puncture

- **Puncture the vein at an angle 30-40 towards the skin and guide the needle following the course of the vein until blood flash-back is observed in the blood collecting chamber at the proximal end of the needle**

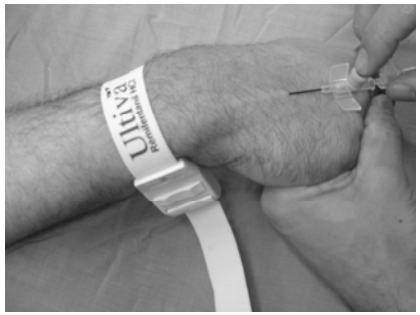




8- Pull back the needle

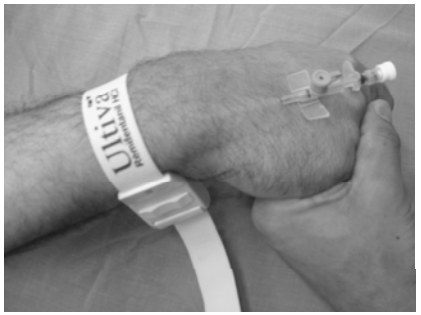
- **pull back the needle approximately 1 cm until blood flows through the plastic catheter to confirm that the catheter is in the vessel.**
- **Train yourself to do this with three fingers of your operating (dominant hand)**

(the index finger holding the canula still, and the thumb and Middle finger pulling back the needle)



9- Advance the catheter

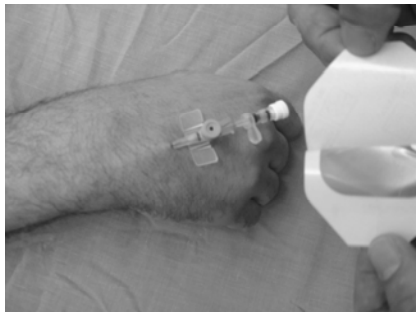
- **With leaving the needle part untouched, advance the plastic catheter –full length- into the vein. This should be accomplished with gentle force, so you can recognize any resistance.**
- **Don't Advance the catheter forcefully and if any resistance is recognized, re-check integrity of insertion.**



10- Release the pressure cuff/Tourniquet:

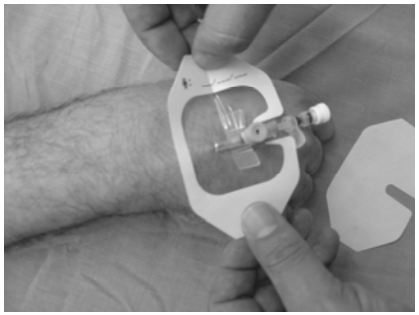
- **Forgetting to do this , ha resulted in Catastrophic consequences!!!**

  (Especially in neonates and small pediatric patients)



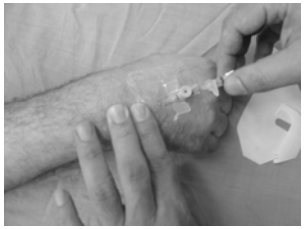
11- Fix the I.V. Canula

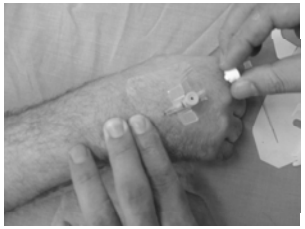
- **using the prepared plaster strips / or ready made canula dressing**



12- Remove the needle

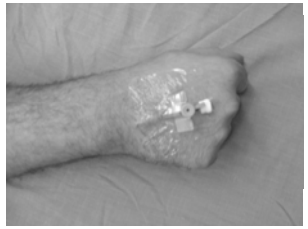
- **first apply a compressing pressure with your non-dominant hand just proximal to the canula (remember its intra-vessel length),**
- **pull the needle out, and apply the canula cover, or connect the infusion line.**

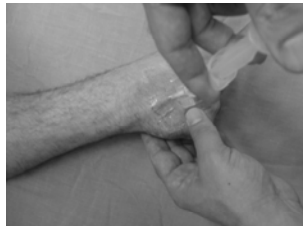




13- Check the patency and function of the canula

- **by injecting few mls of normal saline (or tuning on the infusion) and observing for ease of injection/infusion flow, and swelling formation.**





Needle disposition

- **When handling the needle make sure it is always directed away from patient, yourself, and other staff and quickly and safely discard it in the proper sharps container**

Important Points!!

- **<u>The three most common causes of failure to canulate a vein are:</u>**
  1- Failure to stretch the skin and consequently fix the vein, or prematurely releasing the skin before the canula is fully advanced
  2- Pulling back the guide needle too early, before the plastic catheter enter the vein
  3- Advancing the canula despite resistance

<u>Avoid:</u>

- Infected areas,
- Thrombosed veins: hard and non-compressible on palpation
- Joint areas: risk of kinking of canula
- Paralyzed Limbs: risk of thrombosis
- Limbs with lymphatic blockage (e.g. after breast surgery)
- Vein valves: if you see a lump in the vein course

Maneuvers to make the vein more visible:

- **Applying the tourniquet proximal to the puncture site**
- **Put limb in dependent position**
- **Use muscle pump to fill the vein (ask patient to open and close hand several times)**
- **Tapping over the vein**
- **Warming the puncture site: by rubbing or applying warm water bag**
- **Spray area with alcohol or nitrolingual spray if available**

Canula Care

- **Inspect frequently for development of any erythema, or swelling along the vein course**
- **Change dressing if not clean**
- **Always flush canula with few mls of normal saline after administration of drugs etc through the same port**
- **Change the canula every 2 – 3 days and more frequently if was used for blood or lipid solution transfusion.**

Central venous line

- Indications
- Sites
- Procedure
- Complications

Indications and uses

Indications for the use of central lines include
Monitoring of the central venous pressure (CVP) in acutely ill patients to quantify fluid bal
Long-term Intravenous antibiotics
Long-term Parenteral nutrition especially in chronically ill patients
Long-term pain medications
Chemotherapy
Drugs that are prone to cause phlebitis in peripheral veins (caustic), such as:
Calcium chloride
Chemotherapy
Hypertonic saline
Potassium chloride
Amiodarone
Plasmapheresis
Dialysis
Frequent blood draws
Frequent or persistent requirement for intravenous access
Need for intravenous therapy when peripheral venous access is impossible
Blood
Medication
Rehydration

Sites of insertion

- Subclavian vein
- Internal jugular vein
- Basilic vein
- Femoral vein
- External jugular vein.

Complications of CVL insertion

- Pneumothorax (subclavian,IJV)
- Bleeding and haematoma formation
- Arterial puncture
- Infection
- arrhythmias (subclavian,IJV)

Advantages and disadvantages of central vein approaches

Approach	Advantages	Disadvantages
External jugular	Superficial vessel that is often visible Coagulopathy not prohibitive Minimal risk of pneumothorax (especially with US guidance) Head-of-table access Prominent in elderly patients Rapid venous access	Not ideal for prolonged venous access Poor landmarks in obese patients High rate of malposition Catheter may be difficult to thread
Internal jugular	Minimal risk of pneumothorax (especially with US guidance) Head-of-table access Procedure-related bleeding amenable to direct pressure Lower failure rate with novice operator Excellent target using US guidance	Not ideal for prolonged access Risk of carotid artery puncture Uncomfortable Dressings and catheter difficult to maintain Thoracic duct injury possible on left Poor landmarks in obese/edematous patients Potential access and maintenance issues with concomitant tracheostomy Vein prone to collapse with hypovolemia Difficult access during emergencies when airway control being established
Subclavian	Easier to maintain dressings More comfortable for patient Better landmarks in obese patients Accessible when airway control is being established	Increased risk of pneumothorax Procedure-related bleeding less amenable to direct pressure Decreased success rate with inexperience Longer path from skin to vessel Catheter malposition more common (especially right SCV) Interference with chest compressions
Femoral	Rapid access with high success rate Does not interfere with CPR Does not interfere with intubation No risk of pneumothorax Trendelenburg position not necessary during insertion	Delayed circulation of drugs during CPR Prevents patient mobilization Difficult to keep site sterile Difficult for PA catheter insertion Increased risk of ilofemoral thrombosis

US: ultrasound; SCV: subclavian vein; CPR: cardiopulmonary resuscitation; PA: pulmonary artery.
With permission from: Factor P, Sznajder JI. Vascular cannulation. In: Principles of Critical Care, Hall JB, Schmidt GA, Wood LDH (Eds), McGraw-Hill, New York, 1998. Copyright 1998 McGraw-Hill.

Complications of central venous catheterization

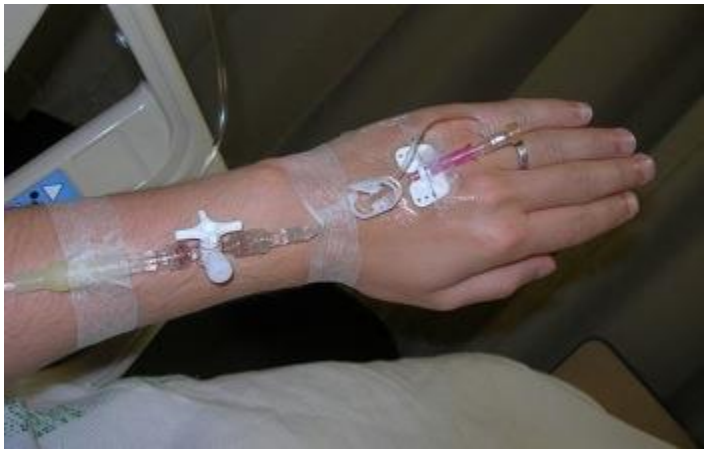
Immediate
Bleeding
Arterial puncture
Arrhythmia
Air embolism
Thoracic duct injury (with left SC or left IJ approach)
Catheter malposition
Pneumothorax or hemothorax
Delayed
Infection
Venous thrombosis, pulmonary emboli
Catheter migration
Catheter embolization
Myocardial perforation
Nerve injury

Graphic 77376 Version 2.0

Peripheral Venous Access

Peripheral venous access, or intravenous (IV) access, is commonly used to administer fluids, medications, or blood.

IV therapy is one of the fastest routes of providing medication, and is the most effective means of providing fluids during periods of dehydration or hemodynamic instability.



Indications

Peripheral IVs are used to:

- administer medications, IV fluids, blood, or parenteral nutrition
- maintain access for the above in a potentially unstable patient
- sample venous blood (when necessary)

Contraindications

The following contraindications all relate to the site chosen rather than the procedure itself.

- existing skin infection in intended IV site
- venous thrombosis proximal to intended site
- arterial-venous shunt (i.e. for hemodialysis) in extremity
- lymphatic obstruction (i.e. following axillary node dissection) in extremity

Procedure

1- Choosing Catheter Size

Teflon/Plastic catheters are the most common type of catheter used. They range in size from 14 - 25g.

- 22-25g - pediatric applications or occasionally in adults with very small veins
- 20g - standard, multipurpose adult IV

- 18g - suitable for higher flow rates in adults and for routine administration of blood products
- 14 - 16g - large (painful) catheters reserved for situations where volume resuscitation is needed or anticipated. i.e. hypovolemic shock, GI bleeding, Some preoperative surgical cases

Butterfly Catheters are generally used only in pediatrics and for very short-term venous access, as they tend to perforate veins easily and are more prone to infection. Sizes 20 - 25g.

Safety IV catheter systems are increasingly used to reduce needle stick injuries by encasing the entire needle within a guard.

2- Choosing the Site

Upper extremities are generally preferred to lower as they are more convenient for staff and patient and pose lesser risk of infection, phlebitis and deep vein thrombosis.

Preferred sites are **dorsal hand and volar forearm**, in the non-dominant arm if possible. Try to pick a long straight section of vein or a bifurcation. Start as far distal on the arm as possible - if you miss you can try more proximally without getting leaks from previous sites.

If the IV is being placed to resuscitate a patient, veins within the antecubital fossa (the inside of the elbow) should be considered, given their relatively bigger size.

Sites to Avoid

- joints - more difficult to stop the IV from kinking or infiltrating when the joint is flexed
- infected skin/cellulitis - for obvious reasons.
- extremities with arteriovenous fistulas (ie hemodialysis patients)
- traumatized extremities - veins may be interrupted or occluded proximally
- extremities with venous insufficiency i.e. post mastectomy
- paretic limbs i.e. stroke, spinal cord injury

3- Materials Required

- povidone - Iodine Swabs
- alcohol Swabs
- tourniquet - Velcro, Elastic or BP Cuff
- IV Catheter (Hopefully just one)
- normal saline Lock
- 10 cc syringe with normal saline for flushing
- tape
- transparent Dressing (i.e. Tegaderm, Opsite)

- gloves
- IV infusion setup
- local Anaesthetic i.e. 1 - 2 % Plain Xylocaine in Tuberculin Syringe
- incontinence Pad - for under the patient's arm so the area nurse won't curse you
- incontinence Pad - for yourself if first try with an IV (Optional)

4- Accessing the Vein

Explain the procedure to the patient and why it is necessary- obtain verbal consent.

Get **everything** ready - open packages, tear your pieces of tape etc.

minimal standard (Universal) precautions for IV starts is gloves.

Ensure the patient is comfortable - lying or sitting.

Prepare normal saline lock by cleaning the port with an alcohol swab and priming it with sterile normal saline flush solution. Leave the saline flush syringe attached.

Apply the tourniquet 5 - 10 cm proximal to the chosen site. If using a BP cuff inflate it to ~ 15 mm Hg below arterial systolic pressure.

Prep the skin with Povidone - Iodine starting over the site and circling outwards. Follow this with an alcohol swab in the same fashion. If you choose to use local anaesthesia, raise a small intradermal weal of Xylocaine over the site.

Using your non-dominant hand, hold the patient's extremity and gently pull the skin taught over the vein. Using the safety IV catheter insert the needle bevel upwards, parallel to the skin surface.

Push the needle into the vein until you feel a pop and blood appears in the flash chamber of the IV Cannula. While holding the needle stationary, advance the cannula into the vein by placing the forefinger of your dominate hand, or the thumb of your non dominate hand, against the push-off tab. Pull the needle back into the needle guard until it clicks.

The needle is now encased in the needle guard to help prevent needle stick injuries.

Remove the tourniquet from the patient's arm.

Attach the primed Normal Saline lock and flush it with 1-3 mls of Normal saline flush solution using the positive pressure technique (the saline injected via a syringe at the same time as the syringe is being removed).

Cover the site with a sterile transparent dressing.

Swab the top of the Normal saline lock again and connect the primed IV tubing. Observe for fluid flow and continue to watch for swelling.

Tape the tubing in place. Properly dispose of sharps, gloves etc

Troubleshooting

Vein Blows or IV solution infiltrates out of vein - Remove it. Apply firm pressure. Try the other arm or more proximal site.

Spider veins (too small) - Dilate veins with warm moist washcloth, gentle tapping, hanging over side of bed (the arm). Some advocate 0.5 cm of nitroglycerine ointment over site.

IV won't run - Make sure cannula in vein, tourniquet is off, drip valve is open, IV solution bag is elevated. Make sure the drip chamber is not too full. May try withdrawing cannula by few mm in case it's up against a venous valve.

Air in IV Line - Wind IV tubing around tightly around barrel of a pen, pushing air back into IV bag Or Aspirate air using needle and syringe in the distal injection port.

Complications

Hematoma - generally caused by passing the catheter through the back wall of the vein. Remove catheter and apply local pressure for 5 - 10 minutes.

Extravasation - recognized by local swelling at the IV site. Majority of extravasations are benign and treated by removing the catheter and applying local pressure.

Extravasation Necrosis - occurs secondary to extravasation of hypertonic solutions (D50W, 10 % CaCl, TPN, etc), toxic substances (chemotherapy, contrast dye) or vasoactive substances (epinephrine, dopamine). Accordingly, these are usually diluted or given centrally. If necrosis occurs, discontinue the infusion immediately and consult the pharmacy - some of these substances have specific treatments to reduce the risk of tissue necrosis.

Irritative Phlebitis - recognized by erythema at the site and possibly along the path of the vein. The majority is caused by irritation of the vein by the catheter or infused substances. Irritating substances may be diluted to reduce this risk. Treatment is removal of the catheter, local heat application and sometimes anti-inflammatories.

Infectious Thrombophlebitis - sometimes difficult to distinguish from irritative phlebitis - look for erythema and tenderness proximally along the vein; may be associated with fever. Usually caused by invasion of skin organisms such as *Staph. Aureus* or *Staph. Epidermidis*. Aseptic technique and a policy of changing IV sites every 2 - 3 days will reduce the risk. Treatment is by removal of the catheter, local heat and anti-staphylococcal antibiotics

Embolization - uncommon problem unless solutions are being infused under pressure. Small bubbles in a line are not dangerous but larger quantities of air should be removed. To avoid shearing off and embolizing small pieces of the Teflon catheter, never withdraw the metal needle then replace it in the catheter again.

Lecture #3

Cadiopulmonary resuscitation

Basic life support algorithm

OBJECTIVES

To know

- ❑ Assessment,airway,breathing, circulation
- ❑ How to perform cardiopulmonary resuscitation in adequate manner
- ❑ How to turn the patient in a recovery position
- ❑ The possible problems that may be associated with the BLS

Introduction

- Initial assessment,Aw maintenance ,expired air ventilation, chest compression
- No equipment is used
- Aim is to keep adequate vent. Circulation until equipment can be obtained .
- In resp failure it can reverse the cause
- The first 3-4 golden min
- O2 content ,available and reserve
- Permanent damage

History

- Hx of first BLS is in the Bible
- First medical BLS is in 1744 by Tossach
- Victorian period
- 1950 mouth to mouth breathing was rediscovered
- 1878 closed chest cardiac massage was first used by Boehm
- Open chest massage became the slandered method of cardiac arrest
- 1960 Jude et al reintroduced the external cardiac massage
- So 1960 is the year of modern cardiopulmonary resuscitation

Theory of chest compression

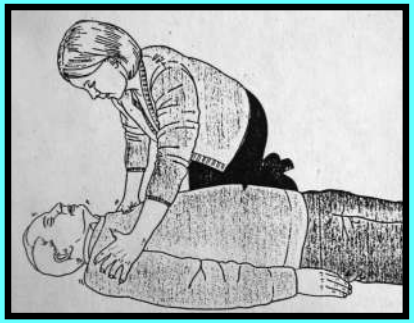
- Cardiac massage and External cardiac massage
- Heart pump theory was criticized in 1970
  - ECHO valve incompetents
  - Coughing produce sustained circulation
- Thoracic pump theory By increasing intrathoracic pressure

BLS algorithm

- Safe to approach
- Check response
- If responsive

  leave in the same position

  check regularly



BLS algorithm

- If no response

1. Ask for help
2. Open the AW

   Head tilt

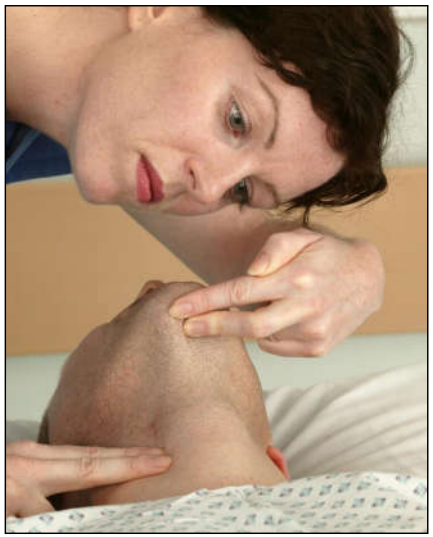
   Chin lift

   Jaw thrust

Open Airway
Look for signs of life

…. to confirm cardiac arrest

- Patient response
- Open airway
- Check for normal breathing
  - (caution agonal breathing)
- Check circulation
- Monitoring



BLS algorithm

- Check for breathing

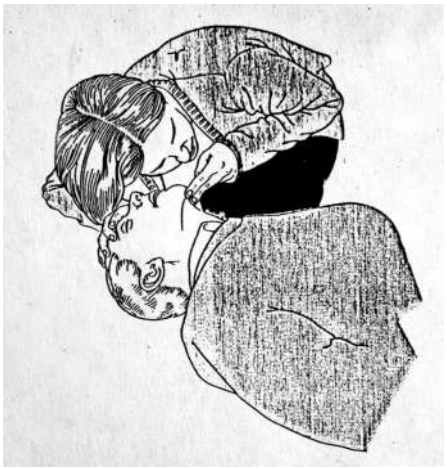
  Lock

  Listen

  Feel

FOR 10 second

(OLD GUIDELINES)



BLS algorithm

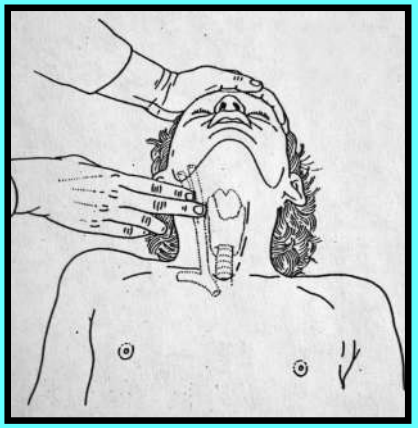
- If no breathing
  send for help
  turn pt onto his back
  remove any visible obstruction
  give 2 breaths

(OLD GUIDELINES)

BLS algorithm

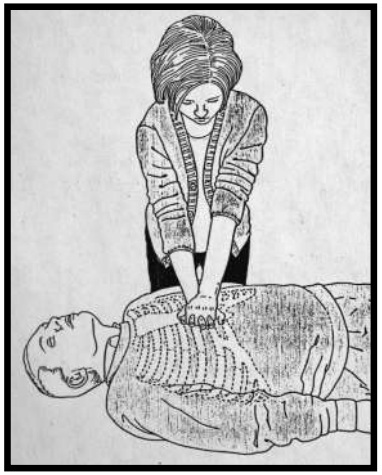
- Check circulation
  at one of the major artery the best is carotid for 5-10 seconds

(OLD GUIDELINES)



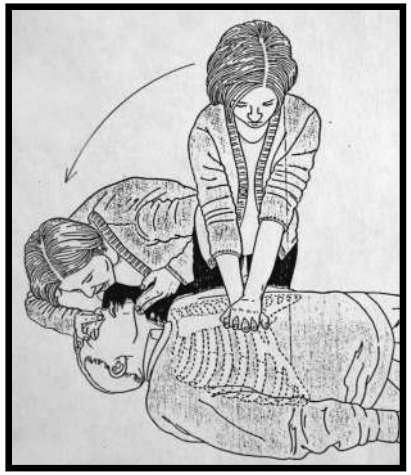
BLS algorithm

- If no circulation
  identify the middle of the sternum
  start ECM 30:2
  use the heel of the hands
  fingers interlocked
  vertical above the pt chest
  arms should be straight
  depress the sternum 1.5-2 inc
  rate is about 100 min



BLS algorithm

- Continue resuscitation until
  The pt shows signs of life
  CPR team arrives
  you become exhausted



When to go for help

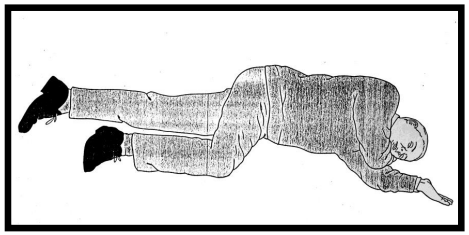
- If 2 persons available 1 is doing BLS
  2 is going for help
- if 1 is available decide what to do
- If the pt is an adult not a child or due to trauma or drowning

Choking

- If the pt is conscious and breathing encourage him to continue coughing
- If the obst is complete or the pt shows signs of exhaustion or cyanosis
  if the pt is conscious start back blows
  If no response do abdominal thrust
- If the pt is unconscious
  finger sweeps
  abdominal thrust while the pt supine

Recovery position

- After breathing and circulation has been restored
- To maintain the opening of the airway
- To prevent inhalation of gastric content



Part 2

Introduction

" SCA is a leading cause of death in the United States and Canada .At the first analysis of heart rhythm, about 40% of victims of out-of-hospital SCA demonstrate ventricular fibrillation (VF)

Introduction

Many SCA victims can survive if bystanders act immediately while VF is still present, but successful resuscitation is unlikely once the rhythm deteriorates to asystole

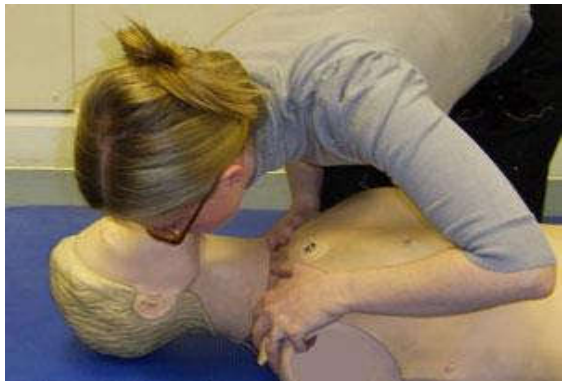
DRsABC

- DRsABCis a useful acronym to help you remember each stage in sequence:
  - Danger
  - Response
  - Shout for help
  - Airway
  - Breathing
- Circulation

Danger

Danger Check for danger to either yourself or the patient, to ensure it's safe to approach. Danger within the community may include: Furniture, pets, utilities Danger within the hospital setting may include: Bedside furniture, jugs of water, vase of flowers, visitors, cables, spillages

Response



Shake and shout

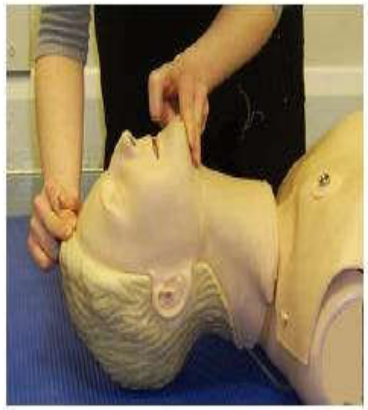
Cardio Pulmonary Resuscitation (CPR)

Shout for help

'Can I have a hand over here *now* please!'

As you have an unresponsive patient, you need some help.

Airway



Open the airway with a head tilt, chin lift manoeuvre
Look for any visible obstructions

Not breathing

Cardio Pulmonary Resuscitation (CPR)

Look, Listen and Feel for Breathing

Look, listen and feel for breathing for 10 seconds, whilst maintaining the head tilt / chin lift

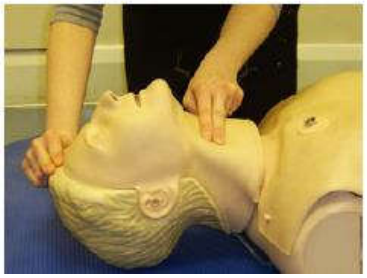
Breathing? Yes

If the patient is unresponsive, but is breathing and has a pulse, they need to be placed in the recovery position

GIVE TWO RESCUE BREATHS

Give two rescue breaths by mouth-to-mouth breathing

Circulation

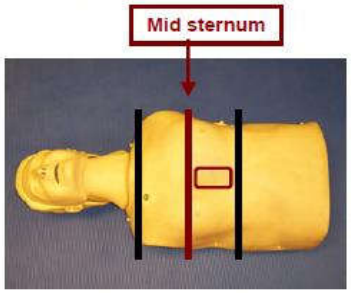


Locate the carotid pulse and feel for 10 seconds

Not Breathing, No Pulse?

Commence CPR at a ratio of: **30 chest compressions : 2 ventilations**

Chest Compressions



The heel of the hand is placed in the middle of the lower half of the sternum, indicated by the rectangle on the picture on the left

Still with me ?

CHECK FOR RESPONSIVENESS — No response

CALL FOR HELP

OPEN AIRWAY

LOOK, LISTEN, FEEL — .Not breathing

GIVE TWO RESCUE BREATHS

CHECK FOR PULSE — No pulse

START CHEST COMPRESSIONS

30 compressions

2 breaths

30 compressions

2 breaths

30 compressions

2 breaths

30 compressions

2 breaths

30 compressions

Check for pulse

If pulse can be felt, continue rescue breaths at one breath every 5 to 6 seconds.

If the victim starts breathing, turn him/her on his/her side. Wait for medical help to

Adult BLS Healthcare Provider Algorithm

Circulation

rican Heart Association

Learn and Live

Chest Compressions – How long?

Continue CPR until:

- The patient shows signs of life
- The resuscitation team / ambulance arrives and tells you to stop
- You are physically exhausted

William Osler, 1919

"Learn to see, learn to hear, learn to feel, learn to smell, and know that by practice alone can you become expert. Medicine is learned by the bedside and not in the classroom. See, and then reason and compare and control. But see first ".

Adult advanced life support

Authors

Jasmeet Soar, Charles Deakin, Andrew Lockey, Jerry Nolan, Gavin Perkins

1. The guideline process

The process used to produce the Resuscitation Council (UK) Guidelines 2015 has been accredited by the National Institute for Health and Care Excellence. The guidelines process includes:

- Systematic reviews with grading of the quality of evidence and strength of recommendations. This led to the 2015 International Liaison Committee on Resuscitation (ILCOR) Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations.[1,2]
- The involvement of stakeholders from around the world including members of the public and cardiac arrest survivors.
- Details of the guidelines development process can be found in the Resuscitation Council (UK) Guidelines Development Process Manual.
- These Resuscitation Council (UK) Guidelines have been peer reviewed by the Executive Committee of the Resuscitation Council (UK), which comprises 25 individuals and includes lay representation and representation of the key stakeholder groups.

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2. Summary of changes in advanced life support since 2010 Guidelines

The 2015 Advanced life support (ALS) guidelines have a change in emphasis aimed at improved care and implementation of these guidelines in order to improve patient outcomes.[3] The key changes since 2010 are:

- Increased emphasis on minimally interrupted high quality chest compressions throughout any ALS intervention.
- Chest compressions must only be paused briefly to enable specific interventions. This includes minimising interruptions in chest compressions to less than 5 seconds when attempting defibrillation or tracheal intubation.
- There is a new section on monitoring during ALS.
- Waveform capnography must be used to confirm and continually monitor tracheal tube placement, and may be used to monitor the quality of CPR and to provide an early indication of return of spontaneous circulation (ROSC).
- There are a variety of approaches to airway management during CPR and a stepwise approach based on patient factors and the skills of the rescuer is recommended.
- The recommendations for drug therapy during CPR have not changed, but there is equipoise for the role of drugs in improving outcomes from cardiac arrest.
- The routine use of mechanical chest compression devices is not recommended, but they may be useful in situations where sustained high quality manual chest compressions are impractical or compromise provider safety.
- Peri-arrest ultrasound may be used to identify reversible causes of cardiac arrest.
- Extracorporeal life support techniques may be used as a rescue therapy in selected patients where standard ALS measures are not successful.
- The ALS algorithm (Figure 1) has been modified slightly to show these changes.

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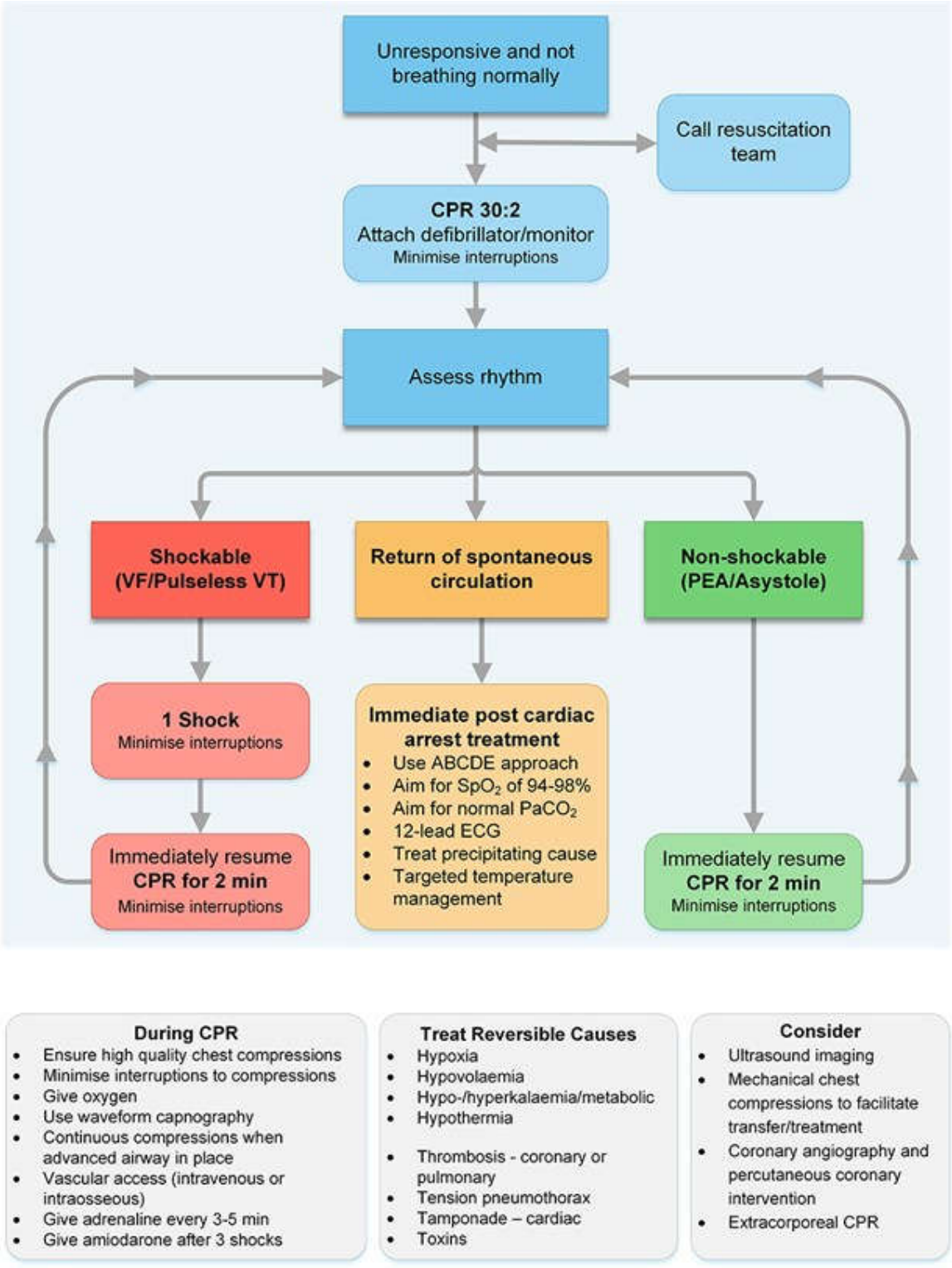

Figure 1. Adult advanced life support algorithm

3. Introduction

This section on adult advanced life support (ALS) adheres to the same general principles as Guidelines 2010, but incorporates some important changes. The guidelines in this section apply to healthcare professionals trained in ALS techniques. Laypeople, first responders, and automated external defibrillator (AED) users are referred to the Adult basic life support and automated external defibrillation section.

Adult ALS includes advanced interventions after basic life support has started and when appropriate an AED has been used. The transition between basic and advanced life support should be seamless as BLS will continue during and overlap with ALS interventions. Post-resuscitation care guidelines are presented in a new section that recognises the importance of the final link in the Chain of Survival.[4]

These guidelines are based on the International Liaison Committee on Resuscitation (ILCOR) 2015 Consensus on Science and Treatment Recommendations (CoSTR) for ALS[2] and the European Resuscitation Council 2015 Advanced Life Support Guidelines.[5] These contain all the reference material for this section.

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4. ALS treatment algorithm

Heart rhythms associated with cardiac arrest are divided into two groups: shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT)) and non-shockable rhythms (asystole and pulseless electrical activity (PEA)). The main difference in the treatment of these two groups is the need for attempted defibrillation in patients with VF/pVT.

Other actions, including chest compression, airway management and ventilation, vascular access, administration of adrenaline, and the identification and correction of reversible factors, are common to both groups. The ALS algorithm provides a standardised approach to the management of adult patients in cardiac arrest.

Drugs and advanced airways are still included among ALS interventions, but are of secondary importance to early defibrillation and high quality, uninterrupted chest compressions. At the time of writing these guidelines, three large randomised controlled trials (RCTs) (adrenaline versus placebo [ISRCTN73485024], amiodarone versus lidocaine versus placebo[6] [NCT01401647] and supraglottic airway (i-gel) versus tracheal intubation [ISRCTN No: 08256118]) are currently ongoing.

Shockable rhythms (VF/pVT)

The first monitored rhythm is VF/pVT in approximately 20% of both in-hospital[7] and out-of-hospital cardiac arrests (OHCAs).[8] Ventricular fibrillation/pulseless ventricular tachycardia will also occur at some stage during resuscitation in about 25% of cardiac arrests with an initial documented rhythm of asystole or PEA.[9,10]

Treatment of shockable rhythms (VF/VT)

1. Confirm cardiac arrest – check for signs of life and normal breathing, and if trained to do so check for breathing and a pulse simultaneously.
2. Call resuscitation team.
3. Perform uninterrupted chest compressions while applying self-adhesive defibrillation/monitoring pads – one below the right clavicle and the other in the V6 position in the midaxillary line.
4. Plan actions before pausing CPR for rhythm analysis and communicate these to the team.
5. Stop chest compressions; confirm VF/pVT from the ECG. This pause in chest compressions should be brief and no longer than 5 seconds.
6. Resume chest compressions immediately; warn all rescuers other than the individual performing the chest compressions to “stand clear” and remove any oxygen delivery device as appropriate.
7. The designated person selects the appropriate energy on the defibrillator and presses the charge button. Choose an energy setting of at least 150 J for the first shock, the same or a higher energy for subsequent shocks, or follow the manufacturer’s guidance for the particular defibrillator. If unsure of the correct energy level for a defibrillator choose the highest available energy.
8. Ensure that the rescuer giving the compressions is the only person touching the patient.
9. Once the defibrillator is charged and the safety check is complete, tell the rescuer doing the chest compressions to “stand clear”; when clear, give the shock.
10. After shock delivery immediately restart CPR using a ratio of 30:2, starting with chest compressions. Do not pause to reassess the rhythm or feel for a pulse. The total pause in chest compressions should be brief and no longer than 5 seconds.

11. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.
12. Pause briefly to check the monitor.
13. If VF/pVT, repeat steps 6–12 above and deliver a second shock.
14. If VF/pVT persists, repeat steps 6–8 above and deliver a third shock. Resume chest compressions immediately. Give adrenaline 1 mg IV and amiodarone 300 mg IV while performing a further 2 min CPR. Withhold adrenaline if there are signs of return of spontaneous circulation (ROSC) during CPR.
15. Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists.
16. Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).
17. If organised electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of ROSC (check for signs of life, a central pulse and end-tidal CO_2 if available).
    1. If there is ROSC, start post-resuscitation care.
    2. If there are no signs of ROSC, continue CPR and switch to the non-shockable algorithm.
18. If asystole is seen, continue CPR and switch to the nonshockable algorithm.

The interval between stopping compressions and delivering a shock must be minimised. Longer interruptions to chest compressions reduce the chance of a shock restoring a spontaneous circulation. Chest compressions are resumed immediately after delivering a shock (without checking the rhythm or a pulse) because even if the defibrillation attempt is successful in restoring a perfusing rhythm, it is very rare for a pulse to be palpable immediately after defibrillation. The duration of asystole before ROSC can be longer than 2 min in as many as 25% of successful shocks.[11] If a shock has been successful immediate resumption of chest compressions does not increase the risk of VF recurrence.[12] Furthermore, the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored.[13]

The use of waveform capnography can enable ROSC to be detected without pausing chest compressions and may be used as a way of avoiding a bolus injection of adrenaline after ROSC has been achieved. Several human studies have shown that there is a significant increase in end-tidal CO_2 when ROSC occurs.[5,14] If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

Regardless of the arrest rhythm, after the initial adrenaline dose has been given, give further doses of adrenaline 1 mg every 3–5 min until ROSC is achieved; in practice, this will be about once every two cycles of the algorithm. If signs of life return during CPR (e.g. purposeful movement, normal breathing or coughing), or there is an increase in end-tidal CO_2, check the monitor; if an organised rhythm is present, check for a pulse. If a pulse is palpable, start post-resuscitation care. If no pulse is present, continue CPR.

Give amiodarone 300 mg IV after three defibrillation attempts irrespective of whether they are consecutive shocks, or interrupted by CPR, or for recurrent VF/pVT during cardiac arrest. Consider a further dose of amiodarone 150 mg IV after a total of five defibrillation attempts. Lidocaine 1 mg kg^{-1} may be used as an alternative if amiodarone is not available but do not give lidocaine if amiodarone has been given already.

Witnessed, monitored VF/pVT

If a patient has a monitored and witnessed cardiac arrest in the catheter laboratory, coronary care unit, a critical care area or whilst monitored after cardiac surgery, and a manual defibrillator is rapidly available:

- Confirm cardiac arrest and shout for help.
- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.
- Rapidly check for a rhythm change and, if appropriate, ROSC after each defibrillation attempt.
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful.

This three-shock strategy may also be considered for an initial, witnessed VF/pVT cardiac arrest if the patient is already connected to a manual defibrillator – these circumstances are rare. Although there are no data supporting a three-shock strategy in any of these circumstances, it is unlikely that chest compressions will improve the already very high chance of ROSC when defibrillation occurs early in the electrical phase, immediately after onset of VF/pVT.

If this initial three-shock strategy is unsuccessful for a monitored VF/pVT cardiac arrest, the ALS algorithm should be followed and these three-shocks treated as if only the first single shock has been given.

Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm.[15-19]Its routine use is therefore not recommended. Consider a precordial thump only when it can be used without delay whilst awaiting the arrival of a defibrillator in a monitored VF/pVT arrest. Using the ulnar edge of a tightly clenched fist, deliver a sharp impact to the lower half of the sternum from a height of about 20 cm, then retract the fist immediately to create an impulse-like stimulus.

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as cardiac arrest in the presence of electrical activity (other than ventricular tachyarrhythmia) that would normally be associated with a palpable pulse.[20]These patients often have some mechanical myocardial contractions, but these are too weak to produce a detectable pulse or blood pressure – this is sometimes described as 'pseudo-PEA' (see below). PEA can be caused by reversible conditions that can be treated if they are identified and corrected. Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated effectively.

Treatment of PEA and asystole

1. Start CPR 30:2
2. Give adrenaline 1 mg IV as soon as intravascular access is achieved
3. Continue CPR 30:2 until the airway is secured – then continue chest compressions without pausing during ventilation
4. Recheck the rhythm after 2 min:

a. If electrical activity compatible with a pulse is seen, check for a pulse and/or signs of life
i. If a pulse and/or signs of life are present, start post resuscitation care
ii. If no pulse and/or no signs of life are present (PEA OR asystole):

1. Continue CPR
2. Recheck the rhythm after 2 min and proceed accordingly
3. Give further adrenaline 1 mg IV every 3–5 min (during alternate 2-min loops of CPR)

b. If VF/pVT at rhythm check, change to shockable side of algorithm.

Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because the patient may respond to cardiac pacing when there is ventricular standstill with continuing P waves. There is no value in attempting to pace true asystole.

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5. Treat reversible causes

Potential causes or aggravating factors for which specific treatment exists must be considered during all cardiac arrests.[21] For ease of memory, these are divided into two groups of four, based upon their initial letter: either H or T:

- Hypoxia
- Hypovolaemia
- Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders
- Hypothermia
- Thrombosis (coronary or pulmonary)
- Tension pneumothorax
- Tamponade – cardiac
- Toxins

The four 'Hs'

Minimise the risk of **hypoxia** by ensuring that the patient's lungs are ventilated adequately with the maximal possible inspired oxygen during CPR. Make sure there is adequate chest rise and bilateral breath sounds. Using the techniques described below, check carefully that the tracheal tube is not misplaced in a bronchus or

the oesophagus.

Pulseless electrical activity caused by **hypovolaemia** is due usually to severe haemorrhage. This may be precipitated by trauma, gastrointestinal bleeding or rupture of an aortic aneurysm. Stop the haemorrhage and restore intravascular volume with fluid and blood products.

Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia and other metabolic disorders are detected by biochemical tests or suggested by the patient's medical history (e.g. renal failure). Give IV calcium chloride in the presence of hyperkalaemia, hypocalcaemia and calcium channel-blocker overdose.

Hypothermia should be suspected based on the history such as cardiac arrest associated with drowning.

The four 'Ts'

Coronary **thrombosis** associated with an acute coronary syndrome or ischaemic heart disease is the most common cause of sudden cardiac arrest. An acute coronary syndrome is usually diagnosed and treated after ROSC is achieved. If an acute coronary syndrome is suspected, and ROSC has not been achieved, consider urgent coronary angiography when feasible and, if required, percutaneous coronary intervention. Mechanical chest compression devices and extracorporeal CPR can help facilitate this (see below).

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolism. If pulmonary embolism is thought to be the cause of cardiac arrest consider giving a fibrinolytic drug immediately. Following fibrinolysis during CPR for acute pulmonary embolism, survival and good neurological outcome have been reported, even in cases requiring in excess of 60 min of CPR. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60–90 min before termination of resuscitation attempts. In some settings extracorporeal CPR, and/or surgical or mechanical thrombectomy can also be used to treat pulmonary embolism.

A **tension pneumothorax** can be the primary cause of PEA and may be associated with trauma. The diagnosis is made clinically or by ultrasound. Decompress rapidly by thoracostomy or needle thoracocentesis, and then insert a chest drain.

Cardiac **tamponade** is difficult to diagnose because the typical signs of distended neck veins and hypotension are usually obscured by the arrest itself. Cardiac arrest after penetrating chest trauma is highly suggestive of tamponade and is an indication for resuscitative thoracotomy. The use of ultrasound will make the diagnosis of cardiac tamponade much more reliable.

In the absence of a specific history, the accidental or deliberate ingestion of therapeutic or **toxic**substances may be revealed only by laboratory investigations. Where available, the appropriate antidotes should be used, but most often treatment is supportive and standard ALS protocols should be followed.

Use of ultrasound imaging during advanced life support

When available for use by trained clinicians, focused echocardiography/ultrasound may be of use in assisting with diagnosis and treatment of potentially reversible causes of cardiac arrest. The integration of ultrasound into advanced life support requires considerable training if interruptions to chest compressions are to be minimised. A sub-xiphoid probe position has been recommended.[22-24] Placement of the probe just before chest compressions are paused for a planned rhythm assessment enables a well-trained operator to obtain views within 10 seconds.

Several studies have examined the use of ultrasound during cardiac arrest to detect potentially reversible causes.[25-27] Although no studies have shown that use of this imaging modality improves outcome, there is no doubt that echocardiography has the potential to detect reversible causes of cardiac arrest. Specific protocols for ultrasound evaluation during CPR may help to identify potentially reversible causes (e.g. cardiac tamponade, pulmonary embolism, hypovolaemia, pneumothorax). Absence of cardiac motion on sonography during resuscitation of patients in cardiac arrest is highly predictive of death although sensitivity and specificity has not been reported.[28-31]

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6. During CPR

High quality chest compressions with minimal interruption

During the treatment of persistent VF/pVT or PEA/asystole, there should be an emphasis on giving high quality chest compression between defibrillation attempts or rhythm checks, whilst recognising and treating

reversible causes (4 Hs and 4 Ts), and whilst obtaining a secure airway and intravascular access. Aim for a chest compression pause of less than 5 seconds for rhythm checks, defibrillation attempts, and tracheal intubation. To achieve this rescuers must plan their actions before pausing compressions.

Monitoring during advanced life support

The following methods can be used to monitor the patient during CPR and help guide ALS interventions:

- Clinical signs such as breathing efforts, movements and eye opening can occur during CPR. These can indicate ROSC and require verification by a rhythm and pulse check, but can also occur because CPR can generate a sufficient circulation to restore signs of life including consciousness.[32]
- Pulse checks when there is an ECG rhythm compatible with an output can be used to identify ROSC, but may not detect pulses in those with low cardiac output states and a low blood pressure.[33] The value of attempting to feel arterial pulses during chest compressions to assess the effectiveness of chest compressions is unclear. A pulse that is felt in the femoral triangle may indicate venous rather than arterial blood flow. There are no valves in the inferior vena cava and retrograde blood flow into the venous system can produce femoral vein pulsations.[34] Carotid pulsation during CPR does not necessarily indicate adequate myocardial or cerebral perfusion.
- Monitoring heart rhythm through pads, paddles or ECG electrodes is a standard part of ALS. Motion artefacts prevent reliable heart rhythm assessment during chest compressions forcing rescuers to stop chest compressions to assess the rhythm, and preventing early recognition of recurrent VF/pVT. We suggest that artefact-filtering algorithms are not used for analysis of ECG rhythm during CPR unless as part of a research programme.[35]
- End-tidal CO_2 with waveform capnography. The use of waveform capnography during CPR has a greater emphasis in Guidelines 2015 and is addressed in more detail below.
- The use of CPR feedback or prompt devices during CPR should be considered only as part of a broader system of care that should include comprehensive CPR quality improvement initiatives [36-38] rather than an isolated intervention.
- Blood sampling and analysis during CPR can be used to identify potentially reversible causes of cardiac arrest. Avoid finger prick samples in critical illness because they may not be reliable; instead, use samples from veins or arteries.
- Blood gas values are difficult to interpret during CPR. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid-base state.[39] Analysis of central venous blood may provide a better estimation of tissue pH.
- Invasive cardiovascular monitoring in critical care settings (e.g. continuous arterial blood pressure and central venous pressure monitoring). Invasive arterial pressure monitoring will enable the detection of low blood pressure values when ROSC is achieved.
- Ultrasound assessment is addressed above to identify and treat reversible causes of cardiac arrest, and identify low cardiac output states (‘pseudo-PEA’).

Waveform capnography during advanced life support

Use waveform capnography whenever tracheal intubation is undertaken. Although the prevention of unrecognised oesophageal intubation is clearly beneficial, there is currently no evidence that use of waveform capnography during CPR results in improved patient outcomes. The role of waveform capnography during CPR includes:

- Ensuring tracheal tube placement in the trachea (although it will not distinguish between bronchial and tracheal placement).
- Monitoring ventilation rate during CPR and avoiding hyperventilation.
- Monitoring the quality of chest compressions during CPR. End-tidal CO_2 values are associated with compression depth and ventilation rate and a greater depth of chest compression will increase the value.[40] Whether this can be used to guide care and improve outcome requires further study.[41]
- Identifying ROSC during CPR. An increase in end-tidal CO_2 during CPR can indicate ROSC and prevent unnecessary and potentially harmful dosing of adrenaline in a patient with ROSC.[14,41-43] If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.
- Prognostication during CPR. Precise values of end-tidal CO_2 depend on several factors including the cause of cardiac arrest, bystander CPR, chest compression quality, ventilation rate and volume, time from cardiac arrest and the use of adrenaline. Values are higher after an initial asphyxial arrest,

with bystander CPR, and decline over time after cardiac arrest.[41,44,45] Low end-tidal CO_2 values during CPR have been associated with lower ROSC rates and increased mortality, and high values with better ROSC and survival.[41,46,47] The inter-individual differences and influence of cause of cardiac arrest, the problem with self-fulfilling prophecy in studies, our lack of confidence in the accuracy of measurement during CPR, and the need for an advanced airway to measure end-tidal CO_2 reliably limits our confidence in its use for prognostication. The Resuscitation Council (UK) recommends that a specific end-tidal CO_2 value at any time during CPR should not be used alone to stop CPR efforts. End-tidal CO_2 values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

Defibrillation

This section predominantly addresses the use of manual defibrillators. Guidelines concerning the use of an automated external defibrillator (AED) are addressed in the Adult basic life support and automated external defibrillation section. The defibrillation strategy for the 2015 Resuscitation Guidelines has changed little from the former guidelines:

- The importance of early, uninterrupted chest compressions remains emphasised throughout these guidelines, together with minimising the duration of pre-shock and post-shock pauses – even 5–10 seconds delay will reduce the chances of the shock being successful.[48-53]
- Continue chest compressions during defibrillator charging, deliver defibrillation with an interruption in chest compressions of no more than 5 seconds and immediately resume chest compressions following defibrillation.
- Place the right (sternal) electrode to the right of the sternum, below the clavicle. Place the apical paddle in the mid-axillary line, approximately over the V6 ECG electrode position. This electrode should be clear of any breast tissue. It is important that this electrode is placed sufficiently laterally.
- Defibrillation shock energy levels are unchanged from the 2010 Guidelines.
    - Deliver the first shock with an energy of at least 150 J.
    - The shock energy for a particular defibrillator should be based on the manufacturer's guidance.
    - Those using manual defibrillators should be aware of the appropriate energy settings for the type of device used, but in the absence of this and if appropriate energy levels are unknown, for adults use the highest available shock energy for all shocks.
    - If an initial shock has been unsuccessful it is worth attempting the second and subsequent shocks with a higher energy level if the defibrillator is capable of delivering a higher energy but, based on current evidence, both fixed and escalating strategies are acceptable.
    - If VF/pVT recurs during a cardiac arrest (refibrillation) give subsequent shocks with a higher energy level if the defibrillator is capable of delivering a higher energy.
- There are no high quality clinical studies to indicate the optimal strategies within any given waveform and between different waveforms.[2] Knowledge gaps include the minimal acceptable first-shock energy level; the characteristics of the optimal biphasic waveform; the optimal energy levels for specific waveforms; and the best shock strategy (fixed versus escalating). It is becoming increasingly clear that selected energy is a poor comparator with which to assess different waveforms as impedance-compensation and subtleties in waveform shape result in significantly different transmyocardial current between devices for any given selected energy. The optimal energy levels may ultimately vary between different manufacturers and associated waveforms. Manufacturers are encouraged to undertake high quality clinical trials to support their defibrillation strategy recommendations.
- No one must touch the patient during shock delivery. Standard clinical examination gloves (or bare hands) do not provide a safe level of electrical insulation.[54]
- Use oxygen safely during defibrillation by:
    - Removing any oxygen mask or nasal cannulae and place them at least 1 m away from the patient's chest during defibrillation.
    - Leaving the ventilation bag connected to the tracheal tube or other airway adjunct. Alternatively, disconnect the ventilation bag from the tracheal tube and move it at least 1 m from the patient's chest during defibrillation.

Airway management and ventilation

The options for airway management and ventilation during CPR vary according to patient factors, the phase of the resuscitation attempt (during CPR, after ROSC), and the skills of rescuers.[55] They include: no airway and

no ventilation (compression-only CPR), compression-only CPR with the airway held open (with or without supplementary oxygen), mouth-to-mouth breaths, mouth-to-mask, bag-mask ventilation with simple airway adjuncts, supraglottic airways (SGAs), and tracheal intubation (inserted with the aid of direct laryngoscopy or videolaryngoscopy, or via a SGA).[2,5,56,57]

In comparison with bag-mask ventilation and use of a SGA, tracheal intubation requires considerably more training and practice and can result in unrecognised oesophageal intubation and increased hands-off time. A bag-mask, a SGA and a tracheal tube are frequently used in the same patient as part of a stepwise approach to airway management but this has not been formally assessed.[56]Patients who remain comatose after initial resuscitation from cardiac arrest will ultimately require tracheal intubation regardless of the airway technique used during cardiac arrest. Anyone attempting tracheal intubation must be well trained and equipped with waveform capnography. Personnel skilled in advanced airway management should attempt laryngoscopy and intubation without stopping chest compressions; a brief pause in chest compressions may be required as the tube is passed through the vocal cords, but this pause should be less than 5 seconds. In the absence of these, use bag-mask ventilation and/or an SGA until appropriately experience and equipped personnel are present.

There is no high quality evidence supporting one particular intervention over another. [2,57] Depending on the circumstances and the skills of the rescuers, use either an advanced airway (tracheal intubation or supraglottic airway (SGA)) or a bag-mask for airway management during CPR.[2,5]

Basic airway manoeuvres and airway adjuncts

Assess the airway. Use head tilt and chin lift, or jaw thrust to open the airway. Simple airway adjuncts (oropharyngeal or nasopharyngeal airways) are often helpful, and sometimes essential, to maintain an open airway. When there is a risk of cervical spine injury, establish a clear upper airway by using jaw thrust or chin lift in combination with manual in-line stabilisation of the head and neck by an assistant.[58,59] If life-threatening airway obstruction persists despite effective application of jaw thrust or chin lift, add head tilt in small increments until the airway is open; establishing a patent airway takes priority over concerns about a potential cervical spine injury.

Oxygen during CPR

During CPR, give the maximal feasible inspired oxygen concentration. There are no data to indicate the optimal arterial blood oxygen saturation (SaO_2) during CPR, and no trials comparing different inspired oxygen concentrations. In one observational study of patients receiving 100% inspired oxygen via a tracheal tube during CPR, a higher measured partial pressure of arterial oxygen (PaO_2) value during CPR was associated with ROSC and hospital admission.[60] The worse outcomes associated with a low PaO_2 during CPR could, however, be an indication of illness severity.

After ROSC, as soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94–98%.[2] Avoid hypoxaemia, which is also harmful – ensure reliable measurement of arterial oxygen saturation before reducing the inspired oxygen concentration.[61] This is addressed in the Post-resuscitation care section.

Ventilation

Provide artificial ventilation as soon as possible in any patient in whom spontaneous ventilation is inadequate or absent. Expired air ventilation (rescue breathing) is effective but the rescuer's expired oxygen concentration is only 16–17%, so it must be replaced as soon as possible by ventilation with oxygen-enriched air. A pocket resuscitation mask enables mouth-to-mask ventilation and some enable supplemental oxygen to be given. Use a two-hand technique to maximise the seal with the patient's face. A self-inflating bag can be connected to a face mask, tracheal tube, or SGA. The two-person technique for bag-mask ventilation is preferable. Deliver each breath over approximately 1 second and give a volume that corresponds to normal chest movement; this represents a compromise between giving an adequate volume, minimising the risk of gastric inflation, and allowing adequate time for chest compression. During CPR with an unprotected airway, give two ventilations after each sequence of 30 chest compressions. Once a tracheal tube or SGA has been inserted, ventilate the lungs at a rate of about 10 breaths min^{-1} and continue chest compression without pausing during ventilation.[2,5]

Alternative airway devices

The tracheal tube has generally been considered the optimal method of managing the airway during cardiac arrest.[62] There is evidence that, without adequate training and experience, the incidence of complications, such as unrecognised oesophageal intubation (2.4–17% in several studies involving paramedics)[63-67] and dislodgement, is unacceptably high.[68] Prolonged attempts at tracheal intubation are harmful; the cessation of chest compressions during this time will compromise coronary and cerebral perfusion. Several alternative airway devices have been used for airway management during CPR.

There are published studies on the use during CPR of the Combitube, the classic laryngeal mask airway (cLMA), the Laryngeal Tube (LT) and the i-gel, and the LMA Supreme (LMAS) but none of these studies has been powered adequately to enable survival to be studied as a primary endpoint. Instead, most researchers have studied insertion and ventilation success rates. The SGAs are easier to insert than a tracheal tube and,[69] unlike tracheal intubation, can generally be inserted without interrupting chest compressions.[70]

Laryngeal mask airway (LMA)

An LMA is relatively easy to insert, and ventilation using an LMA is more efficient and easier than with a bag-mask. If gas leakage is excessive, chest compression will have to be interrupted to enable ventilation. Although an LMA does not protect the airway as reliably as a tracheal tube, pulmonary aspiration is uncommon when using an LMA during cardiac arrest. The original LMA (classic LMA [cLMA]) has been superseded by several second generation SGAs that have more favourable characteristics, particularly when used for emergency airway management.[71]

I-gel

The cuff of the i-gel does not require inflation; the stem of the i-gel incorporates a bite block and a narrow oesophageal drain tube. It is very easy to insert, requiring only minimal training and a laryngeal seal pressure of 20–24 cmH_2O can be achieved.[72,73] The ease of insertion of the i-gel and its favourable leak pressure make it theoretically very attractive as a resuscitation airway device for those inexperienced in tracheal intubation. In observational studies insertion success rates for the i-gel were 93% (n = 98) when used by paramedics for out-of-hospital cardiac arrest (OHCA)[74] and 99% (n=100) when used by doctors and nurses for in-hospital cardiac arrest (IHCA).[75] The i-gel is in widespread use in the UK for both IHCA and OHCA.

LMA Supreme (LMAS)

The LMAS is a disposable version of the Proseal LMA, which is used in anaesthetic practice. In an observational study, paramedics inserted the LMAS successfully and were able to ventilate the lungs of 33 (100%) cases of OHCA.[76]

Tracheal intubation

Tracheal intubation should be attempted only by trained personnel able to carry out the procedure with a high level of skill and confidence. No intubation attempt should interrupt chest compressions for more than 5 seconds. Use an alternative airway technique if tracheal intubation is not possible.

Healthcare personnel who undertake prehospital intubation should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills. Rescuers must weigh the risks and benefits of intubation against the need to provide effective chest compressions. The intubation attempt may require some interruption of chest compressions but, once an advanced airway is in place, ventilation will not require interruption of chest compressions. Personnel skilled in advanced airway management should be able to undertake laryngoscopy without stopping chest compressions; a brief pause in chest compressions will be required only as the tube is passed through the vocal cords. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until ROSC;[77,78] this strategy is being studied in a large prehospital randomised trial.[79] The intubation attempt should interrupt chest compressions for less than 5 seconds; if intubation is not achievable within these constraints, recommence bag-mask ventilation. After intubation, tube placement must be confirmed and the tube secured adequately.

Videolaryngoscopy

Videolaryngoscopes are being used increasingly in anaesthetic and critical care practice.[80,81] In comparison with direct laryngoscopy, they enable a better view of the larynx and improve the success rate of intubation. Preliminary studies indicate that use of videolaryngoscopes improve laryngeal view and intubation success rates during CPR [82-84] but further data are required before recommendations can be made for wider use during CPR.

Confirmation of correct placement of the tracheal tube

The Resuscitation Council (UK) recommends using waveform capnography to confirm and continuously monitor the position of a tracheal tube during CPR in addition to clinical assessment. End-tidal CO_2 detectors that include a waveform graphical display (capnographs) are the most reliable for verification of tracheal tube position during cardiac arrest.[2,5]

Clinical assessment includes observation of chest expansion bilaterally, auscultation over the lung fields bilaterally in the axillae (breath sounds should be equal and adequate) and over the epigastrium (breath sounds should not be heard). Clinical signs of correct tube placement alone (condensation in the tube, chest rise, breath sounds on auscultation of lungs, and inability to hear gas entering the stomach) are not reliable. The reported sensitivity (proportion of tracheal intubations correctly identified) and specificity (proportion of oesophageal intubations correctly identified) of clinical assessment varies: sensitivity 7–100%; specificity 66–

100%.[85-89]

Based on the available data, the accuracy of colormetric CO_2 detectors, oesophageal detector devices and non-waveform capnometers does not exceed the accuracy of auscultation and direct visualisation for confirming the tracheal position of a tube in victims of cardiac arrest. Waveform capnography is the most sensitive and specific way to confirm and continuously monitor the position of a tracheal tube in victims of cardiac arrest and must supplement clinical assessment (auscultation and visualisation of tube through cords). Waveform capnography will not discriminate between tracheal and bronchial placement of the tube – careful auscultation is essential. Existing portable monitors make capnographic initial confirmation and continuous monitoring of tracheal tube position feasible in almost all settings, including out-of-hospital, emergency department and in-hospital locations where intubation is performed.

Cricothyroidotomy

If it is impossible to ventilate an apnoeic patient with a bag-mask, or to pass a tracheal tube or alternative airway device, delivery of oxygen through a cannula or surgical cricothyroidotomy may be life saving. A tracheostomy is contraindicated in an emergency, as it is time consuming, hazardous and requires considerable surgical skill and equipment.

Surgical cricothyroidotomy provides a definitive airway that can be used to ventilate the patient's lungs until semi-elective intubation or tracheostomy is performed. Needle cricothyroidotomy is a much more temporary procedure providing only short-term oxygenation. It requires a wide-bore, non-kinking cannula, a high-pressure oxygen source, runs the risk of barotrauma and can be particularly ineffective in patients with chest trauma. It is also prone to failure because of kinking of the cannula, and is unsuitable for patient transfer. In the 4th National Audit Project of the UK Royal College of Anaesthetists and the Difficult Airway Society (NAP4), 60% of needle cricothyroidotomies attempted failed.[90] In contrast, all surgical cricothyroidotomies achieved access to the trachea. While there may be several underlying causes, these results indicate a need for more training in surgical cricothyroidotomy and this should include regular manikin-based training using locally available equipment.[91]

Drugs for cardiac arrest

The ILCOR systematic reviews found insufficient evidence to comment on critical outcomes such as survival to discharge and survival to discharge with good neurological outcome with any drug during CPR.[2] There was also insufficient evidence to comment on the best time to give drugs to optimise outcome.

Thus,although drugs are still included among ALS interventions, they are ofsecondary importance to high quality uninterrupted chest compressions and early defibrillation.

Adrenaline

Despite the continued widespread use of adrenaline during resuscitation, there is no placebo-controlled study that shows that the routine use of adrenaline during human cardiac arrest increases survival to hospital discharge, although improved short-term survival has been documented.[92-94]

The current recommendation is to continue the use of adrenaline during CPR as for Guidelines 2010. We have considered the benefit in short-term outcomes (ROSC and admission to hospital) and our uncertainty about the benefit or harm on survival to discharge and neurological outcome given the limitations of the observational studies.[2,95,96]

The Resuscitation Council (UK) has decided not to recommend a change to current practice until there are high quality data on long-term outcomes. Dose response and placebo-controlled efficacy trials are needed to evaluate the use of adrenaline in cardiac arrest. There is an ongoing randomised study of adrenaline vs. placebo for OHCA in the UK (PARAMEDIC 2: The Adrenaline Trial, ISRCTN73485024).

Amiodarone

No anti-arrhythmic drug given during human cardiac arrest has been shown to increase survival to hospital discharge, although amiodarone has been shown to increase survival to hospital admission.[97,98] Despite the lack of human long-term outcome data, the balance of evidence is in favour of the use anti-arrhythmic drugs for the management of arrhythmias in cardiac arrest. There is an ongoing trial comparing amiodarone to lidocaine and to placebo designed and powered to evaluate for functional survival.[6]

Vascular access during CPR

The role of drugs during cardiac arrest is uncertain. Some patients will already have intravenous access before they have a cardiac arrest. If this is not the case ensure CPR had started and defibrillation, if appropriate, attempted before considering vascular access.

Peripheral versus central venous drug delivery

Although peak drug concentrations are higher and circulation times are shorter when drugs are injected into a central venous catheter compared with a peripheral cannula,[99] insertion of a central venous catheter requires interruption of CPR and can be technically challenging and associated with complications. Peripheral venous cannulation is quicker, easier to perform and safer. Drugs injected peripherally must be followed by a flush of at least 20 mL of fluid and elevation of the extremity for 10–20 seconds to facilitate drug delivery to the central circulation.

Intraosseous route

If intravenous access is difficult or impossible, consider the intraosseous (IO) route. This is now established as an effective route in adults.[100-108] Intraosseous injection of drugs achieves adequate plasma concentrations in a time comparable with injection through a vein.[109,110] Animal studies suggest that adrenaline reaches a higher concentration and more quickly when it is given intravenously as compared with the intraosseous route, and that the sternal intraosseous route more closely approaches the pharmacokinetics of IV adrenaline.[111] The recent availability of mechanical IO devices has increased the ease of performing this technique.[112] There are several intraosseous devices available as well as a choice of insertion sites including the humerus, proximal or distal tibia, and sternum. The decision concerning choice of device and insertion site should be made locally and staff adequately trained in its use.

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7. CPR techniques and devices

Mechanical chest compression devices

We recommend that automated mechanical chest compression devices are not used routinely to replace manual chest compressions.

Automated mechanical chest compression devices are a reasonable alternative to high quality manual chest compressions in situations where sustained high quality manual chest compressions are impractical or compromise provider safety.[2]

Interruptions to CPR during device deployment should be avoided. Healthcare personnel who use mechanical CPR should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills.

Since Guidelines 2010 there have been three large RCTs enrolling 7582 patients that have shown no clear advantage for the routine use of automated mechanical chest compression for OHCA using the Lund University Cardiac Arrest System (LUCAS)[113,114] and AutoPulse devices.[115] Ensuring high quality chest compressions with adequate depth, rate and minimal interruptions, regardless of whether they are delivered by machine or human is important.[116,117] Mechanical compressions usually follow a period of manual compressions;[118] the transition from manual compressions to mechanical compressions whilst minimising interruptions to chest compression and avoiding delays in defibrillation is therefore an important aspect of using these devices. The use of training drills and 'pit-crew' techniques for device deployment are suggested to help minimise interruptions in chest compression.[119-121]

Extracorporeal cardiopulmonary resuscitation (ECPR)

Extracorporeal CPR (ECPR) should be considered as a rescue therapy for those patients in whom initial ALS measures are unsuccessful and, or to facilitate specific interventions (e.g. coronary angiography and percutaneous coronary intervention (PCI) or pulmonary thrombectomy for massive pulmonary embolism).[122,123] There is an urgent need for randomised studies of ECPR and large ECPR registries to identify the circumstances in which it works best, establish guidelines for its use and identify the benefits, costs and risks of ECPR.[124,125]

Extracorporeal techniques require vascular access and a circuit with a pump and oxygenator and can provide a circulation of oxygenated blood to restore tissue perfusion. This has the potential to buy time for restoration of an adequate spontaneous circulation, and treatment of reversible underlying conditions. This is commonly called extracorporeal life support (ECLS), and more specifically extracorporeal CPR (ECPR) when used during cardiac arrest. These techniques are becoming more commonplace and have been used for both in-hospital and out-of-hospital cardiac arrest despite limited observational data in select patient groups. Observational studies suggest ECPR for cardiac arrest is associated with improved survival when there is a reversible cause for cardiac arrest (e.g. myocardial infarction, pulmonary embolism, severe hypothermia, poisoning), there is little comorbidity, the cardiac arrest is witnessed, the individual receives immediate high quality CPR, and ECPR is implemented early (e.g. within 1 hour of collapse) including when instituted by emergency physicians and intensivists.[126-132]

The implementation of ECPR requires considerable resource and training. When compared with manual or

mechanical CPR, ECPR has been associated with improved survival after IHCA in selected patients.[126,128] After OHCA outcomes with both standard and ECPR are less favourable.[133] The duration of standard CPR before ECPR is established and patient selection are important factors for success.[122,126,130,132,134-136]

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8. Duration of resuscitation attempt

If attempts at obtaining ROSC are unsuccessful the resuscitation team leader should discuss stopping CPR with the team. The decision to stop CPR requires clinical judgement and a careful assessment of the likelihood of achieving ROSC. If it was considered appropriate to start resuscitation, it is usually considered worthwhile continuing, as long as the patient remains in VF/pVT, or there is a potentially reversible cause than can be treated. The use of mechanical compression devices and extracorporeal CPR techniques make prolonged attempts at resuscitation feasible in selected patients. It is generally accepted that asystole for more than 20 minutes in the absence of a reversible cause and with ongoing ALS constitutes a reasonable ground for stopping further resuscitation attempts.[137]

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9. Acknowledgements

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Jasmeet Soar, Jerry P. Nolan, Bernd W. Böttiger, Gavin D. Perkins, Carsten Lott, Pierre Carli, Tommaso Pellis, Claudio Sandroni, Markus B. Skrifvars, Gary B. Smith, Kjetil Sunde, Charles D. Deakin.

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ALS Recertification Course

ALS recertification course learning outcomes

- Standardised CPR for adults
- Update on clinical changes to resuscitation guidelines
- Re-evaluation of knowledge and practical skills acquisition
- Assessment

ALS recertification course format

- Manual
- Lectures
- Skill stations
- Cardiac Arrest Simulation (CAS) training

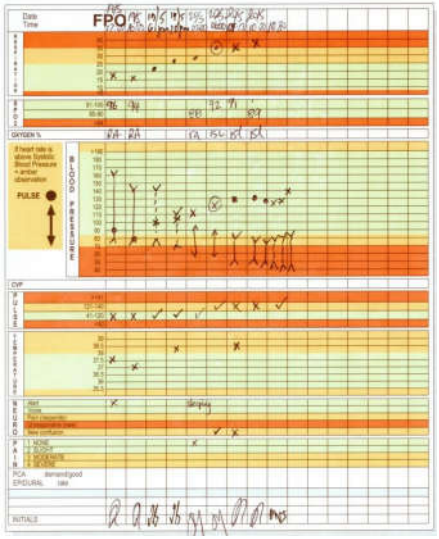
ALS recertification course assessment

- MCQ
- Practical skills (continuous assessment)
  - Airway management
  - Initial assessment and resuscitation
- Cardiac Arrest Simulation (CASTest)
- Provider certificate valid for 4 years

Causes and Prevention of Cardiac Arrest

Early recognition of the deteriorating patient

- Most arrests are predictable
- Deterioration prior to 50 - 80% of cardiac arrests
- Hypoxia and hypotension are common antecedents
- Delays in referral to higher levels of care



Outcome after in-hospital cardiac arrest (UK)

	VF/VT	Non-VF/VT
Number of patients	570 (18%)	2,614 (82%)
ROSC > 20 min	385 (68%)	689 (26%)
Survival to hospital discharge	251 (44%)	179 (7%)

- Source: National Cardiac Arrest Audit (NCAA) 2010
- Based on 3,184 **adults** (aged ≥ 16 y) in 61 hospitals participating in NCAA (increasing numbers of hospitals during Oct 2009 to Oct 2010) with known presenting/first documented rhythm and complete data for ROSC and survival to hospital discharge. All these individuals received chest compressions and/or defibrillation from the resuscitation team in response to a 2222 call. Many in-hospital cardiac arrests do not fulfil these criteria and are not included here.
- For full definitions, see NCAA Dataset Specification

ncaa

Supported by Resuscitation Council (UK) and ICNARC

Recognition of the deteriorating patient - Early Warning Scoring Systems

Score	3	2	1	0	1	2	3
Pulse (min^{-1})		≤ 40	41-50	51-90	91-110	111-130	≥ 131
Respiratory rate (min^{-1})	≤ 8		9-11	12-20		21-24	≥ 25
Temperature (°C)	≤ 35.0		35.1 – 36.0	36.1 – 38.0	38.1-39.0	≥ 39.1	
Systolic BP (mmHg)	≤ 90	91-100	101-110	111-249	≥ 250		
Oxygen saturation (%)	≤ 91	92-93	94-95	≥ 96			
Inspired oxygen				Air			Any oxygen therapy
AVPU				Alert (A)			Voice (V) Pain (P) Unresponsive (U)

Example of early warning scoring (EWS) system*

* From Prytherch et al. ViEWS—Towards a national early warning score for detecting adult in-patient deterioration. Resuscitation. 2010;81(8):932-7

Recognition of the deteriorating patient - Early Warning Scoring Systems

EWS	Minimal observation frequency	Escalation	
		Recorder's action	Doctor's action
3-5	4 hourly	Inform nurse in charge	
6	4 hourly	Inform doctor	Doctor to see within 1 h
7-8	1 hourly	Inform doctor Consider continuous monitoring	Doctor to see within 30 min and discuss with senior doctor and/or outreach team
≥9	30 min	Inform doctor Start continuous monitoring	Doctor to see within 15 min and discuss with senior doctor and ICU team

Example Escalation Protocol based on early warning score (EWS)

The ABCDE approach to the deteriorating patient

Airway

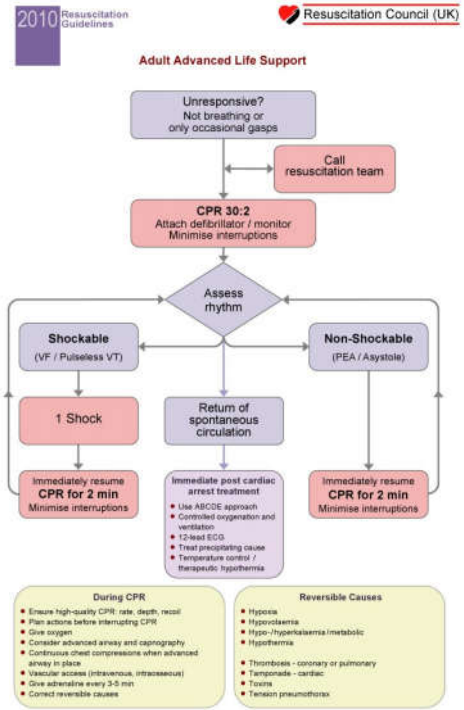
Breathing

Circulatio
n

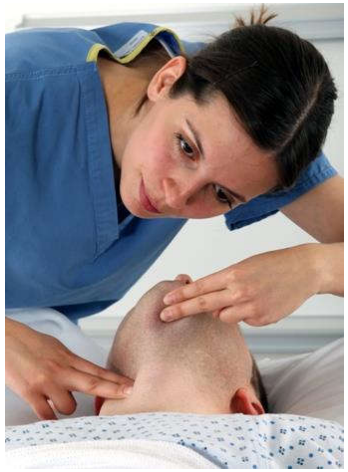
Disability

Exposure

ALS Algorithm



Unresponsive?
Not breathing or
only occasional gasps



To confirm cardiac arrest…

- Patient response
- Open airway
- Check for normal breathing
  - Caution agonal breathing
- Check circulation
- Monitoring

Unresponsive?
Not breathing or
only occasional gasps

Call
resuscitation team

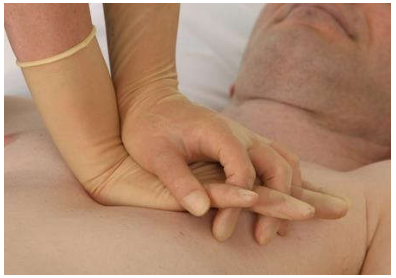
Cardiac arrest confirmed

Unresponsive?
Not breathing or
only occasional gasps

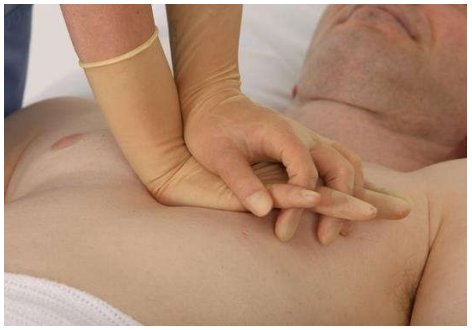
Call
resuscitation team

CPR 30:2
Attach defibrillator / monitor
Minimise interruptions

Cardiac arrest confirmed



Chest compression



- 30:2
- Compressions
  - Centre of chest
  - 5-6 cm depth
  - 2 per second (100-120 min^{-1})
- Maintain high quality compressions with minimal interruptions
- Continuous compressions once airway secured
- Switch CPR provider every 2 min cycle to avoid fatigue

START

PAUSE

CPR

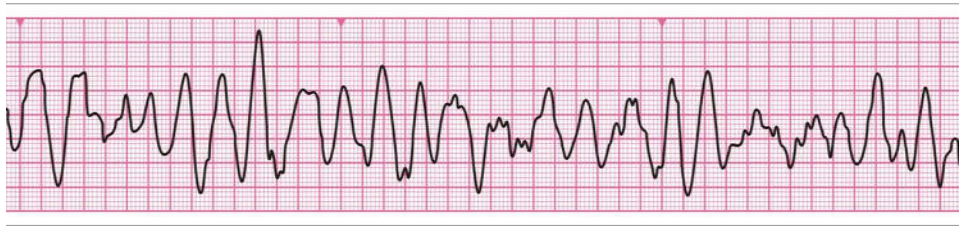
Assess rhythm

Shockable
(VF / Pulseless VT)

Non-Shockable
(PEA / Asystole)

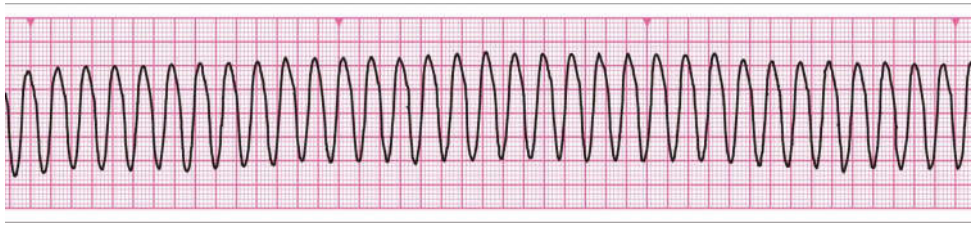
MINIMISE INTERRUPTIONS IN CHEST COMPRESSIONS

Shockable
(VF)



- Bizarre irregular waveform
- No recognisable QRS complexes
- Random frequency and amplitude
- Uncoordinated electrical activity
- Coarse/fine
- Exclude artefact
  - Movement
  - Electrical interference

Shockable
(VT)

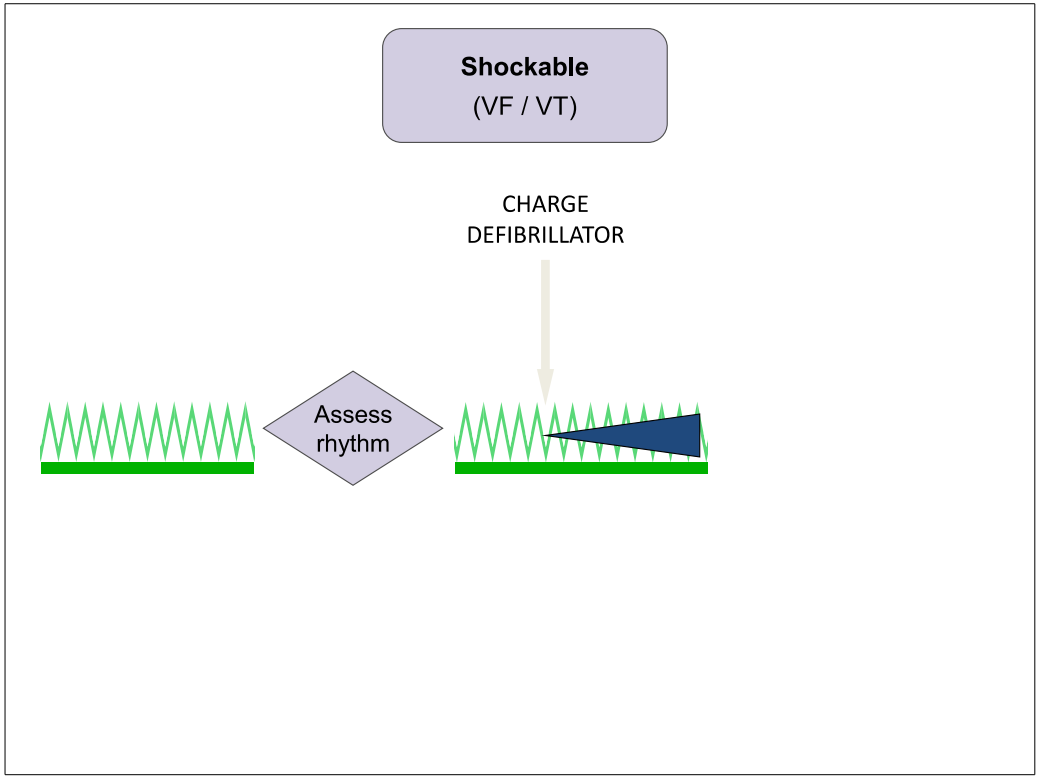


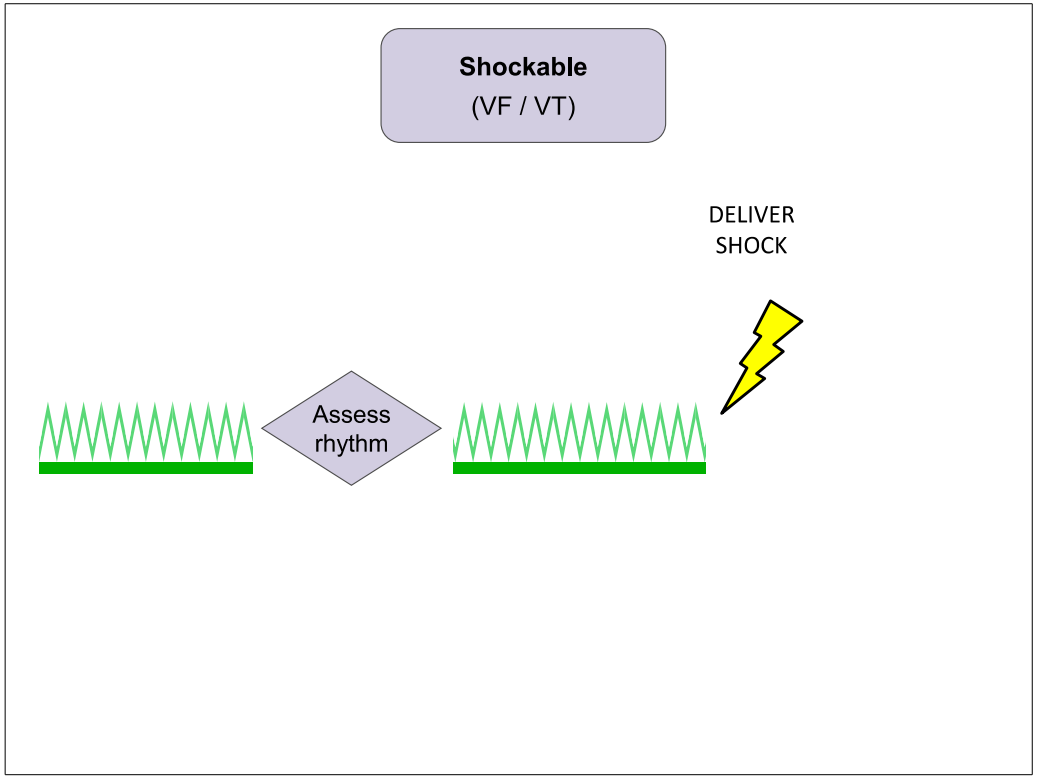
- Monomorphic VT
  - Broad complex rhythm
  - Rapid rate
  - Constant QRS morphology
- Polymorphic VT
  - Torsade de pointes

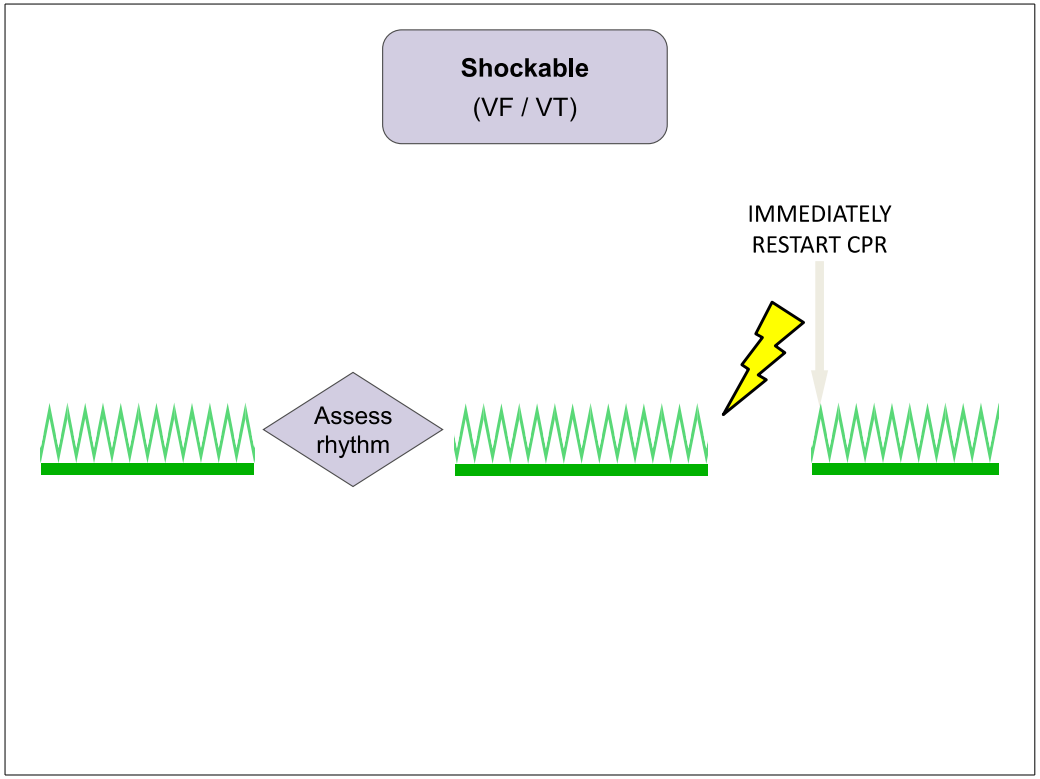
Shockable
(VF / VT)

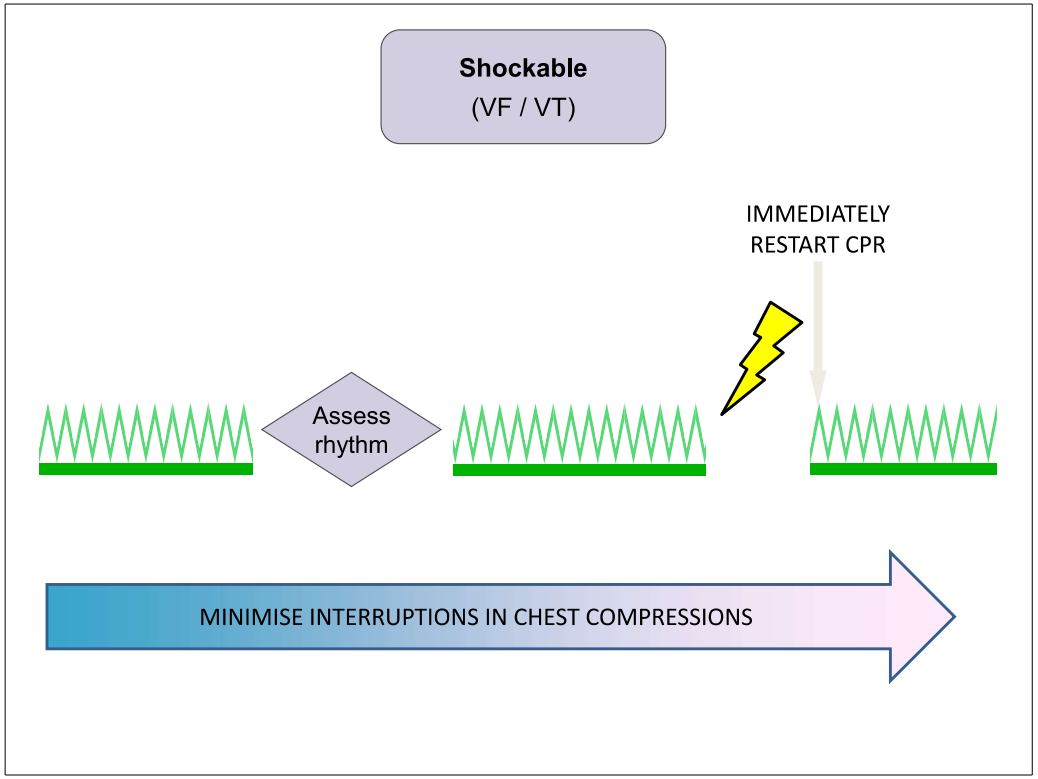
RESTART
CPR

Assess rhythm


Shockable
(VF / VT)
CHARGE
DEFIBRILLATOR
Assess
rhythm


Shockable
(VF / VT)
DELIVER
SHOCK
Assess
rhythm

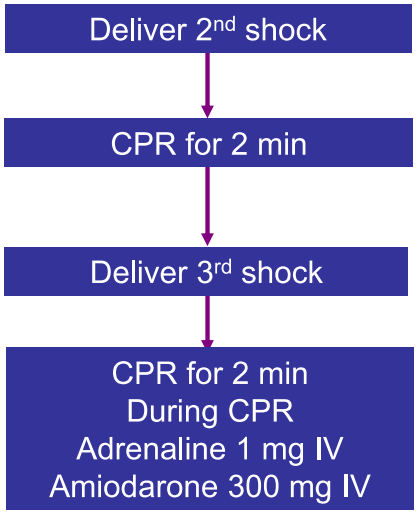

Shockable
(VF / VT)
IMMEDIATELY
RESTART CPR
Assess
rhythm


Shockable
(VF / VT)
IMMEDIATELY
RESTART CPR
Assess
rhythm
MINIMISE INTERRUPTIONS IN CHEST COMPRESSIONS

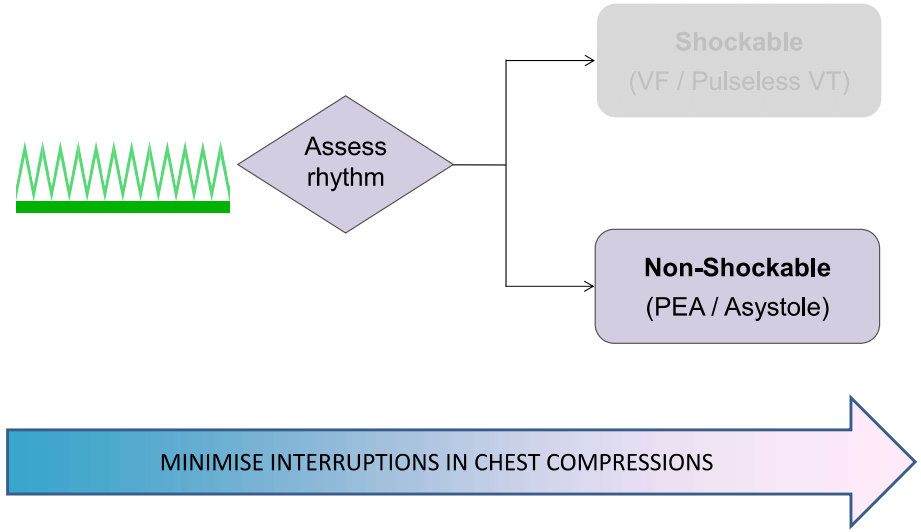
Defibrillation energies

- Vary with manufacturer
- Check local equipment
- If unsure, deliver highest available energy
- DO NOT DELAY SHOCK
- Energy levels for defibrillators on this course…

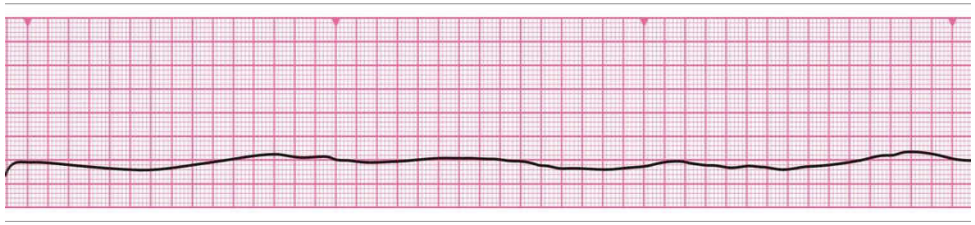
If VF / VT persists



- 2nd and subsequent shocks
  - 150 – 360 J biphasic
  - 360 J monophasic
- Give adrenaline and amiodarone after 3rd shock during CPR



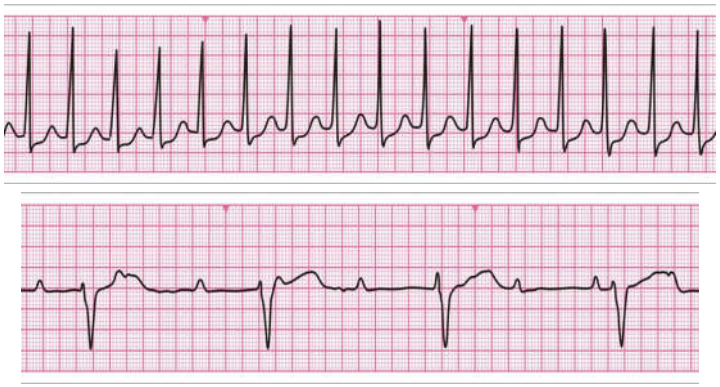
Non-Shockable
(Asystole)



- Absent ventricular (QRS) activity
- Atrial activity (P waves) may persist
- Rarely a straight line trace

- Adrenaline 1 mg IV then every 3-5 min

Non-Shockable
(PEA)



- Clinical features of cardiac arrest
- ECG normally associated with an output
- Adrenaline 1 mg IV then every 3-5 min

During CPR

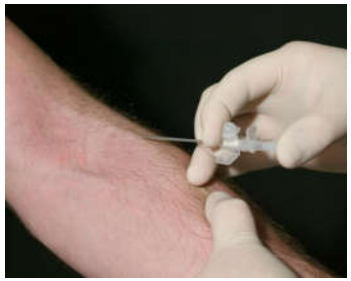
- Ensure high-quality CPR: rate, depth, recoil
- Plan actions before interrupting CPR
- Give oxygen
- Consider advanced airway and capnography
- Continuous chest compressions when advanced airway in place
- Vascular access (intravenous, intraosseous)
- Give adrenaline every 3-5 min
- Correct reversible causes

Airway and ventilation

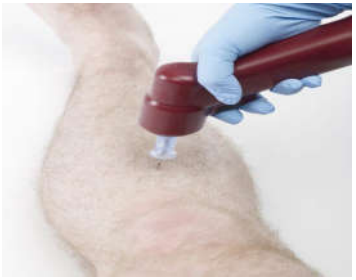
- Secure airway:
  - Supraglottic airway device e.g. LMA, i-gel
  - Tracheal tube
- Do not attempt intubation unless trained and competent to do so
- Once airway secured, if possible, do not interrupt chest compressions for ventilation
- Avoid hyperventilation
- Capnography

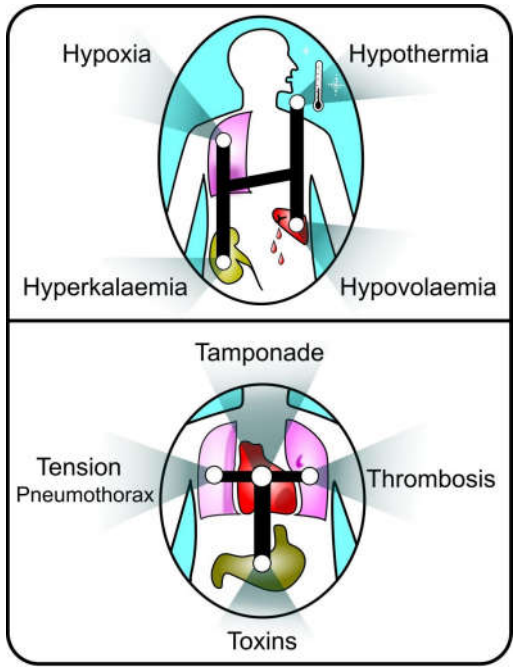
Vascular access

- Peripheral versus central veins



- Intraosseous





Hypoxia

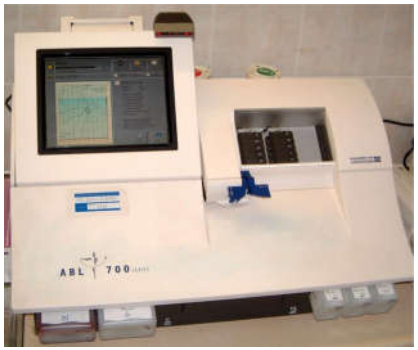
- Ensure patent airway
- Give high-flow supplemental oxygen
- Avoid hyperventilation

Hypovolaemia

- Seek evidence of hypovolaemia
  - History
  - Examination
    - Internal haemorrhage
    - External haemorrhage
    - Check surgical drains
- Control haemorrhage
- If hypovolaemia suspected give intravenous fluids

Hypo/hyperkalaemia and metabolic disorders

- Near patient testing for K^+ and glucose
- Check latest laboratory results
- Hyperkalaemia
  - Calcium chloride
  - Insulin/dextrose
- Hypokalaemia/ Hypomagnesaemia
  - Electrolyte supplementation

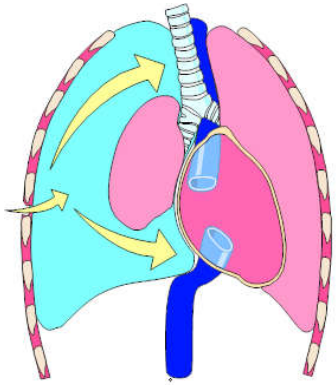


Hypothermia

- Rare if patient is an in-patient
- Use low reading thermometer
- Treat with active rewarming techniques
- Consider cardiopulmonary bypass

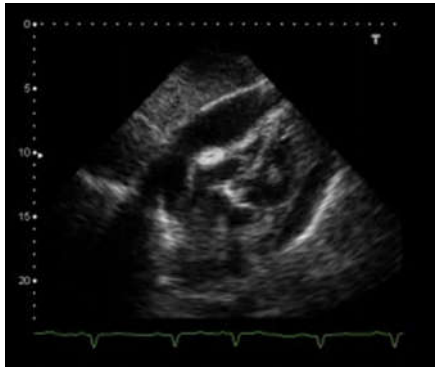
Tension pneumothorax

- Check tube position if intubated
- Clinical signs
  - Decreased breath sounds
  - Hyper-resonant percussion note
  - Tracheal deviation
- Initial treatment with needle decompression or thoracostomy



Tamponade, cardiac

- Difficult to diagnose without echocardiography
- Consider if penetrating chest trauma or after cardiac surgery
- Treat with needle pericardiocentesis or resuscitative thoracotomy

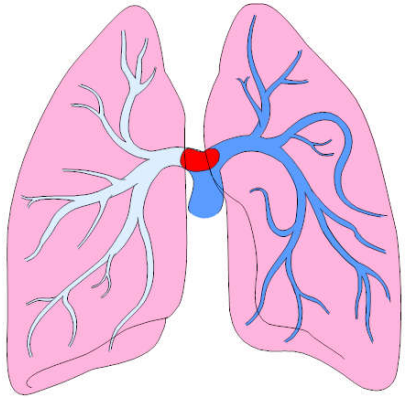


Toxins

- Rare unless evidence of deliberate overdose
- Review drug chart

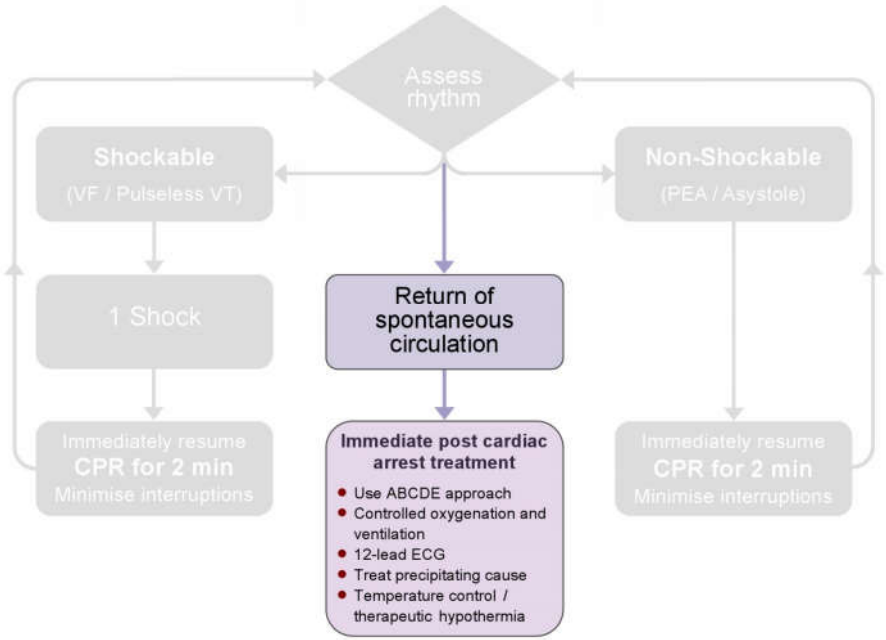
Thrombosis

- If high clinical probability for PE consider fibrinolytic therapy
- If fibrinolytic therapy given continue CPR for up to 60-90 min before discontinuing resuscitation



Ultrasound

- In skilled hands may identify reversible causes
- Obtain images during rhythm checks
- Do not interrupt CPR



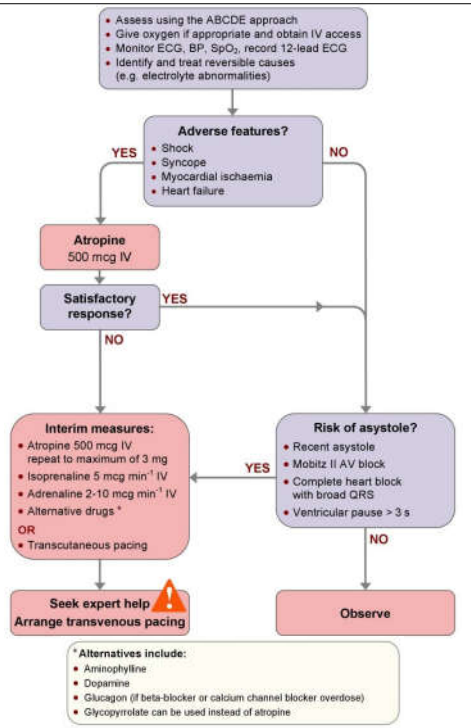
Resuscitation team

- Roles planned in advance
- Identify team leader
- Importance of non-technical skills
  - Task management
  - Team working
  - Situational awareness
  - Decision making
- Structured communication

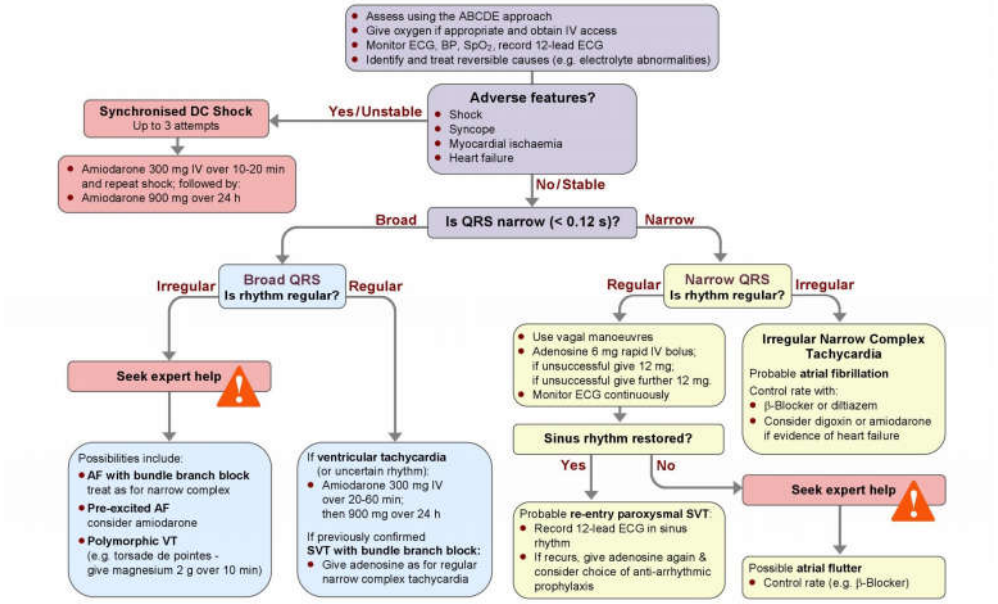
Peri-Arrest

Bradycardia algorithm

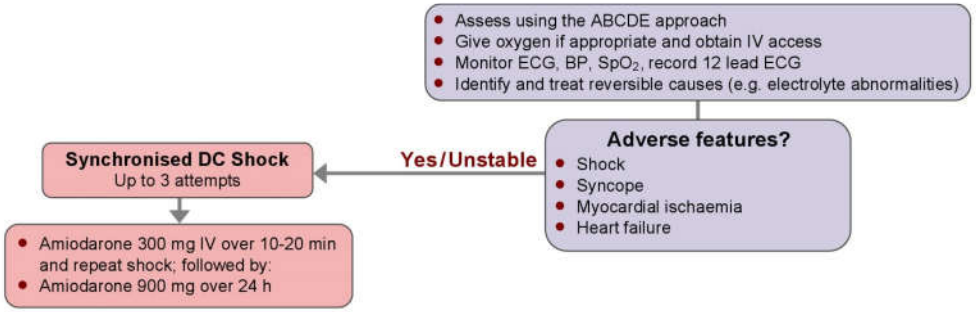
Includes rates inappropriately slow for haemodynamic state



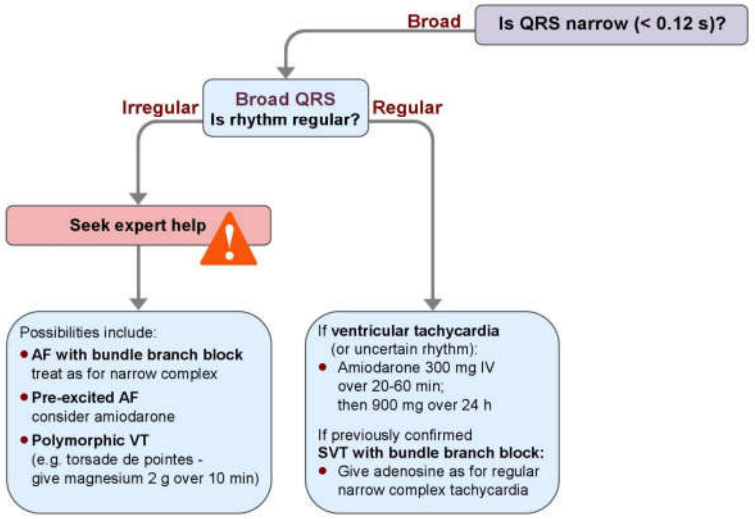
Tachycardia algorithm (with pulse)



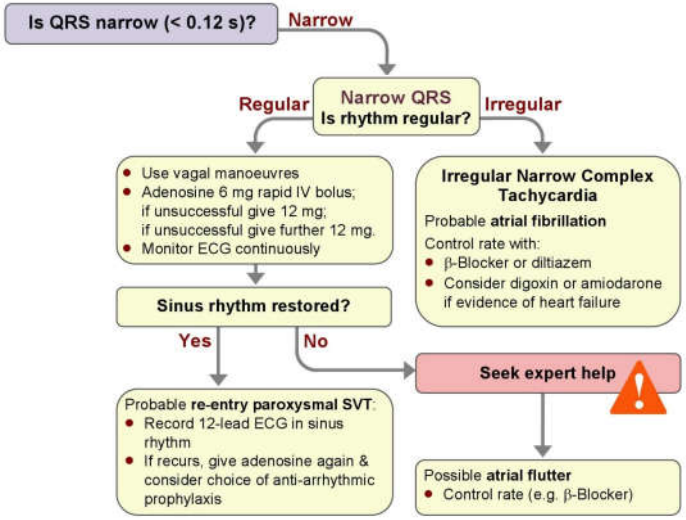
Tachycardia algorithm



Stable broad-complex tachycardia



Stable narrow-complex tachycardia



Summary

- Modifications to ALS are based upon current evidence
- Focus is on standardised CPR for adults

Lecture @ 2 pm

Thursday 1st week

Introduction to Anaesthesia

"Understanding the Concept"

By Dr Ibraheem QUDAISAT
Assist. Proff.
Anaesthesia dept.
Faculty of Medicine
Jordan University

Vocabulary "Jargon" in Anesthesia:

Consciousness:

- *Can perhaps be described as* :

"Our continuing stream of Awareness of either our surroundings or our sequential thoughts"

Anaesthesia
(*American*, **Anesthesia**):
==========

*__Anaesthesia: Loss of feeling or Sensation__

**Latin terminology*: An ====➔ No,
aesthesia ➔ Sensation

ANESTHESIA
- Partial or complete loss of sensation
- with or with out loss of consciousness
- as result of disease, injury, or administration of an anesthetic agent, usually by injection or inhalation

* Can be effected:
- locally :(***local anesthesia***),
- to regions of the body :(***Regional Anesthesia***),
- or Generally :(***general Anesthesia)***

*Sensation of stimuli can be blocked with the patient either conscious ***(Local and Regional Anesthesia)*** or unconscious (***General Anesthesia***).

Hypnosis

"The state of being asleep and consequently unaware of the surrounding"

To the anesthetist it technically implies:

- There is deprivation of critical faculties induced by "hypnotism".

* It is pharmacologically induced to a level at which the patient cannot be roused to consciousness by physical stimuli, but can still react unconsciously by withdrawal or autonomic reflexes if not deep enough to block nociception, or not given sufficient analgesia as well.

Narcosis

* A state of stupor produced by drugs (i.e. it is pharmacologically induced).

- It is more accurate than Hypnosis.
- It is confusingly used for Morphine-like drugs of addiction
  - i.e. They take them for their euphoric action and not to get stuperous.

Sedation

- Sedation : May be used vaguely to imply anything from allaying anxiety to inducing near natural sleep with drugs, by depressing the highest critical cerebral centers of the brain.

Pain vs. Nociception

- The word "Painful Stimulus" should be restricted to conscious patients who are aware of the pain.
- Under General Anesthesia, the word **"Nociceptive stimulus"** is better used.
- A Nociceptive stimulus will cause pain in the conscious patient and reflex response (*e.g. Tachycardia*) in the unconscious one.

Analgesia

- "The state of freedom from pain"
- Can be effected locally, and the patient is still conscious "e.g. by use of local Anesthetics".
- Can be generalized and patient still coconscious
  - Systemic Analgesic Drugs.
  - "stage-one general Anesthesia"…… (*see below)*
- Can be part of deep general Anesthesia.

*****So, Anesthesia and analgesia are not interchangeable words!***

Anxiolysis

- A reduction in anxiety (Fear, apprehension, and stress due to awareness of an impending unpleasant experience).
- Sedation will lead to Anxiolysis, but Anxiolysis can be effected by certain non-sedative drugs (e.g. Tranquilizers).

Tranquillizers and antidepressants

- ***Tranquillizers:*** Drugs which acts at a lower level of the central nervous system than the cerebral cortex to produce a calming effect.*(Major: Neuroleptic or Antipsychotics) &(Minor: anxiolytics)*
- ***Antidepressants***: drugs that alter the mood and mental reactions of patients.

Important Notes !!

*** All hypnotic sedatives, tranquilizers and antidepressants in large doses will cause loss of consciousness, respiratory depression and abolition of the protective reflexes.

- The difference between drugs commonly used for sedation and those used for intravenous induction of general anesthesia is the therapeutic margin.

Muscle Relaxation

" Rendering the muscles less tense by decreasing their tone, or even paralyzing them ".

- Can be obtained in different ways:
    - **By Central depression of the nervous system.**
    - **By local anesthesia of peripheral nerves.**
    - **By blocking the neuromuscular junction.**

*** Any drug which causes a muscle to relax could be called a Muscle Relaxant, but in anaesthesia practice this term is almost exclusively reserved for the group of intravenously administered drugs which block the chemical transmission of a nerve impulse at the Neuromuscular Junction leading to muscle paralysis.*

General Anesthesia

General Anesthesia clinically implies that

the patient has <u>been rendered reversibly unconscious by DRUGS</u>

for the execution of a painful operative or diagnostic test.

Subdivisions of General Anesthesia

Are based on <u>the route</u> by which the drug is introduced into the body and thence via the blood stream to the brain:

- *Intravenous*
- *Inhalational*
- *Intramuscular*
- *Rectal*

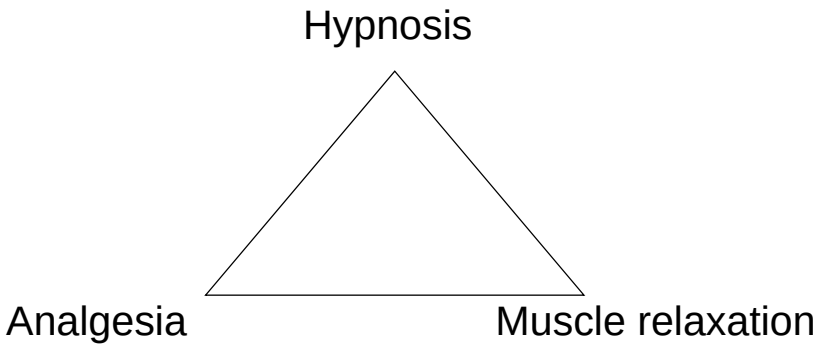
<u>Modern "Balanced" General Anesthesia</u>

- An altered physiologic state, characterized by Reversible loss of Consciousness, analgesia of the entire body, amnesia, and some degree of Muscle Relaxation.
- This is brought about by different groups of drugs that has different specific actions:
  - **<u>Hypnotic drugs</u> for effecting Loss of Consciousness.**
  - **<u>Analgesic Drugs</u> for effecting analgesia.**
  - **<u>Muscle relaxant</u> Drugs for effecting muscle paralysis.**

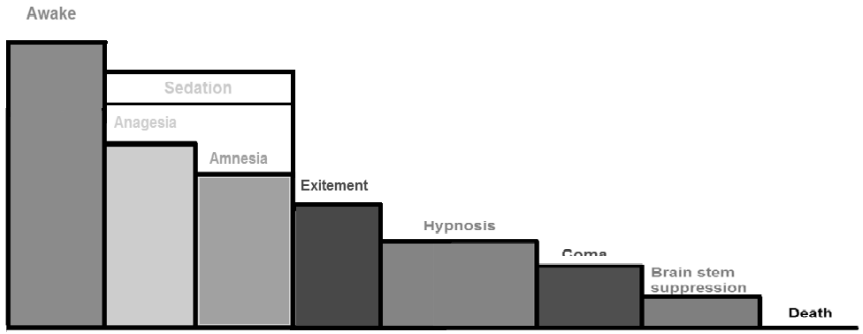
==========

This is in comparison with "Old Anesthesia" where all of the above actions were effected by a single agent (e.g. Ether) at high concentrations with higher incidence of side effects.

Triad of General anaesthesia



- ***General Anesthesia*** *is <u>not</u> a single, all-or-non state of altered Consciousness; rather,*
  - *It is a continuum of alteration of Consciousness, brought about by increasing plasma and CNS levels of the anesthetic agent.*
  - *This continuum can be classified into stages characterized by cumulative development of different clinical states of consciousness.*



<u>Stages of anesthesia</u>

STAGE 1: (Stage of concious sedation)*:*

- Low anesthetic Concentrations in CNS→ Decrease activity of neurons in cells of Substantia Gelatinosa in Spinal Cord → interruption of the function of Spinothalamic tract → some degree of Analgesia.
- *<u>Clinically:</u>* The patient initially experiences analgesia without amnesia. However, later in stage 1 both analgesia and amnesia ensue.

<u>Stages of anesthesia</u>

2- Stage of Excitement:

- At Higher brain concentrations of anesthetic drug:
  - complex neuronal actions take place, including blockade of many small inhibitory neurons such as Golgi type II cells, together with a paradoxic facilitation of excitatory neurotransmitters.
- *<u>Clinically:</u>*

During this stage the patient appears to be delirious and excited but definitely is amnesic.

Also:

- **Respiration is irregular in rate and volume.**
- **Retching and vomiting may occur.**
- **Incontinence and struggling sometimes occur.**

➢ **Regular breathing marks the end of this stage.**

Stages of anesthesia

3- *Stage of Surgical Anesthesia:*

- With increasing Anesthetic concentration, there will be a progressive depression of pathways in the reticular activating system, together with suppression of spinal reflex activity that contributes to muscle relaxation.
- *Clinically:*
  It begins with the recurrence of regular breathing and extends to complete cessation of spontaneous respiration.

** Four planes of stage III have been defined, representing increasing depth of anesthesia:

Plane 1: From the return of regular respirations to the cessation of REM.

Plane 2 :The Surgical Plane: from the cessation of REM to the onset of paresis of the intercostal muscles.

Plane 3 :From the onset to the complete paralysis of the intercostal muscles.

Plane 4: From the paralysis of the intercostal muscles to the paralysis of the diaphragm - at the end of this plane the patient will be apneic.

Stages of anesthesia

4- *Stage of Medullary depression:*

- At high CNS drug concentrations, the activity of neurons in the respiratory and vaso-motor centers in the brain stem - which are relatively insensitive to anesthetic drugs – is depressed, leading to Cardio-Respiratory Collapse.

** Full Respiratory and Circulatory support are a must, Otherwise, Coma and Death will ensue.

Stages of anesthesia

- The stages of General Anesthesia used to be clinically distinct in old anesthesia because of slow onset time of older drugs (Ether, etc…).
- In Modern Anesthesia, the distinctive signs of each of the 4 stages are obscured due to:
  - Use of drugs with rapid onset of action.
  - Use of means of mechanical ventilation.
  - Pre or intra-operative use of drugs that influence the signs of anesthesia.

Scope of Anesthesia

- Work In Every Area Of Medicine
  - OR, PACU, ICU, OB, Peds, Pain Clinic
- Work With The Most Diverse Patient Population
  - Premature Infants To Geriatrics
- Provide Medical Care & Critical Care
  - Prior To, During, And After Surgical Procedures
- Work With Advanced Technology

You Might Like Anesthesia If…

- You Enjoy Performing Procedures
- You Are Interested In Critical Care
- You Enjoyed:
  - Pharm, Physio, Cardiology, Pulmonology
- You Like All Areas Of Medicine
  - You Can Specialize Though
- You Like To See Immediate Results

Role of the Anesthetist

- Anesthetist is the perioperative physician.
- Provides medical care to each patient:
  - Pre-operative evaluation.
  - Patient counceling and informed consent
  - Consultation with surgical team
  - Providing pain control
  - Supporting life functions during surgery.
  - Supervising immediate post-operative care

Scope of anesthesia

I. Anesthesia for surgical procedures in the operating theatres.

I. Anesthesia and sedation in remote areas
   - ✓ *Day case surgery*
   - ✓ *Radiology*
   - ✓ *Endoscopy*
   - ✓ *Dental*
   - ✓ *Shockwave Lithotripsy*
   - ✓ *Cardioversion*
   - ✓ *Electroconvulsive therapy*

II. Cardiopulmonary resuscitation

I. Labour analgesia

I. Pain management: Acute and chronic

Brief History

- Pre-1800: Surgery Without Anesthesia
- 1846: First Ether Anesthetic
- 1847: Chloroform was introduced by James Simpson.
- 1884: Cocaine For Local Anesthesia
- 1910s-1930s: Endotracheal Intubation.
- 1921: Epidural Anesthesia Described
- 1943: Curare Clinical Trial Success (Montreal)
- Mid 1950s: halothane
- 1960: Short Acting Opioids
- 1982: Transesophageal Echo
- 1983: Laryngeal Mask Airway

Ether Day – October 16th, 1846

Before anesthesia:…

..... Surgery was a terrifying last resort in a final attempt to save life.

Dr D J Wilkinson, past Honorary Treasurer and Archivist, Association of Anaesthetists of Great Britain and Ireland

Liston, an eminent surgeon, was once operating for a bladder stone.

The panic stricken patient finally broke loose from the brawny assistants, ran out of the room, down the hall and locked himself in the lavatory.

Liston, hot on his heels and a determined man, broke down the door and carried the screaming patient back to complete the operative procedure.

(Rapier HR. Man against Pain London 1947;49).

Anaesthesia is now very safe, with mortality of less than 1 in 250,000 directly related to anaesthesia.

==========*cf.*==========

The global mortality rate due to traffic accidents
was 19 per 100 000 population (1:5263)

Thank You

THE UNIVERSITY OF JORDAN

Preoperative Assessment& Evaluation& Pre-medications

Objectives

- Determine pt medical status by

(1) Proper history

(2) Physical exam

(3) Indicated Lab. Investigations

(4) Review medical records

(5) Consider if needed further testing or consults to develop anesthesia plan.

- Perform proper airways examination
- ASA Classification: Perform adequate preoperative psychological & pharmacological preparation for adult and pediatric patients.
- Evaluation of patients with known systemic disease

- HTN - DM

-Thyroid disease -Cardiac disease

- Pulmonary disorder.

Objectives

- Identify ASA fasting guidelines
- Identify patients at risk of peri-op. aspiration
- Anti cholinergic premedication
- Perioperative corticosteroid coverage
- DVT / PE prophylaxis
- Antibiotics prophylaxis

History (Know your patient)

- Age / Weight/ Type of surgery/ fasting hours
- Past history

_ Anesthetic Hx : any complications (difficult airways/delayed emergence/PONV) , family hx. (scoline apnea)
_ Surgical / Medical history (DM, HTN , thyroid)

- Focused review of systems

Neuro

Epilepsy -
CVA/TIA -
Denervation disease-

Respiratory

Asthma
COPD
OSA
Recent URTI/LRTI
Cough/ sputum
Smoking

Cardio

IHD (CP /Angina/ stent)
CHF (PNDs/ orthopnea)
Exercise intolerance
Palpitations

Cont…

GIT	**Renal**	
GERD PUD Hiatus Hernia Intestinal obstruction.	CRF ARF On dialysis	Blood disorders Antiplatelet Anticoagulation

- Medications (*e.g.* B-blockers, statins)
- Allergy (*e.g.* penicillin)

Physical Examination

- Vital signs
- General appearance
- Cardiac exam

Check rate and rhythm
Auscultate heart sounds

- Respiratory exam

Look for signs of resp. distress
Respiratory rate
Auscultate lung sounds

- Neuro exam

Mental status
Gross motor/ gross sensory

Airway Examination

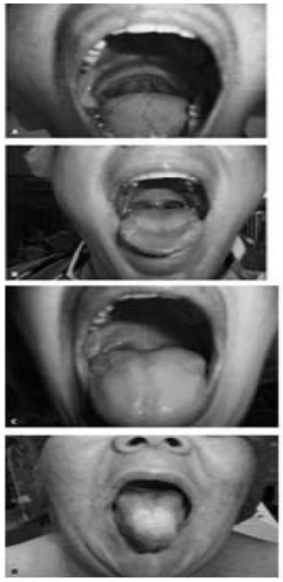
(1)Mallampatti classification

It categorizes the ratio of tongue size to the oropahrynx

Has low positive predictive value

Mallampatti Classification

class	Structures identified when pt seated
1	Tonsilar pillars, uvula , soft & hard palate
2	Uvula ,soft & hard palate
3	Base of uvula ,soft & hard palate
4	Only hard palate is can be seen



Airway exam

(2) Mouth opening

(3)Teeth (prominent upper incisors/ loose or mobile)

(4)Palate (high arched)

(5) Ability to protrude the lower jaw beyond the upper incisors (jaw protrusion)

Airway exam

(6) Neck exam

Look for short or thick neck

Look for neck movements

Look for neck masses ⇨ tracheal shift

(7) Three distances

Tyro-mental distance

-It describes the distance between the mentum & thyroid notch
-It helps in determining how readily the laryngeal axis will fall in line with the pharyngeal axis
-It is normally in adults > 6cm

#Sterno- mental distance

-It describes the distance between the mentum & suprasternal notch
-If this distance less than 12 cm it predicts difficult intubation

#Interincisor distance

-It describes the distance between the upper and lower incisors
-It is normally 4.5 cm

Method of Assessment (L.E.M.O.N)

L Look externally

teeth /tongue / face / mouth opening

E Evaluate the three distances

interincisor / thyromental / thyrosternal distance

M Mallampatti score (3 or 4)

O Obstruction (presence of any obstruction like peri-tonsillar abscess , thyroid mass , VC nodule)

N Neck mobility

Lab Investigations

- Blood tests
- CXR
- ECG
- PFT

Lab Investigations

- CBC — Advanced age/ Anemic pt/ Bleeding /chronic disease (kidney liver heart)
- KFT — Diabetics/ HTN/ chronic disease / on medications like diuretics , digoxin ,ACEI
- Sugar — Diabetics / HTN/ chronic disease / on steroid
- LFT — Liver disease / malnourished pt
- Coagulation studies — Bleeding disorder/ Kidney disease/ Liver disease/ pt on anticoagulants

Cont…

☆CXR

Indicated in patients with respiratory or cardiac disease

Indicated in smokers

Indicated in patients with recent LRTI

☆ECG

Indicated in patients with respiratory or cardiac disease

Advanced Age (M: 55y F: 65y)

Any patient with CAD risk factors

(HTN, DM, Hyperlipidemia , exercise intolerance)

Pulmonary Function test

_ Identifying patients at risk, evaluating the risk, and finding modified factors to decrease risk

Guidelines don't support the routine use of PFT.

(1) Indicated in obstructive lung disorders

(2) Indicated in restrictive lung disorders

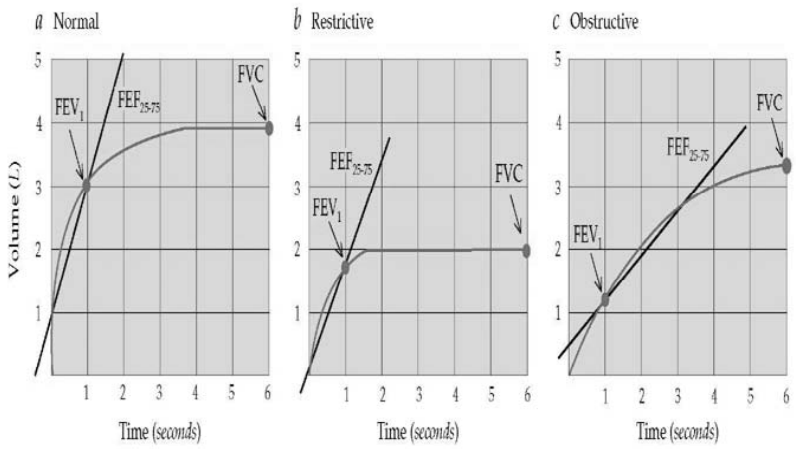
(3) Indicated in neuromuscular disorders

_ Includes mainly

Spirometry

ABGs

Pulmonary Function test



Major surgery

- Defined as highly invasive surgery commonly needs

Blood transfusion

Invasive monitor

Post op. ICU

Categrory	Health status	Comment
ASA 1	Healthy	
ASA 2	Mild systemic disease	Has a well-controlled disease of one body system; cigarette smoking ; mild obesity, pregnancy
ASA 3	Severe systemic disease	Some functional limitation; has a controlled disease of more than one body system or one major system
ASA 4	Severe systemic disease that is constant threat to life	Has at least one severe disease that is poorly controlled or at end stage; possible risk of death
ASA 5	Moribund patients who are not expected to survive without the operation	Not expected to survive > 24 hours without surgery; imminent risk of death
ASA 6	A declared brain-dead patient whose organs are being removed for donor purposes	

Preoperative preparation in adults

- It includes preop. Visit with informative and comforting interview about OR events , anesthesia steps & all patient concerns like fear of death .loss of consciousness which would replace many grams of antidepressants .
- Take your time before the operation to earn the trust and confidence of the patient.
- premedication to achieve sedation & amnesia in selected pts.
- Orally given before 60 min, on the other hand I.V given before few minutes.

Benzodiazepine

- They produce anxiolysis, amnesia and sedation.
- They have little depression on ventilatory and circulatory systems in premedication doses
- Low incidence of toxicity (wide therapeutic index)
- Lack of opioids side effects (nausea & vomiting)

Midazolam

- It is water soluble with rapid metabolism
- Onset 1-2 mins
- Dose 1-2 mg IV given prior to the trip to OR
- Mental function return to normal within 1-4 hours
- Better than lorazepam , diazepam Why??

Rapid onset/// Rapid elimination // rapid clearance

Preoperative preparation of pediatrics

- Age is the most important aspect when psychological preparation is considered.
- A baby younger than 8 months has no separation anxiety so preparation is often directed toward educating the parents.
- Toddlers(1-2) & preschool (3-5) will become upset when separated, and its so difficult to explain for them OR events
- This age group is good candidate for premedication.
- Consider your visit as chance to connect with the child by becoming familiar with his/her toys & games to gain trust .
- It may be helpful for the child to have their parents accompany to the OR after explaining events of induction.

Preoperative preparation of pediatrics

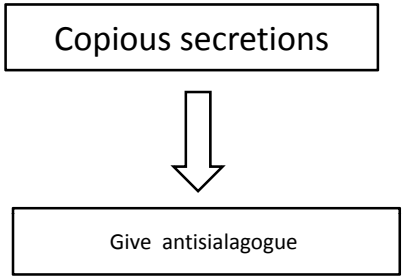
- The goal is to reduce apprehension, produce sedation & amnesia.
- Premedication is not used for children before 8 months.
- Preferred route is oral (older children) or rectal (preschool) esp. if there is no IV access.
- Avoid IM route as you can.
- Premedication use in pediatric patients is controversial ???
- (1) Premedication has failure rate of 20 %
- (2) Premedication hasn't proved to reduce psychological outcome
- (3) Smooth induction is less likely to produce long lasting psychological problems.

Preoperative preparation of pediatrics

- The most commonly used is oral midazolam
- Dose 0.5 - 0.75 mg/kg
- Cherry flavored with bitter after taste
- It produce sedation but not sleep
- Onset within 15 minutes
- Can be given intranasally

Preoperative preparation of pediatrics

- The second commonly used is ketamine
- Route include oral rectal & IM
- Given 30 minutes before induction
- Dose (5-10 mg/kg)
- The disadvantage of ketamine use
-



- Evaluation of patients with known systemic disease

- HTN

- DM

-Thyroid disease

-Cardiac disease

- Pulmonary disorder.

Hypertension

HTN has been divided into three stages

Stage 1	140-159	90-99
Stage 2	160-179	100-109
Stage 3	More than 180	More than 110

Hypertension

- HTN may be associated with

CAD

ECG changes suggesting chronic ischemia

Uncontrolled BP is associated with increased risk of perioperative myocardial infarction and cardiac arrhythmia mainly A fib

Delay or Don't delay

- Delay the surgery if

If SBP >180

IF DBP >110

Delay esp. if there is end organ damage in heart (CAD or LVH) or in the kidney (CRI or border line Cr)

Diabetes

- DM is a disease of

Microvascular
Retinal vessel
Renal arterioles
Neurons

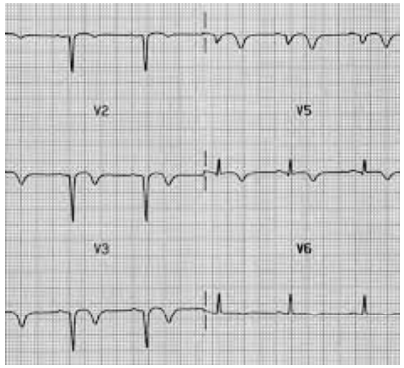
Macrovascular
Brain
Heart
Peripheral vessels

- DM is associated with CAD////…. ECG should be done for all diabetics WHY???

- Answer

Because they are at higher risk of silent MI than non diabetics

Seen on ECG as Q waves

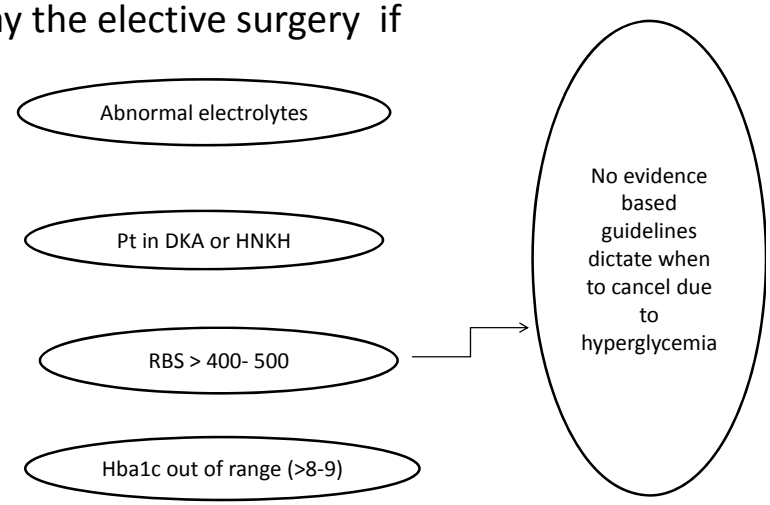


Diabetes Evaluation

- (1) through HX and exam focusing on end organ damage
- (2) compliance to medication
- (3) documentation of sugar readings
- (4) ECG
- (5) KFT , Sugar ,HbA1c

When to delay????

- Delay the elective surgery if



Goals of delay

<u>Why we do focus on preoperative glycemic control????</u>

- (1) Reduce infection rate
- (2) Improve wound healing
- (3) improves postoperative outcomes in term of end organ functions// heart, brain//
- (4) decrease length of stay in hospital or ICU
- (5) Avoid complicated postoperative course of DKA or metabolic derangement.

Diabetes Perioperative Recommendations

(1)Oral hypoglycemic are held on the day of surgery

(2)Discontinue metformin 48 hours preop.

(3) Continue insulin through the evening before the surgery

(4) Check blood sugar on arrival to holding area

(5) Plan the surgery as the first case on schedule

Cont….

(6) For type 1 DM administer half the dose of long acting and intermediate insulin, but hold rapid acting or short acting insulin

(7) Intraoperative glycemic control is needed and the goal is (110-200)

Mix 100 IU actrapid with 100 cc N/S and titrate your infusion accordingly

Thyroid disease

- Look for signs & symptoms of hypothyroidism or hyperthyroidism
- Ask about stridor (upper airway obstruction)
- Ask about medications and compliance
- Look for thyroid masses with possible tracheal shift

Thyroid disease

Why in anesthesia we are concerned about thyroid status???

Hypothyroidism

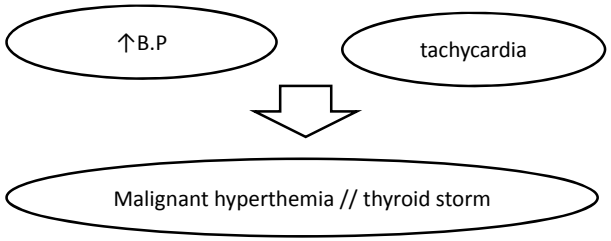
Hypoventilation
Hypoglycemia
Hypothermia
hyponatremia

Hyperthyroidism

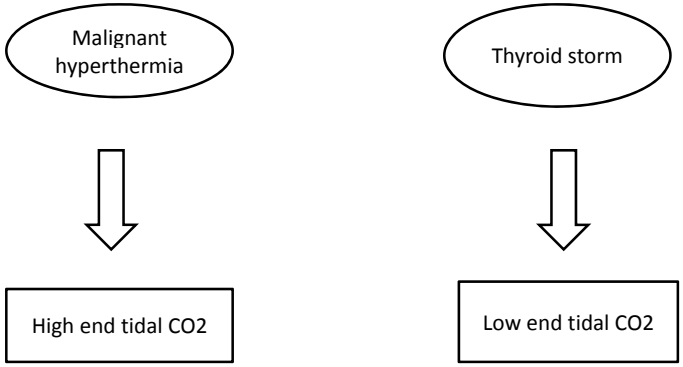
Risk of thyroid storm

Thyroid storm

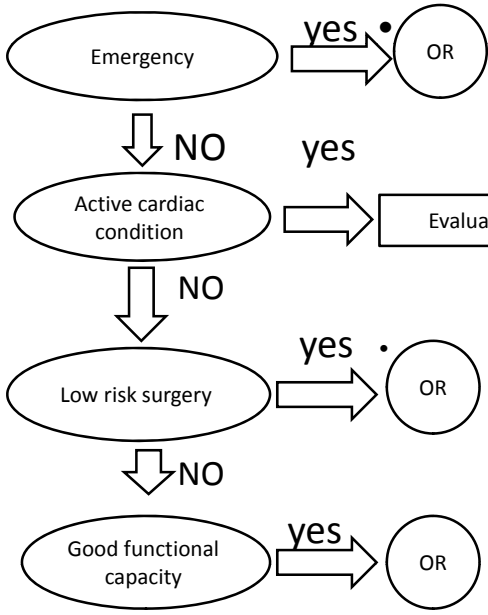
- Hypermetabolic state due to sudden release of T3 or T4 or both.
- Clinically manifested with fever agitation tachycardia , shock ,heart failure
- Intra operatively there is only

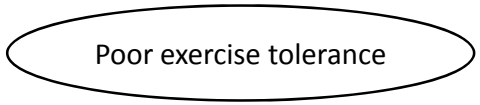


How to differentiate ???

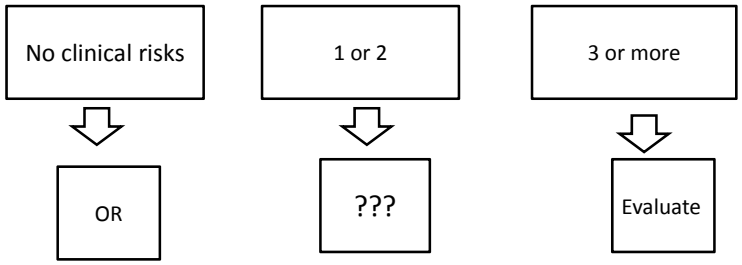


Cardiac Evaluation For Non-Cardiac Surgery





- The decision now depends on presence of clinical risk factors



Active cardiac conditions

- Unstable angina
- Decompensated heart failure
- Significant or new onset arrhythmia
- Severe valvular disease
- Myocardial infarction in the last one month

Types of Non Cardiac surgery

- Low risk surgery(<1%)

Includes superficial , endoscopic , breast surgery

- Intermediate risk surgery (1-5 %)

Includes intraperitoneal , intrathoracic , Head& neck, major ortho. surgery

- High risk surgery (> 5%)

Includes major vessles :Abdominal Aorta
Carotids

Clinical Risk factors

- DM
- Renal impairment
- CHF
- CVA history
- IHD history

Respiratory disease

- Perioperative complication includes

1-Pneumonia
2-Aspiration
3-Pulmonary Embolism
4-Atelectasis
5-Bronchspasm
6-COPD exacerbation
7-Respiratory failure may need mechanical ventilation

Respiratory disease

- These postoperative risks increase with

Upper abdominal surgery// Thoracic surgery
Emergency surgery// duration of surgery

Preexisting diseases COPD // OSA // Asthma // Smoking

Respiratory disease

- To minimize respiratory complications

1- Address preexisting respiratory problems with assessment of - type - severity - reversibility
2- Epidural analgesia
3- DVT prophylaxis
4- Reduce bacterial contamination during endotracheal insertion

Smoking

- Studies showed that smoke cessation for at least 4 to 8 weeks was necessary to reduce post operative complications

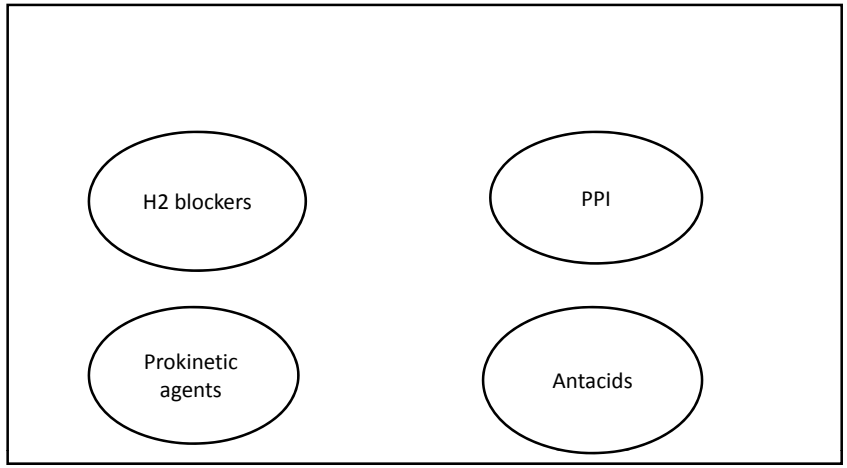
- Airway of smokers showed increased reactivity under GA
- Premedicate with B2 agonist bronchodilator at the morning of surgery.

- Identify ASA fasting guidelines
- Identify patients at risk of peri-op. aspiration
- Anti cholinergic premedication
- Perioperative corticosteroid coverage
- DVT / PE prophylaxis
- Antibiotics prophylaxis

ASA Fasting Guidelines

Clear fluid	**2 hours**	Water , Fruit juice without pulp,
Milk		
Human	**4 hours**	
Infant formula	**6 hours**	
Light Foods	**6 hours**	Fruits , juice with pulp, Vegetables
Heavy foods	**8 hours**	Fatty meals , meats

Perioperative Aspiration



Perioperative Aspiration

- ASA members found that the literature is insufficient to support the effect of any of the drug classes on reduction of incidence of emesis & pulmonary aspiration .
- Guidelines don't recommend routine use of them and limit their use for patients at risk .

Perioperative Aspiration

- Risk factors:
- Anesthesia Factors

  Light anesthesia

  Opioid use
- Patient Related

  Obesity , GERD, Hiatus Hernia, Pregnancy , Gastro paresis , difficult airway
- Surgery Related

  Emergency surgery , Intestinal obstruction

H2 Blockers

- Classes include Cimitidine, Ranitidine (Zantac), Famotidine.
- They block histamine receptor ability to induce acid secretion by proton pump.
- They reduce gastric fluid volume and acidity

Antacids

Given ½ an hour before induction

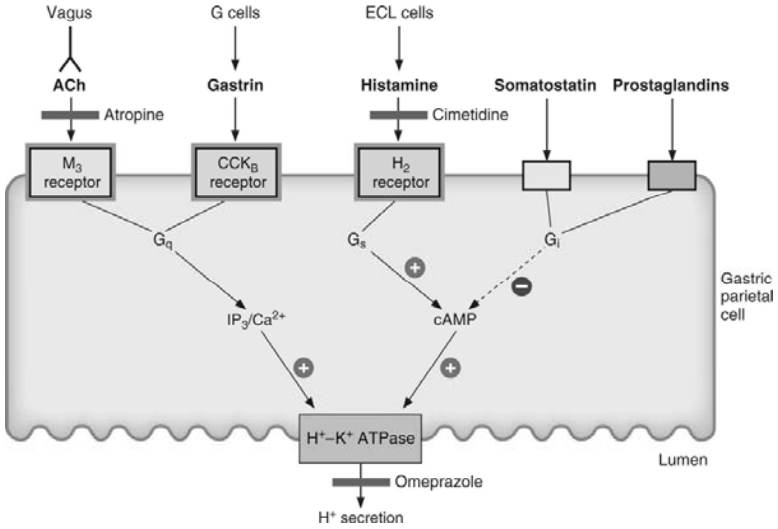
- Reduce gastric acidity only

PPI

-Omeprazole, the first drug in this class, lansoprazole , esomeprazole .
-Binds to H+ / K+ pump on parietal cell.
-Given 40 mg IV 30 min before surgery .
-Reduce both volume and acidity

Metoclopromide

-Act on dopamine receptors
-Increase gastric motility & LES tone
-Reduce gastric fluid volume only



Anticholinergic Premedication

(1) Antisialagogue effects

Was routinely used

Now indicated in awake fiberoptic intubation , intra oral surgeries. Bronchoscope (better visualization+ ????)

(2) Vagolytic effect

It blocks Ach effect on SA node

Used to prevent reflex bradycadia in

- ☆ Traction of viscera or extraocular muscles
- ☆ Carotid sinus stimulation
- ☆ Repetitive doses of succinylcholine

Cont….

	Atropine	Glycopyrrolate	Scopolamine
Antisialagouge	+	+++	+
Increased HR	+++	++	+
Sedation	+	0	+++

Gastric acid secretion & Anticholinergic

Perioperative Steroids

- Any patient taking corticosteroids for long period needs preoperative steroid supplement to cover stress of anesthesia & surgery. Esp, with higher doses & longer duration .
- Any patient on steroid ttt for at least one month needs coverage **WHY ???**

Because it is impossible to identify the **specific** duration or the **specific** dose at which Adrenocortical suppression

Perioperative steroids

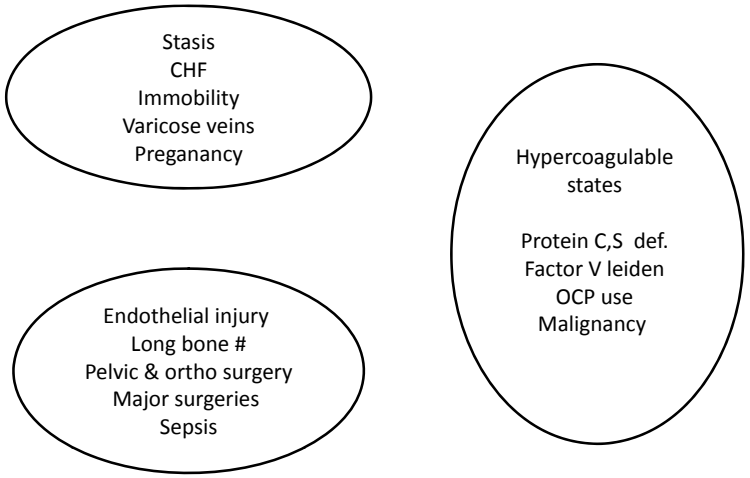
- Coverage depends on type of the surgery :

(1) Minor surgery :
On the morning of the dose 1.5 times his oral dose
No need for IV

(2) *Intermediate surgery* :
On the morning of the dose 2 times his oral dose
Give hydrocortisone (25mg/ 75mg/ 50 mg)

(3) *Major surgery*
On the morning of the dose 2 times his oral dose
Give hydrocortisone (50mg/ 100mg/ then 100 mg Q 8 hours for 1 day)

DVT / PE Prophylaxis



DVT /PE Prophylaxis

Unfractionated heparin (UH)

UH given 5000 IU SC should be given within two hours of operation

And then every 8 hours is probably more effective at preventing VTE with similar risk of major bleeding

Low molecular weight heparin (LMWH)

Start dosing the night before surgery with no other preoperative dosing to decrease the risk of operative bleeding

Dose depends on weight 1mg/kg once daily .

Antibiotics Prophylaxis

- Antibiotics should be given in coordination with the surgeon in contaminated - clean contaminated

Other indications

- Prevention of infective endocarditis
- Prevention of infection in immunocompromised pt.

Best time for administration is within 60 minutes before the surgery.

Two exceptions for this rule

(1)Vanco ⟹ before 2 hours

(2) Tourniquet ⟹ prior to inflation

Re-dosing concept in long surgeries (Cefazolin given every 4 hours)

Fluid Management and Blood Transfusion

Dr. Ahmed Shahin M.D

Outline

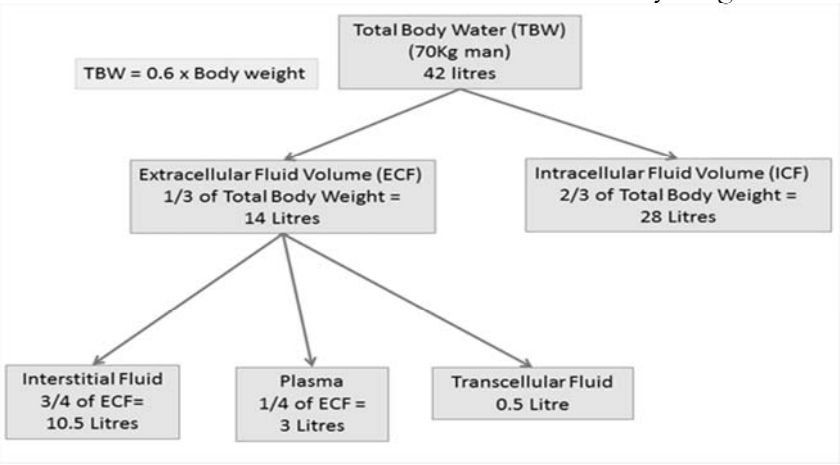
- Body Fluid Compartements
- Body Fluid Composition, Estimated Blood Volume, Allowable Blood Loss
- Osmolality and Tonicity
- Daily Intake and Output
- Types of Fluid
- Blood Transfusion

WATER

- Building material
- Solvent
- Reaction medium and reactant
- Carrier for nutrient and waste products
- Thermoregulation
- Lubricant and shock absorber

Body Fluid Compartments

- TBW: 55-60% of the BW in men and 45-50% in young women



Body Fluid Composition in Age Groups

AGE	TBW AS % OF TOTAL BODY WEIGHT
Neonate	80
6 months	70
1 year	60
Young adult	60
Elderly	50

WHAT IS THE 'NORMAL' DAILY INTAKE AND OUTPUT OF FLUID AND ELECTROLYTES?

INTPUT, OUTPUT

- Input: Oral, Enteral, Intravenous
- Output: 'Sensible': that it is easily seen and measured e.g. urine output and loses from the gastrointestinal tract.

'Insensible': not seen and not easy to quantify e.g. sweat, and water vapor in exhaled gases.

OUTPUT	
Urine	1500 ml
Gastrointestinal(faeces)	200 ml
Skin(sweat)	400 ml
Respiratory	400 ml
Total	2500
INTAKE	
Drinking	1500 ml
Eating	750 ml
Metabolism	250 ml
Total	2500

Maintenance and Deficit

- Maintenance Vs deficit
- Rule of 4 /2/ 1
- Ex. 70 kg patient

  1st 10 kg: 10 kg * 4 ml = 40 ml / kg

  2nd 10 kg: 10 kg * 2 ml = 20 ml / kg

  3rd 10 kg: 50 kg * 1 ml = 50 ml / kg Total=110ml/kg
- Ex. Fasting for 10 hr without any intake: 10*110= 1100ml

Ongoing losses and Blood Loss

- Calculate Anticipated Surgical Fluid Losses
  - Minimal tissue trauma (ex. herniorrhaphy): 0-2 cc/kg/hr
  - Moderate tissue trauma (ex. cholecystectomy): 4-6 cc/kg/hr
  - Severe tissue trauma (ex. bowel resection): 8-10 cc/kg/hr
- Ex. 70kg undergoing major laparotomy

  10 ml * 70 kg = 700 ml/hour as long as surgery is going on

- Measure Blood Losses
- Add up suction volume, lap pads (150 cc each if fully soaked) and 4x4 small pads (10 cc each if fully soaked)
- A common recommendation is to give 3 cc of crystalloid for every 1 cc of blood loss
- Ex. In the 1st hour of laparotomy there was 200ml of pure blood in the suction jar, 2 fully soaked lap pads, and 10 half soaked small pads
- 200 ml + 2 * 150 ml + 10 * 5 ml = 550 ml (in that hour) to be replaced with either 550 * 3 of cryst. or -if indicated- 550 ml of blood

ESSENTIAL PRINCIPLES

Osmolarity and Osmolality

- These are ways of quantifying how much of a solute is dissolved in a solution.

• ***Osmola(R)ity*** No. of osmoles of solute particles per unit VOLUME of solution and has units **osmoles/litre**. In the body we use **milliosmole**

• ***Osmola(L)ity*** No. of osmoles of solute particles per unit WEIGHT of solvent and has units **osmoles/kilogram**.

Plasma Osmolality

Plasma osmolality = 2 (Na + K) + glucose + urea

= 2 (137 + 4.0) + 5.0 + 4

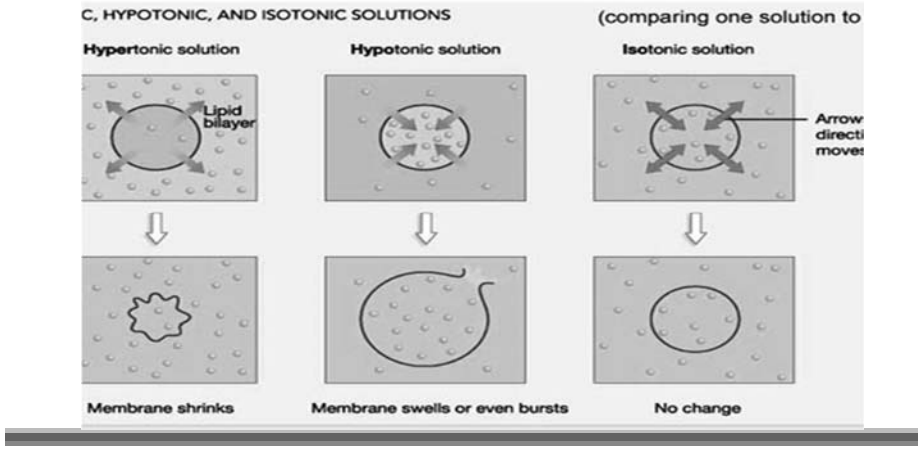
= 291 mosmol/kg H20

Glu: /18

Urea: /2.8

Tonicity

- A way of describing the relative solute concentrations of two solutions which are separated by a selectively-permeable membrane (often called a semi-permeable membrane).



Intravenous Fluids

Types

- Three main types
  - Crystalloids
  - Colloids
  - Blood products

- Solutions that contain a combination of water and electrolytes.
- Divided into "***balanced***" salt solutions (e.g. Ringer's lactate) and ***hypotonic*** solutions (e.g. D5W).

- Classified into three groups based on their predominant use
  - Replacement Solutions
  - Maintenance Solutions
  - Special Solutions

Replacement Solutions

- Used to replace ECF
- All isotonic, usually replace losses that involves both water and electrolytes
- Have a [Na+] similar to that of the ECF which effectively limits their fluid distribution to the ECF compartment.
- Distributes between the ISF ¾ and the plasma ¼ in proportion to their volumes

Ringer's Lactate (Hartman's)

- Na+ = 131 mmol/L
- Cl- = 111 mmol/L
- Lactate = 29 mmol/L
- K+ = 5 mmol/L
- Ca++ = 2 mmol/L
- PH = 6.5
- Osmolality = 279 mosm/L
- Potential problem = potassium may accumulate, lactate metabolism causes alkalosis

Maintenance Solutions

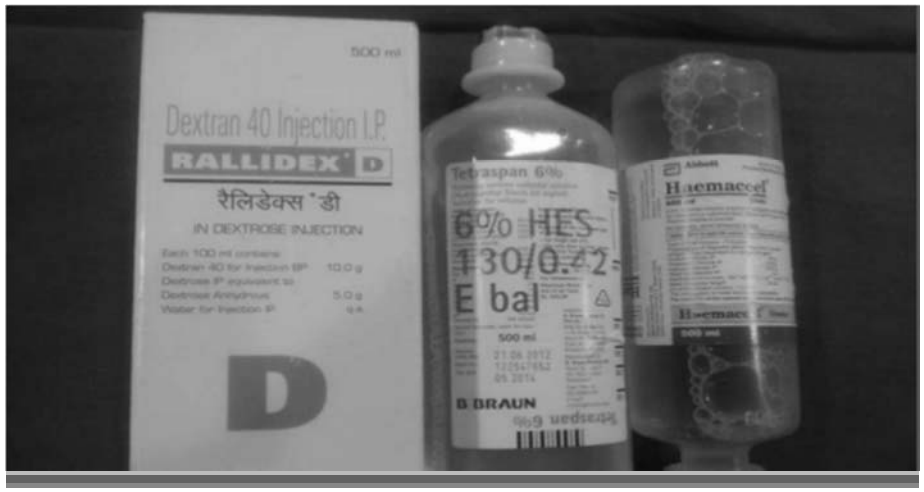
- Isosmotic as administered but not necessarily isotonic
- Usually used when the loss involves mainly pure water
- Ex. D5W, Normal Saline

Normal saline (0.9% saline solution)

- 9 g of NaCl/L water
- 154 mmol/L sodium
- 154 mmol/L chloride
- Osmolality = 308 mosm/L
- PH = 5.0
- Potential problem = hyperchloraemic metabolic acidosis, more likely with renal insufficiency

Special Solutions

- Hypertonic (3%) saline…. hyponatremia
  - 30 gm NaCl, 1027, 4.5 to 7.0
- Half normal saline…. hypernatremia
  - 77 meq/L
- 8.4% Bicarbonate solution… acidosis
- Mannitol 20%….brain oedema, pulmonary oedema



Albumin
Dextran
Gelatins
Starches

Colloids

- Colloid: a large molecule that does not diffuse across semipermeable membranes (capillary)
- Exerts an osmotic pressure in the blood, causing fluid to remain within the vascular system. The result is an increase in intravascular volume.

- Two categories of colloid may be defined:
  - Natural (e.g. human albumin)
  - Artificial (e.g. gelatins, dextran and hydroxyethyl starches [HES]).

Albumin

- Half-life ($t^{1}/_{2}$) = 1.6 hours in plasma
- Stays within the intravascular space unless the capillary permeability is abnormal
- 5% solution - isotonic; 10% and 25% solutions - hypertonic
- Expands volume 5x its own volume in 30 minutes
- Side effects - volume overload, fever (pyrogens in albumin), defects of haemostasis

PERIOPERATIVE BLOOD TRANSFUSION

Estimated Blood Volume (EBV)

- Men 75 ml / kg
- Women 65 ml / kg
- Infants 80 ml / kg
- Neonates 85 ml / kg
- Premature Neonates 96 ml / kg

Allowable Blood Loss (ABL)

- EBV = weight (kg) * Average blood volume
- Allowable Blood Loss = [EBV*(Hi-Hf)]/Hi
- Where:
  - EBV=Estimated Blood Volume
  - Hi= initial hemoglobin (Hct)
  - Hf= final hemoglobin (Hct)

- Normal Hct Values
  - Men 42-52%
  - Women 37-47%

Example

- Q: Before surgery is to take place, what is the EBV of a female patient weighing 50 kg? Also, what is the ABL of this patient if her Hct is 45?
- EBV = 50kg x 65 = 3250, The final lowest acceptable Hct (Hf) = 30%
- (3250 x (45 – 30))/45 = 1083 Using this rough estimate, the patient in this example could loose 1083 mL of blood without needing a transfusion.
- Replacing Blood Loss: "Ideally, blood loss should be replaced with crystalloid or colloid solutions to maintain intravascular volume (normovolemia) until the danger of anemia outweighs the risks of transfusion. For most patients, that point corresponds to a hemoglobin between 7 and 10 g/dL (or a hematocrit of 21-30%). Below a hemoglobin concentration of 7 g/dL, the resting cardiac output has to increase greatly to maintain normal oxygen delivery"

Blood Products

- Whole blood
- Packed Red Blood Cells
- Platelets
- Fresh Frozen Plasma
- Cryoprecipitate
- Human Albumin

Blood Bank Practices

Preparation of Blood Components

- Blood donors:
  - Approximately 17 million units of blood are donated in Europe each year.
  - Each donor is interviewed for medical history of known infectious diseases
  - Each unit is screened for antibodies to:
  - Syphilis
  - Hepatitis B and C
  - HIV 1 and 2
  - +/- CMV

Blood Bank has tests to compare the blood of the donor to the blood of the recipient

So you must know the blood groups

Red Blood Cell Groups

- At least 20 separate blood group antigen systems are known; fortunately, only the ABO and the Rh systems are important in the majority of blood transfusions.

The ABO System

- Simply speaking, the chromosomal locus for this system produces two alleles: A and B. Each represents an enzyme that modifies a cell surface protein.

Incidence	Naturally Occurring Antibodies in Serum	Type
%45	Anti-B	A
%8	Anti-A	B
%4	—	AB (Universal recipient)
%43	Anti-A, anti-B	O (Universal donor)

The Rh System and others

- The Rh system is encoded by two genes located on chromosome 1.
- There are about 46 Rh-related antigens, but in most clinical settings, the five principal antigens (**D**, C, c, E, and e).
- The most common and most immunogenic allele is the D antigen (80–85% of white).
- Other systems include the Lewis, P, Ii, MNS, Kidd, Kell, Duffy, Lutheran, Xg, Sid, Cartright,YK, and Chido Rodgers antigens.
- Fortunately, with some exceptions (Kell, Kidd, Duffy, and S), alloantibodies against these systems rarely cause serious hemolytic reactions.

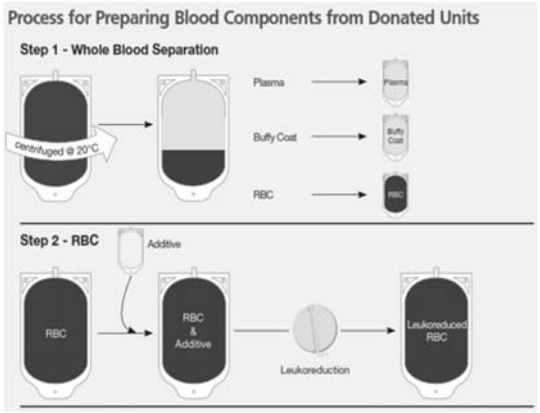
Compatibility Testing

- **ABO-Rh Testing** (Group and Save)
- The patient's RCs are tested with serum known to have anti A and anti B antibodies to determine blood type.
- Confirmation of blood type is then made by testing the patient's serum against RCs with a known antigen type.
- The patient's RCs are also tested with anti-D antibodies to determine Rh.

- **Crossmatching**
- Mimics transfusion: donor cells are mixed with recipient serum.
- (1) confirms ABO and Rh typing (<5 min)
- (2) detects antibodies to the other blood group systems (45 min)
- (3) detects antibodies in low titers or those that do not agglutinate easily(45 min)

Centrifugation

- Collect 500 mL whole blood
- Divert the first 40 mL to reduce risk of bacterial contamination from donor skin
- The 40 mL are used for donor unit testing
- Blood is centrifuged and separated into 3 parts:
- ◆ Red Blood Cells
- ◆ Plasma
- ◆ Buffy coat



- Separated of blood components by 1 unit of **Whole blood**:

- **PRBCS**(hematocrit 70%):
  - 250 mL+saline preservative=350 mL.
  - 1–6°C.

- A preservative–anticoagulant solution is added. The most commonly used solution is **CPDA**-1:
- **C**itrate as an anticoagulant (by binding calcium)
- **P**hosphate as a buffer
- **D**extrose as a RC energy source
- **A**denosine as a precursor for ATP synthesis.
- 35 days
- AS-1 (Adsol) or AS-3 (Nutrice) extends the shelf-life to 6 weeks.
- ADSOL (Adenine, glucose, mannitol and sodium chloride)
- NUTRICE (Adenine, glucose, citrate, phosphate and NaCl)

Packed Red Blood Cells

- Ideal for patients requiring RCs but not volume replacement (eg, anemia pt in compensated CHF).
- Hgb 7-8 g/dL (<6, most people require blood; >10 most people do not)
- Each unit raise Hgb by 1g/dl
- 170-μm filter to trap any clots or debris.
- Warming to 37°C during infusion.
- Hypothermia and low levels of 2,3-diphosphoglycerate (2,3-DPG) in stored blood can cause a marked leftward shift of the hemoglobin–oxygen dissociation curve
- ABO-compatible units are mandatory.

- **Platelet:**
  - 50–70 mL.
  - 20–24°C for 5 days.

Platelets

- Thrombocytopenia or dysfunctional platelets .
- Surgery or invasive procedures: 70,000 x 10^9/L.
- Vaginal delivery and minor surgical procedures: 50,000 x 10^9/L.
- Each unit expected to increase the count by 10,000–20,000 x 10^9/L.
- ABO-compatible platelet transfusions are desirable but not necessary

- **Plasma:**
- The remaining plasma supernatant is further processed and frozen to yield fresh frozen plasma; rapid freezing helps prevent inactivation of labile coagulation factors (V and VIII)
- 200 mL.
- Once thawed it must be transfused within 24 h.

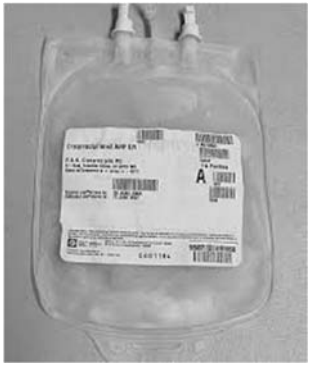
Fresh Frozen Plasma

- FFP contains all plasma proteins, including all clotting factors.
- Indications:
  - Isolated factor deficiencies.
  - Reversal of warfarin therapy.
  - Coagulopathy associated with liver disease.
  - CABG,bleeding+NL ACT.
  - Massive blood transfusions.
  - Antithrombin III def.

- The initial therapeutic dose is usually 10–15 mL/kg
- ABO-compatible units are mandatory.
- Coagulation factors INR 1.4-1.6 (INR>1.6, most people require FFP transfusion for major surgery; INR<1.4, most people do not require)

Cryoprecipitate

- Each unit (15 ml) contains fibrinogen 150 mg, factor VIII 100 units, von Willebrand factor (vWF) (100 units)
- DIC, hemophilia A, von Willebrand disease, quick reversal of thrombolytic therapy
- Fibrinogen (most people require cryoprecipitate for major surgery if fibrinogen < 1 g/dL)



Complications of Blood Transfusion

- Hemolytic reactions
  - Acute Vs. Delayed
- Febrile Non hemolytic reactions
- Transfusion Related Acute Lung Injury (TRALI)
- Infectious complications

Hemolytic Reactions

- Classified as either acute or delayed
- Acute Hemolytic Reactions
- 1:38,000. fatal in 1 in 100,000 (severity depends on volume of incompatible blood)
- The most common cause is misidentification … ABO mostly
- Often severe.
- In awake: chills, fever, nausea, and chest and flank pain.
- In anesthetized: rise in temperature, unexplained tachycardia, hypotension, hemoglobinuria, and diffuse oozing in the surgical field. DIC, shock, and renal shutdown can develop rapidly

Management of hemolytic reactions

1. Once suspected, the transfusion should be stopped immediately.
2. The unit should be rechecked against the blood slip and the patient's identity bracelet.
3. Blood should be drawn to identify hemoglobin in plasma, to repeat compatibility testing, and to obtain coagulation studies and a platelet count.
4. A urinary catheter should be inserted, and the urine should be checked for hemoglobin.
5. Osmotic diuresis should be initiated with mannitol and intravenous fluids.
6. In the presence of rapid blood loss, platelets and FFP are indicated.

• Delayed Hemolytic Reactions

- 1:12,000
- Antibodies to non-D antigens of the Rh system or to foreign alleles in other systems such as the Kell, Duffy, or Kidd antigens.
- 1–1.6% chance even if ABO and Rh compatabale.
- By the time significant amounts of these antibodies have formed (weeks to months), the transfused RCs have been cleared from the circulation. Moreover, the titer of these antibodies subsequently decreases and may become undetectable.
- The hemolytic reaction is therefore typically delayed 2–21 days after transfusion, and mild, consisting of malaise, jaundice, and fever, failure of the patient's Hct to rise in spite of the transfusion and the absence of bleeding, and serum unconjugated bilirubin increases.
- The treatment is primarily supportive.

Febrile non-haemolytic transfusion reactions (FNHTR)

- Acute (<24 hours)
  - Cytokine accumulation during storage of cellular components (especially in platelet units)
  - Recipient antibodies (raised as a result of previous transfusions or pregnancies) reacting to donor human leucocyte antigens (HLA)
- Unexpected temperature rise (≥38°C or ≥1°C above baseline), Chills, rigors, increased respiratory rate, anxiety and a headache
- leucocyte depletion

TRALI

- ARDS following blood transfusion
- High morbidity … mechanical ventilation
- Lung injury is generally transient with PO2 levels returning to pretransfusion levels within 48 -96 hours and CXR returning to normal within 96 hours.
- Mortality rate, often approximated at 5 to 10%
- Treatment as ARDS

Approaches to Fluid Management

The "Classic" Approach

- Step 1: Calculate Ongoing Maintenance Requirements

4/2/1 rule: 4 cc/kg/hr for the first 10 kg, 2 cc/kg/hr for the second 10 kg, and 1 cc/kg/hr for every kg above 20.

Ex. 70kg → 10 * 4 ml = 40 ml
10 * 2 ml = 20 ml
50 * 1 ml = 50 ml
110ml / hour given each hour as oral or enteral intake is stopped

Ex. 15kg → 10 * 4 ml = 40 ml + 5 * 2 ml = 10 ml = 50 ml / hour

- Step 2:Calculate Preoperative Fluid deficit

Maintenance * the time without intake what so ever

Ex. 70kg fasting for 10 hours pre-operatively

M = 110ml/hour * time 10 hour = 1100 ml

- Step 3: Calculate Anticipated Surgical Fluid Losses
- Minimal tissue trauma (ex. herniorrhaphy): 0-2 cc/kg/hr
- Moderate tissue trauma (ex. cholecystectomy): 4-6 cc/kg/hr
- Severe tissue trauma (ex. bowel resection): 8-10 cc/kg/hr

- Ex. 70kg undergoing major laparotomy →

10 ml * 70 kg = 700 ml/hour as long as surgery is going on

- Step 4: Adjust for Blood Losses

- A common recommendation is to give 3-4 cc of crystalloid for every 1 cc of blood loss

- Remember to add up suction volume, lap pads (100-150 cc each if fully soaked) and 4x4 small pads (10 cc each if fully soaked)

Ex. In the 1st hour of laparotomy there was 200ml of pure blood in the suction jar, 2 fully soaked lap pads, and 10 fully soaked small gauses

200 ml + 2 * 150 ml + 10 * 10 ml = 600 ml (in that hour) to be replaced with either 600 * 4 of cryst. or -if indicated- 600 ml of blood

- 1st hour = Maintenance + ½ Deficit + Blood loss + Ongoing loss
- 2nd hour = Maintenance + ¼ Deficit + Blood loss + Ongoing loss
- 3rd hour = Maintenance + ¼ Deficit + Blood loss + Ongoing loss
- Maintenance continued post-operatively as long as fasting
- Blood loss replaced as long as there is bleeding
- Ongoing loss as long as the surgery continues

THANK YOU

I V Anesthetic Agents

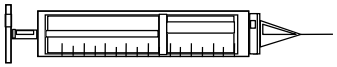
Dr. Abdelkarim Al Oweidi AL-Abbadi

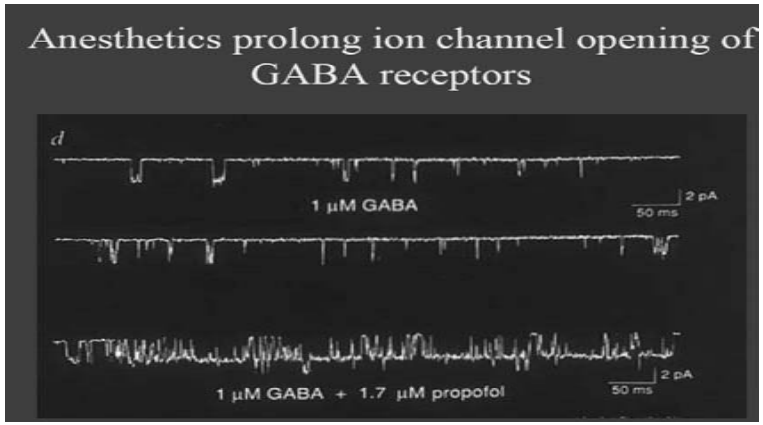
Associate Prof. of Anesthesia

Goals of General Anesthesia

- Hypnosis (unconsciousness)
- Amnesia
- Analgesia
- Immobility/decreased muscle tone
  - (relaxation of skeletal muscle)
- Reduction of certain autonomic reflexes
  - (gag reflex, tachycardia, vasoconstriction)

Intravenous Anesthetics





Ideal Intravenous anesthetic

- Water-soluble, painless on injection
- Rapid onset, rapid recovery, little accumulation.
- Little depression of respiratory/cardiovascular system.
- No nausea or vomiting,
- No interaction with muscle relaxant,
- No histamine release.
- Safe after intra-arterial injection.
- No toxic effects.
- No hypersensitivity reactions.
- Long shelf-life at room temperature.

Pharmacological Principles

- High- perfusion organs (vessel-rich); brain takes up disproportionately large amount of drug compared to less perfused areas (muscles, fat, and vessel-poor groups).
- Drugs bound to plasma proteins are unavailable for uptake by an organ.

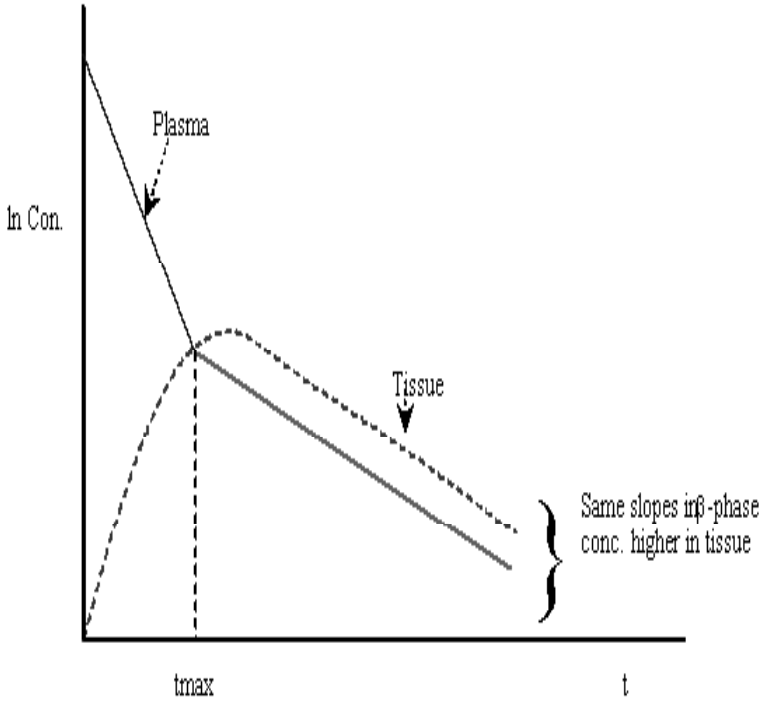
Pharmacological Principles

- After highly perfused organs are saturated during initial distribution, the greater mass of the less perfused organs continue to take up drug from the bloodstream.
- As plasma concentration falls, some drug leaves the highly perfused organs to maintain equilibrium.
- This redistribution from the vessel-rich group is responsible for termination of effect of many anesthetic drugs.

Pharmacological Principles

Compartment Model

- Offers a simple way to characterize the distribution of drugs in the body
- Can be conceptualized as a group of tissues that posses similar pharmacokinetics (Central and peripheral compartments)
- Distribution phase vs. Elimination phase



Protein sites of action

- **Inhibitory channels** :

➢ GABA-A channels (the main inhibitory receptor).

➢ Glycine channels.

- **Excitatory channels :**

➢ Neuronal nicotic.

➢ NMDA.

INTRAVENOUS ANAESTHETIC AGENTS

1- Barbiturate

Sodium thiopental(used for over 40 years)

Methohexital

2. Non barbiturate

Propofol

Ketamine (infrequently used)

Etomidate

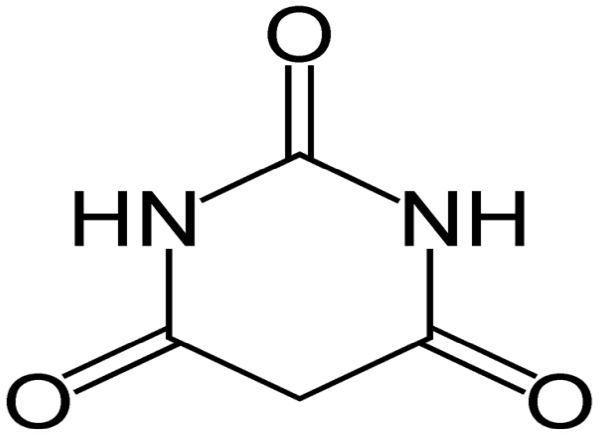
3. Other adjuvant intravenous anesthetic agents (benzodiazepines, midazolam

Intravenous Anesthetics

Barbiturates

- Depress the Reticular activating system located in the brain stem that controls several vital functions & consciousness
- Affect the function of nerve synapses not axons
- Suppress transmission of excitatory neurotransmitters and enhance transmission of inhibitory ones.
- They are barbituric acid derivatives, substitution at the number 5 carbon determines hypnotic potency and anticonvulsant activity

Barbituric Acid



SODIUM THIOPENTAL (PENTOTHAL)

Ultra short acting barbiturate.

Classification:

-IV anesthetic-hypnotic

Physical chemical properties :

-It's a Yellow powder with a sulphuric smell and a bitter taste

-Highly lipid soluble compound

-When combined with sodium

SODIUM THIOPENTAL (PENTOTHAL)

-It is bacteriostatic in water and has a ph of 10.6 to10.8

-When injected ,sodium bicarbonate is neutralized and the thiopental is converted to its lipid soluble non ionized form(40% ionized at ph=7.4)

-+++ protien binding by albumin(75%)

Sodium thiopental (pentothal)

- When dissolved in water it makes a 2.5% solution (25mg/ml)
- Thiopental is a *core* medicine in the World Health Organization's "Essential Drugs List", which is a list of minimum medical needs for a basic health care system

SODIUM THIOPENTAL (PENTOTHAL)

Pharmacokinetics

- **Dose of 3-5 mg/kg results in loss of consciousness..**
- Time required to render the patient unconscious is generally ***30-60 secs*** after administration. This is called the **"arm brain"** circulation time.
- Arm brain circulation time is the time required for the drug to pass from site of injection to the brain as it passes through the right heart, pulmonary circ., and the left heart

Sodium thiopental (pentothal)

Pharmacokinetics

- Solo: anesthetic state persists for 5-10 mins
- So is most commonly used in the ***induction phase*** of general anesthesia
- As with all lipid soluble anesthetic drugs, the **short duration of action** of Sodium thiopental is almost entirely due to its redistribution away from central circulation towards muscle and fat tissue

Sodium thiopental (pentothal)

Pharmacokinetics

- Sulphur containing drug.. acidosis, NSAIDS may displace thiopental from albumin.
- Liver & renal disease may be associated with low albumin levels so result in an increase in free thiop… toxicity.
- Metabolism: primarily in the liver with approximately 10 to 15%of the drug level metabolized per hour.
- Desulfuration: Reaction in liver produces pentobarbital which undergoes oxidative metabolism yielding 2 compounds with no anesthetic activity.
- Less than 1% of the drug is excreted unchanged in the urine.

SODIUM THIOPENTAL (PENTOTHAL)

pharmacodynamics

- **CNS**: barbiturates interact with chloride ion channels by altering the duration they spend in an open state.
- This **facilitates inhibitory neurotrasmitters** such as Gama Amino Butyric Acid (GABA)as well as **blocking excitatory NTM** actions such as Glutamic acid.
- Thiopental will decrease both cerebral: electrical **&** metabolic activity so it can be used ***to stop seizures activity in emergency situations.***
- To maintain depression of cerebral electrical activity very high dose are required but to maintian seizure control & avoid CV depression from high dose of thiopental other drugs are used (e.g. benzodiazepines)

SODIUM THIOPENTAL (PENTOTHAL)

pharmacodynamics

- **CNS**: **Elevated ICP** can quickly be reduced by thiopental BUT The improvement of ICP requires high dose of thiopental to be maintained.
- The reduction of ICP is due to cerebral vasoconstriction, reduced cerb. metabolism & O2 requirements.
- thiopental has an **an anti-analgesic effect**,,, low dose may decrease pain threshold.
- **Intraocular pressure decreases** up to 25% with 3-5mg/kg of thiopental and persists for 3 to 5 minutes.

SODIUM THIOPENTAL (PENTOTHAL)

Pharmacodynamics

- **CVS:-**thiopental causes a dose related **depression of myocardial function** as measured by CO, SV & BP.

- Coronary blood flow, HR & myocardial O2 uptake all increase following thiopental administration.

- Venous tone decreases (decreased preload) and contributes to the increase in **HR** and decrease in **BP.**

-little change in total peripheral resistance

SODIUM THIOPENTAL (PENTOTHAL)

Pharmacodynamics

- **Respiratory**: 2 or 3 large breaths followed by apnea for less than 1min.

-There is dose related **depression** of the **respiratory response** to **hypercarbia and hypoxia.**

-**Laryngospasm and bronchoconstriction** may be associated with *light levels* of thiopental and with airway manipulation or intubation.

-**FRC is reduced** by 20% with induction of anaesthesia

>(FRC) is the volume of air present in the lungs at the end of passive expiration

SODIUM THIOPENTAL (PENTOTHAL)

Pharmacodynamics

- **GI:** Enzyme induction may occur with prolonged high dose therapy.

- **Hypoalbuminemia** will result in an increase in free thiopental and an **increase in the potency of thiopental.**

- **GU/pregnancy/fetus:**

- Thiopental has little or **no effect** on the kidneys or gravid uterus although thiopental crosses the placenta it has no significant effect on the fetus when used for c/sections (dose used is limited to 4mg/kg).

SODIUM THIOPENTAL (PENTOTHAL)

Dose & administration

-Thiopental should be used with caution in *shock* status because normal dose may lead to *rapid death.*

-For a short procedure (e.g.cardioversion) a dose of 2 mg/kg is generally sufficient.

-For elderly women with hip fracture . A small minimal dose induces anesthesia.

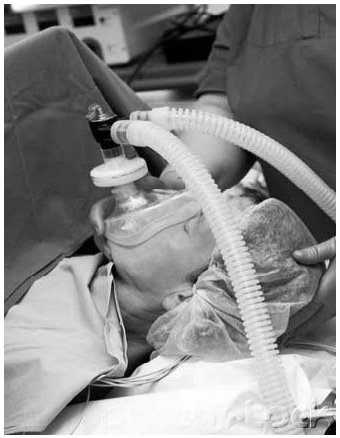
SODIUM THIOPENTAL (PENTOTHAL)
INDICATIONS

1- Induction of anesthesia.

2- Maintenance of anesthesia for short procedures.

3-Control of convulsive states

4- To supplement regional anesthesia.



SODIUM THIOPENTAL (PENTOTHAL)

Absolute Contraindications

1- Airway obstruction

2- Porphyria

3- Previous hypersensitivity

PRECUATIONS

1- CVS disease

2- Severe hepatic disease

3- Renal diseases

SODIUM THIOPENTAL (PENTOTHAL)
SIDE EFFECTS

1- Hypotension esp. if thiopental is administered to hypovolemic, shocked ones.

2- Respiratory depression: when excessive doses are used.

3- Tissue necrosis.

4- Laryngeal spasm.

5- Bronchospasm: unusual but in asthmatics pts.

6- Allergic reactions: from coetaneous rashes to severe anaphylactic shock with CVS collapse

SODIUM THIOPENTAL (PENTOTHAL)
SIDE EFFECTS

7- **Rarely, Intra-Arterial injection can occur.**

The consequences of accidental arterial injection may be severe.

Degree of injury is related to the concentration of the drug.

***Treatment* consists of**

1. Dilution of the drug by the administration of saline into the artery.

2. Heparinization to prevent thrombosis.

3. Brachial plexus block, stellate G block, vasodilators

Overall, the proper administration of thiopental intravenously is remarkably free of local toxicity

PROPOFOL

- Intravenous anaesthetic/hypnotic.
- Akylphenol.
- Propofol is a sweet drug in the OR, but definitely not for home use.

PROPOFOL
PHYSICAL AND CHEMICAL PROPERTIES

Emulsion consists of:

1% propofol 10mg/ml.

10% soyabean oil.

2.25 %glycerol.

1.2% purified egg phosphatide.

PROPOFOL
PHYSICAL AND CHEMICAL PROPERTIES

- Propofol is a highly lipid soluble oil that's combined with glycerol, egg, and soya bean oil for IV administration.
- It's appearance is similar to that of a 2% milk.
- It has a pH of 7 and is supplied in 20 ml ampoules with a concentration of 10 mg/ml.
- Neither precipitates histamine release nor triggers malignant hyperthermia.
- Has no effects on muscle relaxants.
- Associated with low incidence of nausea & vomiting…..

PROPOFOL
DOSAGE

- For healthy unpremedicated 2.5-3 mg/kg.
- For premedicated 1.5-2 mg/kg.
- Elderly patients <= 1 mg/kg.
- Maintenance of anesthesia (50-150 mcg/kg/min) combined with Opioids (Continuous Infusion: Total intravenous Anesthesia TIVA)
- For IV conscious sedation for operative procedures with local anaesthesia 25-75

PROPOFOL
EFFECTS ON ORGAN SYSTEMS

Cerebral:

decreases cerebral blood flow and intracranial pressure. Propofol has antiemetic, antipruritic, and anticonvulsant properties.

Cardiovascular:

decrease in arterial blood pressure secondary to a drop in systemic vascular resistance, contractility, and preload. Hypotension is more pronounced than with thiopental. Propofol markedly impairs the normal arterial baroreflex response to hypotension

PROPOFOL
EFFECTS ON ORGAN SYSTEMS

Respiratory:

Propofol causes profound respiratory depression. Propofol induced depression of upper airway reflexes exceeds that of thiopental.

Venous irritation:

- Pain on injection is more common than with thiopental esp. if given in a small vein in the hand.
- To solve this problem:

1. small doze of lidocaine *with* propofol.
2. administering propofol through a fast flowing more proximal IV catheter.

- It decreases cerebral metabolic rate (CMRO2), intracranial pressure (ICP), and cerebral blood flow (CBF) (by increasing CVR) ----- neuroprotetive .
- It may decrease cerebral perfusion pressure (CPP)----limitation.
- Anti-convulsant (it is used for treatment of status epilepticus) at higher induction doses, so that it is not the agent of choice for ECT .
- Some case reports of delayed onset of grand mal seizures in patients with epilepsy .

PROPOFOL
CONTRAINDICATIONS

1. Egg allergy.
2. Lack of resuscitation equipment or knowledge of the drug.
3. Inability to maintain a patent airway.
4. Conditions in which reduction in blood pressure can't be tolerated. e.g. patients with fixed cardiac output (severe aortic or mitral stenosis, pericardial tamponade) and those in shock status.

Advantages of propofol over thiopentone

- Rapid and smooth recovery.
- Completely eliminated from body in 4 hours so early ambulation.
- Anti-emetic.
- Anti-pruritic.
- Bronchodilator.

Disadvantages

- Apnea is more profound and longer.
- Hypotension is more severe.
- Injection is painful.
- Solution is less stable (6 hrs).
- Chances of sepsis with contaminated solution is high.
- Myoclonic activity.
- Sexual fantasies and hallucination.
- More expensive than thiopentone.
- Allergic reactions in individuals who are allergic to egg lecitin.
- Propofol addiction has also been reported.
- Propofol infusion syndrome: It is very rare but is a lethal complication. Usually seen if infusion is continued for more than 48 hrs & is much more

-Propofol Infusion Syndrome (PRIS)

✓ It is a rare complication Noticed in children when propofol is used for prolonged sedation in the ICU .

✓ It is both dose, and duration dependent (recommended maximum infusion dose is 4 mg/kg/h) .

✓ Characterized by an un-explained metabolic acidosis, hyperkalemia, hyperlipidemia, hepatomegaly, renal failure, rhabdomyolysis, and most importantly ECG changes, arrhythmias, and heart failure .

✓ Mortality is 50% .

✓ Don't use propofol for prolonged sedation in

Etomidate

- Etomidate is a carboxylated imidazole derivative. Etomidate has anesthetic and amnetic properties, but has no analgesic properties

Uses

- Etomidate is commonly used in the emergency setting as part of a rapid sequence induction to induce anesthesia or for conscious sedation. It is often used in this setting since it has a rapid onset of action and a low cardiovascular risk profile, and therefore is less likely to cause a significant drop in blood pressure than other induction agents
- It is the agent IV anesthetic agent of choice for aneurysm surgery & patients with cardiac disease.

Dosage

- The anaesthetic induction dose for adult humans is 0.3 mg/kg intravenously, with a typical dose being 20 mg. In common with all induction agents, etomidate causes loss of consciousness after one **arm-brain circulation time**.
- At the typical dose, anesthesia is induced for about 5–10 minutes even though the half-life of drug metabolism is approximately 75 minutes. This is because etomidate is redistributed from the plasma to other tissues.

Metabolism

- Etomidate is highly protein bound in blood plasma and is metabolised by hepatic and plasma esterases to inactive products with a redistribution half-life of 2–5 minutes and an elimination half-life of 68–75 minutes.

Actions and effects

- Etomidate does not cause significant cardiovascular or respiratory depression, but may cause a brief period of apnea.
- The decrease in cerebral blood flow produced by etomidate is approximately the same as that produced by thiopental
- Etomidate slightly lowers intracranial pressure and it usually causes a moderate decrease in intraocular pressure

Side effects (disadvantages)

- Adrenocortical suppressioin on long term infusion.
- Nausea & vomiting. (40 %)
- Has very high incidence of myoclonus.
- High incidence of thrombophlebitis.
- It can cause vitamin C deficiency & platelet dysfunction.
- May produce pain on injection
- No analgesia.
- Hiccups are common.

Contraindications and precautions

- Use of etomidate is not recommended since data are insufficient to support its use in obstetrics, including cesarean section deliveries
- It is not known whether etomidate is distributed into breast milk. However, problems in humans have not been documented

- Appropriate studies with etomidate have not been performed in children up to 10 years of age . Safety and efficacy have not been established.

- Elderly patients are more sensitive to the effects of etomidate than are younger patients. In addition, geriatric patients are more likely to have age-related hepatic function impairment, which may require reduction of dosage in patients receiving etomidate

Intravenous Anesthetics

- **Ketamine**
- Has multiple effects through the CNS including blocking polysynaptic reflexes in the spinal cord and inhibiting neurotransmitter effects in selected areas of the brain
- Ketamine dissociates the thalamus from the limbic cortex
- N-methyl-D-aspartae receptor antagonist
- Structurally analogue to phencyclidine
- Can cause hallucinogenic effects and nightmares
- Dose : Induction IV 1-2 mg/kg, IM 3-5 mg/kg

Intravenous Anesthetics

Absorption

- Administered IM or IV with peak plasma level within 10-15 min after IM injection

Distribution

- More lipid soluble and less protein bound than thiopental
- Distribution half-life is 10-15 min

Biotransformation and excretion

- Biotransformed in the liver to several metabolites some retain anesthetic properties (norketamine)
- Short elimination half-life (2h)
- Excreted renally

Intravenous Anesthetics

- **Effect on organ systems**

Cardiovascular

- Increases Blood pressure, heart rate, and cardiac output
- Increases pulmonary artery pressure and myocardial work
- Avoid in patient with coronary artery disease

Respiratory

- Minimal effect on the ventilatory drive
- Potent bronchodilator

Intravenous Anesthetics

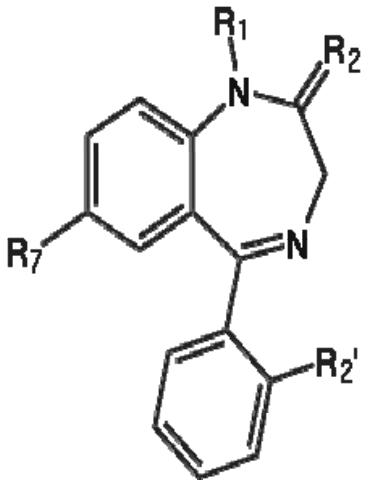
Cerebral

- Increase cerebral oxygen consumption, cerebral blood flow and intracranial pressure
- Myoclonic activity is associated increased subcortical electrical activity
- Undesirable psychotomimetic effects (illusions, disturbing, dreams and delirium)
- Have analgesic effects

Intravenous Anesthetics

Benzodiazepines

- Interact with specific receptors in the CNS mainly in the cortex
- Binding to receptors enhances the inhibitory effects of various neurotransmitters (GABA)
- Flumazenil is a specific benzodiazepine-receptor antagonist that effectively reverses most of the CNS effect
- Chemical structure includes a benzene ring and a 7-member diazepine ring, substitution ay various positions on these rings affect potency and biotransformation



Agent	Use	Route	Dose
Diazepam	Premedication	Oral	0.2-0.5 mg/kg upto 15 mg
	Sedation	IV	0.04-0.2 mg/kg
	Induction of hypnosis	IV	0.3-0.6 mg/kg
Midazolam	Premeditation	IM	0.07-0.15 mg/kg
	Sedation	IV	0.01-0.1 mg/kg
	Induction of hypnosis	IV	0.1-0.4 mg/kg
Lorazepam (not Recommended in children)	Premedication	Oral	0.05 mg/kg
		IM	0.03-0.05 mg/kg
	Sedation	IV	0.03-0.04 mg/kg

Intravenous Anesthetics

Absorption

- Administered orally, IM and IV for sedation or induction of GA
- Diazepam and Lorazepam well absorbed from GI tract, peak plasma level in 1-2 h respectively
- Dose Midazolam : premedication IM 0.07-0.15 mg/kg, sedation IV 0.01-0.1 mg/kg, Induction IV 0.1-0.4 mg/kg

Distribution

- Diazepam is lipid soluble and rapidly cross the blood brain barrier, water soluble at low pH
- Redistribution is rapid for benzodiazepines (3-10 min)
- Highly protein bound (90-98%)

Intravenous Anesthetics

Biotransformation

- Rely on the liver for transformation into water-soluble glucoronide end products
- Slow hepatic extraction, long half-life for diazepam (30h)

Excretion

- Metabolites are excreted mainly in the urine
- Entrohepatic circulation produces a second peak in diazepam plasma concentration 6-12h following administration

Intravenous Anesthetics

- **Effect on organ systems**

Cardiovascular

- Minimal CVS depressant effects
- Arterial BP, Cardiac output, and PVR slightly decreased
- Heart rate sometimes increased

Respiratory

- Depresses ventilatory response to CO2
- Ventilation must be monitored

Intravenous Anesthetics

Cerebral

- Reduces cerebral oxygen consumption
- Decreases cerebral blood flow and intracranial pressure
- Effective in preventing and controlling grand mal seizures
- Sedative dosages cause antegrade amnesia

Intravenous Anesthetics

Opioids

- Classically known as narcotic analgesics
- Name derived from Poppy juice (opium), first obtained from the capsules of the unripe oriental Poppy seed (*papaver somniferum*), of which Morphine is the principal active ingredient.
- "Opiates": a term generally used for naturally occurring substances with properties similar to Morphine.
- "Opioids" : refers to all naturally occurring and synthetic drugs with an affinity for opioid receptors, and actions that can be stereospecifically antagonized by Naloxone

Intravenous Anesthetics

Mode of Action

- By interaction with Specific opioid receptors in the CNS (brain and Spinal Cord) and peripheral tissues (somatic and sympathetic nerves)
- Opioids Modify the complex emotional experience of pain as well as affecting its transmission as a sensory modality.
- Their influence on the reactive component of pain (i.e. anxiety, Fear and suffering) can greatly influence the patients' ability to tolerate pain.

Intravenous Anesthetics

Opioid Receptors

Four major Types:

1- μ (mu): with μ-1 and μ-2 subtypes
2- K (kapa)
3- б (delta)
4- σ (sigma)

- The pharmacodynamic properties of specific opioids depend on which receptor is bound, the binding affinity, and whether the receptor is activated.
- Opioid receptors can also be activated by some endogenous peptides (Endorphins, enkephalins, and dynorphins)
- Opioid receptor activation inhibits the presynaptic release and post-synaptic response to excitatory neurotransmitters (e.g. Acetyl-choline, substance P).

Intravenous Anesthetics

Opioids classification

1- **Agonists**:

- ***Strong***: Morphine, pethidine, Methadone, Fentanyl,…
- ***Moderate:*** Codeine, Oxycodone, Hydrocodone
- ***Weak***: Propoxyphene

2- **Mixed agonist/antagonist:** Pentazocine, butorphanol, nalbuphene, Buprenorphine, Nalorphine,…

3- **Antagonists**: Naloxone, Naltrexone, Doxapram, …

Intravenous Anesthetics

- **<u>Agonist opioid drugs:</u>**
  - Have linear Dose-Response relationship
  - Stimulate μ and K receptors
  - Antagonized by Naloxone and Nalorphine
- **<u>Agonist/Antagonist opioid drugs:</u>**
  - Have a plateau or bell shaped Dose response curve
  - Antagonists at μ receptor above low dose.
  - Full or partial agonists at **K** receptor.
  - Antagonized by Naloxone but not by Nalorphine.

Intravenous Anesthetics

- **<u>Antagonist opioid drugs</u>**

- Naloxone has a higher affinity for μ receptor than for other opioid receptors.
- Doxapram is used to treat the respiratory depression caused by Buprenorphine since its effects are only partially reversed by Naloxone.

Intravenous Anesthetics

Receptor	Clinical effect	Agonist examples
μ (mu)	**- Supra-spinal analgesia (μ-1) - Respiratory Depression (μ-2) - Physical dependence - Muscle rigidity**	**Morphine Met-enkephalin Beta endorphin Fentanyl**
К (kapa)	**- Sedation - Spinal analgesia**	**Morphine Nalbuphene Butorphanol Dynorphins Oxycodone**
б (delta)	**- Analgesia - Behavioral - Epileptogenic**	**Leu-Enkephalin Beta endorphin**
σ (sigma)	**- Dysphoria - Hallucinations - Respiratory stimulation**	**Pentazocine Nalorphine Ketamine?**

Intravenous Anesthetics

Pharmacokinetics

- Distribution half lives of all opioids are fairly rapid: 5-20 minutes.
- Re-distribution is responsible for termination of action of small doses
- Morphine has low fat solubility accounting for its slow onset and prolonged duration of action.
- Most opioids depend on the liver for biotransformation, with high hepatic extraction ratio.
- Morphine has both active and inactive metabolites
- Pethidine (Meperidine) is metabolized to the active noremeperidine.
- Remifentanyl has a unique ester structure ➔ rapid ester hydrolysis; terminal elimination half

Intravenous Anesthetics

- Excretion of end products of opioids metabolism is mainly through the kidney
- Noremeperidine has an excitatory effect on CNS leading to Myoclonic activity and seizures that are not reversed by Naloxone
- A late secondary peak in Fentanyl plasma level may occur 4 hours after last IV dose due to enterohepatic recirculation and release of sequestered drug.
- Morphine-3-glucuronide is partly excreted in bile and can be broken down by intestinal bacteria, releasing morphine that may be reabsorbed by Enterohepatic recirculation.

Intravenous Anesthetics

CVS

In general, Opioids do not seriously impair the cardiovascular System.

•

Meperidine	↑H/R, ↓cardiac contractility, Histamine release in some individuals
Morphine	↓H/R at high doses (vagus mediated), histamine release
Fentanyl, sufentanyl, Remifentanyl alfentanyl	↓H/R at high doses
Combination	? Significant myocardial depression

Intravenous Anesthetics

Respiratory

- Depress ventilation, particularly respiratory rate.
- ↑Resting PaCO2 with blunted ventilatory response to CO2 challenge.
- Apneic threshold is elevated.
- Hypoxic drive is decreased.
- Histamine release - bronchospasm: (Morphine and Meperidine).
- Chest wall rigidity: Fentanyl, sufentanyl, alfentanyl.
- Opioids effectively blunt the airway reflexes to airway management.

Intravenous Anesthetics

Cerebral

- Opioids are the mainstay of intra-operative pain management (powerful analgesics).
- In normal brains, Opioids- in general- reduce cerebral Oxygen Consumption, blood flow, and intracranial pressure, but to a much lower degree than barbiturates or benzodiazepines.
- Meperidine:? EEG activation.
- Stimulation of CRTZ➔ Nausea & Vomiting.
- Opioids do not reliably produce amnesia.
- Opioids are recently effectively used in intra- thecal and epidural spaces for analgesia.
- Meperidine: has local anesthetic qualities and effectively used to treat shivering.

Intravenous Anesthetics

Gastro-intestinal

- Contraction of sphincter of Oddi and biliary spasm .
- Constipation.

Genitourinary: Urine retention

Endocrine:

- More effective than inhalational anesthetics in blocking the stress response to surgical stimulation.

Ophthalmic: Miosis.

Intravenous Anesthetics

Concurrent use of:

- MAOI drugs➔ Respiratory arrest, hypertension or Hypotension, coma and Hyperpyrexia. (esp. with Meperidine).
- Other Hypnotic and sedative drugs➔ synergism in sedative, cardiovascular, and respiratory effects.
- Erythromycin: impairment of alfentanyl biotransformation.

Intravenous Anesthetics

- **Dexmedetomidine**
- **Is a sedative medication used by intensive care units and anesthesiologists, and is marketed under the brand name Precedex**
- It is relatively unique in its ability to provide sedation without causing respiratory depression
- Its mechanism of action is agonism of alpha-2 receptors in certain parts of the brain
- It is the S-enantiomer of medetomidine
- Has sedative, analgesic, sympatholytic, and anxiolytic effects

Intravenous Anesthetics

- Reduces the volatile anesthetic, sedative and analgesic requirements of the patient without causing significant respiratory depression
- Effective treatment for the dangerous cardiovascular symptoms of cocaine intoxication and overdose
- Has an opiod sparing effect
- The recommended dosage is 1 µg/kg IV over 10 min
- Maintenance infusion rate of 0.2-0.7 µg/kg/hr
- Metabolized in the liver and its metabolite is eliminated in the urine
- Side effects include bradycardia, heart block and hypotension

Summary of Intravenous Anesthetic Agents

Drug	Speed of Induction and Recovery	Main Unwanted Effects	Notes
Thiopental	Fast (accumulation occurs, giving slow recovery) Hangover	Cardiovascular and respiratory depression	Used as induction agent declining. Decreases cerebral blood flow and O2 consumption.
Etomidate	Fast onset, fairly fast recovery	Excitatory effects during induction and recovery, Adrenocortical suppression	Less cardiovascular and respiratory depression than with thiopental, Causes pain at injection site
Propofol	Fast onset, very fast recovery	Cardiovascular and respiratory depression. Pain at injection site.	Most common induction agent. Rapidly metabolized; possible to use as continuous infusion.
Ketamine	Slow onset, after-effects common during recovery	Psychotomimetic effects following recovery, Postoperative nausea, vomiting and salivation	Produces good analgesia and amnesia
Midazolam	Slower than other agents		Little respiratory or cardiovascular depression

Table 8–8. Summary of nonvolatile anesthetic effects on organ systems.

Agent	Cardiovascular HR	Cardiovascular MAP	Respiratory Vent	Respiratory B'dil	Cerebral CBF	Cerebral $CMRO_2$	Cerebral ICP
Barbiturates							
Thiopental	↑↑	↓↓	↓↓↓	↓	↓↓↓	↓↓↓	↓↓↓
Thiamylal	↑↑	↓↓	↓↓↓	↓	↓↓↓	↓↓↓	↓↓↓
Methohexital	↑↑	↓↓	↓↓↓	0	↓↓↓	↓↓↓	↓↓↓
Benzodiazepines							
Diazepam	0/↑	↓	↓↓	0	↓↓	↓↓	↓↓
Lorazepam	0/↑	↓	↓↓	0	↓↓	↓↓	↓↓
Midazolam	↑	↓↓	↓↓	0	↓↓	↓↓	↓↓
Opioids							
Meperidine*	↑	*	↓↓↓	*	↓	↓	↓
Morphine*	↓	*	↓↓↓	*	↓	↓	↓
Fentanyl	↓↓	↓	↓↓↓	0	↓	↓	↓
Sufentanil	↓↓	↓	↓↓↓	0	↓	↓	↓
Alfentanil	↓↓	↓↓	↓↓↓	0	↓	↓	↓
Remifentanil	↓↓	↓↓	↓↓↓	0	↓	↓	↓
Ketamine	↑↑	↑↑	↓	↑↑↑	↑↑↑	↑	↑↑↑
Etomidate	0	↓	↓	0	↓↓↓	↓↓↓	↓↓↓
Propofol	0	↓↓↓	↓↓↓	0	↓↓↓	↓↓↓	↓↓↓
Droperidol	↑	↓↓	0	0	↓	0	↓

*The effects of meperidine and morphine on MAP and bronchodilation depend upon the extent of histamine release.
HR = heart rate; MAP = mean arterial pressure; Vent = ventilatory drive; B'dil = bronchodilation; CBF = cerebral blood flow; $CMRO_2$ = cerebral oxygen consumption; ICP = intracranial pressure.
0 = no effect.
0/↑ = no change or mild increase.
↓ = decrease (mild, moderate, marked).
↑ = increase (mild, moderate, marked).

Thank you

Department of anesthesia and intensive care

The University of Jordan

2017

INHALATIONAL ANESTHETICS

Dr. Mustafa Alrabayah

Click to add title

- ***Anesthesia*** defined as the abolition of sensation
- ***Analgesia*** defined as the abolition of pain

- "Triad of General Anesthesia"
  - need for unconsciousness
  - need for analgesia
  - Need for amnesia
  - (±) need for muscle relaxation

Signs and Stages of Anesthesia

Analgesia. Mild CNS depression. Suitable for surgical procedures not requiring muscle relaxation. All anesthetics do not produce analgesia.

Delirium: An excited state resulting from cortical motor depression. This can be avoided with rapidly acting, potent anesthetics. This stage extends from the lack of consciousness in stage 1 to surgical anesthesia in stage 3.

Surgical Anesthesia: Further subdivided into stages representative of increasing muscle relaxation, the final stage is disappearance of muscle tone.

Deep anesthesia andRespiratory paralysis: Generally not desirable.

Inhalational Anesthetic Agents

- Inhalational anesthesia refers to the delivery of *gases or vapors* from the respiratory system to produce or maintain anesthesia

- Exposure to the pulmonary circulation allows a more rapid appearance in arterial blood than intravenous administration

Advantages of inhalational anesthesia

- Completely painless induction
- No IV (intravenous) access needed
- Rapid appearance of drug in arterial blood
- Safe: as long as patient is breathing satisfactorily, elimination of agent and emergence from anesthesia is essentially guaranteed.

1840, William Morton publically administered ether

1847, James Simpson introduced chloroform

It was more potent but could have severe side effects such as sudden death and late onset severe liver damage

1877, introduction of local anesthesia

1920's intravenous induction agents were introduced

1940's Muscle relaxants were introduced

Minimum Alveolar Concentration

- Minimal alveolar concentration of inhalational agent that prevent movement in 50% of the patients in response to surgical stimulation (skin incision)
- Equivalent to ED_{50}
- For the same agent, varies with age, temperature and other drugs on board

Minimum Alveolar Concentration

- **The rationale for this measure of anaesthetic potency is,**
  - a. alveolar concentration can be easily measured
  - b. near equilibrium, alveolar and brain tensions are virtually equal
  - c. the high cerebral blood flow produces rapid equilibration
- **Factors which support the use of this measure are,**
  - a. MAC is invariant with a variety of noxious stimuli
  - b. individual variability is small
  - c. sex, height, weight & anaesthetic duration do not alter MAC
  - d. doses of anaesthetics in MAC's are additive

Factors affecting MAC

PHYSIOLOGIC & PHARMACOLOGIC FACTORS AFFECTING MAC

Increase in MAC:-

- Hyperthermia
- Hypernatraemia
- Drug induced elevation of CNS catecholamine stores
- Chronic alcohol abuse & chronic opioid abuse
- Increases in ambient pressure (experimental)
- Cyclosporine
- Excess pheomelanin production(red hair)

Decrease in MAC:-

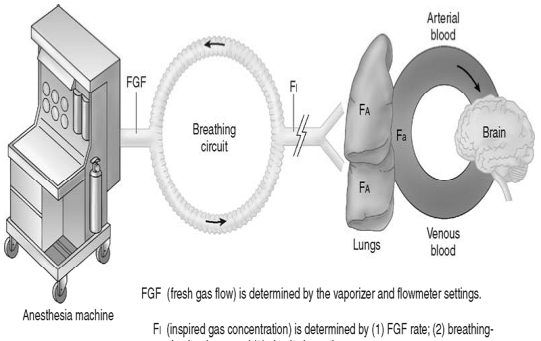
- Hypothermia & Hyperthermia (if >42◦ C)
- Hyponatraemia
- Drug induced decrease in CNS catecholamine level
- Increasing age (6% decrease/decade)
- Preoperative medication
- Hypoxaemia (PaO2< 38mmHg)
- Hypotension(<40 mm hg- MAP)
- Anaemia (Haematocrit<10%)
- Pregnancy (progesterone)
- Postpartum(returns to normal in 24-72 hrs)
- CNS depressant drugs – Opioids,Benzodiazepines TCA's etc.
- other drugs–lithium, Lidocaine,Magnesium
- acute alcohol abuse
- Cardiopulmonary bypass

Uptake and Distribution

The depth of general anesthesia depends on the partial pressure (or gas fraction) exerted by the inhalational agent in the patientbrain (b).

This brain partial pressure depends on arterial (a) blood partial pressure which depends on alveolar (A) partial pressure which depends on partial pressure of agent in the inspired gas (I):

p_I -> p_A -> p_a -> p_b



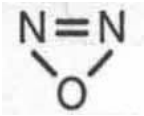
FGF (fresh gas flow) is determined by the vaporizer and flowmeter settings.

FI (inspired gas concentration) is determined by (1) FGF rate; (2) breathing-circuit volume; and (3) circuit absorption.

FA (aveolar gas concentration) is determined by (1) uptake (uptake = λb/g x C(A-V) x Q); (2) ventilation; and (3) the concentration effect and second gas effect:
a) concentrating effect
b) augmented inflow effect

Fa (arterial gas concentration) is affected by ventilation/perfusion mismatching.

Nitrous Oxide



- The only inorganic anesthetic gas in clinical use
- Characterized by inert nature with minimal metabolism
- Colorless, odorless, tasteless, and does not burn
- Week Anesthetic good analgesic agent
- Major difference is low potency
- MAC value is 104%
- Needs other agents for surgical anesthesia
- Low blood solubility

Nitrous Oxide

Nitrous Oxide Systemic Effects

Cardiovascular

- Depress myocardial contractility
- Arterial BP, CO, HR: unchanged or slightly ↑ due to stimulation of catecholamines
- Constriction of pulmonary vascular smooth muscle → increase pulmonary vascular resistance
- Peripheral vascular resistance: not altered
- Higher incidence of epinephrine-induced arrhythmia

Nitrous Oxide

Nitrous Oxide Systemic Effects

Respiratory

- Respiratory rate: ↑
- Tidal volume: ↓
- Minute ventilation, resting arterial CO2: minimal change
- Hypoxic drive (ventilatory response to arterial hypoxia): ↓

Cerebral

- CBF, cerebral blood volume, ICP: ↑
- Cerebral oxygen consumption (CMRO2): ↑

Nitrous Oxide

Nitrous Oxide Systemic Effects

Neuromuscular

- Not provide significant muscle relaxation
- Not a triggering agent of malignant hyperthermia

Renal

- Increase renal vascular resistance
- Renal blood flow, glomerular filtration rate, U/O: ↓

Hepatic

- Hepatic blood flow: ↓

Gastrointestinal

- Postoperative nausea and vomiting

Nitrous Oxide

Nitrous Oxide Side Effects

- Beginning of case: second gas effect
- End of case: diffusion hypoxia
- Diffusion into closed spaces
- Inhibits methionine synthetase (precursor to DNA synthesis)
- Inhibits vitamin B-12 metabolism

Nitrous Oxide

Biotransformation & toxicity

- Almost all eliminated by exhalation
- Biotransformation < 0.01%
- Irreversibly oxidize Co in vit.B12 → inhibit vit.B12-dependent enzymes → interfere myelin formation, DNA synthesis
- Prolonged exposure → bone marrow suppression, neurological deficiencies
- Avoided in pregnant patients

Nitrous Oxide

Contraindications

- N2O diffuse into the cavity more rapidly than air (principally N 2) diffuse out
- Pneumothorax, air embolism, acute intestinal obstruction, intrac ranial air, pulmonary air cysts, intraocular air bubbles, tympanic membrane grafting
- Avoided in pulmonary hypertension

Drug interactions

- Due to high MAC, combination with more potent agents → de crease the requirement of other agents
- Potentiates neuromuscular blockade

Halothane

Halothane

- Halogen substituted ethane
- Volatile liquid easily vaporized, stable, and nonflammable
- Most potent inhalational anesthetic
- MAC of 0.75%
- Efficacious in depressing consciousness
- Very soluble in blood and adipose
- Prolonged emergence

Halothane

Halothane Systemic Effects

Cardiovascular

- Direct myocardial depression → dose-dependent reduction of a rterial BP
- Coronary artery vasodilator, but coronary blood flow ↓ due to systemic BP ↓
- Blunt the reflex: hypotension inhibits baroreceptors in aortic arc h and carotid bifurcation → vagal stimulation ↓ → compensator y rise in HR
- Sensitzes the heart to the arrhythmogenic effects of epinephrin e
- Systemic vascular resistance: unchanged

Halothane

Halothane Systemic Effects

Respiratory

- Rapid, shallow breathing
- Alveolar ventilation: ↓
- Resting PaCO2: ↑
- Hypoxic drive: severely depressed
- A potent bronchodilator, reverses asthma-induced bronchospas m
- Depress clearance of mucus → promoting postoperative hypoxi a and atelectasis

Halothane

Halothane Systemic Effects

Cerebral

- Dilating cerebral vessels → cerebral vascular resistance ↓ → CBF ↑
- Blunt autoregulation (the maintenance of constant CBF during changes in arterial BP)
- ICP: ↑, prevented by hyperventilation prior to administration of halothane
- Metabolic oxygen requirement: ↓

Neuromuscular

- Relaxes skeletal muscle
- A triggering agent of malignant hyperthermia

Halothane

Halothane Systemic Effects

Renal

- Renal blood flow, GFR, U/O: ↓
- Part of this can be explained by a fall in arterial BP and CO, preoperative hydration limits these changes

Hepatic

- Hepatic blood flow: ↓

Biotransformation & toxicity

- Oxidized in liver by cytochrome P-450

Halothane

Halothane Side Effects

Halothane Hepatitis" -- 1/35,000 cases

- oxidized in liver by cytochrome P-450 2EI to trifluroacetic acid
- fever, jaundice, hepatic necrosis, death
- immunologically mediated assault
- exposure dependent

Halothane

Halothane Side Effects

Malignant Hyperthermia-- 1/60,000

- **Classic** -- rapid rise in body temperature, muscle rigidity, tachy cardia, rhabdomyolysis, acidosis, hyperkalemia, DIC
- **physiology**--hypermetabolic state by inhibition of calcium reup take in sarcoplasmic reticulum
- **Diagnosis** -- previous symptoms, increase CO2, rise in CPK lev els, myoglobinuria
- autosomal dominant inheritance
- **Treatment** -- early detection, d/c agents, hyperventilate, bicarb, IV dantrolene (2.5 mg/kg), ice packs/cooling blankets, lasix/m annitol/ fluids.
- **ICU monitoring**

Halothane

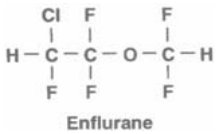
Contraindications

- Unexplained liver dysfunction following previous exposure
- No evidence associating halothane with worsening of preexisting liver disease
- Intracranial mass lesion, hypovolemic, severe cardiac disea se…

Drug interactions

- Myocardial depression is exacerbation by β-blockers and CCB
- With aminophylline → serious ventricular arrhythmia

Enflurane



Enflurane

- Developed in 1963 by Terrell, released for use in 1972
- Stable, nonflammable liquid
- MAC 1.68%
- Haloginated methyl ethyl ether.

Enflurane

Enflurane Systemic Effects

Cardiovascular:

- Inhibits sympathetic baroreflex response
- Sensitizes myocardium to effects of exogenous catecholamines – arrhythmias
- Potent inotropic and chronotropic depressant and decreases syste mic vascular resistance-- lowers blood pressure and conduction dramatically

Enflurane

Enflurane Systemic Effects

Respiratory

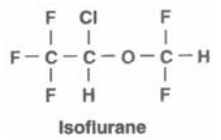
- drive is greatly depressed -- central and peripheral responses
- increases dead space
- widens A-a gradient
- produces hypercarbia in spontaneously breathing patient
- bronchodilator

Enflurane

Enflurane Side Effects

- Metabolism one-tenth that of halothane-- does not release quantity of hepatotoxic metabolites
- Metabolism releases fluoride ion-- renal toxicity
- Epileptiform EEG patterns

Isoflurane



Isoflurane

- Nonflammable,pungent
- MAC of 1.30 %
- Haloginated methyl ethyl ether
- A chemical isomer of enflurane

Isoflurane

Isoflurane Systemic Effects

Cardiovascular

- Minimal cardiac depression
- HR: ↑ due to partial preservation of carotid baroreflex
- Systemic vascular resistance: ↓ (Produces most significant reduction in systemic vascular resistance) → BP: ↓
- Dilates coronary arteries → coronary steal syndrome or drop in per fusion pressure → regional myocardial ischemia → avoided in patie nts with CAD
- Sensitizes myocardium to catecholamines -- less than halothane or enflurane

Isoflurane

Isoflurane Systemic Effects

Respiratory

- Respiratory depression, minute ventilation: ↓
- Blunt the normal ventilatory response to hypoxia and hypercap nia
- Irritate upper airway reflex
- A good bronchodilator

Cerebral

- CBF, ICP: ↑, reversed by hyperventilation
- Cerebral metabolic oxygen requirement: ↓

Neuromuscular

- Relaxes skeletal muscle

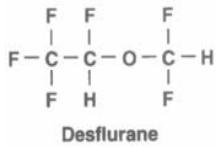
Isoflurane

Isoflurane Systemic Effects

Renal
- Renal blood flow, GFR, U/O: ↓

Hepatic
- Total hepatic blood flow: ↓

Desflurane



Desflurane

- Structure is similar to isoflurane
- High vapor pressure
- requires special vaporizer
- Low solubility → ultrashort duration of action
- Moderate potency
- MAC 6%

Desflurane

Desflurane systemic effects

Cardiovascular
- Systemic vascular resistance: ↓ → BP: ↓
- CO: unchanged or slightly depressed
- Rapid increases in concentration lead to transient elevation in HR, BP, catecholamine levels
- Not increase coronary artery blood flow

Desflurane

Desflurane systemic effects

Respiratory
- Tidal volume: ↓, respiratory rate: ↑
- Alveolar ventilation: ↓, resting PaCO2: ↑
- Depress the ventilatory response to ↑PaCO2
- Pungency and airway irritation

Cerebral
- Vasodilate cerebral vasculature → CBF, ICP: ↑, lowered by hyperventilation
- Cerebral metabolic rate of oxygen: ↓

Desflurane

Desflurane systemic effects

Neuromuscular
- Dose-dependent decrease in the response to train-of-four and tetanic peripheral nerve stimulation

Renal
- No evidence of any nephrotoxic effects

Hepatic
- No evidence of hepatic injury

Desflurane

Desflurane side effects

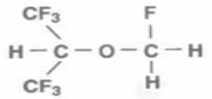
Degraded by desiccated CO2 absorbent into **carbon monoxide**

Desflurane

Contraindications
- Severe hypovolemia, malignant hyperthermia, intracranial hypert ension

Drug interactions
- Potentiate nondepolarizing NMBAs
- Emergence associated with delirium in some pediatric patients

Sevoflurane



Sevoflurane

- Nonpungency
- Rapid increase in alveolar anesthetic concentration
- Smooth and rapid inhalation inductions in pediatric and adult patients

Sevoflurane

Sevoflurane systemic effects

Cardiovascular
- Mildly depress myocardial contractility
- Systemic vascular resistance, arterial BP: ↓
- CO: not maintained well due to little rise in HR
- Prolong QT interval

Respiratory
- Depress respiration
- Reverse bronchospasm

Sevoflurane

Sevoflurane systemic effects

Cerebral
- CBF, ICP: slight ↑
- Cerebral metabolic oxygen requirement: ↓

Neuromuscular
- Adequate muscle relaxation for intubation of children

Renal
- Renal blood flow: slightly ↓
- Associated with impaired renal tubule function

Hepatic
- Portal vein blood flow: ↓
- Hepatic artery blood flow: ↑

Sevoflurane

Sevoflurane side effects

Biotransformation & toxicity
- Liver microsomal enzyme P-450
- Degraded by alkali (barium hydroxide lime, soda lime), producing nephrotoxic end products **(compound A)**

- accumulation of compound A increases with
  - increased respiratory gas temperature
  - low-flow anesthesia
  - dry barium hydroxide absorbent
  - high sevoflurane concentrations
  - time

Sevoflurane

Contraindications
- Severe hypovolemia, susceptibility to malignant hyperthermia, intracranial hypertension

Drug interactions
- Potentiate NMBAs
- Not sensitize the heart to catecholamine-induced arrhythmias

Xenon

- Nonexplosive, nonpungent, odorless and chemically inert
- No metabolism and low toxicity
- High cost
- MAC 71%
- It has some analgesic effect.
- Reduces anesthesia-emergent nausea and vomiting
- Very close to the 'ideal agent'
- Minimal haemodynamic effects.
- Seems not to trigger malignant hyperthermia.

Click to add title

Quantitation of Depth of Anesthesia in Humans

Anesthetic	MAC (% atm)	MAC with 60% N_2O (% atm)	MAC-Awake (% atm)	MAC-Intubation (% atm)	MAC-BAR (% atm)
Nitrous oxide	104	NA	66	>120	ND
Xenon	71	ND	31	ND	ND
Desflurane	7.25	4.0	2.60	ND	9.42
Sevoflurane	1.85	0.66	0.67	4.52	4.15
Isoflurane	1.15	0.50	0.37	1.76	1.50
Halothane	0.74	0.29	0.38	1.12	1.07

Respiratory Effects

- All cause respiratory depression
- Increased respiratory rate
- Decreased tidal volume
- CO_2 retention
- Decreased alveolar minute ventilation
- Abolish the hypoxic response at less than half MAC concentrations
- Fantastic bronchodilators by direct action on smooth muscle

Respiratory Effects

Type of breathing
- Rapid shallow breathing
- Decrease minute ventilation
- Increase PaCO2

Response to CO2
- Desflurane and Sevoflurane cause profound decrease in ventilat ion and CO2 accumulation

Hypoxic drive
- 0.1 MAC produce 50% depression
- 1.1 MAC produce 100% depression

Airway resistance
- They cause bronchodilation
- They can cause airway irritation

Cardiovascular Effects

- All cause cardiac depression
- Isoflurane and desflurane cause increased heart rate which may ma sk depression

Systemic vascular resistance

- Isoflurane and desflurane decrease (great for starting IVs)
- Halothane and nitrous oxide do not change
- Steal phenomena

Cardiovascular Effects

MAP

- N2O cause no or modest increase
- Halothane cause decrease by cardiac depression
- Others causes decrease by causing decrease SVR

HR

- Sevoflurane cause increase at > 1.5 MAC
- Halothane does not cause tachycardia.
- Others increase HR

CO & SV

- Halothane cause dose dependent decrease
- N2O cause slight increase (sympathomimetic)

CNS Effects

- Hypnotic, not analgesic
- Cause unconsciousness
- Spinal cord depression (lack of reflexes)

CNS Effects

EEG:

- <0.4 MAC increase the frequency and voltage
- >0.4 MAC abrupt shift of high voltage occures from posterior t o anterior of the brain.

CBF:

- >0.6 MAC in normocapnic pt. produce cerebral vasodilation an d results in dose dependent increase in CBF.

O2 requirements:

- All decrease

ICP

- All increase

Renal Effects

- All decrease arterial pressure
- They cause a dose related decrease in renal blood flow, glomerular filtration rate and urine output.
- RBF and GFR will be maintained until threshold of autoregulation
- Urine output is variable, depending on ADH and aldosterone and o ther agents
- Enflurane ..... nephrotoxic

Hepatic effects

Circulation

- Hepatic blood flow is maintained

Hepatic function

- Transient increase in liver enzymes

Skeletal muscle effects

NMJ

- They cause dose dependent potentiation of NMBD (except for N2O)

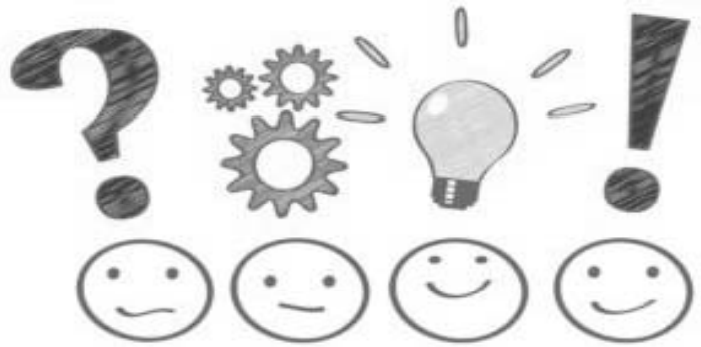
Malignant hyperthermia

Obstetric effects

- Produce dose dependent decrease in uterine contractility and bloo d flow
- may cause uterine atony
- They rapidly cross the placenta and reach the fetus

Ideal Characteristics

1. Be pleasant to inhale, permitting a smooth induction and emergenc e.
2. Be potent to allow the concomitant administration of high oxygen.
3. Rapid induction and emergence (low solubility).
4. Be easy to administer and analyze
5. Be easily and cheaply prepared in pure form.
6. Be stable in storage and with soda-lime, not flammable, not metab olized.
7. Act at specific CNS sites to cause unconsciousness.
8. No CV or respiratory effects, non-toxic to organ systems.
9. Provide postop pain relief.



MUSCLE RELAXANTS

Dr Walid Zuabi
FCA RCSI

MUSCLE RELAXANTS

-Neuromuscular blocking agents.

-It DOES NOT ensure amnesia, analgesia or loss of consciousness.

-Neuromuscular transmission:

The region between the motor neuron and the muscle cell.

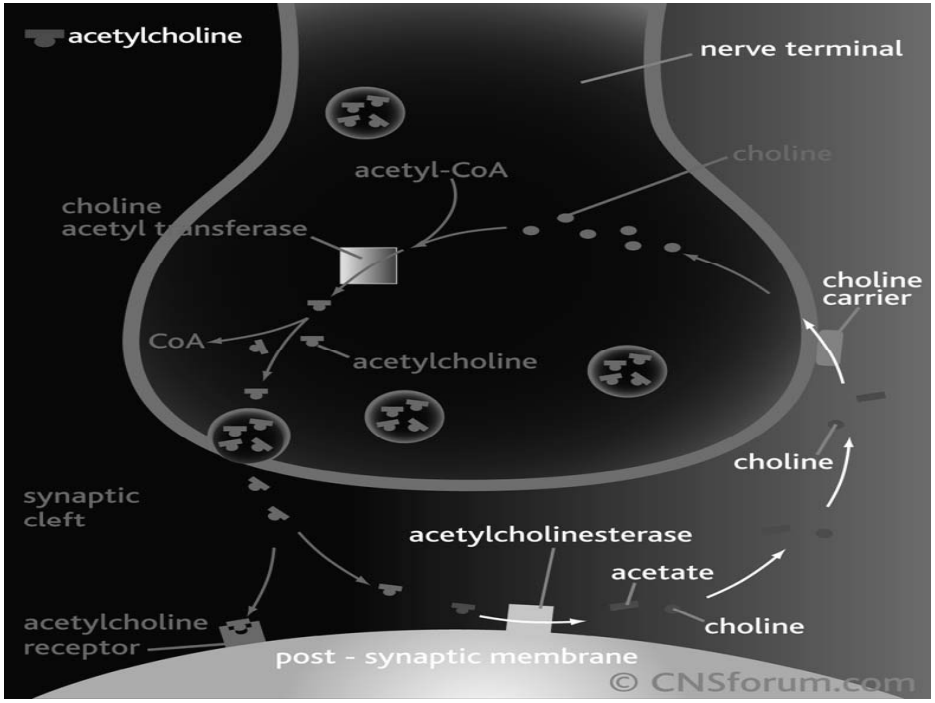
-Synaptic cleft: a narrow gap separates the cell membranes of the neuron and the muscle fiber.

-ACH.

-Nicotinic Cholinergic receptors.

-Action potential, ACH release ,channel opening [Na in and Ca release]…muscle contraction.

-ACH esteraze.. ACH hydrolysis, channels close..repolarization..relaxation.



Mechanism of action

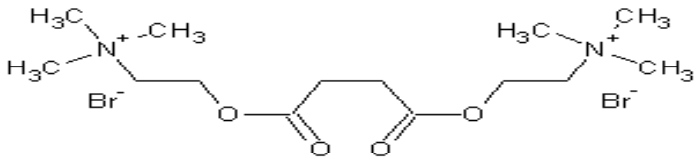
DMR: 2 ACH molecules,genereate action potential that cause prolonged depolarization and muscle relaxation.

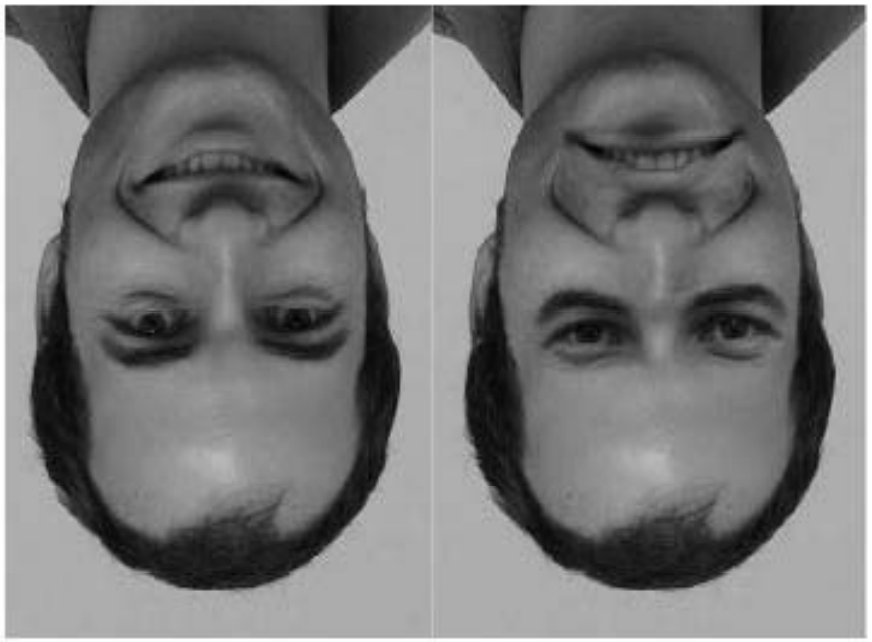
NDMR:it is a competitive ACH antagonist.. binds to the ACH receptors and prevent its action on the post- junctional plate…no action potential.

INDICATIONS

- Anaesthetic: when intubation indicated: full Stomach.
- Surgical: Long operations
  Microscopic operations
- N.B :DON'T GIVE THE MR BEFORE ENSURING ADEQUTE VENTILATION.

Suxamethonium
mg/ml





DMR

- Succinylcholine

  2 joined ACH molecules
- Rapid onset, short duration: use for RSI in emergencies.

Rapid metabolism by pseudocholinesterase: *low level in liver disease,pregnancy,renal failure.*

Suxamethonium mg/ml

Dose

- IV:1-1.5 mg/kg for dults
- 1-2 mg/kg in children due to higher ECF.
- IM: 5mg/kg.
- SIDE EFFECTS:

  Bradycardia Risk of **cardiac arrest, rhabdomyolysis hyperkalaemia** in patients with myopathies.
- Bradycardia Occurs due to stimulation of **muscarinic** receptors in the SA node…more common in children and after repeated doses of the drug. .

Suxamethonium mg/ml

- Fasciculations: disorganized visible motor unit contractions.
- Post-op.myalgia.
- Hyperkalaemia
- Increased intrgastric pressure.
- Increase of IOP
- Triggering of malignant hyperthermia
- Scoline apnea

NDMR

-Steroids: pancuronium,vecuronium,rocuronium.

-Benzyl isoquinolines : Atracurium, cisatracurium,mivacurium.

histamine release, extrahepatic metabolism.

-The glottic muscles is the most resistant one to block.

-Patients with liver cirrhosis or renal failure need adjustment of the drug type and dose.

ATRACURIUM:

-Metabolism by nonspecific esterases and hoffman elimination.

-Dose: iv 0.5 mg/kg.

-Duration: 30 minutes, more in hypothermic patients.

-Storage :2-8 c.

-Side effects: hypotension ,tachycardia, bronchospasm, laudanosine toxicity.

CISATRACURIUM

Stereoisomer of atracurium

Hoffman elimination

no histamine release.

-Dose : 0.15 mg/kg , intubation within 2 minutes, duration ½ hour.

MIVACURIUM

-Metabolized by pseudocholinesterase.

-Its action is prolonged in patients with liver failure,renal failure,pregnacy.

-Dose: 0.15-0.2 mg/kg.

-Onset: 2-3 minutes, duration: 20 minutes.

-Side effects: histamine release.

-Good choice when intubation is needed for short procedures.

PANCURONIUM

-Steroid ring with 2 ACH molecules.

-Hepatic metabolism.

-Renal and bile excretion.

-It is not the best in renal failure and cirrhosis.

-Dose: 0.1 mg/kg, duration: 45 min.

-S.E: Hypertension and tachycardia due to vagolytic effect,
catecholamine release…arrhythmias.
Allergic reactions.

VECURONIUM

-Steroid monoquaternary relaxant.

-Excretion mainly biliary then renal.

-prolonged action in RF.

-Dose: 0.1 mg/kg acts for 30-40 min.

-Store at room temp as powder.

+ve: stable CVS.
duration of action is not significantly prolonged in pts with cirrhosis.

ROCURONIUM
Esmeron

-Monoquaternary steroid analogue of vecuronium.

-Rapid onset of action, use for modified RSI.

-Liver and renal elimination.

-Prolonged action in hepatic failure and pregnancy.

-No active metabolite ..so can be used for infusions.

-Dose:0.6-1mg/kg. iv
2mg/kg IM for children.

-Duration of action depends on the dose.

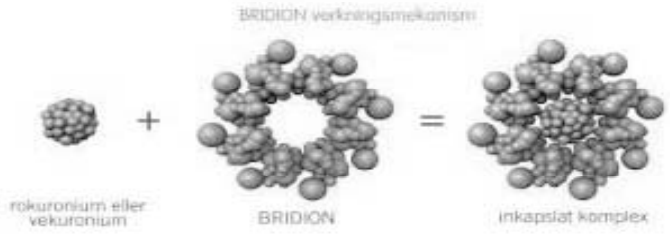
-Painful on injection, precipitation.

bridion®
sugammadex

bridion®
sugammadex

•Modified γ-cyclodextrin, with a lipophilic core and a hydrophilic periphery

bridion®
sugammadex



As you lecture, you keep watching the faces, and information keeps coming back to you all the time.

As you lecture, you keep watching the faces, and information keeps coming back to you all the time.

HYPOXIA AND OXYGEN THERAPY

Dr. Sami Abu- Halaweh
Prof.
The University of Jordan
Jordan University Hospital

HYPOXIA
(1) Definition

HYPOXIA:- is reduced Oxygen for tissue respiration

***Anoxia*:-** complete absence of oxygen in tissues

Hypoxia Vs Hypoxemia

PHYSIOLOGICAL PRINCIPLES
(1) Oxygen Delivery to the Tissues

This depends on three important factors:

1- The SUPPLY of oxygen during inspiration

2- The TRANSFER of oxygen from the alveoli to the pulmonary capillaries

3- The TRANSPORTATION of oxygen by the blood to the tissues

PHYSIOLOGICAL PRINCIPLES (CONT.)
(2) Oxygen Cascade

This represents the series of partial pressure of oxygen (PO2) decline from Atmosphere (at sea level) to the Mitochondria in the cell. It is formed by the following pressures:

1- Atmospheric Air = 160 mmHg (21 KPa)
2- Humidified Tracheal Gas = 150 mmHg (19.8 KPa)
3- The Alveolar Gas = 106 mmHg (14 KPa)
4- The Arterial Blood = 100 mmHg (13.3 KPa)
5- The Capillary Blood = 45-55 mmHg (6.7 KPa)
6- The Mitochondria = 7.5-40 mmHg (1-5 KPa)

Reduction of PO2 at any stage in the cascade causes reduction in subsequent steps. This risks inadequate mitochondrial PO2 for AEROBIC METABOLISM.

PHYSILOGICAL PRINCIPLES (CONT.)
(3) Oxygen Flux

Is the same as Available Oxygen or Oxygen Delivery, which is the amount of oxygen delivered to the tissues per unit time (usually per minute). This equals:

COxO2 bound to Haemoglobin + O2 dissolved in plasma

= CO/100(HbxSaO2x1.34)+(PaO2x0.003)

Where: CO = Cardiac Output in ml/min

Hb = Haemoglobin Concentration in g/dL

SaO2 = Arterial Oxygen Saturation of Haemoglobin

1.34 = Huefner`s Constant (amount of oxygen in ml bound to each gram of haemoglobin)

PaO2 = Partial Pressure of Oxygen in Arterial Blood in mmHg

0.003 = Amount of Oxygen in ml dissolved in plasma per mmHg PaO2

THE AMOUNT OF OXYGEN FLUX IS NORMALLY 850-1200 ML/MIN OR 500-700 ML/MIN/M^2

PHYSIOLOGICAL PRINCIPLES (CONT.)
(4) Oxygen Consumption

Is the amount of Oxygen consumed by the body per unit time (usually per minute). It is calculated as fallows:

O2 consumption = CO x (Arterial Oxygen Content – Mixed Venous Oxygen Content)

O2 Consumption = 240-270 ml/min AT REST (120-160 ml/min/m^2).

It is increased in : Fever, Sepsis, Shivering, Restlessness, Hypercatabolism

It is decreased in : Cooling, Paralysis, Mechanical Ventilation

HYPOXIA (CONT.)
(3) Acute Hypoxia

It can be due to :

1- Respiratory depression

2- Airway obstruction

3- Atelectasis

4- Ventilation/Perfusion mismatch

5- Reduced Functional Residual Capacity (FRC)

HYPOXIA (CONT.)
(4) Direct Effects of Acute Hypoxia

1- Cyanosis

2- Confusion, Drowsiness

3- Excitement

4- Headache

5- Nausea

6- Myocardial Depression

7- Arrhythmias

8- Bradycardia

9- Renal Impairment

HYPOXIA (CONT.)
(5) Indirect Effects of Acute Hypoxia

These are mediated through stimulation of Carotid and Aortic Bodies :

1- Tachycardia

2- Hypertension

3- Hyperventilation

HYPOXIA (CONT.)
(6) Degrees of Acute Hypoxia

Acute Hypoxia can cause the following according to the degree of oxygen saturation in arterial blood:

1- 85 % Saturation = Mental Impairment

2- 75 % Saturation = Severe Mental Impairment

3- 65 % Saturation = Unconsciousness

HYPOXIA (CONT.)
(7) Chronic Hypoxia

This develops after adaptation for high altitude and chronically developing lung diseases affecting oxygen transfer in the lung.

HYPOXIA (CONT.)
(8) Effects of Chronic Hypoxia

1- **Hyperventilation**

2- Polycythaemia

3- Increased 2-3-DPG

4- Proliferation of peripheral capillaries

5- Alteration in Intracellular Oxidative Enzymes

HYPOXIA (CONT.)
(2) Types of Hypoxia

1- **Hypoxic Hypoxia in inadequate arterial oxygenation**

2- **Anemic Hypoxia in inadequate hemoglobin content**

3- **Circulatory Hypoxia in inadequate perfusion**

4- **Histotoxic Hypoxia in inability of the cell to utilize oxygen**

Hypoxic Hypoxia

- Normal O2 carrying capacity and blood flow
- Low PaO2

Causes:
- Low PO2 in inspired air- high altitude
- Decreased pulmonary ventilation- airway obstruction, paralysis of respiratory muscle, narcotics
- Defect in exchange of gases through the membrane
- A-V shunts- cyanotic congenital heart disease

Characteristic features:

- PaO2- 40 mm Hg; PvO2- 2 mm Hg
- %O2 saturation- arterial 75%; venous 45%
- O2 content- arterial 14 ml/dL; venous 9 ml/ dL
- O2 utilization- 5 ml/ dL
- A-V PO2 difference- 15 mm Hg

- Hypoxic hypoxia

Via peripheral chemoreceptors

Respiratory center

Increased pulmonary ventilation

Reduced PaCO2

Shift to left of O2-Hb curve

Reduced O2 release from Hb

Tissue hypoxia

Anemic Hypoxia

- Arterial PO2 is normal
  - Reduced Hb content
  - Causes:

  (iv) Anemia

  (v) Carbon monoxide poisoning

- Characteristic features:
- → PaO2- 95 mm Hg; PvO2- 40 mm Hg
- → O2 content of blood is reduced
- Hypoxia is not severe at rest because of increase in 2,3 DPG
- Severe hypoxia during exercise

Carbon monoxide poisoning

- CO combines with Hb at the same point where O2 combines
- CO has 250 times more affinity than O2
- Shifts O2-Hb dissociation curve to left
- Treated with 100% O2

Stagnant Hypoxia

Normal Hb% and PaO_2

- Reduced blood flow to tissues
- Causes:

(iv) Circulatory failure

(v) Hemorrhage

Histotoxic Hypoxia

- Tissue utilization of O2 is impaired
- Normal O2 supply to tissues
- Cause: cyanide poisoning

- Characteristic features:
  - → PO2- arterial 95 mm Hg; venous 90 mm Hg
  - → %O2 saturation- arterial 97%; venous 96%
  - → O2 content- arterial 19 ml/dL; venous 18.5 ml/dL

Treatment of hypoxia

Oxygen administration is of importance in hypoxic hypoxia.

A. Inhalation of 100% O2 at normal atmospheric pressure

B. Hyperbaric oxygen therapy

OXYGEN THERAPY (CONT.)
(2) Oxygen Therapy Apparatus Requirements

1- Control of Fractional Inspired Oxygen Concentration (FiO2)
2- Prevention of CO2- Accumulation
3- Minimal Resistance to Breathing
4- Acceptable to the Patient

OXYGEN THERAPY
(1) Indications

1- **Cardio Pulmonary Resuscitation (CPR)**
2- **Respiratory Failure**
3- **Cardiac Failure**
4- **Shock of any Cause**
5- **Increased Metabolic Demands**
6- **Carbon Monoxide (CO)-Poisoning**
7- **Postoperative States**

Normobaric 100% O_2 therapy

It is useful in hypoxic hypoxia.
Dangers of inhaling 100% oxygen: -

- **Produce nasal congestion, throat pain, cough, substernal discomfort, etc by stimulating irritant receptors.**
- **Cause bronchopneumonia if given for more than 24 hrs by inhibiting alveolar macrophages**
- **Newborns should not be given more than 40% oxygen**

Hyperbaric O_2 Therapy

Useful in anemic, stagnant and histotoxic hypoxia

- **Inhalation of 100% O2 at 1 atm can increase arterial PO2 to 673 mm Hg as it includes PCO2 of 40 mm Hg and PH2O of 47 mm Hg**
- **At 1 atmospheric pressure, oxygen dissolved in plasma is 2ml/dL (i.e 673 x 0.003)**
- **3 atm of pure O2 will deliver resting O2 needs**

- **Oxygen toxicity will occur early**
- **Inhibits tissue enzyme activity**
- **Cerebral vasoconstriction**
- **Muscular twitches, tinnitus, convulsions and coma**

OXYGEN THERAPY (CONT.)
(3) Types of Oxygen Therapy Apparatus

1- Fixed Performance Systems : FiO2 is independent of patients factors :
- **A- High Flow Venturi -Type Mask**
- **B- Low Flow Anesthesia Breathing System (with CO2-absorber)**

2- Variable Performance Systems : FiO2 depends upon O2-flow and patient`s factors :
- **A- No Capacity Systems - Nasal Catheter with Low Flow**
- **B- Small Capacity Systems –**
  - **a- Nasal Catheter with High Flow**
  - **b- Simple Face Mask**
  - **c- Tracheostomy Mask or T-Piece**
  - **d- Face Tent**
- **C- Large Capacity Systems –**
  - **a- Soft Plastic Mask**
  - **b- Oxygen Head Box**
  - **c- Oxygen Tent**
  - **d- Incubator**

Acute Respiratory Failure

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RESPIRATORY FAILURE

- "inability of the lung to meet the metabolic demands of the body. This can be from failure of tissue oxygenation and/or failure of CO_2 homeostasis."

ACUTE RESPIRATORY FAILURE

DEFINITION

IT IS ASYNDROME OF INADEQUATE GAS EXCHANGE DUE TO DYSFUNCTION OF ONE OR MORE ESSENTIAL COMPONENTS OF RESPIRATORY SYSTEM.

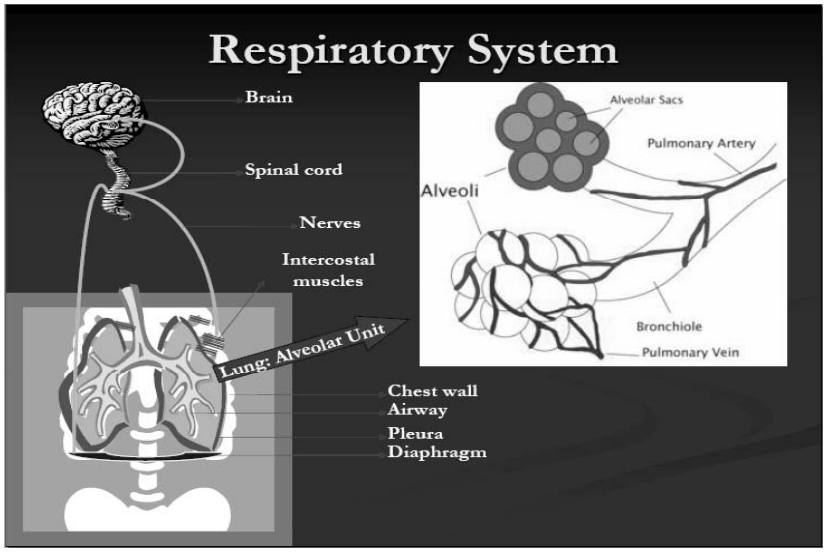
ACUTE RESPIRATORY FAILURE

Definitions

Hypoxemia is reduction in the oxygen content in the arterial blood system.

Tissue hypoxia is reduction in the oxygen delivery to the tissues, caused by reduction *oxygen content* and or reduction in *cardiac output.*

ACUTE RESPIRATORY FAILURE



RESPIRATORY FAILURE

Mechanisms of respiratory failure:

- the incapacity of the thoracic-pulmonary system to achieve a normal gas exchange at the pulmonary level (pulmonary respiratory failure);
- the incapacity of the cardio-vascular system to maintain an optimal tissue perfusion
  (e.g. referring to the shock states);
- the incapacity of tissues to use the oxygen brought by the arterial blood at the cellular level
  (e.g. septic shock, cyanide poisoning);

ACUTE RESPIRATORY FAILURE

- CLASSIFICATION

TYPE I OR HYPOXEMIC (PaO2 < 60 at sea level) : FAILURE OF OXYGEN EXCHANGE

INCREASED SHUNT FRACTION (QS/QT)

- *DUE TO ALVEOLAR FLOODING*
- *HYPOXEMIA REFRACTORY TO O2 SUPPLEMNT*

TYPE II OR HYPERCAPNIC PaCO2>50
FAILURE TO REMOVE OR TO EXCHANGE CO2
DECREASE ALVEOLAR MIN. VENTIL.(VA)
OFTEN ACCOMPANIED BY HYPOXEMIA

ACUTE RESPIRATORY FAILURE

TYPE III RESP.FAILURE : PERIOPERATIVE RESP FAILURE
INCREASED ATELECTASIS DUE TO LOW (FRC) IN SITTING OR ABNORMAL ABD.MECHANICS
RESLUT IN TYPE I OR II RESP FAILURE
AMELIORATED BY ANESTHETICS ,OP TECHNIQUE,POSTURE. INCENTIVE SPIROMETRY. ANALGESIA
TYPE IV RESP FAILURE : IN SHOCK
PATIENT VENTLATED , TO STABILIZE GAS EXCHANGE. TO LOWER RESP MUSCLE O2 CONSUMPTION

Classification of Respiratory Failure

- Type I
  - Oxygenation failure
    - ↓PaO_2,
    - NL - ↓PCO_2
    - ↑$PA\text{-}aO_2$
  - May be caused by any disorder leading to V/Q inequality, shunt, or diffusion impairment

Classification of Respiratory Failure

- Type I
  - Pneumonia
  - ARDS
  - Asthma
  - COPD
  - IPF

Classification of Respiratory Failure

- Type II
  - Ventilation failure
    - ↑PCO_2
    - ↓PaO_2
    - NL $PA\text{-}aO_2$
  - May be caused by any disorder leading to a decrease in minute ventilation

Classification of Respiratory Failure

- Type II
  - Drug intoxication
  - ALS
  - Guillain-Barre syndrome
  - Myasthenia Gravis
  - Polymyositis

Classification of Respiratory Failure

- Type III
  - Combined Oxygenation - Ventilation failure
    - ↓PaO_2
    - ↑PCO_2
    - ↑$PA\text{-}aO_2$
  - May be caused by any disorder leading to Type I failure, but only a few are common

Classification of Respiratory Failure

- Type III
  - ARDS
  - Asthma
  - COPD

Classification of Respiratory Failure

- Type IV
  - IN SHOCK
  - PATIENT VENTLATED , TO STABILIZE GAS EXCHANGE. TO LOWER RESP MUSCLE O2 CONSUMPTION

Classification of Respiratory Failure

- Type IV
  - Cardiogenic shock
  - Septic shock
  - Hypovolemic shock
  - Anaphylactic

ACUTE RESPIRATORY FAILURE

CLASSIFICATION

of Respiratory failure according to the evolution :

ACUTE

CHRONIC

ACUTE ON CHRONIC

Ex. Acute Exacerbation of COPD

Management of Respiratory Failure

- Establish a patent airway
  - Oral or nasal airway
  - Endotracheal intubation
- Maintain adequate oxygenation
  - Supplemental oxygen
- Maintain adequate ventilation
  - Mechanical ventilation
- Treat the underlying cause

HYPOXEMIC RESPIRATORY FAILURE(TYPE 1)

- PaO_2 <60mmHg with normal or low $PaCO_2$ $\rightarrow$ normal or high pH
- Most common form of respiratory failure
- Lung disease is severe to interfere with pulmonary O_2 exchange, but over all ventilation is maintained
- Physiologic causes: V/Q mismatch and shunt

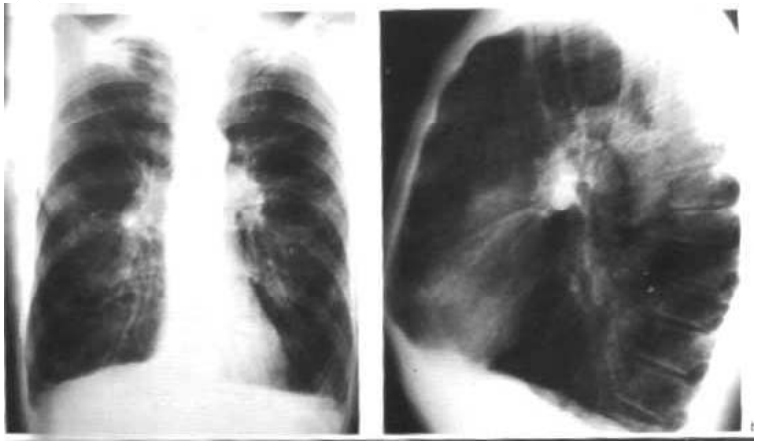
HYPOXEMIC RESPIRATORY FAILURE CAUSES OF ARTERIAL HYPOXEMIA

1. $\downarrow FiO_2$
2. Hypoventilation ($\uparrow PaCO_2$) — Hypercapnic Respiratory failure
3. V/Q mismatch (eg.COPD) — Hypercapnic Respiratory failure
4. Diffusion limitation ?
5. Intrapulmonary shunt
   - pneumonia
   - Atelectasis
   - CHF (high pressure pulmonary edema)
   - ARDS (low pressure pulmonary edema)

Causes of Hypoxemic Respiratory failure

- Caused by a disorder of heart, lung or blood.
- Etiology easier to assess by CXR abnormality:
  - Normal Chest x-ray
    - Cardiac shunt (right to left)
    - Asthma, COPD
    - Pulmonary embolism

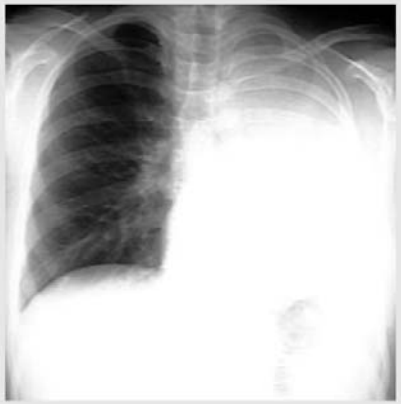
Hyperinflated Lungs : COPD



Causes of Hypoxemic Respiratory failure (cont'd.)

- Focal infiltrates on CXR
  - Atelectasis
  - Pneumonia

An example of intrapulmonary shunt

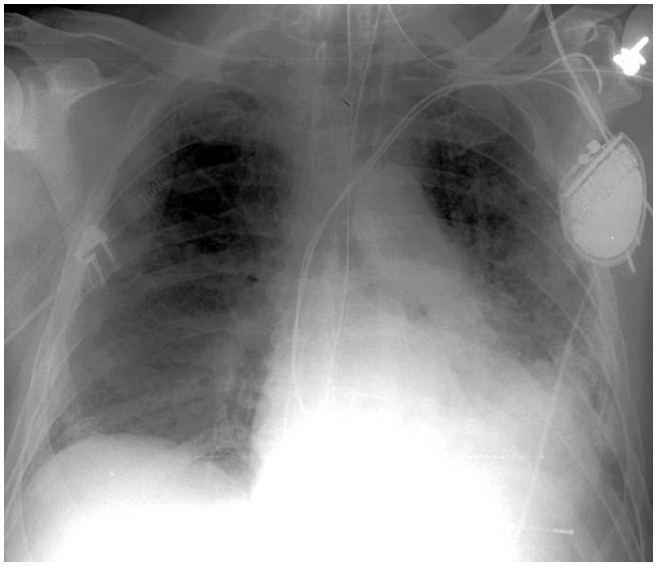


Causes of Hypoxemic Respiratory Failure (cont'd.)

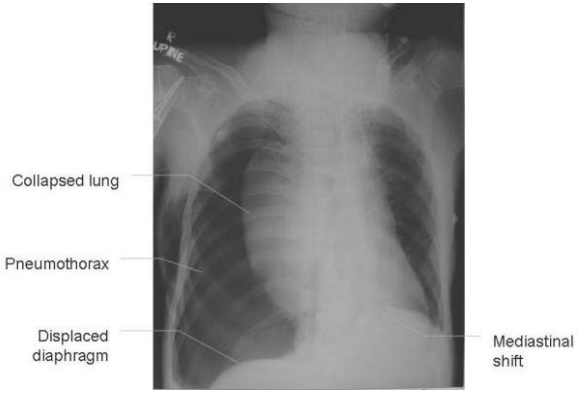
Diffuse infiltrates on CXR

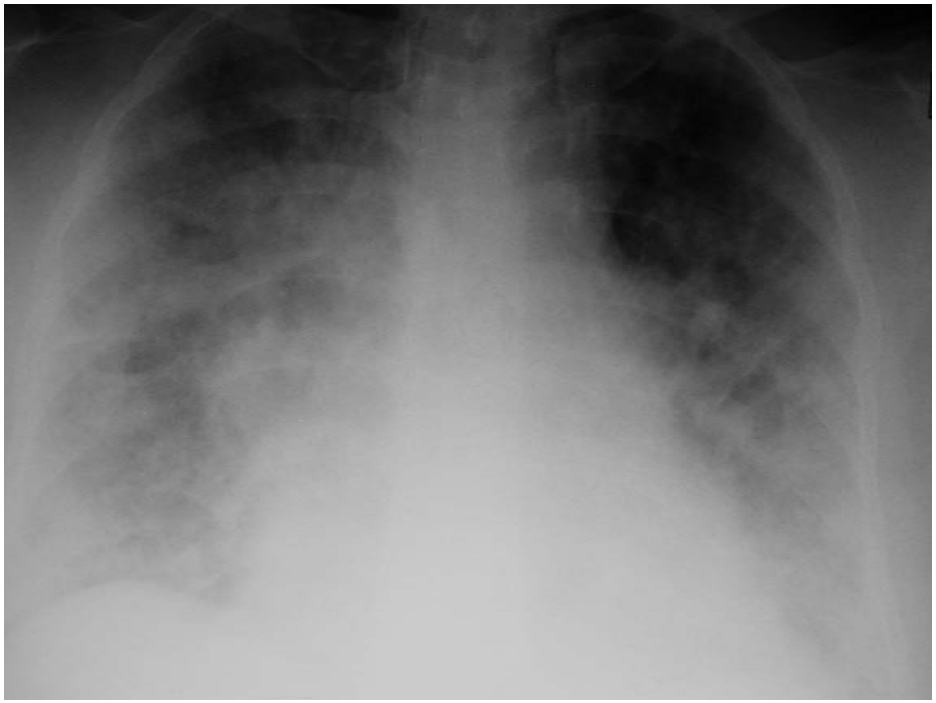
- Cardiogenic Pulmonary Edema
- Non cardiogenic pulmonary edema (ARDS)
- Interstitial pneumonitis or fibrosis
- Infections

Diffuse pulmonary infiltrates



TENSION PNEUMOTHORAX





Hypercapnic Respiratory Failure (Type II)

- $PaCO_2$ >50 mmHg
- Hypoxemia is always present
- pH depends on level of HCO_3
- HCO_3 depends on duration of hypercapnia
- Renal response occurs over days to weeks

Acute Hypercapnic Respiratory Failure (Type II)

- Acute
- Arterial pH is low
- Causes
  - sedative drug over dose
  - acute muscle weakness such as myasthenia gravis
  - severe lung disease: alveolar ventilation can not be maintained (i.e. Asthma or pneumonia)
- Acute on chronic:
- This occurs in patients with chronic CO_2 retention who worsen and have rising CO_2 and low pH.
- Mechanism: respiratory muscle fatigue

Causes of Hypercapnic Respiratory failure

- Respiratory centre (medulla) dysfunction
- Drug over dose, CVA, tumor, hypothyroidism,central hypoventilation
- Neuromuscular disease
  Guillain-Barre, Myasthenia Gravis, polio, spinal injuries
- Chest wall/Pleural diseases
  kyphoscoliosis, pneumothorax, massive pleural effusion
- Upper airways obstruction
  tumor, foreign body, laryngeal edema
- Peripheral airway disorder
  asthma, COPD

ACUTE RESPIRATORY FAILURE

Diagnosis : History

- Sepsis suggested by fever, chills
- Pneumonia -cough , Sputum , chest pain
- P E – dyspnea , chest pain
- COPD – Smoking. Cough. Sputum
- Cardiogenic pulmonary edema- chest pain. PND. Orthopnea

ACUTE RESPIRATORY FAILURE

Noncardiogenic Respiratory failure –
sepsis,Aspiration . Blood .Transfusion

Weakness ***– suggest Neuromuscular respiratory failure or toxins***

Exposure History – ASTHMA . Aspiration, Inhalational injury, Interstitial lung disease

Clinical and Laboratory Manifestation

(non-specific and unreliable)

- Cyanosis
  - bluish color of mucous membranes/skin indicate hypoxemia
- - unoxygenated hemoglobin 50 mg/L
  - not a sensitive indicator
- Dyspnea
  - secondary to hypercapnia and hypoxemia
- Paradoxical breathing
- Confusion, somnolence and coma
- Convulsions

ACUTE RESPIRATORY FAILURE

- DIAGNOSIS- *PHYSICAL FINDING*
- Circulatory changes
  - tachycardia, hypertension, hypotension
- Polycythemia
  - chronic hypoxemia - erythropoietin synthesis
- Pulmonary hypertension
- Cor-pulmonale or right ventricular failure

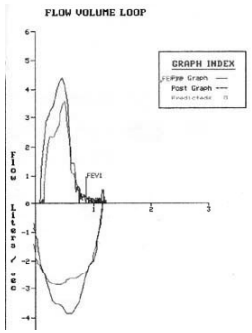
ACUTE RESPIRATORY FAILURE

- **Wheezing – Suggest A/W obstruction :**
  Bronchospasm
  upper or lower airway pathology
  Secretion
  Pulmonary edema
- ***Stridor suggests upper airway obstruction***
- ***Elevated jugular venous pressure suggests right ventricular dysfunction due to accompanying pulmonary hypertension***

ASSESSMENT OF PATIENT

- ABG analysis
  -classify RF and help with cause

  Lung function
- Chest Radiograph
- EKG
- Echocardiography



ACUTE RESPIRATORY FAILURE

- ***CBC***
- ***Cardiac serologic markers***
  Troponin I, (CK-MB)
- ***Microbiology***
  Cultures, Sputum , tracheal aspirate

- ***Blood , urine and body fluid***
- ***Bronchoscopy***

ACUTE RESPIRATORY FAILURE

- ***<u>Management</u>***

- **ABC's**
- **Ensure airway is adequate**
- **Oxygen therapy and assisted ventilation if needed**
- **Support circulation**

ACUTE RESPIRATORY FAILURE

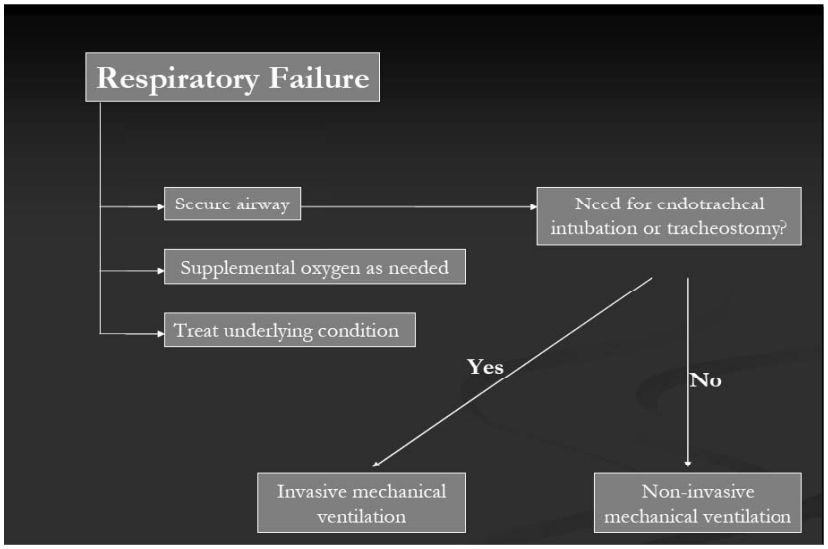
- ***Treatment of a specific cause when possible***
- ***Infection***
- ***Airway obstruction***
- ***Improve cardiac function***

  Positive airway pressure, diuretics, inotropy

ACUTE RESPIRATORY FAILURE

- **Mechanical ventilation**
- **Non-invasive (if patient a/w is protected and hemodynamically stable)**
- **Mask**
- **Invasive (Endotracheal tube)**

ACUTE RESPIRATORY FAILURE



ACUTE RESPIRATORY FAILURE

- **Indication for mechanical ventilation**
- Cardiac or respiratory arrest
- Tachy/bradypnea
- Respiratory acidosis
- Refractory hypoxemia
- Depressed level of consciousness
- Shock
- Inability to excrete secretions

ACUTE RESPIRATORY FAILURE

- ***Neuromuscular disease with V/C less than 10-15 ML/kg***
- ***Increased ICP***

ACUTE RESPIRATORY FAILURE

- Invasive vs. Non-Invasive ventilation
- Non-invasive in :

  COPD

  Cardiogenic pulmonary edema

  Obesity hypoventilation syndrome

  Asthma

- Pressure Limited Ventilators
  - ❖ CPAP
  - ❖ BIPAP

ACUTE RESPIRATORY FAILURE

- Goals of Mechanical Ventilation
- Improve ventilation by augmenting respiratory rate and tidal volume
- Assistant for neural or muscle dysfunction
- Sedated, paralyzed or comatose patient
- Neuropathy
- Intra-operative ventilation
- Correct respiratory acidosis
- Match respiratory demand
- Rest respiratory muscles

ACUTE RESPIRATORY FAILURE

- Correct hypoxemia

  High FIO2

  PEEP
- Improve cardiac function

  Decrease preload

  Decrease afterload

  Decrease metabolic demand

ACUTE RESPIRATORY FAILURE

- Permissive Hypercapnia

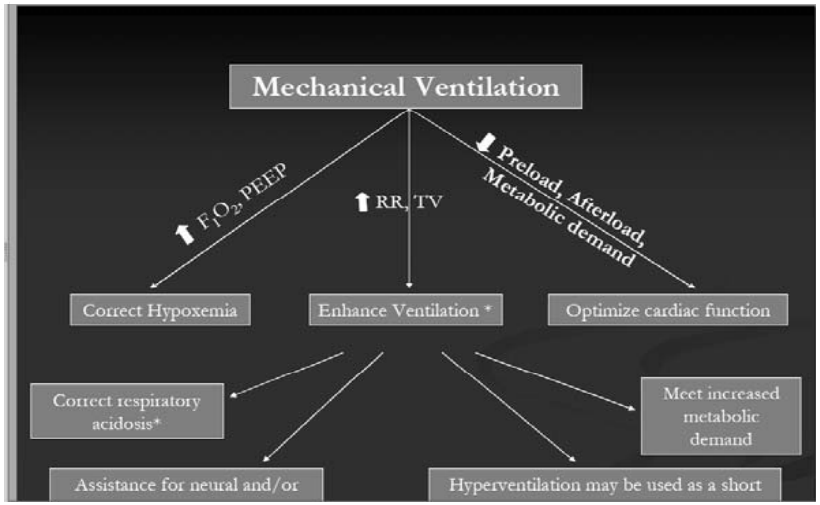
  Strategy that allows PaCO2 to rise by accepting lower alveolar minute ventilation to avoid specific risks:

  Dynamic hyperinflation (Auto-PEEP) and barotrauma in patients with asthma

  Ventilator associated with lung injury in ARDS

  Contraindicated in ICP

ACUTE RESPIRATORY FAILURE



ACUTE RESPIRATORY FAILURE

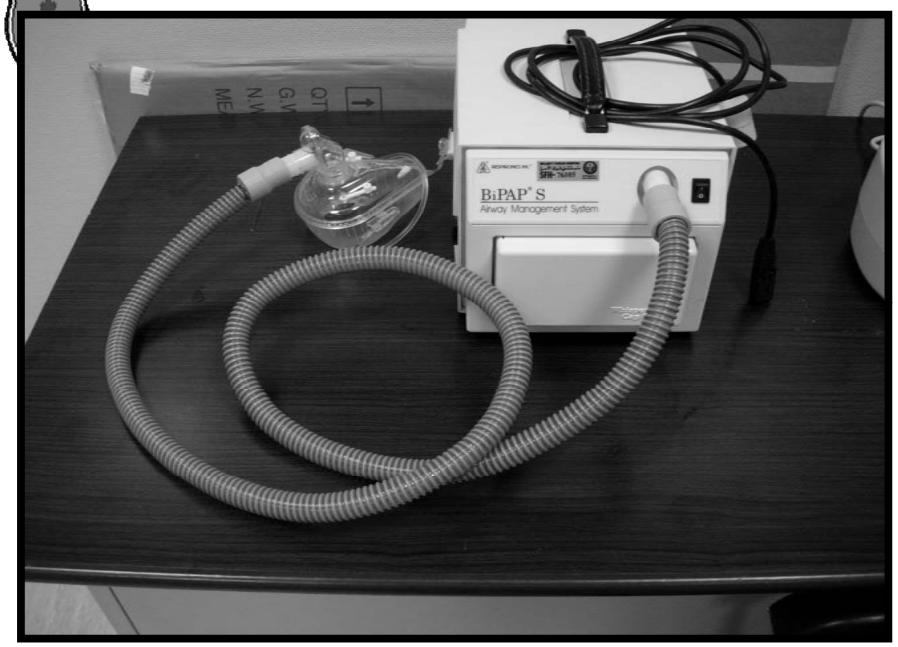
Other issues to consider when initiating Mechanical Ventilation

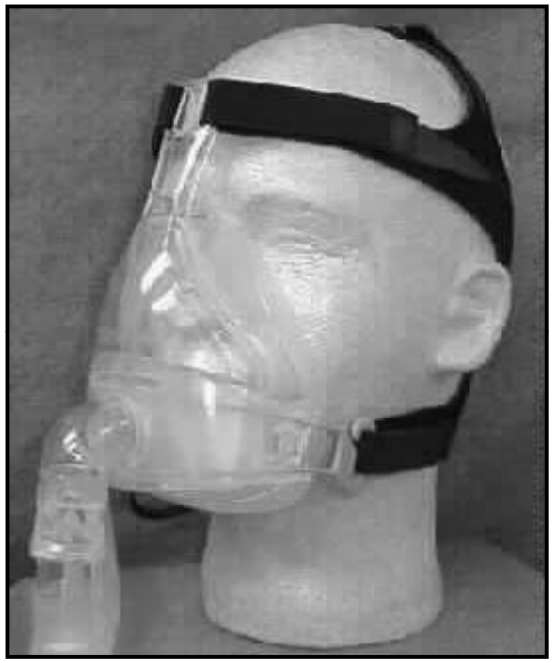
- Don't wait respiratory acidosis with evidence of :
- Inability to protect airway
- RR > 35 /minute
- Respiratory muscle fatigue
- Consider risks and benefits of mechanical vetilation

ACUTE RESPIRATORY FAILURE

Other issues in intubated and Ventilated patients

- Elevate head > 30 degree
- Ulcer and DVT prophylaxis
- In patient with ALI use small TV (6 ml/kg and pressure of 30 cmH2O
- Modify ventilation according to the patient





ACUTE RESPIRATORY FAILURE

- **Monitoring**

  Routine Monitoring
  ECG
  Blood Pressure
  Pulse Oximetry
  Temperature
  Capnography
  Blood gases

ACUTE RESPIRATORY FAILURE

CARE OF VENTILATED PATIENTS

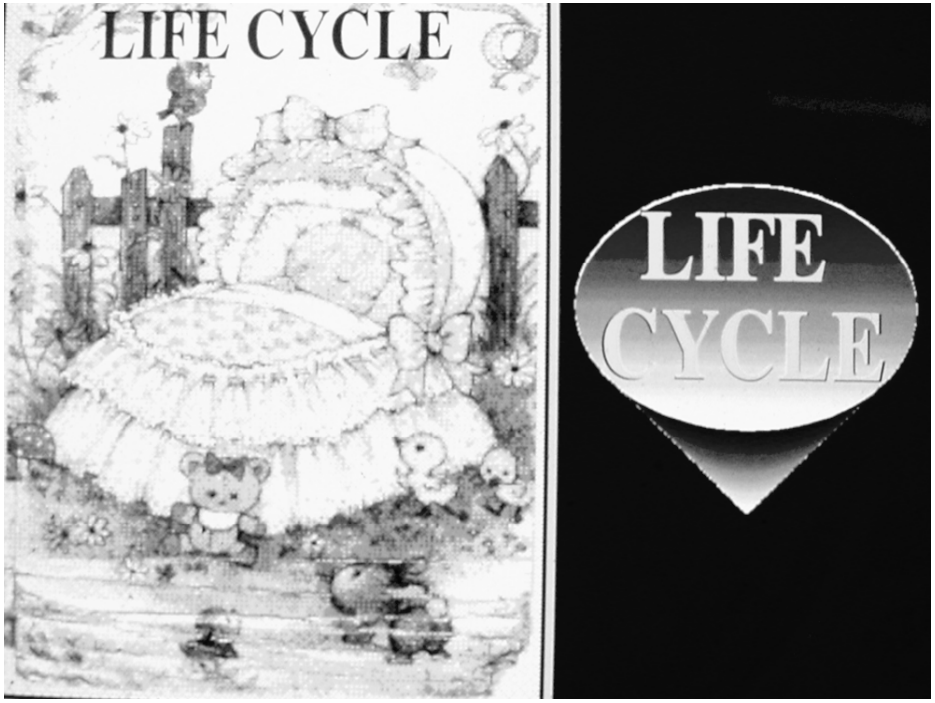
- SEDATION
- ANALGESIA
- NURSING CARE
- MUSCLE RELAXANTS ??? WEAKNESS.MYOPATHY AND (POST PARALYTIC SNDROME)

ACUTE RESPIRATORY FAILURE

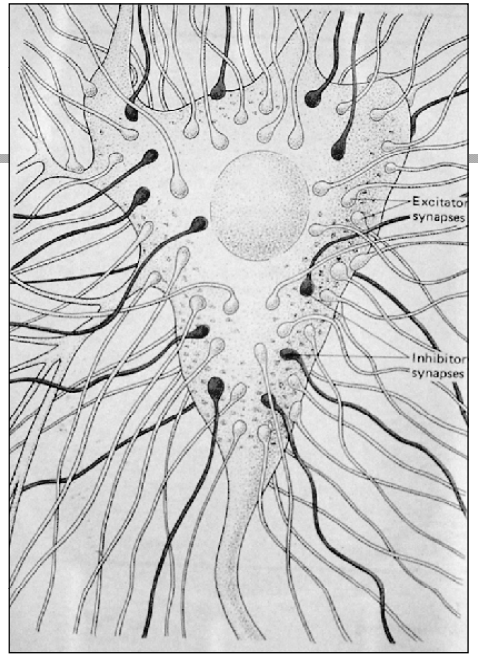
- Criteria for Extubation from Mechanical Ventilation

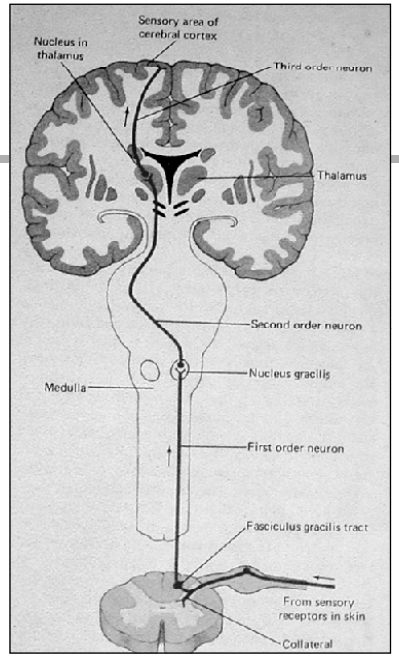
Parameter	Value
Pulmonary mechanics	
Vital capacity	> 10–15 mL/kg
Resting minute ventilation	> 10 L/min
(tidal volume × rate)	
Spontaneous respiratory rate	< 33 breaths/min
Lung compliance	> 100 mL/cm water
Negative Inspiratory force (NIF)	> –25 cm water
Oxygenation	
A–a gradient	< 300–500 mm Hg
Shunt fraction	< 15%
P_{O_2} (on 40% F_{IO_2})	> 70 mm Hg
P_{CO_2}	< 45 mm Hg

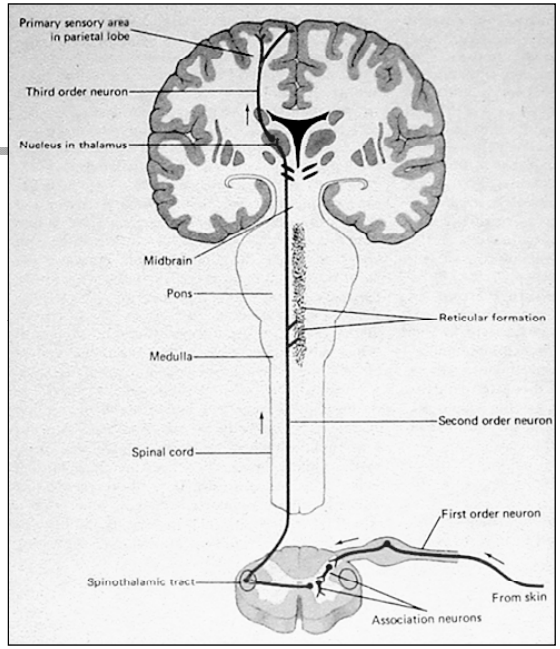
Thank you

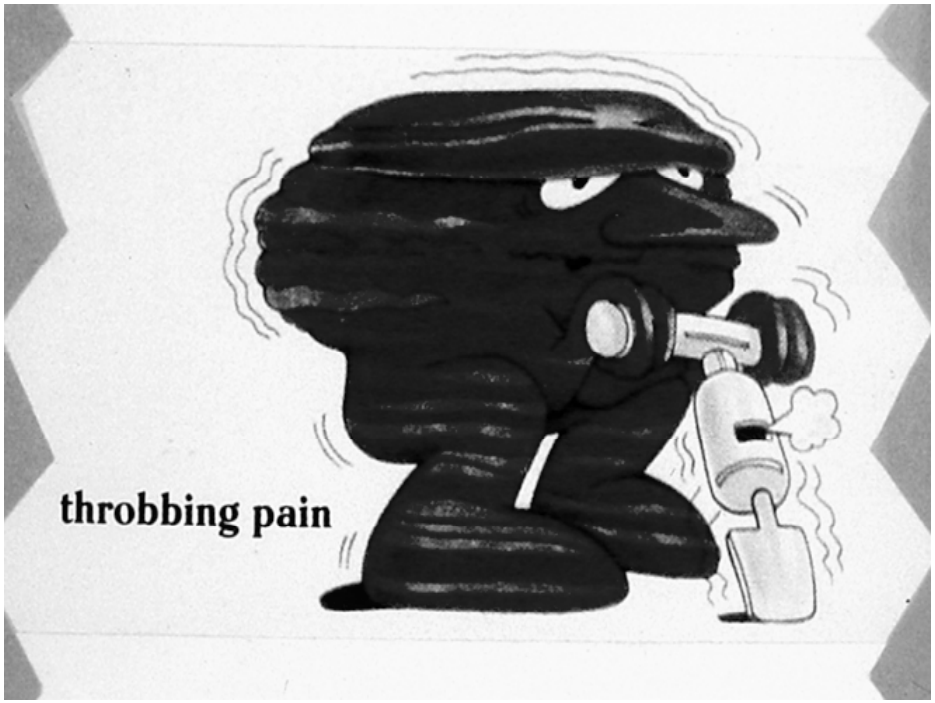

LIFE CYCLE
LIFE
CYCLE


Fear
Helplessness
PAIN
Sleep
Deprivation
Anxiety

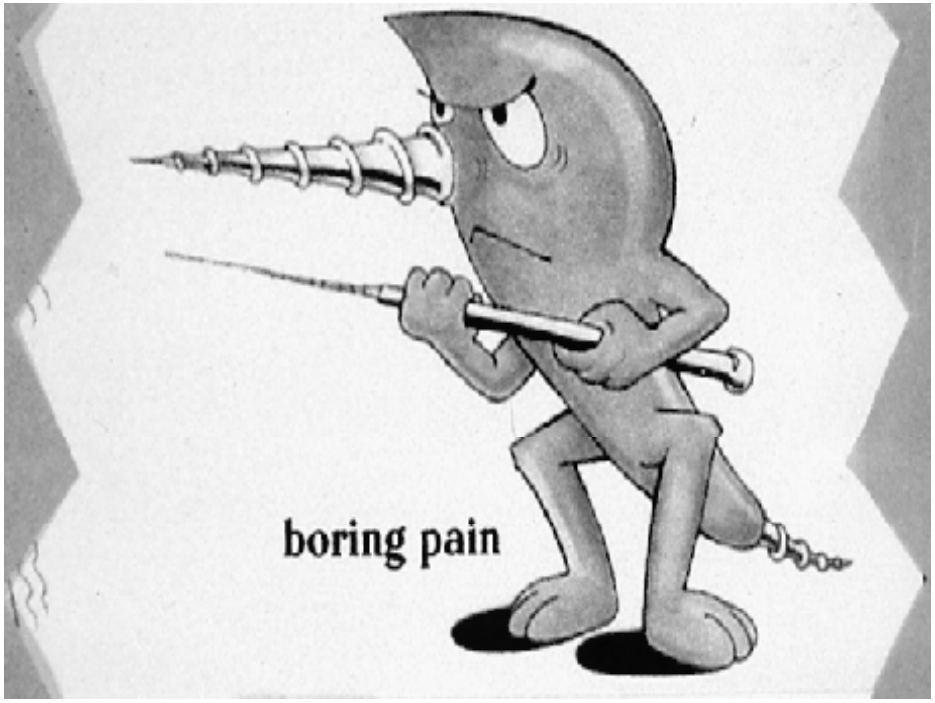

Excitator
synapses
Inhibitor
synapses


Sensory area of cerebral cortex
Nucleus in thalamus
Third order neuron
Thalamus
Second order neuron
Nucleus gracilis
Medulla
First order neuron
Fasciculus gracilis tract
From sensory receptors in skin
Collateral


Primary sensory area in parietal lobe
Third order neuron
Nucleus in thalamus
Midbrain
Pons
Reticular formation
Medulla
Second order neuron
Spinal cord
First order neuron
Spinothalamic tract
From skin
Association neurons

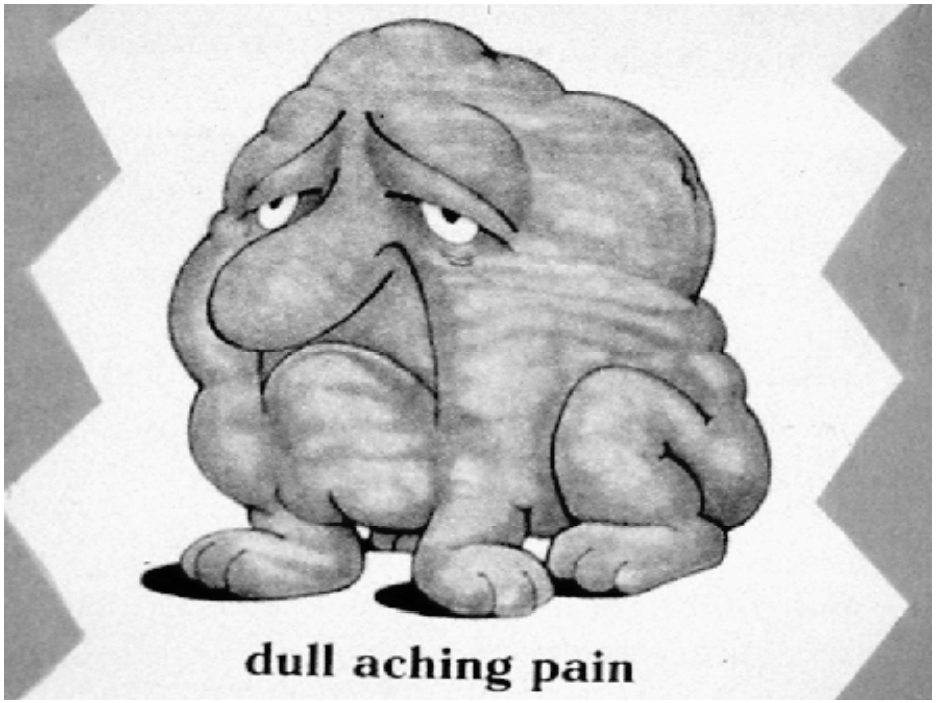

throbbing pain


stabbing pain


boring pain


nagging pain


sharp pain


dull aching pain

Visual analogue scale

No Pain — Worst Pain

The visual analogue scale provides a simple way of measuring pain.
You make amark on the line according to how bad your pain feels.

Treatment of pain

Pharmacologic Management

1-NSAIDS
2-OPIOID
3-ADJUVANT ANALGESICS

Non Drug Methods of Pain Relief

= Surgery
= TENS
= Acupuncture
= Hypnosis

Care of the Soul

Department of anesthesia and intensive care

The University of Jordan

2017

Anesthesia Monitoring in the Operating Room and ICU

Dr. Mustafa Alrabayah

Monitoring in the Past

- **Visual monitoring of respiration and overall clinical appearance**
- **Finger on pulse**
- **Blood pressure**

Introduction

Why do we need intraoperative monitoring???

- To maintain the normal pt physiology & homeostasis throughout anesthesia and surgery:
  induction, maintenance & recovery
  as much as possible. To ensure the well being of the pt.
- Surgery is a very stressful condition → severe sympathetic stimulation, HTN, tachycardia, arrhythmias.
- Most drugs used for general & regional anesthesia cause hemodynamic instability, myocardial depression, hypotension & arrhythmias.
- Under GA the pt may be **hypo** or **hyperventilated** and may develop **hypothermia**.
- Blood loss → anemia, hypotension. So it is necessary to recognise when the pt is in need of blood transfusion **(transfusion point)**.

Introduction

The most critical 2 times during anesthesia are:

INDUCTION - RECOVERY.

Exactly like "***flying a plane***" induction (= take off) & recovery (= landing).

The aim is to achieve a **smooth** induction , a **smooth** recovery & a **smooth** intraoperative course.

Introduction

Any monitor consists of:

1) **Sensor.**
2) **System for data collection.**
3) **System for interpretation.**

Introduction

Degree of invasiveness of monitoring

Non invasive	**ECG**
Penetrating	**ECHO (TEE)**
Invasive	**Arterial cannula**
Highly invasive	**Brain, heart canulation**

Introduction

Limitation of monitoring

1) **Delay.**
2) **Danger.**
3) **Decrease skill.**
4) **Doubt of results.**
5) **Distracting set up.**

ASA Monitoring Guidelines

STANDARD I

Qualified anesthesia personnel shall be present in the room throughout the conduct of all general anesthetics, regional anesthetics and monitored anesthesia care.

ASA Monitoring Guidelines

STANDARD II

During all anesthetics, the patient's oxygenation, ventilation, circulation and temperature shall be continually evaluated.

Respiratory system monitors

CVS monitors

Monitoring of metabolism

Neuromuscular Function

Invasive monitoring

CNS monitors

Respiratory system monitors ⇨

CVS monitors

Monitoring of metabolism

Neuromuscular Function

Invasive monitoring

CNS monitors

- **Clinical monitors.**
- **Airway pressure measurement.**
- **Disconnection alarm.**
- **Stethoscope**
- **Spirometery.**
- **O2 monitoring.**
- **Co2 monitoring.**
- **Anesthetic gas analysis.**
- **H+ ions measurement.**

Respiratory system monitors

CVS monitors ⇨

Monitoring of metabolism

Neuromuscular Function

Invasive monitoring

CNS monitors

- **Peripheral pulse.**
- **Tissue perfusion.**
- **ECG.**
- **Arterial blood pressure.**
- **Central venous catheterization**
- **Pulmonary artery catheterization.**
- **Cardiac output measurement.**
- **TEE.**
- **Blood loss measurement.**

Respiratory system monitors

CVS monitors

Monitoring of metabolism ⇨

Neuromuscular Function

Invasive monitoring

CNS monitors

- **Temperature monitoring.**
- **Tissue oxygenation monitoring.**
- **Indirect calorimetry.**
- **Fluid & electrolyte status**
- **Blood gases & acid base status monitoring.**

Respiratory system monitors

CVS monitors

Monitoring of metabolism

Neuromuscular Function ⇨

Invasive monitoring

CNS monitors

<u>Peripheral nerve stimulation:</u>

1) **Single twitch.**
2) **Train of four twitches.**
3) **Tetanic stimulation.**
4) **Double burst stimulation.**

Respiratory system monitors

CVS monitors

Monitoring of metabolism

Neuromuscular Function

Invasive monitoring ⇨

CNS monitors

- **Arterial line**
- **Central venous pressure**
- **Pulmonary artery catheterization**
- **ICP monitoring**

Respiratory system monitors

CVS monitors

Monitoring of metabolism

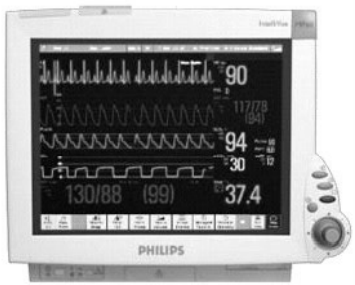
Neuromuscular Function

Invasive monitoring

CNS monitors ⇨

- **Clinical monitoring.**
- **EEG.**
- **Evoked potentials.**
- **Cranial nerve monitoring.**
- **Cerebral blood flow**
- **cerebral oxygenation.**
- **depth of anesthesia.**

How to select a monitor



Depend on

1) **Aim.**
2) **Experience.**
3) **Type of anesthesia.**
4) **Facilities & availability.**
5) **Nature of surgery.**
6) **General condition of the patient.**

Respiratory system monitors

Oxygenation

Ensure adequate oxygen concentration in inspired gas and blood

Methods

- inspired gas oxygen analyzer with alarms (GA)
- pulse oximetry
- exposure to assess color

Respiratory system monitors

Pulse Oximetry

Definition: % of oxy-Hb / oxy + deoxy-Hb.

Timing of SpO2 monitoring: throughout the surgery: before induction till after extubation & recovery.

It is the **LAST** monitor to be removed off the pt before the pt is transferred outside the operating room to recovery room.

SpO2 monitoring should be continued in recovery room.

❑Optical plethysmography
- detects pulsatile changes in blood volume

❑Spectrophotometry
- measures pulsatile hemoglobin saturation

Respiratory system monitors

Pulse Oximetry

Value:

- **SpO2**: arterial O2 saturation (oxygenation of the pt).
- **HR.**
- **Peripheral perfusion status** (loss of waveform in hypoperfusion states: hypotension & cold extremeties).
- Gives an idea about the **rhythm** from the plethysmography wave (arterial waveform). (Cannot identify the type of arrhythmia but can recognize if irregularity is present).
- **Cardiac arrest.**

N.B. Pulse oximeter tone changes with desaturation from high pitched to low pitched (deep sound). So just by listening to the monitor you can recognize: (1) HR (2) O2 saturation.

Respiratory system monitors

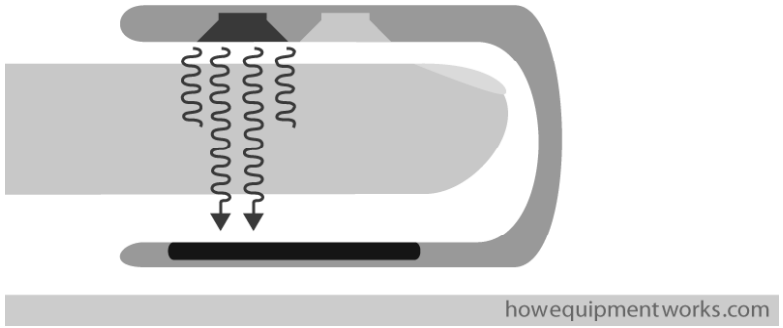
Pulse Oximetry

How to attach/apply saturation probe:

- To the **finger** or **toe**. The red light is applied to the nail.
  Nail polish and stains should be removed → false readings and artefacts
- Usually attached to the limb with the IV line (opposite the limb with the blood pressure cuff).

Respiratory system monitors

Pulse Oximetry



Respiratory system monitors

Pulse Oximetry

Readings:

•Normal person on room air (O2 = 21%) **> 96%.**

•Patient under GA (100% O2) = **98-100%.**

•It is not accepted for O2 saturation to ↓ below **96%** with 100% O2 under GA. Must search for a cause.

•**< 90%** = hypoxemia.

•**< 85%** = severe hypoxemia.

Respiratory system monitors

Pulse Oximetry

Inaccuracies occur when:

- Misplaced on the pts finger, slipped.
- Pt movement, shivering.
- Poor tissue perfusion (cold extremities)
- Poor tissue perfusion (hypotension & shock)
- Cardiac arrest.
- Sometimes by electrical interference from cautery in some monitors.

Respiratory system monitors

Pulse Oximetry

Disadvantages:

Inaccuracy…….if O2 sat less than 70%

Insensitivity …..significant decrease in PaO2 before significant decrease in SaO2 is detected.

Interference…..

- **Intrinsic** e.g. co-Hb, Met-Hb, I.V dyes, bilirubine, fetal Hb……
- **Extrinsic** e.g. motion, cautery, nail bed infection, polish……

Respiratory system monitors

O2 monitoring

(1) Monitor O2 delivery to the patient:

O2 failure alarm.

O2 conc. In the gas mixture

(2) Monitor O2 delivery to the tissues

- **clinical monitoring:**
  cap. refilling, state of extremities…
- **O2 transport monitoring through measurement of:**
  Hb level & SaO2 & PaO2
- **O2 uptake monitoring through:**
  $S\bar{v}O_2$ by pulmonary artery oximetry.
  serum lactic acid level.

Respiratory system monitors

Ventilation

Objective
- ensure adequate ventilation of patient

Methods

qualitative clinical signs
- chest excursion
- observation of reservoir bag
- auscultation of breath sounds

quantitative measurement
- end tidal carbon dioxide
- volume of expired gas
- continuous circuit disconnect monitor for mechanical ventilation

Respiratory system monitors

Capnography

Definition:

– What is Capnography?

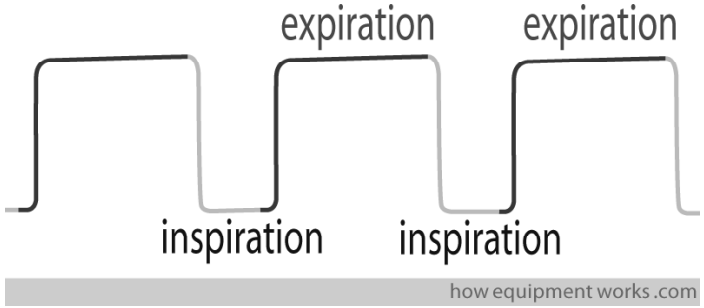
Continuous CO2 measurement displayed as a **waveform** sampled from the patient's airway during ventilation.

– What is EtCO2?

A **point** on the capnogram. It is the final measurement at the endpoint of the pts expiration before inspiration begins again. It is usually the highest CO2 measurement during ventilation.

Respiratory system monitors

Capnography

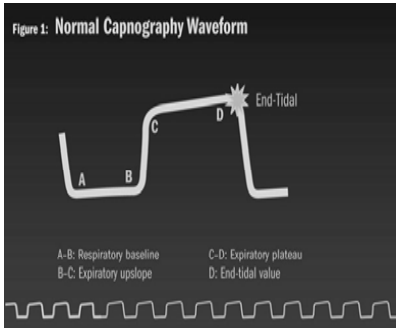


Respiratory system monitors

Capnography

Phases of the capnogram :

- Balseline: A-B
- Upstroke: B-C
- Plateau: C-D
- End-tidal: point D
- Downstroke



Respiratory system monitors

Capnography

- ❑ Theory
  - main or sidestream sampling
- ❑ Applications
  - confirmation of intubation
  - monitoring for circuit disconnection
  - identification of airway obstruction
  - rebreathing/metabolic monitoring

Respiratory system monitors

Capnography

Normal range:
30-35 mmHg. (Usually lower than arterial PaCO2 by **5-6 mmHg** due to dilution by dead space ventilation).

Value (data gained from capnography & ETCO2):

- **ETT**: esophageal intubation.
- **Ventilation**: hypo & hyperventilation, curare cleft (spontaneous breathing trials).
- **Pulmonary perfusion**: pulmonary embolism.
- **Breathing circuit**: disconnection, kink, leakage, obstruction, unidirectional valve dysfunction, rebreathing, exhausted soda lime.
- **Cardiac arrest:** adequacy of resuscitation during cardiac arrest, and prognostic value (outcome after cardiac arrest).

CVS monitors

Circulation

Objective

- ensure adequacy of circulatory function

Methods

- continuous electrocardiogram monitoring
- arterial blood pressure and heart rate q 5 min

CVS monitors

ECG

Value:

- Heart rate.
- Rhythm (arrhythmias) usually best identified from lead II.
- Ischemic changes & ST segment analysis.

Timing of ECG monitoring:

Throughout the surgery: before induction until after extubation & recovery

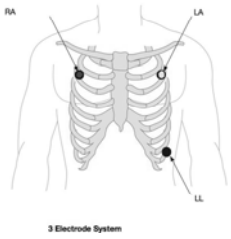
Types & connections of ECG cables:

- **3-leads**: **R**ed=**R**ight Ye**LL**ow=**L**eft **B**lack=A**p**ex (can read leads: I, II, III)
- **5-leads**: **R**ed=**R**ight Ye**LL**ow=**L**eft Black=under red Green=under yellow White=central (can read any of the 12 leads: I, II, III, avR, avL, avF, V1-V6).

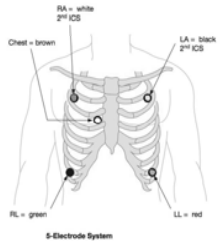
CVS monitors

ECG

- ❑ Heart rate measurement
  - R wave counting (any lead)
- ❑ Ischemia Monitoring
  - lead II and V_5 are 90% sensitive
  - lead II, V_5 and V_4 up to 98% sensitive
- ❑ Arrhythmia monitoring
  - lead II for supraventricular arrhythmias
  - all leads for ventricular arrhythmias



3 Electrode System



5-Electrode System

CVS monitors

ECG

RULES:

- QRS **beep ON** must be heard at all times. NO silent monitors.
- Remember that your **clinical judgement** is much more superior to the monitor. Check peripheral pulsations.
- Cautery → artefacts in ECG (noise/ electrical interference) → check radial (peripheral) pulse
- Arrythmias → check radial (peripheral) pulsations.

CVS monitors

Noninvasive Blood Pressure

- **Methodology**
  - oscillometric algorithms
  - automated
- **Limitations**
  - cuff size
    - oversize erroneously low measurements
    - too small erroneously high

CVS monitors

Noninvasive Blood Pressure

NIBP:
(non-invasive ABP monitoring = automated). Gives readings for:
systolic BP, diastolic BP & MAP

Value:
to avoid and manage extremes of hypotension & HTN.

Avoid ↓ MAP **< 60 mmHg** (for **cerebral** & **renal** perfusion)
avoid ↓ **diastolic pressur**

Risks of HTN episodes: → (CVS): myocardial ischemia, pulmonary edema, (CNS): hemorrhagic stoke, hypertensive encephalopathy.

While hypotensive episodes: (CVS): myocardial ischemia, (CNS): ischemic stroke, hypoperfusion state metabolic acidosis, delayed recovery, renal shutdown.

CVS monitors

Noninvasive Blood Pressure

Timing of BP monitoring:
throughout the surgery: before induction till after extubation & recovery.

Frequency of measurement:
- By default every **5** minutes.
- Every **3** minutes: immediately after spinal anesthesia, in conditions of hemodynamic instability, during hypotensive anesthesia.
- Every **10** minutes: eg. In awake pts under local anesthesia: "monitored anesthesia care" (minimal hemodynamic changes).

Monitoring of metabolism

Temperature

- **Objective**
  - aid in maintaining appropriate body temperature
- **Application**
  - readily available method to continuously monitor temperature if changes are *intended, anticipated or suspected*
- **Monitoring Sites**

- Tympanic
- Esophagus
- Bladder
- Rectum
- Blood (PA catheter)
- Skin

Monitoring of metabolism

Temperature

- Normal heat loss during anesthesia averages **0.5-1 C** per hour, but usually not more that **2-3 C**
- Temperature below 34C may lead to significant morbidity

(complications of hypothermia):

- Cardiac arrhythmias: VT & cardiac arrest.
- Myocardial depression.
- Delayed recovery (delays drug metabolism).
- Delayed enzymatic drug metabolism.
- Metabolic acidosis (tissue hypoperfusion → anerobic glycolysis → lactic acidosis)
- hyperkalemia
- Coagulopathy.

Monitoring of metabolism

Temperature

Hyperthermia Causes

- Malignant hyerthermia
- Endogenous pyrogens
- Excessive environmental warming
- Increases in metabolic rate secondary to:
  - Thyrotoxicosis
  - Pheochromocytoma

Neuromuscular Function

Evaluation of block

Peripheral nerve stimulation:

1) **Single twitch.**
2) **Train of four twitches.**
3) **Tetanic stimulation.**
4) **Double burst stimulation**.

Neuromuscular Function

Evaluation of Reversal of Blockade

❑ Clinical Criteria

- head lift > 5 seconds
- sustained hand grip
- negative inspiratory force
  - at least -55 cmH_2O for adults
  - at least -32 cmH_2O for children
- vital capacity 15 ml/kg
- absence of nystagmus or diplopia

Invasive monitoring

– Arterial line
– Central venous pressure
– Pulmonary artery catheterization

Invasive monitoring

Arterial Line

IBP: (invasive arterial blood pressure monitoring)

It is beat to beat monitoring of ABP via an **arterial** cannula.

Indications:

– Rapid moment to moment BP changes
– Frequent blood sampling
– Circulatory therapies: bypass, IABP, vasoactive drugs, deliberate hypotension
– Failure of indirect BP: burns, morbid obesity

Invasive monitoring

Arterial Line

Radial Artery Cannulation

• Technically easy
• Good collateral circulation of hand
• Complications uncommon except:
  – vasospastic disease
  – prolonged shock
  – high-dose vasopressors
  – prolonged cannulation

Invasive monitoring

Arterial Line

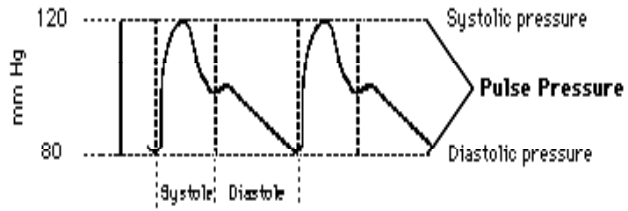
Alternative Sites

Brachial:
– Use longer catheter to traverse elbow joint
– Postop keep arm extended
– Collateral circulation not as good as hand

Femoral:
– Use guide-wire technique
– Puncture femoral artery below inguinal ligament (easier to compress, if required)

Invasive monitoring

Arterial Line



Invasive monitoring

Arterial Line

Complications of arterial cannulation

- **Hematoma.**
- **Vasospasm.**
- **Thrombosis.**
- **Embolization of air or thrombus.**
- **Skin necrosis, infection…..**
- **Nerve damage.**
- **Disconnection and fatal blood loss…..**

Invasive monitoring

Central Venous Line

Indications:

- CVP monitoring
- Advanced CV disease + major operation
- Secure vascular access for drugs
- Secure access for fluids
- Aspiration of entrained air: sitting craniotomies
- Inadequate peripheral IV access
- Pacer, Swan Ganz

Invasive monitoring

Central Venous Line

Advantages of RIJ

- Consistent, predictable anatomic location
- Readily identifiable landmarks
- Short straight course to SVC
- Easy intraop access for anesthesiologist at patient's head
- High success rate, 90-99%

Invasive monitoring

Central Venous Line

Alternative Sites

Subclavian:
- Easier to insert v. IJ if c-spine precautions
- Better patient comfort v. IJ
- Risk of pneumothorax - 2%

External jugular:
- Easy to cannulate if visible, no risk of pneumothorax
- 20%: cannot access central circulation

Femoral
- High infection rate
- No access for CVP readings

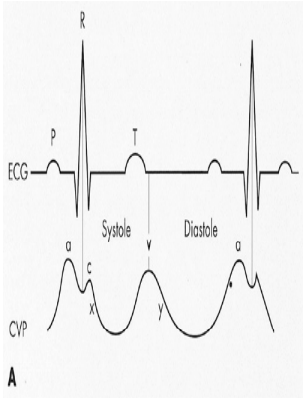
Invasive monitoring

Central Venous Line

- Reflects pressure at junction of vena cava + RA
- CVP is driving force for filling RA + RV
- CVP provides estimate of:
  - Intravascular blood volume
  - RV preload
- Trends in CVP are very useful
- Measure at end-expiration
- Zero at mid-axillary line

Invasive monitoring

Central Venous Line



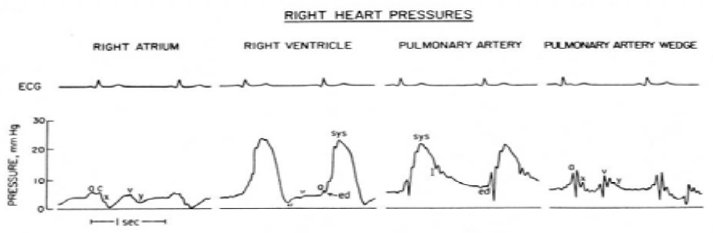
Component	Phase of Cycle	Event
a wave	End diastole	Atrial cont
c wave	Early systole	Isovol vent cont
x descent	Mid systole	Atrial relaxation
v wave	Late systole	Filling of atrium
y descent	Early diastole	Vent filling

Invasive monitoring

Pulmonary Artery Catheter

- Introduced by Swan + Ganz in 1970
- Allows accurate bedside measurement of important clinical variables:

  CO, PAP, PCWP, CVP to estimate LV filling volume, + guide fluid / vasoactive drug therapy



Invasive monitoring

Pulmonary Artery Catheter

Complications

•Minor in 50%, e.g., arrhythmias

•Transient RBBB- 0.9-5%
- External pacer if pre-existing LBBB

•Serious: 0.1-0.5%: pulmonary infarction, PA rupture (e.g., overwedge), endocarditis, structural heart damage

•Death: 0.016%

CNS monitors

Clinical monitoring:

Clinical monitoring:
Signs of pt awareness:
•Movement, grimacing (facial expression).
•Pupils dilated.
•Lacrimation.
•Tachycardia.
•HTN.

Sweating: is always an alarming/warning sign.
Causes
- Awareness.
- Hypoglycemia.
- Hypercapnia.
- Thyroid storm (thyrotoxic crisis).
- Fever.

Monitoring After Extubation & Recovery

BP:
within 20% of baseline.
SpO2:
> 92%
Breathing:
regular, adequate tidal volume.
Muscle power:
sustained head elevation for 5 seconds, good hand grip, tongue protrusion.
Level of consciousness:
fully conscious = 1) obeying orders, 2) eye opening 3) purposeful movement.

MOST IMP: Pt MUST be able to **protect his own airway**.

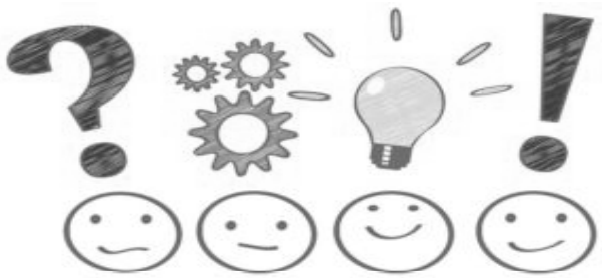
RULES NEVER to FORGET:

Never start induction with a missing monitor: ECG, BP, SpO2.

Never remove any monitors before extubation & recovery.

NEVER ignore an alarm

ALWAYS
- remember that ur **clinical sense** & judgement is be tter than & superior to any monitor.
- U are a doctor u are not a robot.
- The monitor is present to help u not to be ignored and not to cancel ur brain.



Anesthesia for Trauma Patients

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Anesthesia for Trauma

- Trauma is the leading cause of death between the ages of 1 and 45
- In the US preventable deaths decreased from 13% to 7% over the past decades because of more efficient systems of trauma care
- Anesthesia Care
  - Airway and Resuscitation in Emergency Department
  - Operating Room Care
  - Management in Intensive Care Unit

INTRODUCTION

The initial assessment of the trauma patient can be divided into :

1. Primary survey
2. Secondary survey
3. Tertiary survey

Prioritizing Trauma Care

- Do you know your ABC's ?

PRIMARY SURVEY

- ABCDE The primary survey should take 2–5 min

- **Airway**
  - Vocal Response, Auscultation
  - Chin Lift, Bag-Valve-Mask, 100% O2, Intubation, Cricothyriodotomy, Tracheostomy

- **Breathing**
  - Pulse Oximetry, Arterial Blood Gas, CXR
  - Mechanical Ventilation, Tube Thoracostomy

PRIMARY SURVEY

- **Circulation**
  - Vital Signs, Capillary Refill, Response to Fluid Bolus, CBC, Coagulation Studies, FAST, X-Ray
  - Adequate IV Access, Fluid Bolus, Pressure to Open Wounds, Thoracotomy, Transfusion, Surgery

- **Neurologic Disability**
  - GCS, Motor/Sensory Exam, Head, Neck, and Spine CT, Cervical Spine Films
  - Support Oxygenation/Perfusion, ICP Monitoring

- **Exposure**

Airway/Breathing

- Verification of adequate airway and acceptable respiratory mechanics is of primary importance

- Hypoxia is the most immediate threat to life

- Inability to oxygenate a patient will lead to permanent brain injury and death within 5 to 10 Minutes

PRIMARY SURVEY : Airway

- Establishing and maintaining an airway is always the first priority
- Important signs of obstruction include snoring or gurgling, stridor, and paradoxical chest movements.
- The presence of a foreign body should be considered in unconscious patients.
- Advanced airway management (such as endotracheal intubation, cricothyrotomy, or tracheostomy) is indicated if there is apnea, persistent obstruction, severe head injury, maxillofacial trauma, a penetrating neck injury with an expanding hematoma, or major chest injuries.

Airway obstruction

- Direct injury
  - Face, Mandible, or Neck
- Hemorrhage
  - Pharynx, Sinuses, and Upper airway
- Diminished Consciousness
  - Traumatic Brain injury, Intoxication, Analgesic medications
- Aspiration
  - Gastric contents, Foreign body
- Misapplication of Airway/Endotracheal Tube
  - Esophageal Intubation

Inadequate Ventilation

- Diminished Respiratory Drive
  - Traumatic Brain injury, Shock, Intoxication, Hypothermia, Over Sedation
- Direct Injury
  - Cervical Spine, Chest Wall, Pneumo/Hemothorax, Trachea, Bronchi, Pulmonary Contusion
- Aspiration
  - Gastric contents, Foreign body
- Bronchospasm
  - Smoke, Toxic Gas Inhalation

Indications for Endotracheal Intubation

- Cardiac or Respiratory Arrest
- Respiratory Insufficiency
- Airway Protection
- Deep Sedation or Analgesia
  - General Anesthesia
- Transient Hyperventilation
  - Space Occupying Intracranial Lesion/Increased ICP
- Delivery of 100% O2
  - Carbon Monoxide Poisoning
- Facilitation of Diagnostic Workup
  - Uncooperative or Intoxicated Patient

Anesthesia for traumatic patients is different from routine OR practice

- Most urgent cases occur during off-hours, when the most experienced OR and anesthesia personnel may not be available.
- In small hospital there are limited resources and equipments.
- Patient informations are limited; allergies, previous surgical and anesthetic history, which may create sudden crises.
- Patients are usually full stomach and the potential of cervical spine instability.

PRIMARY SURVEY :

Cervical spine injury is unlikely in alert patients without neck pain or tenderness. Five criteria increase the risk for potential instability of the cervical spine:

1. Neck pain
2. Severe distracting pain
3. Any neurological signs or symptoms
4. Intoxication
5. Loss of consciousness at the scene.

Cervical Spine Injury

- Trauma Patients
  - No Radiological Studies
    - Alert, Awake, and Oriented
    - No Neurological Deficits
    - No Distracting Pain
  - MRI Cervical Spine
    - Neck Pain
    - Cervical Tenderness to Palpation

Cervical Spine Injury

- All Other Trauma Patients
  - Lateral radiograph of cervical spine
  - Anteropostererior spinous process C2-T1
  - Open mouth odontoid view
  - Axial CT with reconstruction
    - Regions of questionable injury
    - Inadequate visualization

Protection of the Cervical Spine

- All blunt trauma victims should be assumed to have an unstable cervical spine until proven otherwise
- Direct laryngoscopy causes cervical motion and the potential to exacerbate spinal cord injury
- An “uncleared” cervical spine mandates In-line Stabilization (Not Traction)
- The front of the cervical collar may be removed for greater mouth opening and jaw displacement

Protection of the Cervical Spine

- Emergency Awake Fiberoptic Intubation
  - Requires less manipulation of the neck
  - Generally very difficult
    - Airway Secretions
    - Hemorrhage
    - Rapid Desaturation
    - Lack of Patient cooperation

PRIMARY SURVEY : Breathing

- Assessment of ventilation is best accomplished by the **look, listen, and feel** approach.
- **Look** for cyanosis, use of accessory muscles, flail chest, and penetrating or sucking chest injuries.
- **Listen** for the presence, absence, or diminution of breath sounds.
- **Feel** for subcutaneous emphysema, tracheal shift, and broken ribs.

PRIMARY SURVEY : Circulation

- **Adequacy of circulation** is based on pulse rate, pulse fullness, blood pressure, and signs of peripheral perfusion.
- **Signs of inadequate circulation** include tachycardia, weak or unpalpable peripheral pulses, hypotension, and pale, cool, or cyanotic extremities.
- **The first priority** in restoring adequate circulation is to stop bleeding
- **The second priority** is to replace intravascular volume.

Circulation

- Hemorrhage is the next most pressing concern
- Ongoing blood loss will be fatal in minutes to hours
- Shock is presumed to be a consequence of hemorrhage until proven otherwise

ATLS CLASSIFICATION OF HEMORRHAGIC SHOCK

	Class I	Class II	Class III	Class IV
Blood loss (ml)	≤750	750–1500	1500–2000	>2000
% blood loss	≤15	15–30	30–40	>40
Heart rate (bpm)	<100	>100	>120	>140
SBP	N	N	D	D
Pulse pressure	N or I	D	D	D
Capillary refill	N	I	I	I
Resp rate/ min	14–20	20–30	30–40	<35
Urine output (ml/hr)	>30	20–30	5–15	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious and confused	Confused and lethargic
Fluid replacement	crystalloid	Crystalloid and blood	Crystalloid and blood	Crystalloid, and blood

Symptoms of Shock

- What are the symptoms of shock ?

Symptoms of Shock

- Pallor
- Diaphoresis
- Agitation or Obtundation
- Hypotension
- Tachycardia
- Prolonged Capillary Refill
- Diminished Urine Output
- Narrow Pulse Pressure

Early Resuscitation

- Maintain SBP at 80-100 mm Hg
- Maintain Hematicrit at 25-30%
- Maintain PT/PTT in normal range
- Maintain Platelet count > 50,000
- Maintain Normal serum ionized calcium
- Maintain core temperature > 35° C
- Prevent increase in serum lactate
- Prevent Acidosis

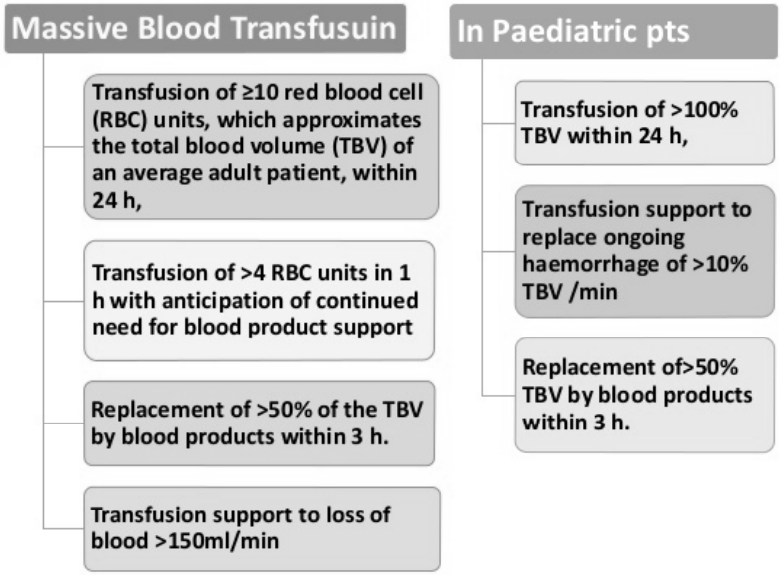
Intravenous Access

- Order of Desirability
  - Large-bore (16g or greater) antecubital vein
  - Other large-bore peripheral veins
  - Subclavian vein
  - Femoral vein
  - Internal jugular vein (Requires removal of cervical collar and neck manipulation)
  - Intraosseous (Tibia or distal end of femur)

Fluid Infusion System

- Active fluid administration up to 1500 ml/min
- Compatible with crystalloid, colloid, RBC, plasma, washed/salvaged blood (Not platelets)
- Reservoir allows for mixing of products
- Controlled temperature (38° -40° C)
- Able to pump through multiple IV lines
- Fail safe detection system to prevent infusion of air
- Accurate recording of volume administered
- Portable to travel with patients between units

Definition of Massive Transfusion



Rule (1:1:1)

- When the patient is in shock, however, and blood loss is likely to be substantial, platelets should be empirically administered in proportion to RBC and plasma.

- Platelets, RBCs, Plasma (1:1:1)

Risks of Aggressive Volume Replacement

- Increased blood pressure
- Decreased blood viscosity
- Decreased hematocrit
- Decreased Clotting factors
- Greater transfusion requirements
- Electrolyte imbalance
- Direct immune suppression
- Premature reperfusion

PRIMARY SURVEY

- **Disability**

  Evaluation for disability consists of a rapid neurological assessment. Because there is usually no time for a Glasgow Coma Scale, the **AVPU system** is used: *a*wake, *v*erbal response, *p*ainful response, and *u*nresponsive.

- **Exposure**

  The patient should be undressed to allow examination for injuries. In-line immobilization should be used if a neck or spinal cord injury is suspected.

Glasgow Coma Score

- What is the Glasgow Coma Score ?

Glasgow Coma Score

- Eye Opening Response
  - 4=Spontaneous
  - 3=To Speech
  - 2=To Pain
  - 1=None
- Verbal Response
  - 5=Oriented to Name
  - 4=Confused
  - 3=Inappropriate Speech
  - 2=Incomprehensible Sounds
  - 1=None

Motor Response

6=Follows Commands
5=Localizes to Pain
4=Withdraws from Pain
3=Abnormal Flexion (Decorticate Posturing)
2=Abnormal Extension (Decerebrate Posturing)
1-None

SECONDARY SURVEY

- The secondary survey begins only when the ABCs are stabilized.
- In the secondary survey, the patient is evaluated from head to toe and the indicated studies (eg, radiographs, laboratory tests, invasive diagnostic procedures) are obtained.
- Head examination includes looking for injuries to the scalp, eyes, and ears.
- Neurological examination includes the Glasgow Coma Scale and evaluation of motor and sensory functions as well as reflexes.
- The chest is auscultated and inspected again for fractures and functional integrity (flail chest).
- Examination of the abdomen should consist of inspection, auscultation, and palpation.
- The extremities are examined for fractures, dislocations, and peripheral pulses.
- A urinary catheter and nasogastric tube are also normally inserted.

SECONDARY SURVEY

- Basic laboratory analysis includes a complete blood count (or hematocrit or hemoglobin), electrolytes, glucose, blood urea nitrogen (BUN), and creatinine.
- Arterial blood gases may also be extremely helpful.
- A chest X-ray should be obtained in all patients with major trauma.
- The possibility of cervical spine injury is evaluated by examining all seven vertebrae in a cross-table lateral radiograph and a swimmer's view.
- Depending on the injuries and the hemodynamic status of the patient, other imaging techniques (eg, chest computed tomography [CT] or angiography) or diagnostic tests such as diagnostic peritoneal lavage (DPL) may also be indicated.

Tertiary Survey

- A tertiary survey is defined as a patient evaluation that identifies and catalogues all injuries after initial resuscitation and operative interventions.
- Many trauma centers also advocate a tertiary trauma survey (TTS) to avoid missed injuries.
- Between 2% and 50% of traumatic injuries may be missed by primary and secondary surveys, particularly following blunt multiple trauma
- The tertiary survey occurs prior to discharge to reassess and confirm known injuries and identify occult ones.

Traumatic Brain Injury

- Anesthetic Management
  - Avoidance of Hypoxemia
    - Intubation
      - Airway protection
      - Controlled Hyperventilation
      - Uncooperative/Combative Patient
      - GCS < 8
  - Control Hemodynamics
    - Avoid Hypotension
    - Fluid Administration
    - Vasopressors
    - Arterial Line

Traumatic Brain Injury

- Management of Cerebral Circulation
  - Hyperventilation
    - PaCO2 at 35 mmHg
    - PaCO2 at 30 mmHg for episodes of elevated ICP
  - Mannitol
    - 0.5-1g/kg
  - Barbiturate

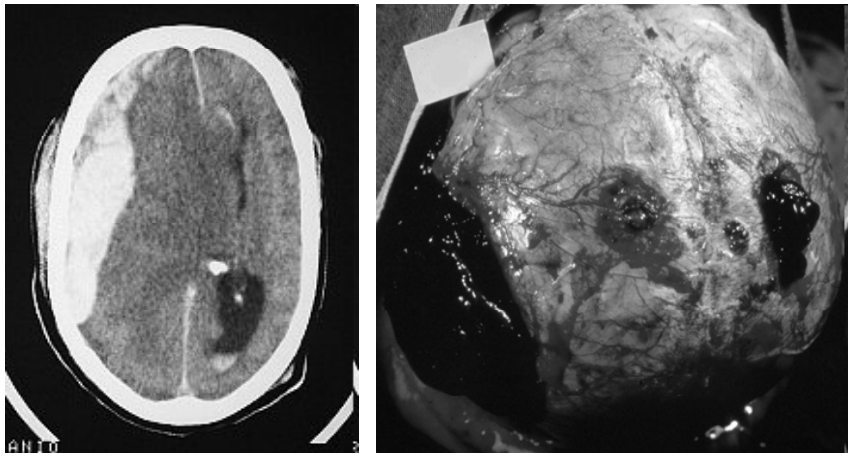
Traumatic Brain Injury

- Temperature
  - Avoid Severe Hypothermia
  - Do not warm aggressively
    - Hyperthermia increases CMRO2
- Position Therapy
  - Elevation of Patients Head
    - Facilitate venous drainage
    - Lower ICP
    - Improved Ventilation/Perfusion

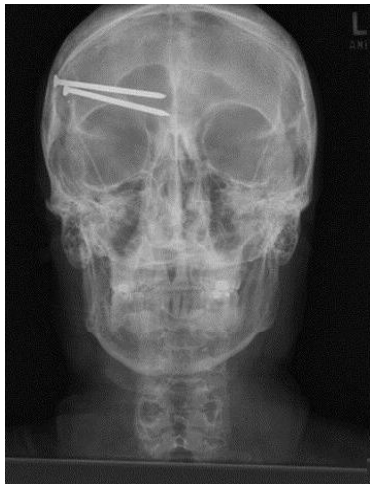
Head & Spinal Cord Trauma

- Any trauma victim with altered consciousness must be considered to have a brain injury
- The level of consciousness is assessed by serial Glasgow Coma Scale evaluations
- Common injuries requiring immediate surgical intervention include epidural hematoma, acute subdural hematoma, and some penetrating brain injuries and depressed skull fractures.
- Intracranial hypertension is controlled by a combination of fluid restriction (except in the presence of hypovolemic shock), diuretics (eg, mannitol, 0.5 g/kg), barbiturates, and deliberate hypocapnia (PaCO2 of 28–32 mm Hg).

SUBDURAL HEMATOMA



PENETRATING BRAIN INJURY

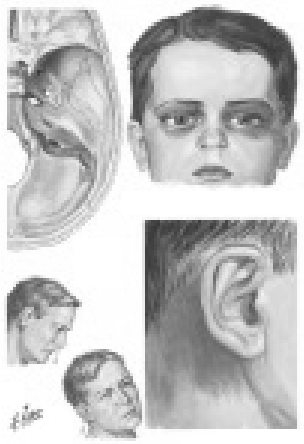


Depressed skull fracture

Head & Spinal Cord Trauma

- Nasal passage of an endotracheal tube or nasogastric tube in patients with basal skull fractures risks cribriform plate perforation and CSF infection.
- Because autoregulation of cerebral blood flow is usually impaired in areas of brain injury, arterial hypertension can worsen cerebral edema and increase intracranial pressure.
- Patients with severe head injuries are more prone to arterial hypoxemia from pulmonary shunting and ventilation/perfusion mismatching.

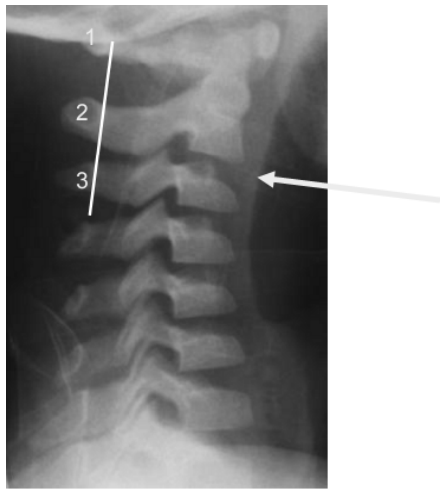
Signs of base of skull fracture



Head & Spinal Cord Trauma

- Lesions of the cervical spine may involve the phrenic nerves (C3–C5) and cause apnea.
- High thoracic injuries will eliminate sympathetic innervation of the heart (T1–T4), leading to bradycardia.
- Acute high spinal cord injury can cause **spinal shock,** a condition characterized by loss of sympathetic tone in the capacitance and resistance vessels below the level of the lesion, resulting in hypotension, bradycardia, areflexia, and gastrointestinal atony.
- Succinylcholine is reportedly safe during the first 48 h following the injury but is associated with life-threatening hyperkalemia afterward.

Pseudosubluxation



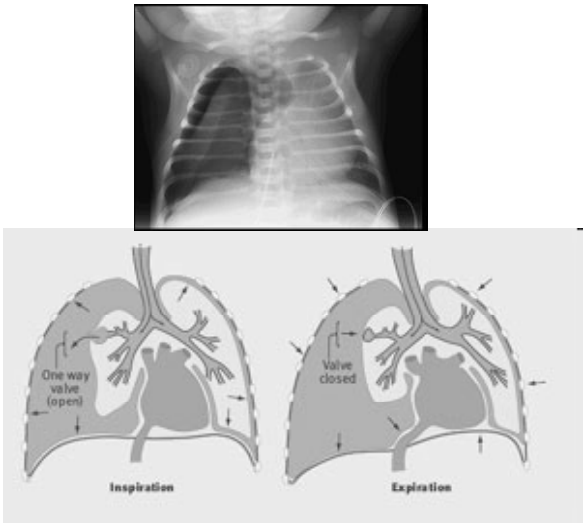
Head & Spinal Cord Trauma

- Short-term high-dose corticosteroid therapy with methylprednisolone (30 mg/kg followed by 5.4 mg/kg/h for 23 h) improves the neurological outcome of patients with spinal cord trauma.
- **Autonomic hyperreflexia** is associated with lesions above T5 but is not a problem during acute management.

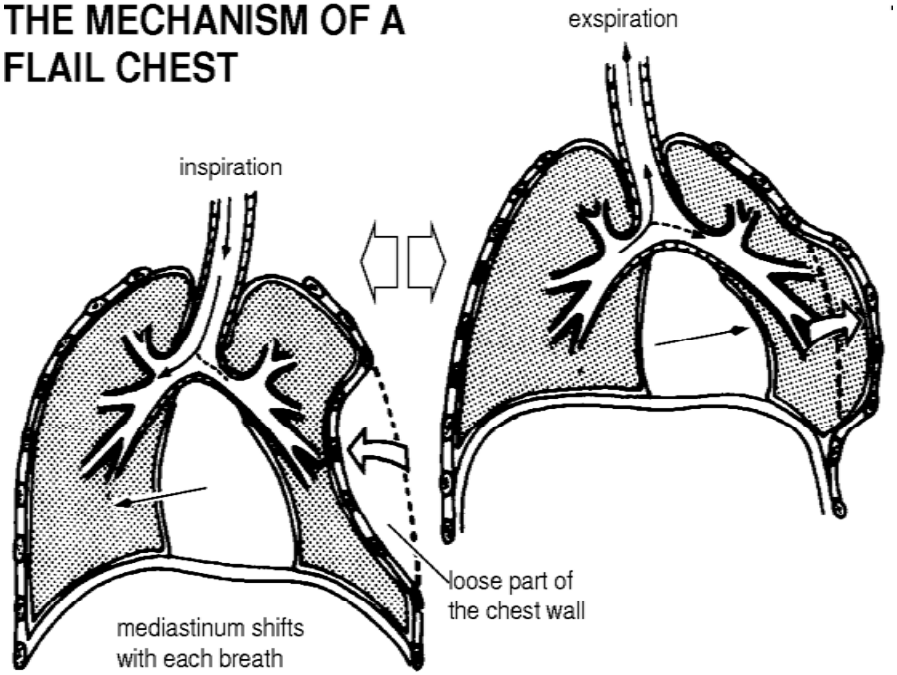
CHEST TRAUMA

- Trauma to the chest may severely compromise the function of the heart or lungs, leading to cardiogenic shock or hypoxia.
- A tension pneumothorax develops from air entering the pleural space through a one-way valve in the lung or chest wall. As a result, the ipsilateral lung completely collapses and the mediastinum and trachea are shifted to the contralateral side.
- Multiple rib fractures may compromise the functional integrity of the thorax, resulting in flail chest.
- Pulmonary contusion results in worsening respiratory failure over time.
- Hemothorax is differentiated from pneumothorax by dullness to percussion over silent lung fields.

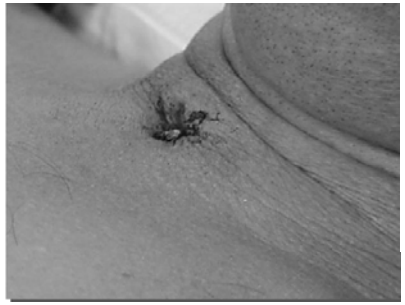
TENSION PNEUMOTHORAX

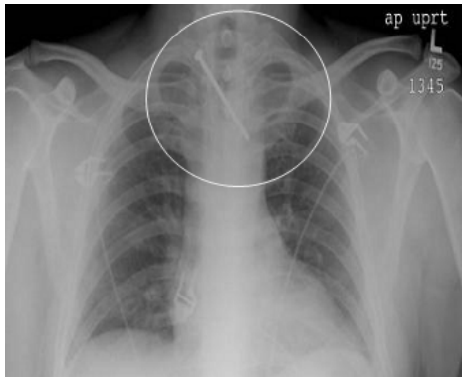


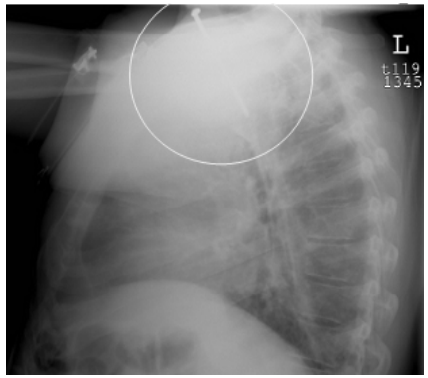
THE MECHANISM OF A FLAIL CHEST



Nail Gun







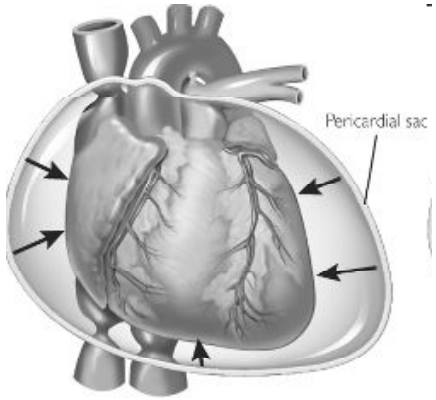
CHEST TRAUMA

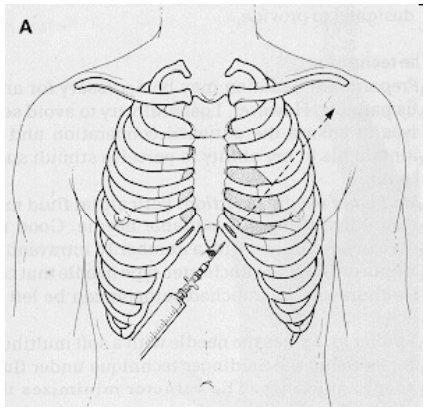
- Hemomediastinum, like hemothorax, can also result in hemorrhagic shock.
- Massive hemoptysis may require isolation of the affected lung with a double-lumen tube (DLT) to prevent blood from entering the healthy lung.
- Air leakage from traumatized bronchi can track an open pulmonary vein causing pulmonary and systemic air embolism.
- Cardiac tamponade is a life-threatening chest injury that must be recognized early. The presence of Beck's triad (neck vein distention, hypotension, and muffled heart tones), **pulsus paradoxus** (a > 10 mm Hg decline in blood pressure during spontaneous inspiration), and a high index of suspicion will help make the diagnosis.

CHEST TRAUMA

- Pericardiocentesis provides temporary relief.
- Myocardial contusion is usually diagnosed by electrocardiographic changes consistent with ischemia (ST-segment elevation), cardiac enzyme elevations (creatine kinase MB or troponin levels), or an abnormal echocardiogram.
- Other possible injuries following chest trauma include aortic transection or aortic dissection, avulsion of the left subclavian artery, aortic or mitral valve disruption, traumatic diaphragmatic herniation, and esophageal rupture.
- Acute respiratory distress syndrome (ARDS) is usually a delayed pulmonary complication of trauma
- The mortality rate of ARDS approaches 50%.

Cardiac Tamponade





ABDOMINAL TRAUMA

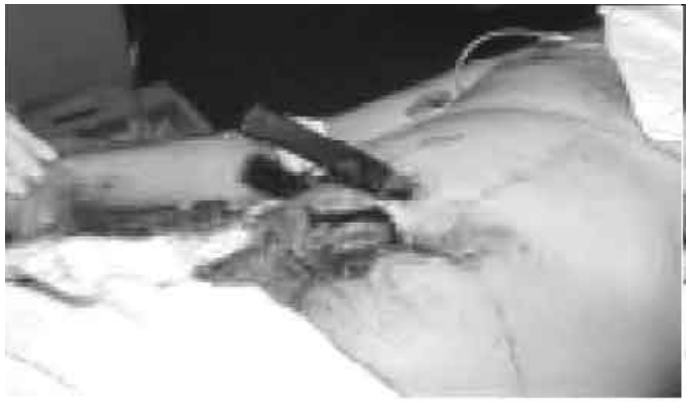
- Up to 20% of patients with intraabdominal injuries do not have pain or signs of peritoneal irritation (muscle guarding, percussion tenderness, or ileus) on first examination.
- Large quantities of blood (acute hemoperitoneum) may be present in the abdomen (eg, hepatic or splenic injury) with minimal signs.
- Abdominal trauma is usually divided into penetrating (eg, gunshot or stabbing) and nonpenetrating (eg, deceleration, crush, or compression injuries).

ABDOMINAL TRAUMA

- **Penetrating abdominal injuries** are usually obvious with entry marks on the abdomen or lower chest.
- The most commonly injured organ is the liver.
- Patients tend to fall into three subgroups:

1. Pulseless
2. Hemodynamically unstable
3. Stable

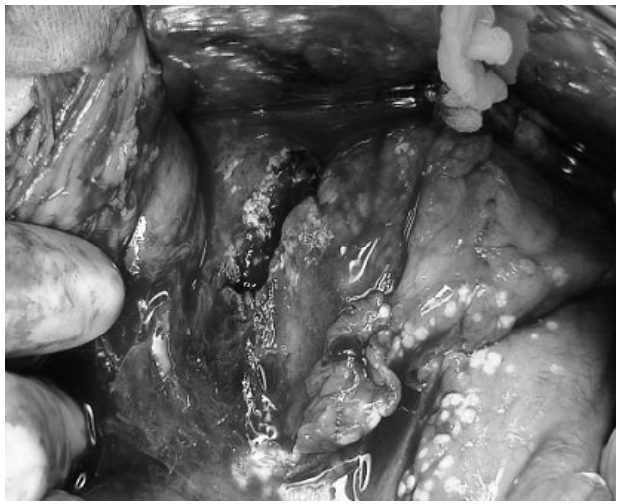
Penetrating abdominal injuries



ABDOMINAL TRAUMA

- Blunt abdominal trauma is the leading cause of morbidity and mortality in trauma, and the leading cause of intraabdominal injuries.
- Splenic tears or ruptures are most common.
- A positive FAST scan in a hemodynamically unstable patient with blunt abdominal trauma is an indication for immediate surgery.
- Profound hypotension may follow opening of the abdomen as the tamponading effect of extravasated blood (and bowel distention) is lost.
- Nitrous oxide is avoided to prevent worsening of bowel distention.
- A nasogastric tube (if not already present) will help prevent gastric dilation but should be placed orally if a cribriform plate fracture is suspected.

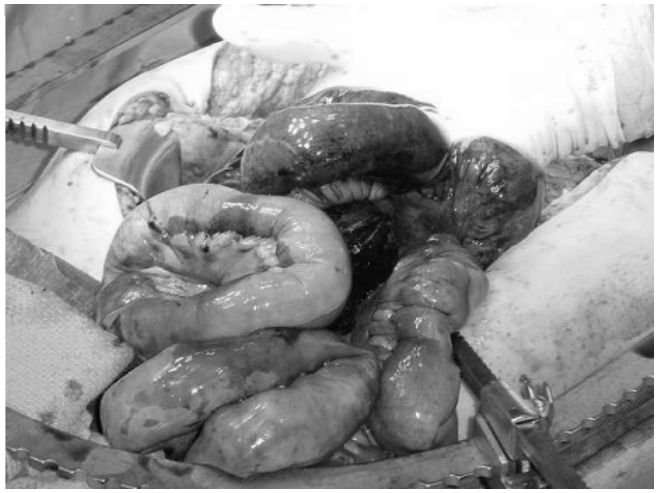
Injury Pancreatic



ABDOMINAL TRAUMA

- The potential for massive blood transfusion should be anticipated, particularly when abdominal trauma is associated with vascular, hepatic, splenic, or renal injuries, pelvic fractures, or retroperitoneal hemorrhage.
- Massive abdominal hemorrhage may require packing of bleeding areas and/or clamping of the abdominal aorta until bleeding sites are identified and the resuscitation can catch up with the blood loss.
- Prolonged aortic clamping leads to ischemic injury to the liver, kidneys, intestines, and, in some instances, a compartment syndrome of the lower extremities.
-

Injury Hollow Viscus

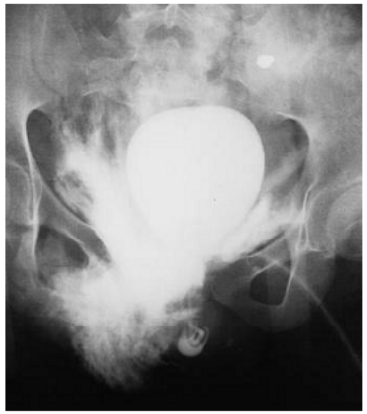


ABDOMINAL TRAUMA

- Progressive bowel edema from injuries and fluid resuscitation may preclude abdominal closure at the end of the procedure.
- Tight abdominal closures markedly increase intraabdominal pressure, resulting in an abdominal compartment syndrome that can produce renal and splanchnic ischemia.
- Oxygenation and ventilation are often severely compromised, even with complete muscle paralysis.
- Oliguria and renal shutdown follow.

Urethral Injury

- Pelvic fractures in 95% of cases
- 5-25% of pelvic fractures have a urethral injury
- Test: retrograde urethrogram

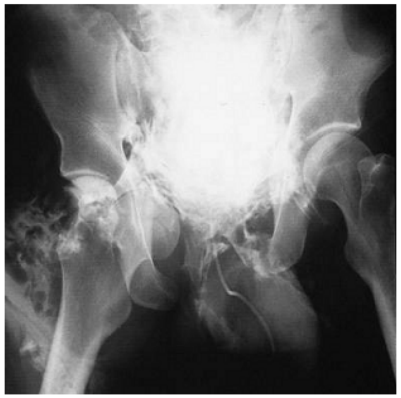


EXTREMITY TRAUMA

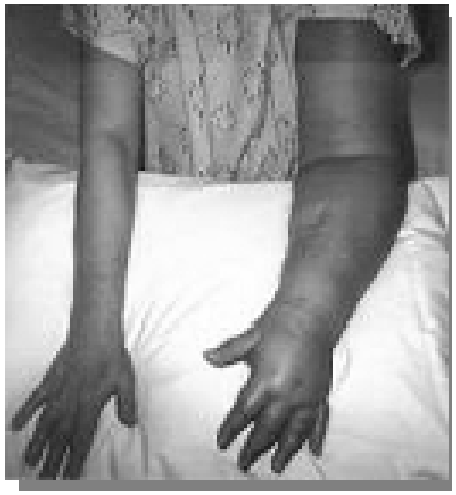
- Extremity injuries can be life-threatening because of associated vascular injuries and secondary infectious complications.
- Vascular injuries can lead to massive hemorrhage and threaten extremity viability.
- **Fat emboli** are associated with pelvic and long-bone fractures and may cause pulmonary insufficiency, dysrhythmias, skin petechiae, and mental deterioration within 1–3 days after the traumatic event
- A compartment syndrome can also occur following large intramuscular hematomas, crush injuries, fractures, and amputation injuries.

Bladder Injury

- Pelvic fracture in 70 to 95%
- 4-8% of pelvic fractures have a bladder injury
- Test: Retrograde cystogram

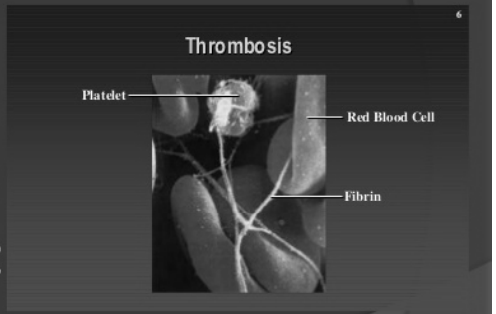


Compartment Syndrome



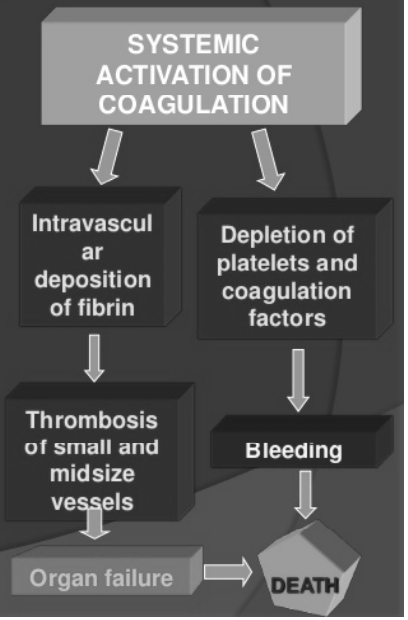
DIC

- It is an acute, subacute, or chronic thrombohemorrhagic disorder.
- It occurs as secondary complication of various diseases.
- An acquired syndrome characterized by **systemic intravascular coagulation**
- Coagulation is always the initial event.



DIC

Most morbidity and mortality depends on extent of intravascular thrombosis



Laboratory diagnosis

- Thrombocytopenia
  - platelet count <100,000 or rapidly declining
- Prolonged clotting times (PT, APTT)
- Presence of Fibrin degradation products or positive D-dimer
- Low levels of coagulation inhibitors
  - AT III, protein C
- Low levels of coagulation factors
  - Factors V,VIII,X,XIII
- Fibrinogen levels not useful diagnostically

Treatment of DIC

- **Stop the triggering process** .
  - *The only proven treatment!*
- Supportive therapy
- No specific treatments
  - Plasma and platelet substitution therapy
  - Anticoagulants
  - Physiologic coagulation inhibitors

ANESTHETIC CONSIDERATIONS

General Considerations

- Regional anesthesia is inappropriate in hemodynamically unstable patients with life-threatening injuries.
- If the patient arrives in the operating room already intubated, correct positioning of the endotracheal tube must be verified.
- If the patient is not intubated the same principles of airway management described above should be followed in the operating room. If time permits, hypovolemia should be at least partially corrected prior to induction of general anesthesia.

General Considerations

- Invasive monitoring (direct arterial, central venous, and pulmonary artery pressure monitoring) can be extremely helpful in guiding fluid resuscitation, but insertion of these monitors should not detract from the resuscitation itself.
- Serial hematocrits (or hemoglobin), arterial blood gas measurement, and serum electrolytes (particularly K+) are invaluable in protracted resuscitations.

General anaesthesia for trauma

Advantages	Disadvantages
Speed of onset	Impairment of global neurological examination
Duration can be maintained as long as needed	Requirement for airway instrumentation
Allows multiple procedures for multiple injuries	Hemodynamic management more complex
Greater patient acceptance	Increased potential for barotrauma
Allows positive pressure ventilation	

Regional anaesthesia for trauma

Advantages	Disadvantages
Allows continued assessment of mental status	Peripheral nerve function difficult to assess
Increased vascular flow	Patient refusal common
Avoidance of airway instrumentation	Requirement for sedation
Improved postoperative mental status	Hemodynamic instability with placement
Decreased blood loss	Longer time to achieve anaesthesia
Decreased incidence of DVT	Not suitable for multiple body lesion
Improved post operative analgesia	May wear off before procedures conclude
Better pulmonary toilet	
Earlier mobilization	

Prophylaxis against Aspiration

- Trauma patients are always considered to have full stomach
  - Ingestion of food or liquids before injury
  - Swallowed blood from oral or nasal injury
  - Delayed gastric emptying
  - Administration of liquid contrast medium
- Reasonable to administer nonparticulate antacid prior to induction
- Cricoid pressure/Sellick Maneuver should be applied continuously during airway management
- Rapid Sequence Induction
  - Avoidance of ventilation between administration of medication and intubation

H2 blockers

- Classes include Cimitidine, Ranitidine (Zantac), Famotidine.
- They block histamine receptor ability to induce acid secretion by proton pump.
- They reduce gastric fluid volume and acidity

Antacids

Given ½ an hour before induction

- Reduce gastric acidity only

PPI

-Omeprazole, the first drug in this class, lansoprazole , esomeprazole .
-Binds to H+ / K+ pump on parietal cell.
-Given 40 mg IV 30 min before surgery .
-Reduce both volume and acidity

Metoclopromide

-Act on dopamine receptors
-Increase gastric motility & LES tone
-Reduce gastric fluid volume only

Induction of Anesthesia

- Propofol/Thiopental
  - Vasodilator, Negative Inotropic effect
  - May Potentate hypotension/Cardiac Arrest
- Etomidate
  - Increased cardiovascular stability
- Ketamine
  - Direct myocardial depressant
  - Catecholamine release
  - Hypertension/Tachycardia
- Midazolam
  - Reduced Awareness
  - Hypotension
- Scopolamine (Tertiary Amine)
  - Inhibits memory formation
- Muscle relaxants alone
  - Recall of Intubation/Recall of Emergency procedures

Neuromuscular Blocking Drugs

- Succinylcholine
  - Fastest onset <1 min
  - Shortest Duration5-10 min
  - Potassium increase 0.5-1.0mEq/L
  - Potassium increase >5mEq/L
    - After 24 hours
    - Safe in acute airway management
    - Burn Victims
    - Muscle Pathology
      - Direct Trauma
      - Denervation
      - Immobilization
  - Increase intraocular pressure
    - Caution in patients with ocular trauma
  - Increase ICP
    - Controversial in head trauma

Criteria for extubation or keep ventilation in ICU

- MENTAL STATUS
Able to follow comands
Pain controlled
Resolution of intoxication

- Airway reflexes
Appropriate cough and gag
Ability to protect airways
From aspiration
No airway edema

* RESPIRATORY MECHANICS
Adequate Tidal volume &RR
Normal Motor strength
FiO2 <0.5%

*Systemic stability
no urgency to return to OR
Normothermia
No signs of Sepsis
Stable vital signs

The End

shock

Definition

Hypotension

“Hypoperfusion can be present in the absence of significant hypotension.”

Pathophysiology

ATP production
Na-K pump
Anaerobic metabolism
acidosis

Physiological Response

Clinical picture

Signs of Organ Hypoperfusion

Multiorgan Dysfunction Syndrome (MODS)

Result is end organ failure

Goals of Shock Resuscitation

- Approach
- Monitoring

Airway

Control Work of Breathing

Optimizing Circulation

-Crystalloids vs Colloids

End Points of Resuscitation
"Goal-directed therapy"

Use objective hemodynamic and physiologic values to guide therapy

1. Urine output > 0.5 mL/kg/hr
2. CVP 8-12 mmHg
3. MAP 65 to 90 mmHg
4. Central venous oxygen concentration > 70%

In general, treat the cause...

Categories

Hypovolemic Shock

-Causes: Non-hemorrhagic
Hemorrhagic
-Signs

Classes of Hypovolemic Shock

	Class I	Class II	Class III	Class IV
Blood Loss	< 750	750-1500	1500-2000	> 2000
% Blood Vol.	< 15%	15 – 30%	30 – 40%	> 40%
Pulse	< 100	**> 100**	> 120	> 140
Blood Pressure	Normal	Normal	**Decreased**	**Decreased**
Pulse Pressure	Normal	**Decreased**	**Decreased**	**Decreased**
Resp. Rate	14 – 20	**20 – 30**	**30 – 40**	**> 40**
UOP	> 30	**20 – 30**	**5 – 15**	**negligible**
Mental Status	sl. Anxious	mildly anx	**confused**	**lethargic**
Fluid	crystalloid	crystalloid	**blood**	**blood**

Cardiogenic Shock

-Causes
-Signs

Coronary PP = DBP - PAOP

GOAL - Coronary PP > 50 mm Hg

- AMI
  - Aspirin, beta blocker, morphine, heparin
  - If no pulmonary edema, IV fluid challenge
  - If pulmonary edema
    - Dobutamine
    - Dopamine – will ↑ HR and thus cardiac work
  - PCI or thrombolytics
- RV infarct
  - Fluids and Dobutamine (no NTG)
- Acute mitral regurgitation
  - Pressors (Dobutamine and Nitroprusside)

If inotropes and vasopressors fail, intra-aortic balloon pump

Distributive Shock

-Types :

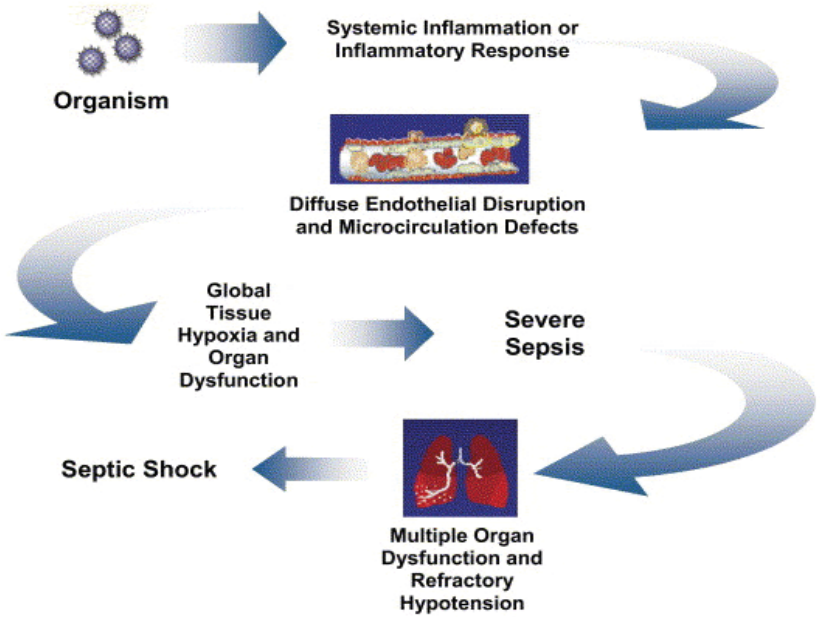
-Signs :

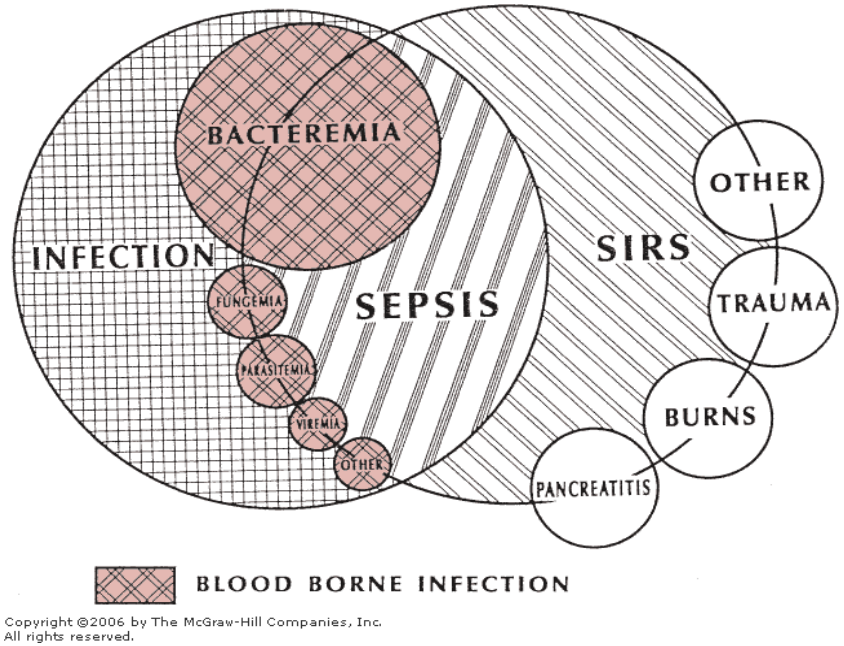
Sepsis

- Two or more of SIRS criteria
  - Temp > 38 or < 36 C
  - HR > 90
  - RR > 20
  - WBC > 12,000 or < 4,000

Plus

- presumed existence of infection





Treatment

Septic Shock

Sepsis Plus refractory hypotension

- After bolus of 20-40 mL/Kg.

-Consider Vasopressors

-Consider adrenal insufficiency

Vasopressors

- Assure adequate fluid volume
- Administer via central venous line
- Do not use dopamine for renal protection
- Requires arterial line placement
- Vasopressin:
  - Refractory shock

Anaphylaxis

IgE Mediated Hypersensitivity (type 1)

-causes:

-Anaphylactoid reaction:

Signs

- Biphasic phenomenon occurs in up to 20% of patients :

  Symptoms return 3-4 hours after initial reaction has cleared
- Symptoms usually begin within 60 minutes of exposure
- Faster the onset of symptoms = more severe reaction
- A “lump in my throat” and “hoarseness” heralds life-threatening laryngeal edema

Treatment

- ABC's
- IVFs, oxygen
- Epinephrine
- Bronchodilators

  Magnesium sulfate
- Second line
  - Corticosteriods
  - H1 and H2 blockers

Neurogenic shock

-Occurs after acute spinal cord injury

-Any injury above T1 can disrupt the entire sympathetic system

-usually lasts from 1 to 3 weeks

Treatment

A,B,Cs

Fluid resuscitation

Vasopressors

Treat bradycardia

Methylprednisolone

Adrenal Crisis

Causes

Autoimmune adrenalitis

Adrenal apoplexy = hemorrhage or infarct

Treatment

Obstructive Shock

-Causes

-Signs

Summary

Type	PAOP	C.O.	SVR
HYPOVOLEMIC	↓	↓	↑
CARDIOGENIC	↑	↓	↑
DISTRIBUTIVE	↓ or N	**varies**	↓
OBSTRUCTIVE	↑	↓	↑

The End

Anaesthesia for Emergency Surgery

Dr. Ahmed Shahin M.D

Outline

- Definitions
- Types
- Preparation
- Intra-operative management
- Post-operative management
- Examples:
  - ATLS
  - Caeserian Section

Definitions

- Elective … ahead of time … planned
- Ex. Inguinal hernia, Cataract …… Breast implants

- EMERGENCY
- Ex. Intestinal Obstruction

- Urgency
- Ex. Kidney stone

Types

- Traumatic
- Non-traumatic:
  - Obstetrical
  - Neurological
  - Vascular
  - Burns
  - Genito-Urinary
  - Gastrointestinal … etc…

Preoperative preparation

- Anesthesia for emergency situations is unplanned, hence the time available to evaluate the patient preoperatively and prepare for surgery is short.
- At bare minimum, one needs to have answers to the following questions:
  - Why patient needs emergency surgery?
  - How much time is available for resuscitation?
  - Are there are signs indicating presence of hemorrhage?
  - What are the results of quick airway assessment for difficult intubation?
  - Whether patient will need ventilatory support postoperatively, ICU care, etc.?

- Full history and examination if possible

- S – Symptoms
- A – Allergies
- M – Medications
- P – Past medical history
- L – Last oral intake
- E – Events prior to incident

- Lab investigations if possible … usually done as resuscitation is carried on

- Prepare to manage any uncontrolled co-morbidities
  - Ex. D.M, HTN, ASTHMA

- Consent for anaesthesia mentioning high risk of awareness and another one for blood transfusion

- Major trauma surgery … 11-43% !!!!

O.R

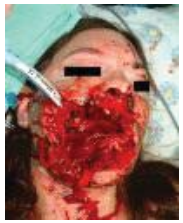
- Warm as is practical

- Intravenous fluid warmers and rapid infusion devices

- Good Suction device … full stomach … aspiration

- Difficult intubation equipment … C-collar … (e.g., fiber optic bronchoscopes, video laryngoscopes)

Risk of aspiration



- Inadequate fasting time
- Head & neck trauma
- Unable to protect airway [head or spinal injury ,vocal cord injury]
- Pregnancy
- Intestinal obstruction
- Pain
- Intra abdominal mass
- Obesity

Complications of difficult airway

- Aspiration
- Hypoxemia
- Trauma to upper airway
- Potential spinal cord injury in cervical injury

Upon arrival

- 2 large caliber peripheral intravenous lines if not already established … 16G or 14G … if difficult… central line
- Routine monitors … Invasive monitoring per case

Anesthetic Induction & Maintenance

- General anaesthesia
- Neuro-axial anaesthesia Ex for LSCS
- Combined anaesthesia
- Peripheral nerve block

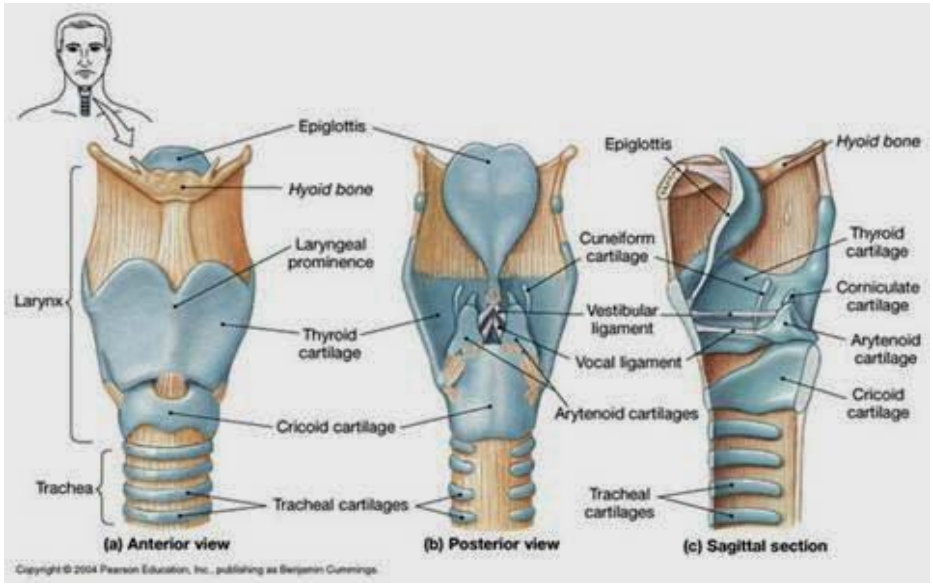
Anesthetic Induction & Maintenance

- Hemodynamic unstable conscious patient …. Nightmare
- Etomidate … Ketamine … Scopolamine
- Always much less doses, and slowly

Rapid sequence induction (RSI)

- Minimizes the risk of aspiration
- The availability of suction must be confirmed before induction.
- Preoxygenation with 100% oxygen for 3-5 minutes or 4 vital breaths.
- Predetermined rapid IV induction agent.
- Followed by rapid acting muscle relaxant without waiting to assess the effect of induction agent.
- Combined with cricoid pressure to reduce the risk of aspiration.
- Patient is not artificially ventilated.

Why Cricoid Cartilage?



Damage Control Surgery

- Stop hemorrhage and limit gastrointestinal contamination of the abdominal compartment
- Definitive repair of complex injuries is not part of DCS

Analgesia

- Effective analgesia ASAP
- Titrated to the desire of the Patient
- Respiratory depression
- No NSAIDS for hyopovolemic patients
- Regional Anaesthesia (Hemodynamic instability, Coagulopathy)

- Heat Loss should be prevented in all ages
- Invasive monitoring should be applied when required
- Local or Regional Anaesthesia should be used when feasible

Summary

- Inadequate History and Investigations
- Inadequate Preparation

a- Not Fasting – Requires Rapid Sequence Induction of Anaesthesia

b- Untreated Pre-Existing Diseases – Requires Resuscitation and Careful Choice of Anaesthetic Drugs and Techniques

c- Unavailability of Appropriate Investigations – Requires Depending on Clinical Impression and Minimal Investigations

d- Unavailability of Appropriate Cross-Matched blood – Requires use of Type-Specific blood or Group O-Neg blood transfusion in life saving procedures until proper Cross-Matched blood is available

Post-operative Management

- Decision for extubation depends on patient's haemodynamic status
- In stable patient, before extubation Direct laryngoscopy is performed and secretion or debris are removed. If nasogastric tube is in situ, it is aspirated.

- Atropine and neostigmine are given and patient will breathe in 100% oxygen.
- Because of the risk of aspiration, extubation is performed only when there is recovery of airway reflexes.

- Some patients may require continuation of ventilatory assistance postoperatively.
- They will be sent to ICU for further resuscitation and ventilation.

Indications for Postoperative ICU Admission

- Severe chest injury
- Evidence of aspiration pneumonia
- Unstable hemodynamic status
- Severe head injury for cerebral protection
- Massive blood loss with massive blood transfusion with DIC
- Polytrauma

ATLS

TRAUMA

- Trauma is a leading cause of morbidity and mortality in all age groups

ATLS

- PRIMARY SURVEY
- SECONDARY SURVEY
- RESUSCITATION
- DEFINITIVE TRAUMA INTERVENTIONS

PRIMARY SURVEY

- A….. It is rather an Ac
- B…..
- C…..
- D…..
- E…..

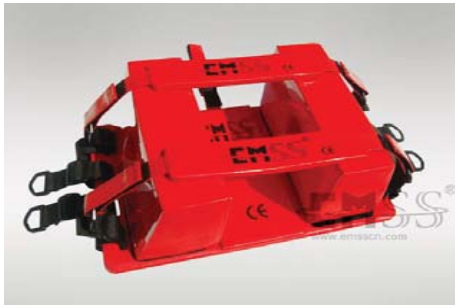
Ac

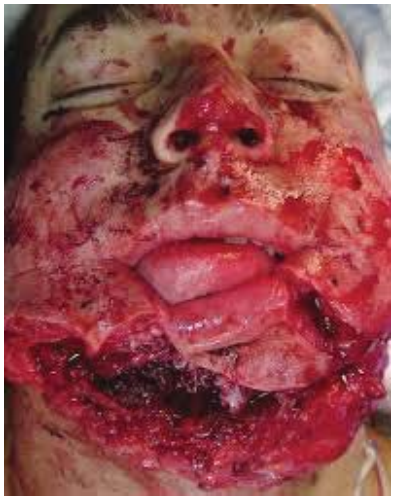
(1) BLS … hypoxia … hypercapnia

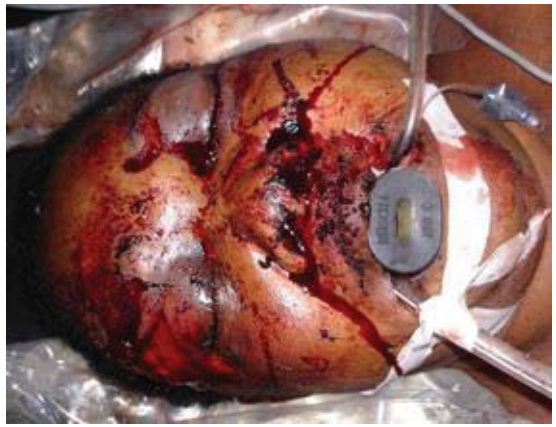
(2) Presumed cervical spinal cord injury until proven otherwise … neck pain … any significant head injury … neurological signs or symptoms … intoxication … loss of consciousness … cervical collar ("C-collar") … MILS.

During intubation do not remove c-collar or stop MILS

(3) Potential failed tracheal intubation … alternative airway devices are now approved for resuscitation







B

- Adequate gas exchange … chest rise … auscultation … vapor … capnography
- Life Threatening pulmonary injury … ex. Tension pneumo-thorax

C

- Pulse and Blood pressure
- Capillary refill
- Urine output

D

- L.O.C … pupillary size and reaction … lateralizing signs … signs of spinal cord injury
- Glucose level
- Alcohol intoxication … illicit or prescribed medications … hypoperfusion … hypercapnia

E

- Full exposure … But …

SECONDARY SURVEY

- Head to Toe Examination
- Scalp .. Face .. Ears .. Neck
- Chest
- Abdomen .. Pelvis
- Extremities

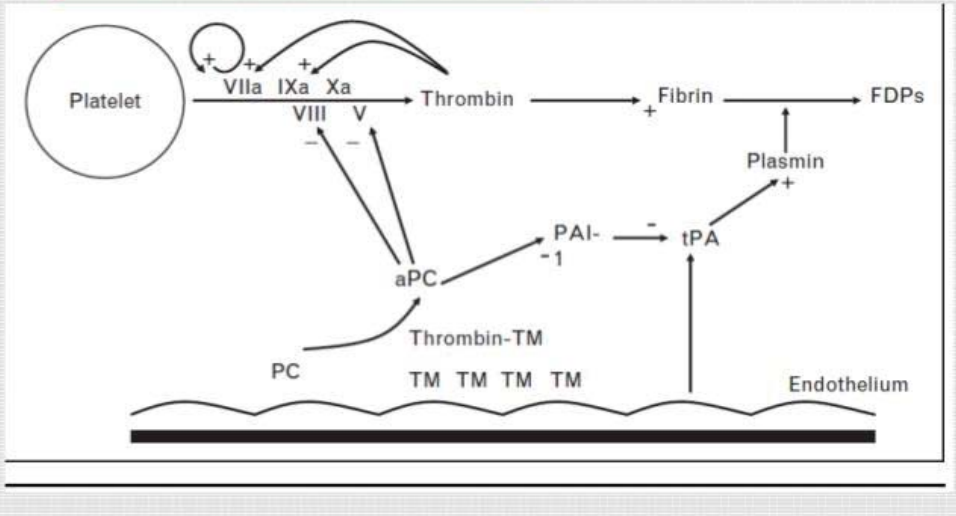
Goals for Resuscitation of The Trauma Patient

PARAMETER	GOAL
Blood pressure	Systolic 80 mmHg, mean 50-60mmHg
Heart rate	< 120 bpm
Oxygenation	SaO2 > 95%
Urine output	0.5ml/kg/h
Mental status	Following commands
Lactate level	<1.6mmol/l
Base deficit	> -5
Haemoglobin	>8.0g/dl

Trauma-Induced Coagulopathy

- Common following major trauma
- Trauma-induced coagulopathy is an independent risk factor for death
- Acute traumatic coagulopathy is only related to severe metabolic acidosis (base deficit ≥6 mEq/L)

Feed back for hemostasis & Hyperfibrinolysis



Complications of Coagulopathy

- Uncontrolled bleeding
- Hemorrhagic shock
- Death

Haemostatic Resuscitation

- Damage control resuscitation
- Blood products in equal portions early in resuscitation has become an accepted approach
- 1 : 1 : 1
- O-negative ☐ type-specific ☐ cross matched

DEFINITIVE TRAUMA INTERVENTIONS

Caesarean Section

Special Precautions

- Supine Hypotensive Syndrome
- Pregnancy Induced (Associated) Hypertension
- Fetal Hypoxia
- Acidic Resting Stomach Juice
- Regurgitation

- C/S rate 14-15% at US
- Anesthesia: 3-12% maternal death
- Majority during G/A: failed intubation, ventilation, oxygenation and pulmonary aspiration of gastric content

- Risk factors:
  - Obesity
  - Hypertensive disorder of pregnancy
  - Emergently performed procedure

Thank You for

listening

PRINCIPLES OF PEDIATRIC ANESTHESIA

Department of anesthesia and ICU
Dr Omar Ababneh
Pediatric Anesthesiologist

Children are not little adults!

Different Anatomy

Different Physiology

Different Pharmacology

Different psychology

Different Approach and preparation

Introduction

- Pediatric anesthesia involves more than simply adjusting drug doses and equipment for smaller patients.
- Neonates (0–1 months), infants (1–12 months), toddlers (12–24 months), and young children (2–12 years of age) have differing anesthetic requirements.

- Safe anesthetic management depends on full appreciation of the physiological, anatomic, and pharmacological characteristics of each group.
- Indeed infants are at much greater risk of anesthetic morbidity and mortality than older children; risk is generally inversely proportional to age.
- In addition, pediatric patients are prone to illnesses that require unique surgical and anesthetic strategies.

DEVELOPMENTAL CONSIDERATIONS:

- A. THE CARDIOVASCULAR SYSTEM:

❖ Anatomic:

- Noncompliant left ventricle
- Residual fetal circulation
- Difficult venous and arterial cannulation

❖ Physiological:

1.Heart-rate-dependent cardiac output(Cardiac stroke volume is relatively fixed)

CO=SV x HR

High Heart Rate to maintain CO

2.Increased heart rate

3. Parasympathetic(ANS)is more dominant

4.Reduced blood pressure

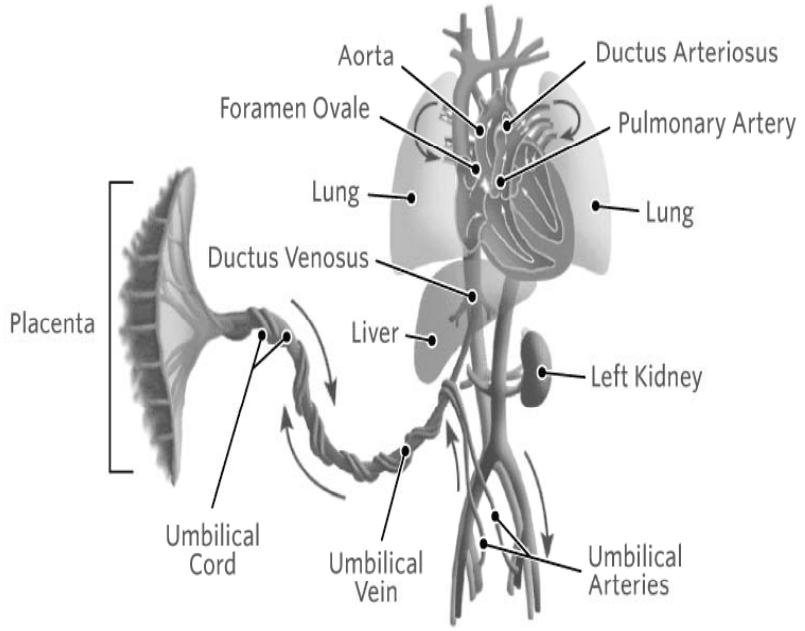
5. The vascular tree is less able to respond to hypovolemia with compensatory vasoconstriction. Intravascular volume depletion in neonates and infants may be signaled by hypotension without tachycardia.

Normal heart rate

Age (days)	Rate
1-3	100-140
4-7	80-145
8-15	110-165
Age (months)	**Rate**
0-1	100-180
1-3	110-180
3-12	100-180
Age (years)	**Rate**
1-3	100-180
3-5	60-150
5-9	60-130
9-12	50-110
12-16	50-100

NOTE: Activation of the parasympathetic nervous system by:
anesthetic overdose,
or hypoxia can quickly trigger bradycardia and profound reductions in cardiac output, that can lead to hypotension, asystole, and
intraoperative death!!!

Transitional Circulation? And flip-flop?
It is the period between mechanical and anatomic closure of the connections(foramen ovale, ductus arteriosus, and ductus venosus)



Many factors (e.g., hypoxia, hypercapnia, anesthesia-induced changes in peripheral or pulmonary vascular tone and parasympathetic stimulation) can affect this precarious balance and result in a sudden return to the fetal circulation.
When such **a flip-flop** occurs, pulmonary artery pressure increases to systemic levels, blood is shunted past the lungs via the patent foramen ovale, and the ductus arteriosus may reopen and allow blood to shunt at the ductal level. A rapid downhill spiral may occur and lead to severe hypoxemia, which explains why hypoxemic events may be prolonged, despite adequate pulmonary ventilation with 100% oxygen.

B. The Respiratory System:
(Almost all cardiac arrest due to respiratory problem!)

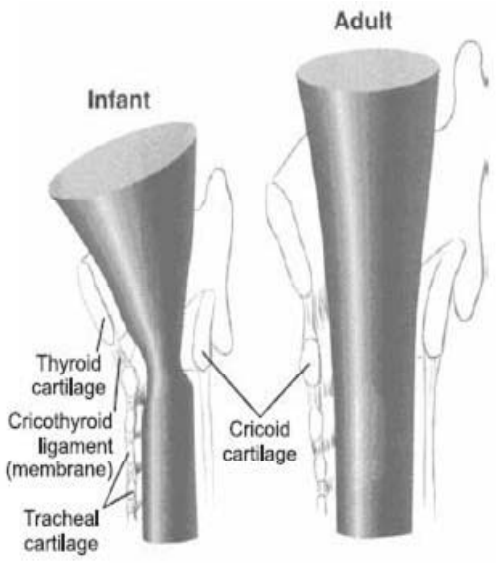
The pulmonary system is not capable of sustaining life until both the pulmonary airways and the vascular system have sufficiently matured to allow the exchange of oxygen from air to the bloodstream across the pulmonary alveolar-vascular bed.

***Independent life is not generally possible until a gestational age of 24 to 26 weeks

At Birth the respiratory system of infants differs from adults in:

- Large head and tongue, short neck
- Narrow nasal passages and small diameter of the airways
- More cephalad and anterior larynx,C4.
- The narrowest point of the A/W is the cricoid cartilage till 5 years
- Long and stiff epiglottis, U to Omega shape ,touch the soft palate(easy airway obstruction)
- The vocal cords are angled; consequently, a blindly passed tracheal tube may easily lodge in the anterior commissure rather than slide into the trachea.
- Short trachea, 5 cm in neonates.
- The chest wall is highly compliant, therefore the ribs provide little support for the lungs; that is, negative intrathoracic pressure is poorly maintained.

- Obligate nose breathers until 5 months
- Horizontal ribs so ventilation is mainly diaphragmatic
- Small number of alveoli, low lung compliance,
- Low FRC but still they high minute ventilation and O2 consumption(oxygen consumption is two to three times higher).
- Hypoxic and hypercapnic ventilatory drive are not well developed in neonates and infants….



That mean:

1. More likely potential for technical airway difficulties in infants than in teenagers or adults.

   ***Difficult intubation has been estimated to occurs in 0.5- 1% in pediatrics population.

2. Increased work of breathing. Example: In preterm infants, the work of breathing is approximately three times that in adults, and this work can be significantly increased by cold stress (i.e., increased metabolic demand for oxygen) or any degree of airway obstruction.
3. Risk of edema and airway resistance.
4. The resulting decrease in functional residual capacity (FRC) limits oxygen reserves during periods of apnea (eg,intubation attempts) and readily predisposes neonates and infants to atelectasis and hypoxemia.

5.
Small FRC
Alveoli numbers is 10 % of adults
Higher O2 Consumption 6ml-7ml/kg Adults (3-4ml/kg)
Diaphragm in neonates and infants<2y easy fatigue (lacks the Type I muscle fibers)

⇩

Rapid desaturation

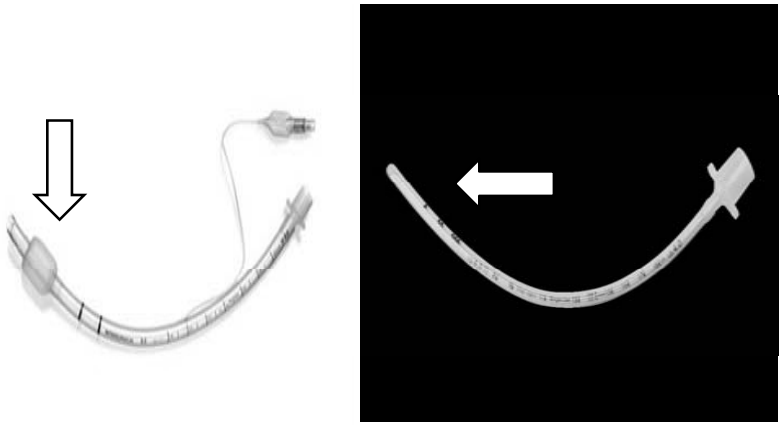
6. Risk of endobronchial Intubation

Age	Size—Internal Diameter (mm)
Newborns	3.0–3.5
Newborn–12 months	3.5–4.0
12–18 months	4.0
2 years	4.5
>2 years	ETT size = (16 + age)/4

Neonates have reduced incidence of subglottic stenosis:

- ❑Immature cartilage
- ❑High water content in cartilage
- ❑Less susceptible for ischemic injuries

Cuffed and uncuffed tracheal tubes

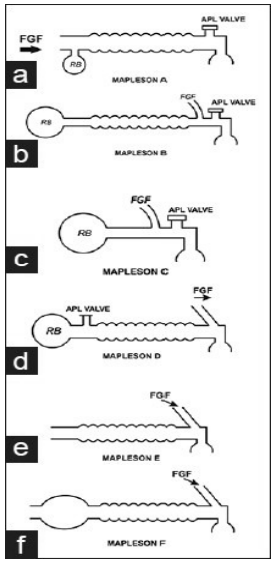


Breathing system

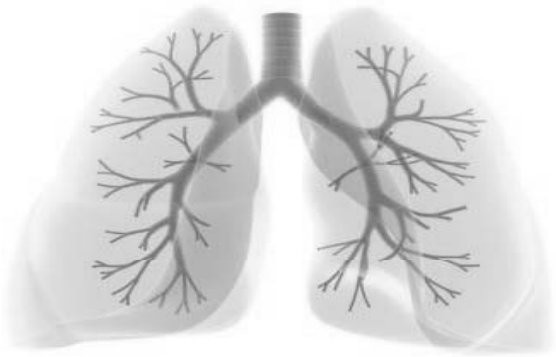
Jackson-Rees' modification of the Mapleson F system

Advantages of T-piece systems

- ❑ Compact
- ❑ Inexpensive
- ❑ No valves
- ❑ Minimal dead space
- ❑ Minimal resistance to breathing
- ❑ Economical for controlled ventilation



Aspiration Risk



Children < 3 years at greater risk of aspiration

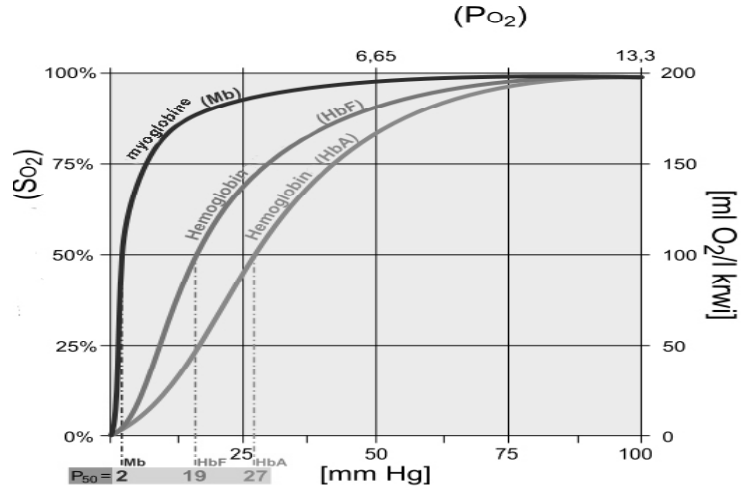
- Higher incidence of GERD
- Short esophagus
- Limited stomach compliance
- Baby trust
- Excessive air swallowing during crying
- No muscle relaxants Inadequate anesthesia

PREOPERATIVE FASTING RECOMMENDATIONS IN INFANTS AND CHILDREN

Type	Fasting Time (hrs)
Clear liquids	2
Breast milk	4
Infant formula	6
Solid (fatty or fried) foods	8

1. Less dehydration
   (better induction hemodynamic profile)
2. Less agitation and crying
   Promotes motility
3. Decrease gastric volume and PH

Neonatal period the HB is HBF .HBF has high affinity to O2 ……P50 is ….. HBF decline with age
HBA peaks at 9 month



Thermoregulation

- Greater heat loss
  - Thin skin
  - Low fat content
  - High surface area/weight ratio
- No shivering until 1 yo
- Thermogenesis by brown fat
- More prone to iatragenic hypo/hyperthermia

Large surface area relative to body weight(2-2.5x BW)

Thin skin and subcutaneous fat(less insulation)

Neonates no shivering

Immature thermoregulation center

Maintenance Fluid Therapy:

Replace Deficits, losses, and bleeding by isotonic fluid like **Lactated Ringer** (not glucose containing fluid)Risks of Hyperglycemia

Term Newborn (ml/kg/day)

Day 1 50-60 D10W

Day 2 100 D10 1/2 NS

>Day 7 100-150 D5-D10 1/4 NS

Older Child: 4-2-1 rule:

4 ml/kg/hr 1st 10 kg +

2 ml/kg/hr 2nd 10 kg +

1 ml/kg/hr for each kg > 20

Include dextrose in the maintenance hydration fluid (Dextrose 1% or Dextrose 2.5%)

*Risk of Hypoglycemia is higher in Premature

**Sick babies(malnutrition , cardiac)

****Regional anesthesia! Why?

*****Glucose infusion

- **Immature Kidney and liver functions** more free fraction of medication leads to greater effect of the high protein bounded drugs:
  - Barbiturates
  - Bupivacaine
  - Alfentanil
  - Lidocaine
- Water soluble Drugs will distribute more,so a higher loading dose to achieve desired serum levels is required:
  - Muscle relaxants
  - Antibiotics
- Drugs that redistribute to fat have larger initial peak levels (Opioids are more potent)
- Less muscle mass (more sensitive to muscle relaxants)
- **Delayed metabolism and excretion**

Induction of GA

IV[better] or inhalational?

HIGHER MAC

Highest MAC in infants 6 months and 1 year

Fast induction !How?

- Greater Alveolar ventilation to FRC ratio
- High cardiac out put to vessel rich organs(brain)
- Reduced tissue blood solubility

⇧ SVOFLURANE
HALOTHANE

⇩ ISOFLURANE
DESFLURANE

URTI

.Symptoms new or chronic?

– Infectious vs allergic or vasomotor

● Viral infection within 2 - 4 weeks of GA with intubation increases perioperative risk

– Wheezing risk increased 10x

– Laryngospasm risk increased 5x

– Hypoxemia, atelectasis, recovery room stay, admissions and ICU admissions all increased

● If possible, delay nonemergent surgeries

Intravenous access may be DIFFICULT!!or even impossible!!!

Keep Intraosseous option in your mind can be used for

-drug administration

-And fluid replacement

-blood sampling

Laryngospasm

Etiology

● Involuntary spasm of laryngeal musculature

– Superior laryngeal nerve stimulation

● Risk increased

– Extubated while lightly anesthetized

– Recent URI

– Tobacco exposure

Treatment

● Positive pressure ventilation (PEEP>10cmH2o)

● Laryngospasm notch

● Propofol

– 0.5–1 mg/kg IV

● Succinylcholine

– 0.2-0.5 mg/kg IV

– 2-4 mg/kg IM

And intubation

Perioperative pain control

● Regional

● Acetaminophen

– PO 10-15 mg/kg, PR 40 mg/kg

• NSAIDS

● Ketorolac 0.5-0.75 mg/kg IM/IV

● Opioids

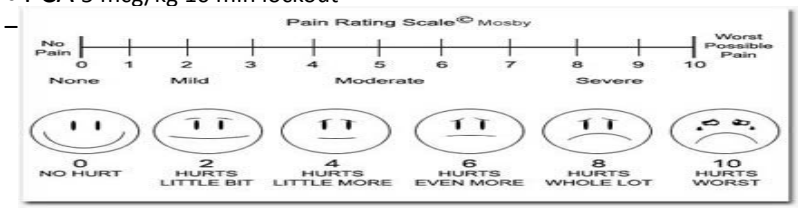
– Morphine 50-100 mcg/kg

● PCA 20 mcg/kg 10 min lockout(>8 years old)

– Hydromorphone 10-20 mcg/kg

● PCA 5 mcg/kg 10 min lockout

–



Regional Anesthesia:

- it decrease anesthetic requirements
- Operative and postoperative utility
- **Caudal block is the most common**
- Options in adults available for children:

– Peripheral blocks and catheters

- Epidural
- Spinal

Monitoring:

-BP

-blood sugar for neonates (Neonates have low glycogen stores ..risk of hypoglycemia)

-a precordial stethoscope

-ECG

-pulse oximeter and capnography

-Temperature: rectal, esophageal, nasopharynx.

-A/W pressure monitoring.

Malignant hyperthermia

- Acute hypermetabolic state in muscle tissue
- Triggering agents

– Volatile agents

– Succinyl Choline

- Incidence

– 1:15,000 peds

– 1:40,000 adults

- MH may occur at any point during anesthesia or emergence
- Recrudescence despite treatment

MH anesthesia

- Family history

– Muscle bx → caffeine contracture test

– +/- Ryanodine receptor abnormality

- High flow O2 flush circuit x 20 min
- Nontriggering

– TIVA, Nitrous

<u>Increased risk of MH:</u>

- Duchenne's muscular dsytrophy
- Central core disease
- Osteogenesis imperfecta
- King Denborough syndrome

Classic signs of MH

Specific

- Rapid rise in EtCO2 early sign
- Rapid increase in temp late sign
- Muscle rigidity +/-
- Rhabdomyolosis
  - Increase CK
- Myoglobinuria

Nonspecific

- Tachycardia
- Tachypnea
- Acidemia
  - Metabolic
  - Respiratory
- Hyperkalemia
- Dysrhythmias

MH treatment

- Discontinue triggering agents
- Hyperventilate with 100% FiO2
- NaHCO3 1-2 mEq/kg IV
- Dantrolene 2.5 mg/kg IV
- Cool patient
- Support as indicated → intropes, dysrhythmias
- Monitor labs
- Consider invasive monitoring
- 1 800-MH-HYPER

Questions?

THANK YOU ALL

LOCAL ANESTHETICS AND REGIONAL ANESTHESIA

Dr Walid Zuabi

FCA RCSI

Local Anesthetics- History

- 1860 - cocaine isolated from *erythroxylum coca.*
- Koller - 1884 uses cocaine for topical anaesthesia.
- Halsted - 1885 performs peripheral nerve block with LA.
- Bier - 1899 first spinal anesthetic .

Local Anesthetics - Definition

A substance which reversibly inhibits nerve conduction when applied directly to tissues at non-toxic concentrations.

- Local anesthetics block generation, propagation, and oscillations of electrical impulses in electrically excitable tissue.

By: acting on Na channels.

PHARMACOLOGY AND PHARMACODYNAMICS

Clinically used local anesthetics consist of lipid-soluble, substituted benzene ring linked to amine group via alkyl chain containing an amide or ester linkage.

• Type of linkage separates local anesthetics into either aminoamides (metabolized in liver) or aminoesters (metabolized in liver or by plasma cholinesterase).

PHARMACOKINETICS

Systemic toxicity depends on blood levels of LA:

- End organs of main concern for toxicity are CV and CNS.
- Determinants of systemic absorption:

» Site of injection (intercostal > caudal > brachial plexus > sciatic/femoral)

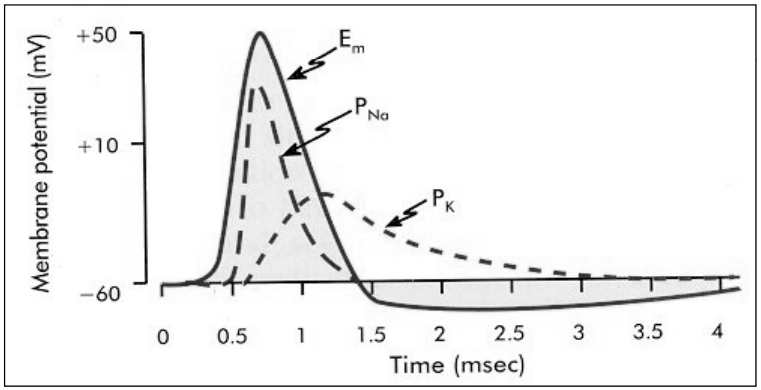
» Dose.

» Physiochemical properties (lipid solubility, protein binding)

» Addition of epinephrine

Local anesthetics - Mechanism

Limit influx of sodium, thereby limiting propagation of the action potential.

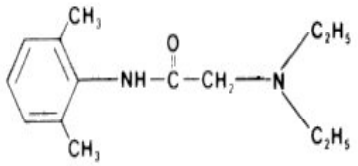


Local Anesthetics - Classes

Amides

Lidocaine

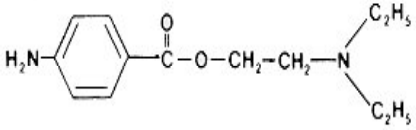
Xylocaine, etc



Esters

Procaine

Novocain



Local anesthetics - Classes

Esters	**Amides**
Cocaine	Bupivacaine
Chloroprocaine	Lidocaine
Procaine	Ropivacaine
Tetracaine	Etidocaine
	Mepivacaine

Bupivacaine

Amide

- Infiltration: use 0.25%, fast onset, 2- to 8-hr duration, max dose 175 mg (225 mg with epinephrine)
- Peripheral nerve block: use 0.25-0.5%, slow onset, 4- to 12-hr duration, max dose 175 mg (225 mg with epinephrine

Bupivacaine

Epidural anesthesia: Use 0.5-0.75%, moderate onset, 2- to 5-hr duration, max dose 175 mg (225 mg with epinephrine)

- Spinal anesthesia: Use 0.5-0.75%, fast onset, 1- to 4-hr duration, max dose 20 mg
- levo (-) bupivacaine less cardiotoxic than racemic bupivacaine, same profile.

Lidocaine

Amide

- Infiltration: use 0.5-1%, fast onset, 2- to 8-hr duration, max dose 300 mg (500 mg with epinephrine)
- Peripheral nerve block: use 1.0-1.5%, fast onset, 1- to 3-hr duration, max dose 300 mg (500 mg with epinephrine)

Lidocaine

Epidural anesthesia: use 1.5-2%, fast onset, 1- to 2-hr duration, max dose 300 mg (500 mg with epinephrine)

- Spinal anesthesia: use 1.5-2%, fast onset, 0.5- to 1-hr duration, max dose 100 mg
- Topical anesthesia: use 4%, fast onset, 0.5- to 1-hr duration, max dose 300 mg
- IV regional: Use 0.25-0.5%, fast onset, 0.5-1 hr duration, max dose 300 mg

Ropivacaine

Amide

- Infiltration: use 0.2-0.5%, fast onset, 2- to 6-hr duration, max dose 200 mg
- Peripheral nerve block: use 0.5-1.0%, slow onset, 5- to 8-hr duration, max dose 250 mg
- Epidural anesthesia: Use 0.5-1.0%, moderate onset, 2- to 6-hr duration, max dose 200 mg
- ***less cardiac toxicity than bupivacaine***

Local anesthetics - Formulation

Biologically active substances are frequently administered as very dilute solutions which can be expressed as *parts of active drug per 100 parts of solution (grams percent)*

Ex.: 2% solution =

$$\frac{2 \text{ grams}}{100 \text{ cc's}} = \frac{2000 \text{ mg}}{100 \text{ cc's}} = \frac{20 \text{ mg}}{1 \text{ cc}}$$

Local Anesthetics - Allergy

- True allergy is very rare.
- Most reactions are from ester class - ester hydrolysis (normal metabolism) leads to formation of PABA - like compounds.
- Patient reports of "allergy" are frequently due to previous intravascular injections.

Local Anesthetics - Toxicity

Tissue toxicity - Rare

- Can occur if administered in high concentrations (greater than those used clinically)
- Usually related to preservatives added to solution

Systemic toxicity - Rare

- Related to blood level of drug secondary to absorption from site of injection.
- Range from lightheadedness, tinnitus to seizures and CNS/cardiovascular collapse

Local anesthetics - Duration

- Determined by rate of elimination of agent from the site injected
- Factors include lipid solubility, dose given, blood flow at site, addition of vasoconstrictors (does not reliably prolong all agents)
- Some techniques allow multiple injections over time to increase duration, e.g. epidural catheter

Local anesthetics - vasoconstrictors

Ratios

Epinephrine is added to local anesthetics in *extremely* dilute concentrations, best expressed as a ratio of grams of drug:total cc's of solution. Expressed numerically, a 1:1000 preparation of epinephrine would be

$$\frac{\text{1 gram epi}}{\text{1000 cc's solution}} = \frac{\text{1000 mg epi}}{\text{1000cc's solution}} = \frac{\text{1 mg epi}}{\text{1 cc}}$$

Local anesthetics - vasoconstrictors

Therefore, a 1 : 200,000 solution of epinephrine would be

$$\frac{\text{1 gram epi}}{\text{200,000 cc's solution}} = \frac{\text{1000 mg epi}}{\text{200,000 cc's solution}}$$

or

$$\frac{\text{5 mcg epi}}{\text{1 cc solution}}$$

Local anesthetics - vasoconstrictors

Vasoconstrictors should not be used in the following locations

- Fingers
- Toes
- Nose
- Ear lobes
- Penis

REGIONAL ANESTHESIA

Regional anesthesia - Definition

Rendering a specific area of the body, e.g. foot, arm, lower extremities, insensate to stimulus of surgery or other instrumentation

Regional anesthesia - Uses

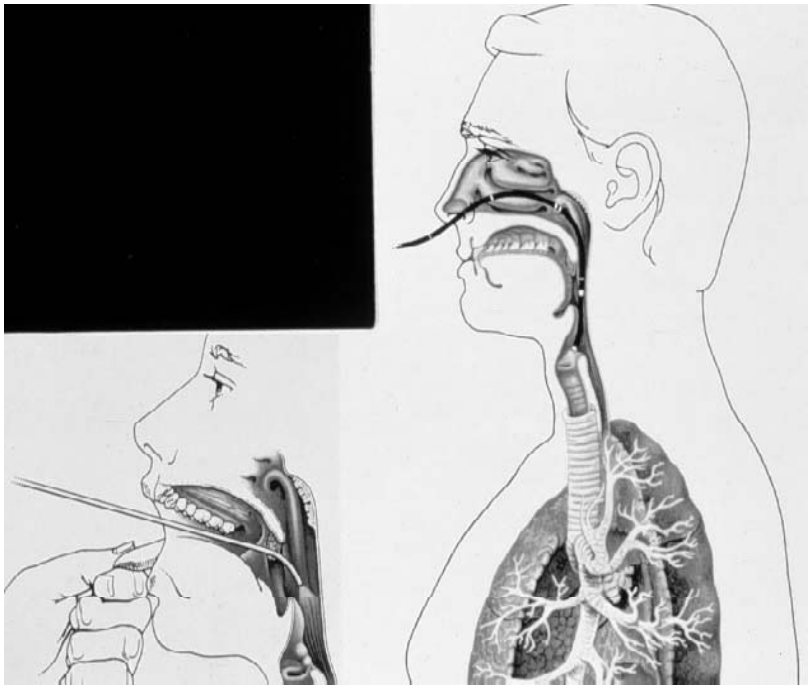
- Provide anesthesia for a surgical procedure
- Provide analgesia post-operatively or during labor and delivery
- Diagnosis or therapy for patients with chronic pain syndromes

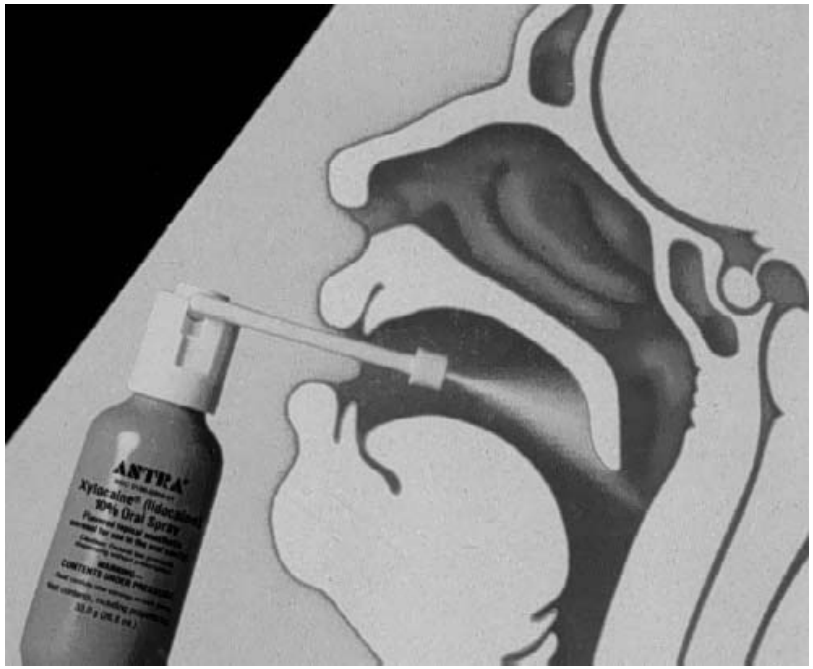
Regional anesthesia - types

- Topical
- Local/Field
- Intravenous block (“Bier” block)
- Peripheral (named) nerve, e.g. radial n.
- Plexus - brachial, lumbar
- Central neuraxial - epidural, spinal

Topical Anesthesia

- Application of local anesthetic to mucous membrane - cornea, nasal/oral mucosa
- Uses :
  - awake oral, nasal intubation, superficial surgical procedure
- Advantages :
  - technically easy
  - minimal equipment
- Disadvantages :
  - potential for large doses leading to toxicity



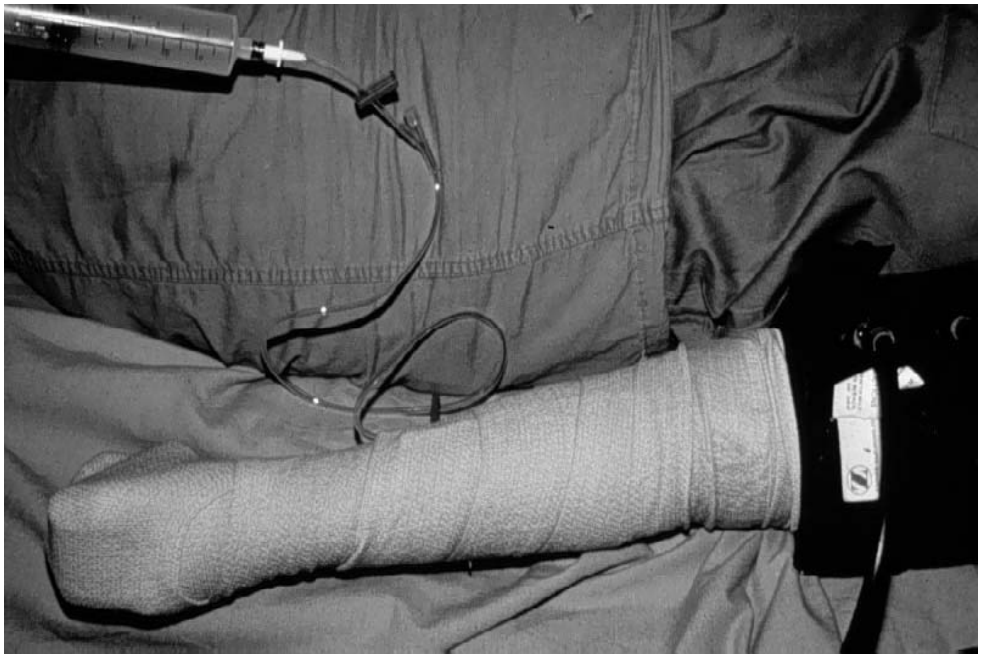


Local/Field Anesthesia

- Application of LA subcutaneously to anesthetize distal nerve endings
- Uses:
  - Suturing, minor superficial surgery, line placement, more extensive surgery with sedation
- Advantages:
  - minimal equipment, technically easy, rapid onset
- Disadvantages:
  - potential for toxicity if large field

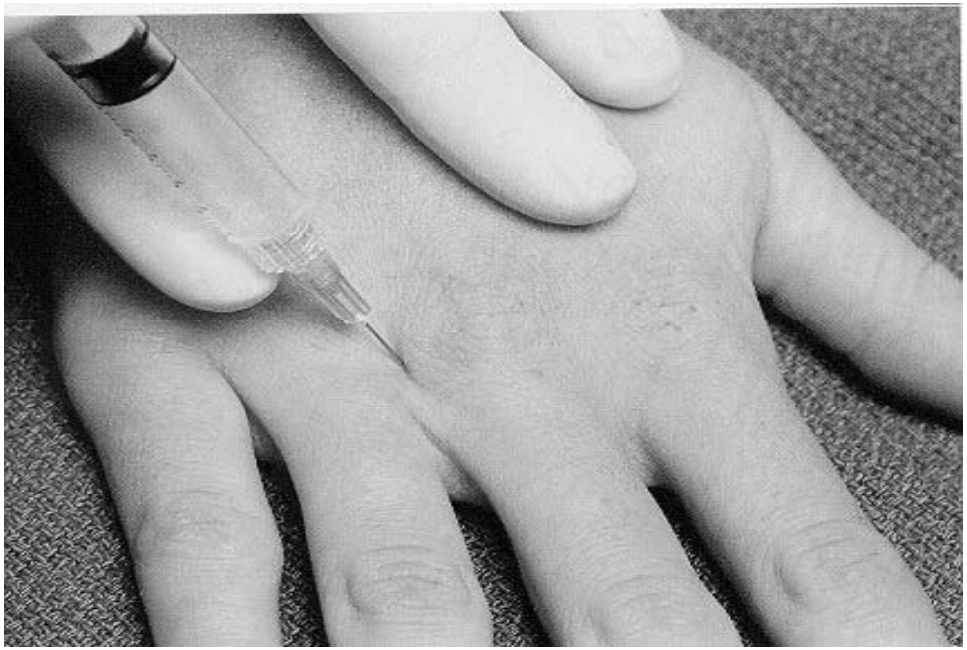
IV Block - “Bier” block

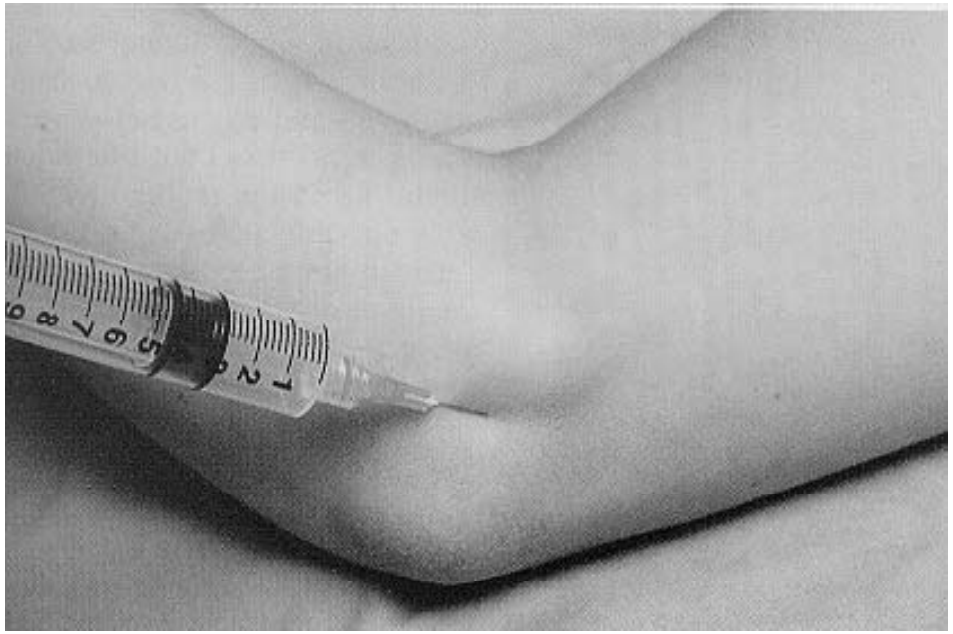
- Injection of local anesthetic intravenously for anesthesia of an extremity
- Uses
  - any surgical procedure on an extremity
- Advantages:
  - technically simple, minimal equipment, rapid onset
- Disadvantages:
  - duration limited by tolerance of tourniquet pain, toxicity



Peripheral nerve block

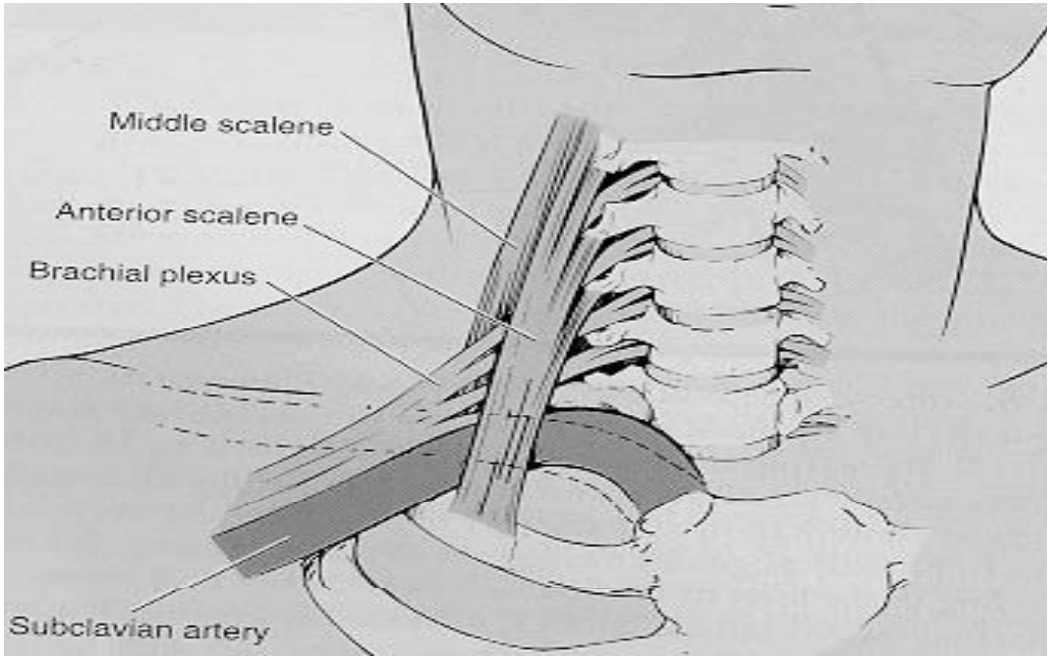
- Injecting local anesthetic near the course of a named nerve
- Uses:
  - Surgical procedures in the distribution of the blocked nerve
- Advantages:
  - relatively small dose of local anesthetic to cover large area; rapid onset
- Disadvantages:
  - technical complexity, neuropathy

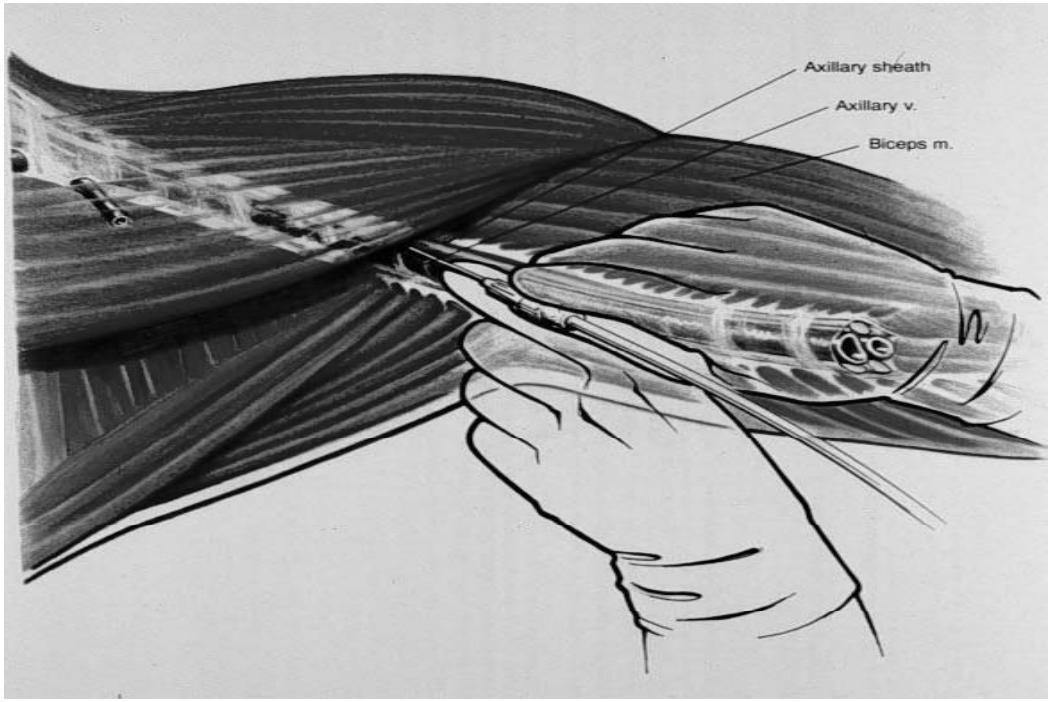




Plexus Blockade

- Injection of local anesthetic adjacent to a plexus, e.g cervical, brachial or lumbar plexus
- Uses :
  - surgical anesthesia or post-operative analgesia in the distribution of the plexus
- Advantages:
  - large area of anesthesia with relatively large dose of agent
- Disadvantages:
  - technically complex, potential for toxicity and neuropathy.





Central neuraxial blockade - “Spinal”

- Injection of local anesthetic into CSF
- Uses:
  - profound anesthesia of lower abdomen and extremities
- Advantages:
  - technically easy (LP technique), high success rate, rapid onset
- Disadvantages:
  - “high spinal”, hypotension due to sympathetic block, post dural puncture headache.

Central Neuraxial Blockade - “epidural”

- Injection of local anesthetic in to the epidural space at any level of the spinal column
- Uses:
  - Anesthesia/analgesia of the thorax, abdomen, lower extremities
- Advantages:
  - Controlled onset of blockade, long duration when catheter is placed, post-operative analgesia.
- Disadvantages:
  - Technically complex, toxicity, “spinal headache”

TOXICITY OF LOCAL ANESTHETICS

Rx of systemic toxicity is primarily supportive:

» Stop injection

» Administer oxygen

» Support ventilation

» Tracheal intubation and control ventilation if necessary

» Suppress seizure activity (thiopental, midazolam, propofol)

SPINALS, EPIDURALS AND CAUDALS

Dr Walid Zuabi

FCA RCSI

Introduction

- Two main types of anaesthesia – general and regional.
- REGIONAL anaesthesia – Drugs administered directly to the spinal cord or nerves to locally block afferent and efferent nerve input.
- Indications
- Contraindications
- Equipment
- Technique
- Complications

Definitions

- **Regional anaesthesia –** The use of local anaesthetic either alone or to supplement general anaesthesia aiming to prevent or reduce nerve conduction of painful impulses to higher centres.
- **Spinal anaesthesia** – Injection of a local anaesthetic directly into the CSF within the sub-arachnoid space.
- **Epidural anaesthesia** – Injection of a local anaesthetic into the potential space ***outside*** the dura.
- **Caudal anaesthesia –** Injection of local anaesthetic into the caudal canal producing block of the sacral and lumbar nerve roots.

Advantages of regional anaesthesia

- Analgesia / anaesthesia provided predominantly in area required, thereby avoiding the systemic administration of drugs.
- Spontaneous ventilation can be preserved – benefit in those with respiratory disease.
- Generally less disturbance of co-existing systemic disease requiring medical therapy.
- Airway reflexes preserved.
- May improve access and facilitate surgery.
- Reduced blood loss with controlled hypotension.
- Less equipment required and reduced cost.
- Less volatile agent required if used in conjunction with GA.
- Some techniques can be continued postoperatively.

Indications – Spinal anaesthesia

- Surgical procedures to the lower body below dermatome T10.
- Analgesia for upper abdominal surgery in combination with general anaesthesia.
- "*circumstances where a skilled anaesthetist is not readily available to administer a general anaesthetic*" Oxford Medical dictionary 1996

Indications – Epidural anaesthesia

- Surgery – can be done using epidural as sole anaesthetic but normally combined with light GA.
  - Thoracic
  - Pulmonary
  - Cardiac
    - Vascular
  - Abdominal
  - Gynaecological / Labour
  - Urological
  - Orthopaedic
- Acute pain relief
  - Post op
  - Trauma
  - Miscellaneous (eg pancreatitis)
- Chronic pain states

Indications – Caudal Anaesthesia

- Provides a way of administering a sacral epidural.
- Popular in paediatrics…Inguinal hernia repair
  Surgery to genitalia
- Adults - Surgery (anorectal, gynaecological, orthopaedics)
- Obstetric (episiotomy, removal of placenta)
- Chronic pain

Contraindications to regional techniques

ABSOLUTE

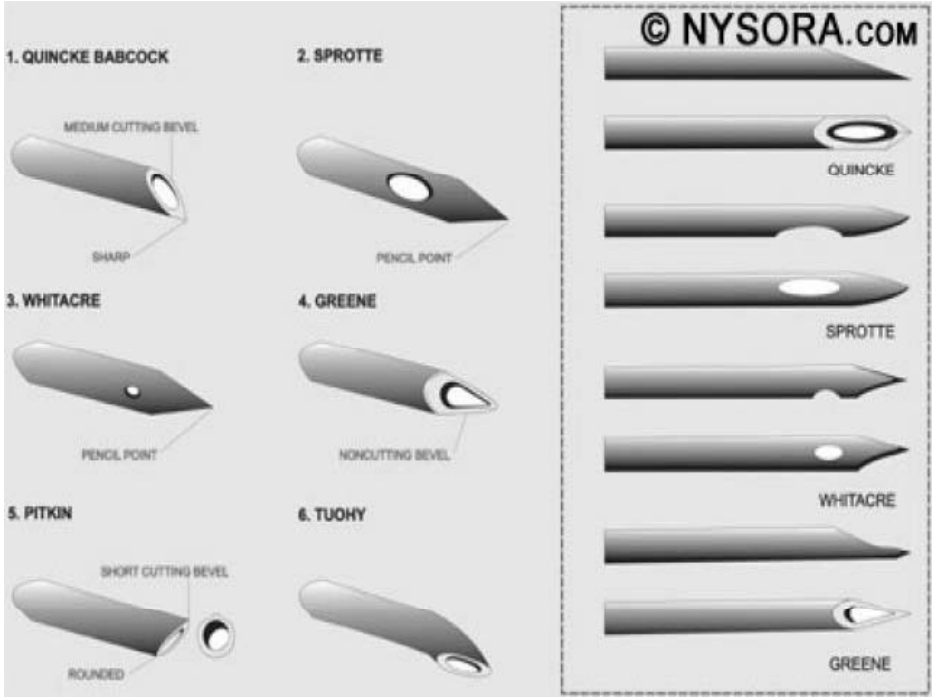
- Patient refusal
- Anticoagulation / coagulopathy
- Local anaesthetic allergy
- Localised infection
- Untreated hypovolaemia

RELATIVE

- Systemic sepsis
- Raised ICP
- Skeletal anomalies
- Neurological disease
- Previous local surgery
- Fixed cardiac output state eg aortic stenosis
- Unco-operative patient

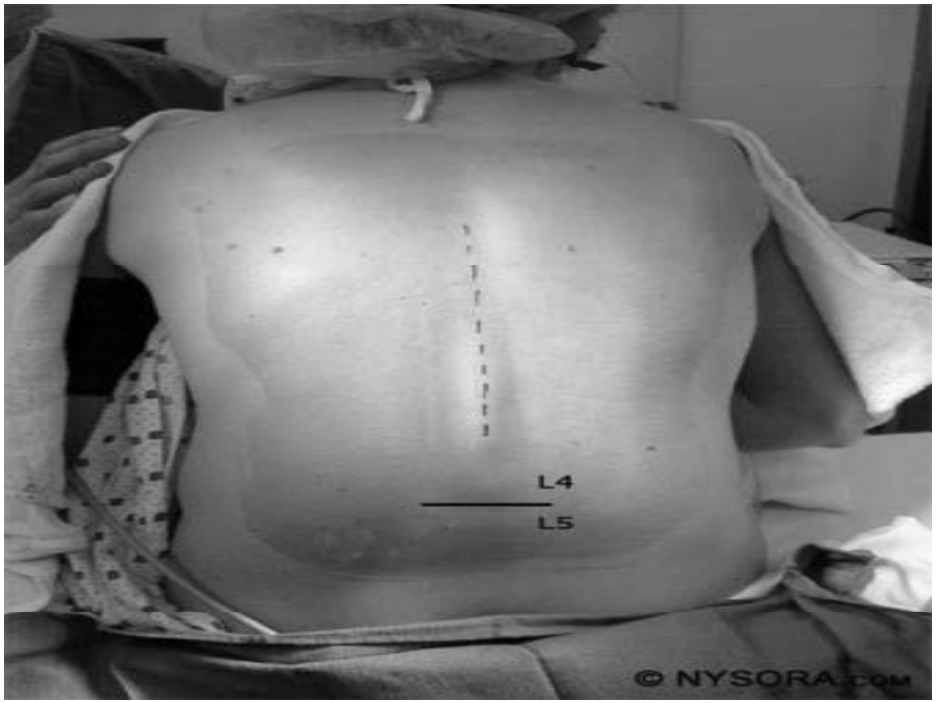
Equipment - spinals

- Needle choice – Quinke / Whitacre / Sprott
  - Gauge eg 25G
- Introducer
- Syringes
- Sterile drapes, swabs, betadine etc
- Local anaesthetics
  - 0.5% hyperbaric bupivicaine 2.5ml – 3.25ml



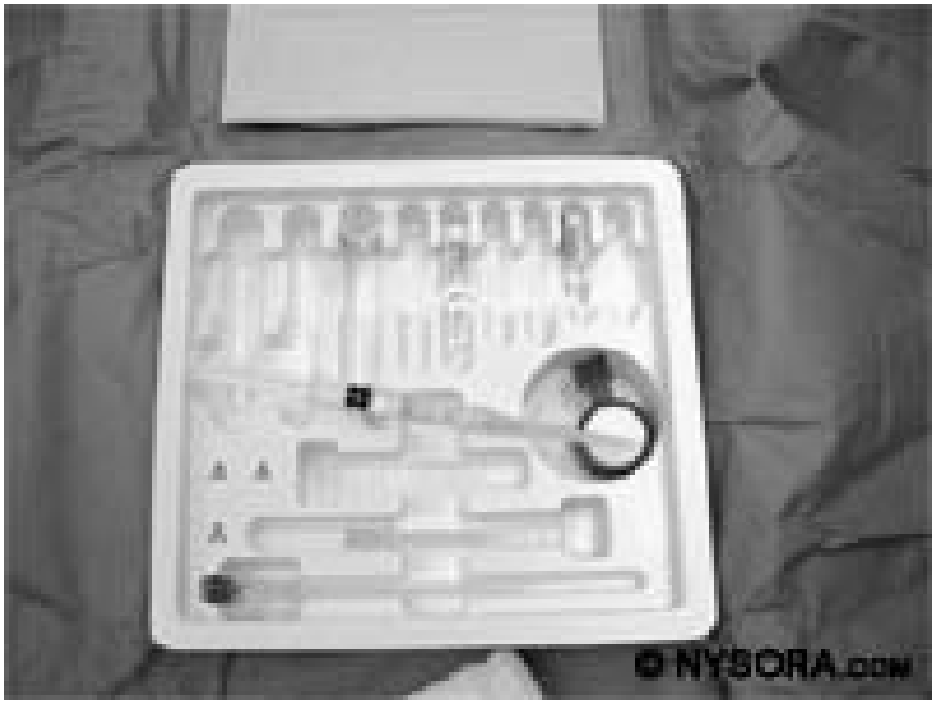
Technique - spinals

- **Consent and explanation**
- Prepare equipment, IV access, monitoring, scrub
- Positioning – Sitting v Lateral
- Approach – midline / paramedian
- Clean skin, drape, identify position
- Skin anaesthesia, introducer, spinal needle, inject local



Equipment - Epidurals

- Sterile drapes, swabs, betadine etc
- Tuohy needle (16-18G)
  - curved tip
  - markings
- Plastic catheter
- Syringes
- Local anaesthetics



Technique - epidurals

- Consent and explanation
- Prepare equipment, IV access, monitoring, scrub
- Positioning – Sitting v Lateral
- Clean skin, drape, identify position
- Skin anaesthesia, Tuohy needle, loss of resistance technique, air v water.
- Catheter insertion, test dose then main dose

Technique - Caudals

- Consent and explanation
- Prepare equipment, IV access, monitoring, scrub, GA
- Clean skin, drape, identify position
- Identify sacral hiatus, advance needle through sacrococcygeal membrane and inject local.
- Catheter may be inserted.

Complications of regional anaesthesia

Complications	Estimated frequency	Comments
Direct nerve damage	1:10,000 – 1:30,000	No effective treatment
Spinal Haematoma	1:150,000 – 1:220,000	Requires urgent evacuation
Spinal infection	1:100,000 – 1:150,000	Aggressive Abs +/- evacuation
Drug error	Unknown	Avoidable, may be fatal
Systemic toxicity	Unknown	May be fatal without treatment
Respiratory depression	Unknown	Especially using opiods
Hypotension	Common	Early treatment needed
Confusional states	Common in elderly	Especially using opiods
Pruritis / nausea / urinary retention	Up to 16% incidence	Treat effectively
Technical failure	5-25%	Accept failure Consider alternative

Complications – Spinal anaesthesia

- Physiological consequences – Hypotension, urinary retention, bradycardia.
- Headache – dural puncture.
- Neurological sequelae

  - usually temporary, secondary to pressure or stretching, permanent damage rare.
  - anterior spinal artery syndrome.
  - infection.

Complications – Epidural anaesthesia

- Similar to spinal (physiological, headache, neurological)
- Shearing or migration of catheter
- Total spinal

  - inadvertent injection of large volume of LA into CSF.
  - profound motor block, may reach cervical cord affecting arms.
  - may produce respiratory failure as phrenic nerves blocked.
  - if reaches cerebral CSF unconsciousness, hypotension, arrest.
  - Management - CALL FOR HELP
    - ABC, supportive including vasopressors, atropine, fluids, head down tilt.

Complications – Caudal anaesthesia

- Incorrect needle placement
  - Too superficial leads to failed block.
  - Too deep can pass into pelvic cavity, may puncture viscera or enter birth canal.
- Intravascular injection into rich plexus of sacral veins.
- Infection
- Dural puncture

Conclusions

- Safe, effective anaesthesia and analgesia.
- Not just for those not fit enough for GA!
- Thorough knowledge of indications, contraindications and techniques required.
- Serious complications rare – must be recognised and managed early.
- Less serious complications more common and usually treatable.

Regional anesthesia

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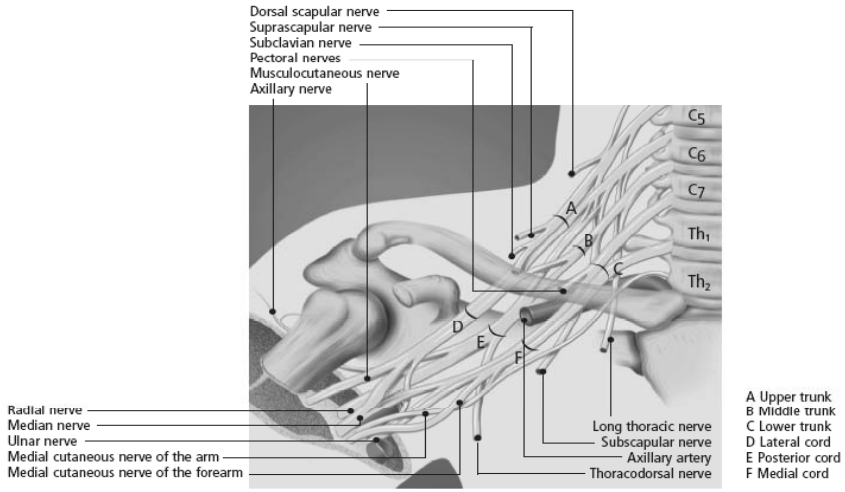
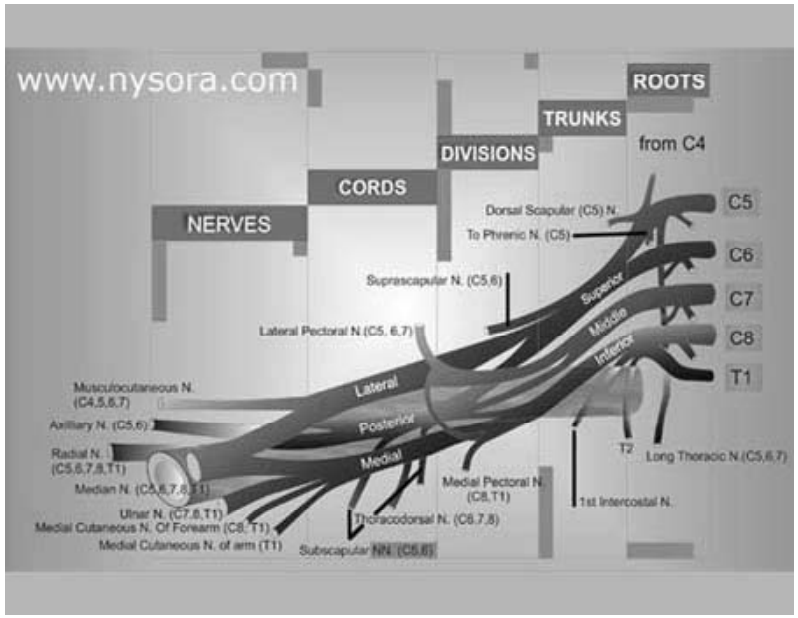
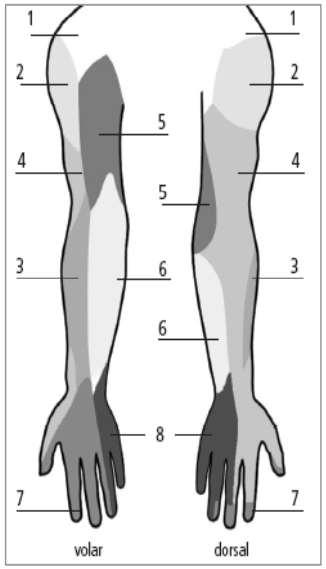
ANATOMY of BRACHIAL PLEXUS:



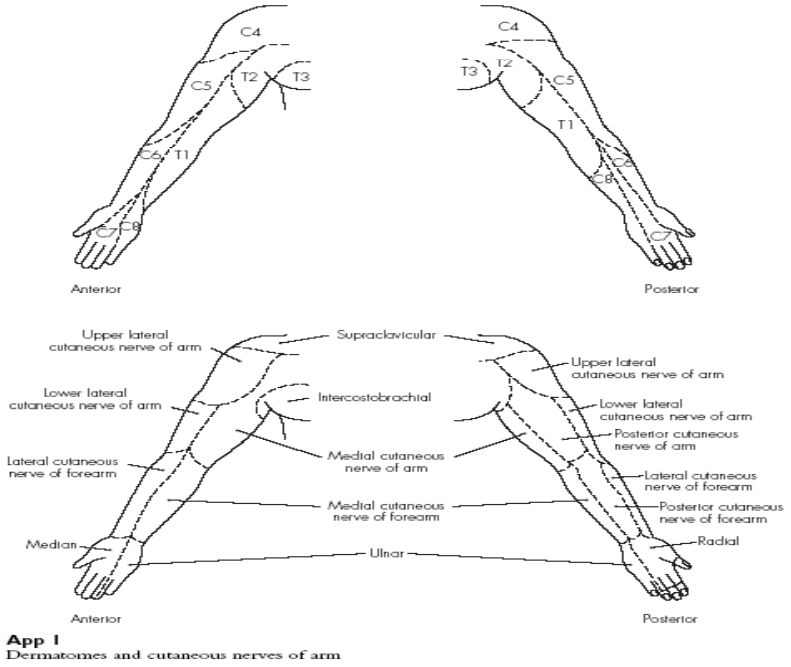


Motor supply areas

Peripheral nerve	Muscle	Function
Suprascapular nerve	Supraspinatus/ infraspinatus muscles	Forms parts of the rotator muscles
Axillary nerve	Deltoid muscle	Abduction of the arm in the shoulder joint
Musculocutaneous nerve	Biceps brachii muscle Brachial muscle	Bends the elbow in supination
	Flexor pollicis brevis muscle	Pronates the forearm (flexes proximal phalanx of thumb)
Median nerve	Flexor carpi radialis muscle	Flexes and abducts wrist radialward
	Flexor digitorum profundus muscle (I-III)	Flexes and adducts the thumb, flexes fingers I-III
Radial nerve	Triceps brachii muscle	Extends elbow
	Extensor carpi radialis (brevis) muscle	Extends and abducts wrist radialward
	Extensor digitorum muscle	Extends and flexes the hand dorsally Extends and spreads the fingers
Ulnar nerve	Flexor carpi ulnaris muscle	Flexes and abducts wrist ulnarward
	Flexor digitorum profundus muscle (IV-V)	Flexes fingers (IV-V)



1 Supraclavicular nerves (from the superficial cervical plexus)
2 Axillary nerve
3 Musculocutaneous nerve
4 Radial nerve
5 Medial cutaneous nerve of the arm
6 Medial cutaneous nerve of the forearm
7 Median nerve
8 Ulnar nerve



App I
Dermatomes and cutaneous nerves of arm

Electrical nerve stimulation

- Peripheral nerves :they contain either sensory or motor, sometimes both.
- Electrical impulses reaching a nerve.

- The electrical current required to trigger muscle contractions correlates with the distance of the tip of the needle to the nerve.
- The closer the needle is to the Nerve, the lower the electrical current that is required to induce contractions or sensory responses.
- In routine clinical practice, an initial electrical current, called threshold current, of 1 mA is used to elicit a response.
- The stimulation needle has reached the desired position at the nerve when contractions of the effecter muscle are induced at a threshold current of 0.2 - 0.3 mA (pulse duration of 0.1 ms).
- Lower pulse amplitudes may cause injury to the nerve.
- Pain fibers are not affected at this pulse duration.

STIMULATION NEEDLES:

- COMPLETELY INSULATED, EXCEPT FOR THE TIP.
- HAVE NO SHARP EDGES.
- THE ELECTRICAL CURRENT HAS A VERY SMALL EXIT OPENING.
- HIGHER CURRENT DENSITY AT THE TIP OF THE NEEDLE.
- EXACT LOCALIZATION, RISK OF INJURY AT A MINIMUM.

Fig. 6: Nerve stimulator: Stimuplex® HNS 12 (B. Braun Melsungen AG)

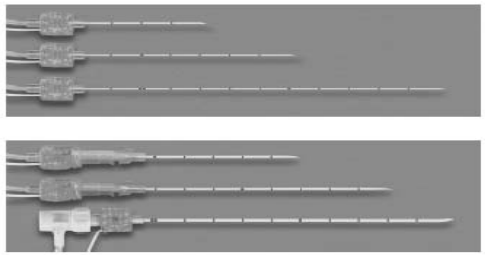


Fig. 7/8: Stimulation needles: Stimuplex® D / Contiplex® D / Contiplex® Tuohy (B. Braun Melsungen AG)

BLOCKS OF UPPER EXTREMITY:

- INTERSCALENE BRACHIAL PLEXUS BLOCK
- INFRACLAVICULAR BRACHIAL PLEXUS BLOCK
- AXILLARY BRACHIAL PLEXUS BLOCK
- WRIST BLOCK

INTERSCALENE BRACHIAL PLEXUS BLOCK:

- MOST CRANIAL
- TWO APPROACHES, ANT. & POST.
- SAME INDICATIONS & CONTRAINDICATIONS
- ANT., LESS TIME
- POST. USEFUL IN ANATOMICAL DIFFICULTY (NO NECK)
- **INDICATIONS:**

➢ SINGLE-SHOT TECHNIQUE

❑ SURGICAL INTERVENTIONS ON THE SHOULDER INCLUDING SHOULDER TOTAL ARTHROPLASTY, PROXIMAL HUMERUS, LATERAL CLAVICLE.

➢ CATHETER TECHNIQUE

❑ POSTOPERATIVE ANALGESIA REQUIREMENTS, E.G., ARTHROPLASTY OF THE SHOULDER JOINT

❑ SUPPORTIVE PHYSIOTHERAPY OF THE SHOULDER JOINT.

- **CONTRAINDICATIONS**

➢ CONTRALATERAL RECURRENT PARESIS

➢ CONTRALATERAL PHRENIC PARESIS

- **SIDE EFFECTS/COMPLICATIONS**

➢ PHRENIC PARESIS

➢ HORNER SYNDROME

➢ RECURRENT PARESIS

➢ VESSEL PUNCTURE (EXTERNAL JUGULAR VEIN, INTERNAL JUGULAR VEIN, COMMON CAROTID ARTERY)

➢ PNEUMOTHORAX (RARE)

LIMITATIONS:

➢ USEFUL ONLY IN SHOULDER SURGERY

Puncture site, Technique: Video 1

- Ant. Approach

Anatomical landmarks
Superior thyroid notch, sternocleidomastoid muscle (posterior scalene gap)

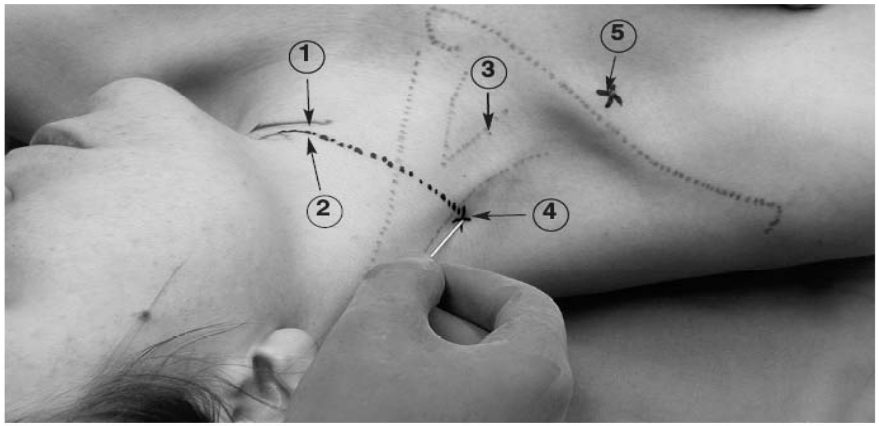


Fig. 11: Interscalene nerve block: Modification according to G. Meier

1. Cricoid
2. Superior thyroid notch
3. Sternocleidomastoid muscle
4. Puncture site for anterior access
5. Vertical, infraclavicular puncture site

Note:

- Pt supine (no pillow)
- Head tilted to other side
- External jugular vein
- Direction of puncture is caudad & dorsal to body axis
- Contractions of biceps indicates proximity to upper trunk
- Usually at 3-4 cm depth
- La injected at threshold current 0.2-0.3 mA
- Allow 15 minutes for adequate block

Post Approach: Video 2

Anatomical landmarks
Processus spinosus C7 (vertebra prominens), Hinterrand M. sternocleidomastoideus, Cricoid

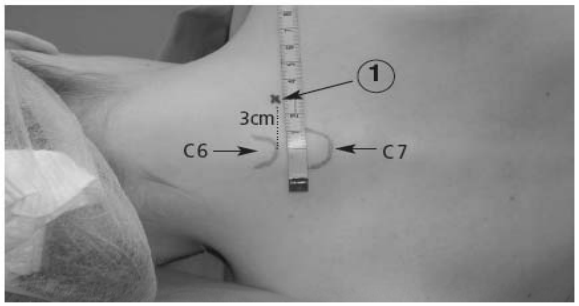


Fig. 13: Posterior access - Positioning

1. Puncture site

- Head placed on pillow
- Cervical spine flexed

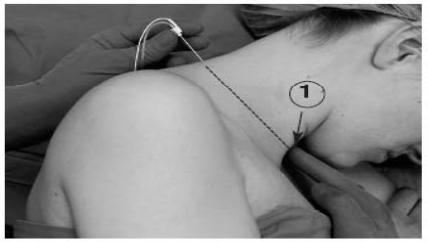


Fig. 14: Posterior access - Puncture technique

1. Cricoid

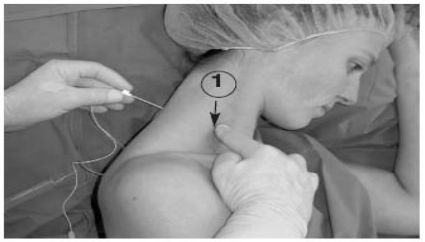


Fig. 15: Posterior access -Puncture technique

1. Posterior edge of the sternocleidomastoid muscle

- Puncture (10-15)° laterally towards post edge of SCM at level of cricoid
- Upon reaching transverse process of C7, adjust cranially
- Contraction of biceps indicates proximity to upper trunk

Infra clavicular brachial plexus block:

- Easy, safe, simple, low risk
- **Indications**
  - procedures in the region of the distal upper arm, on the forearm and the hand.
- **Contraindications**
  - Chest deformities
  - dislocated fracture of the clavicle
  - Uncertainity in puncture site identification

Side effects/complications

- Horner syndrome
- Vessel puncture (cephalic vein, subclavian artery and vein and their branches)
- Pneumothorax
- Phrenic nerve paresis (very rare)

Puncture site & technique:

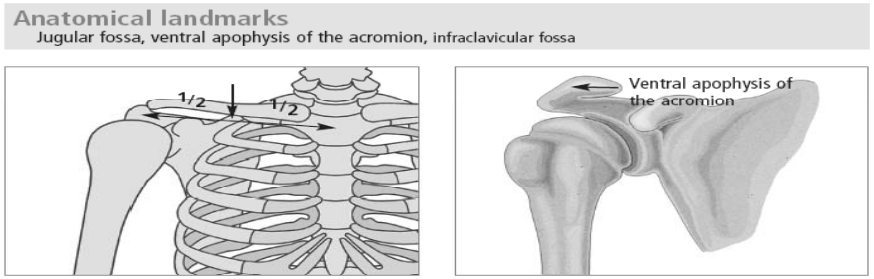


Fig. 16: IVBP- Principal structures for orientation

Fig. 17: IVBP - Lateral limit

- midway between sternal notch & acromion process not coracoid process
- 4 cm is the maximum depth of plexus
- Puncture should be vertical & immediately under clavicle
- Medial direction results in injury to axillary vessels

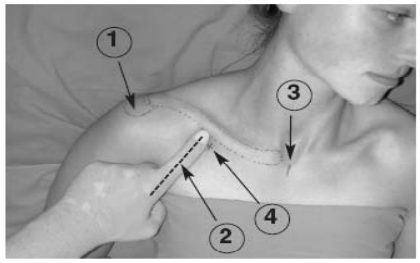


Fig. 20: VIP - Puncture site

1. Ventral apophysis of the acromion
2. Infraclavicular fossa
3. Jugular fossa
4. Puncture site

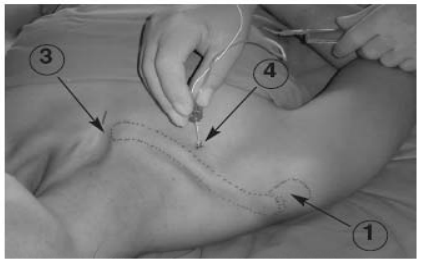


Fig. 21: VIP - Puncture technique

- Pt supine
- Ipsilateral hand relaxed, preferably on abdomen
- Thin pts are more at risk of pneumothorax
- Local injected when desired muscle group are stimulated (flexors or extensors of the fingers)
- **Video 3**

Sources of error:

- Precise & exact localisation of land marks is extremely important to avoid complications
- Puncture should be vertical to supporting surface (Bed) not to the pt.
- Stay close to clavicle
- If in doubt, re-localize ur landmarks

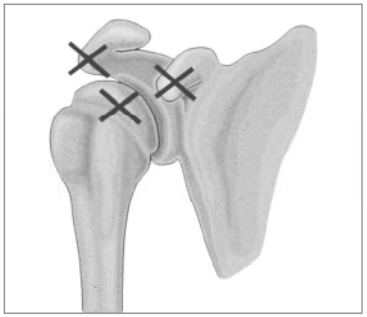


Fig. 22: Sources of error and risks

Advantages of the infraclavicular vertical brachial plexus block

- Clearly defined guide points - clearly defined puncture direction
- Simple to learn - high success rate
- No anaesthetic gaps resulting from the procedure
- No problems with the Esmarch tourniquet
- Comfortable positioning of the patient

Axillary brachial plexus block:

- performed in an area in which cords have already formed the peripheral nerves of the arm
- axillary nerve and musculocutaneous nerve emerge from the plexus above the puncture site.
- widespread technique because it is simple to use and has few complications.

- **Indications**

➢ procedures on the elbow, forearm and hand.

- **Contraindications**

➢ no specific contraindications

Side effects/complications

➢ no specific side effects

- **Advantages**

➢ simple and low-risk and can be performed with or without the nerve stimulator.

- **Disadvantages**

➢ upper arm tourniquet may be poorly tolerated because the medial upper arm is supplied by the intercostobrachialis nerves (Th2) and the lateral upper arm by the axillary nerve (which is usually not blocked).

➢ Frequent gaps in the area of the musculocutaneous nerve and the radial nerve are another disadvantage.

➢ These technique-related weaknesses can be compensated by carrying out secondary peripheral blocks of isolated nerves.

Landmarks & Technique:

Anatomical landmarks

Axilla, axillary artery, medial bicipital groove, pectoralis major muscle, coracobrachialis muscle

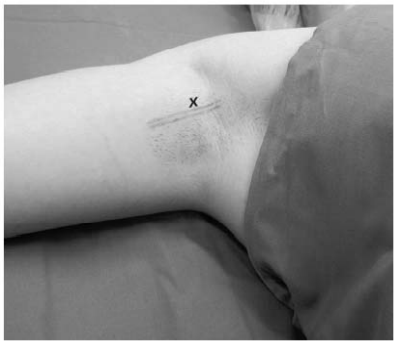


Fig. 23:
Axillary nerve block - Puncture site

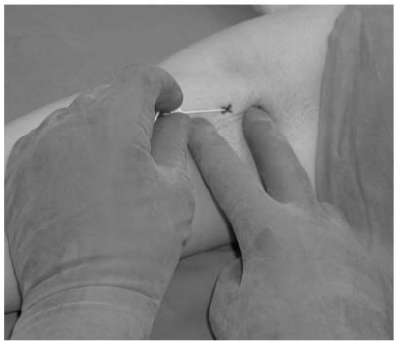
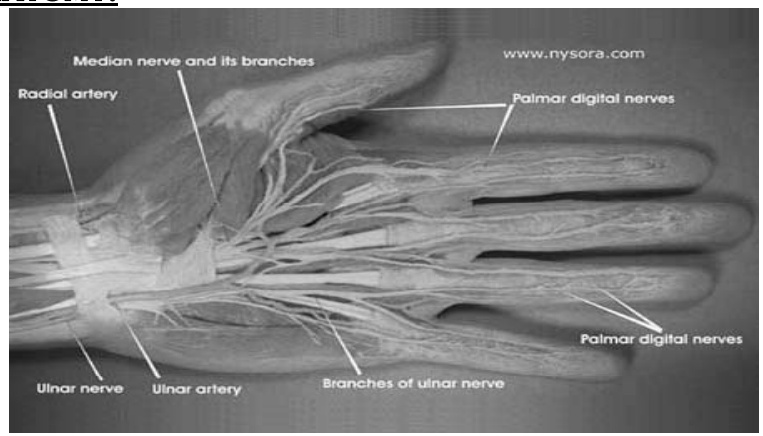


Fig. 24:
Axillary nerve block -Puncture technique

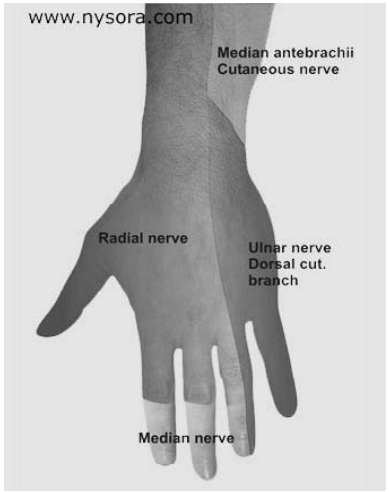
- Supine pt
- Arm abducted 90 degrees, relaxed.
- Axillary artery is felt dorsal to bicipital groove
- Puncture is slightly above axillar artery, high in the axilla, bneath pectoralis muscle
- Needle is inserted parallel to the axillary artery at a 30°-angle to the skin.
- Desired response is area of radial or ulnar nerve
- Video

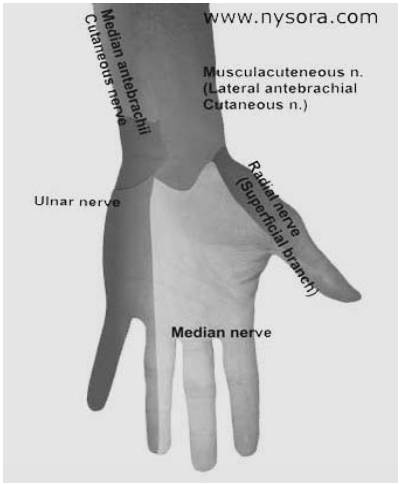
Wrist block:

- **Indications:** surgery on hand & fingers
- **Nerves**: Ulnar, Radial & Median (terminal branches)
- **Anatomy:**



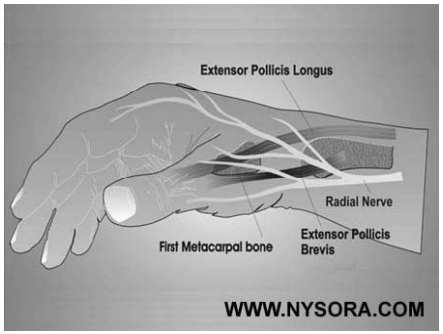
Distribution of Anesthesia





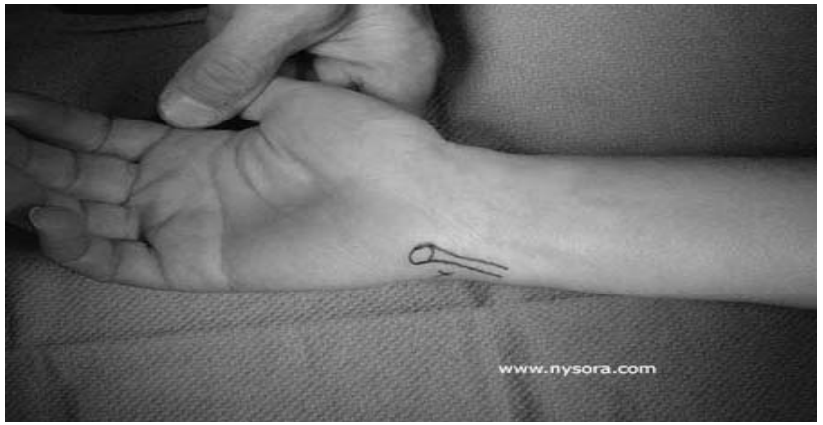
- **Landmarks:**

➢ **Radial Nerve**



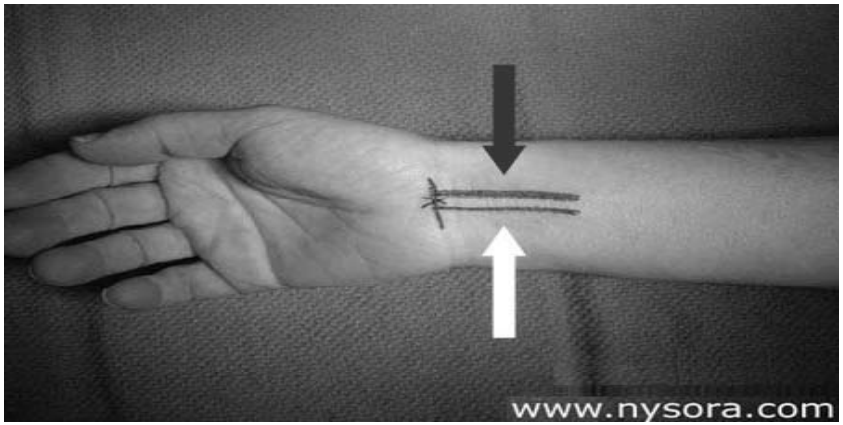
➢ **Ulnar Nerve:**

between the ulnar artery and tendon of the flexor carpi ulnaris.



➢ **Median Nerve:**

between the tendons of the palmaris longus and the flexor carpi radialis



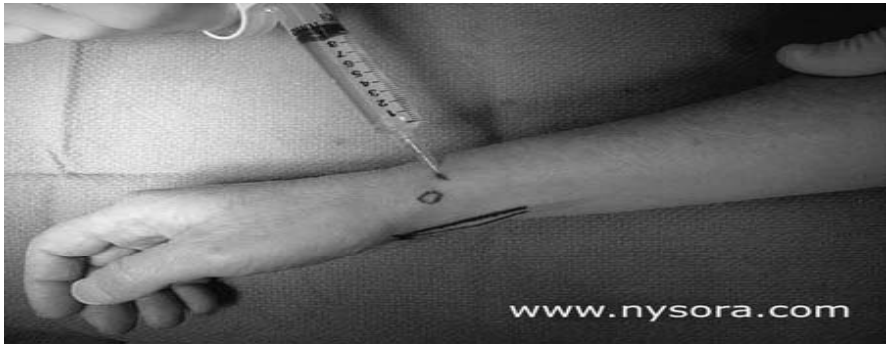
Technique:

➢ The radial nerve: field block, extensive infilt.

➢ less predictable anatomic location

➢ and division into multiple smaller branches.

➢ above the radial styloid, aiming medially. The infiltration is then extended laterally,



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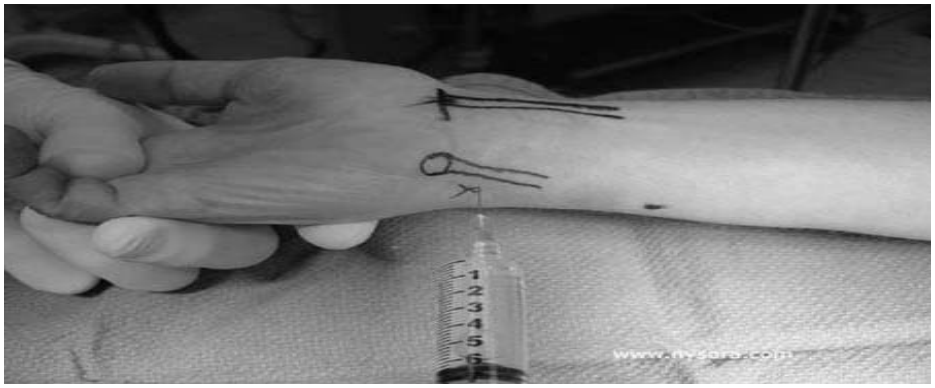
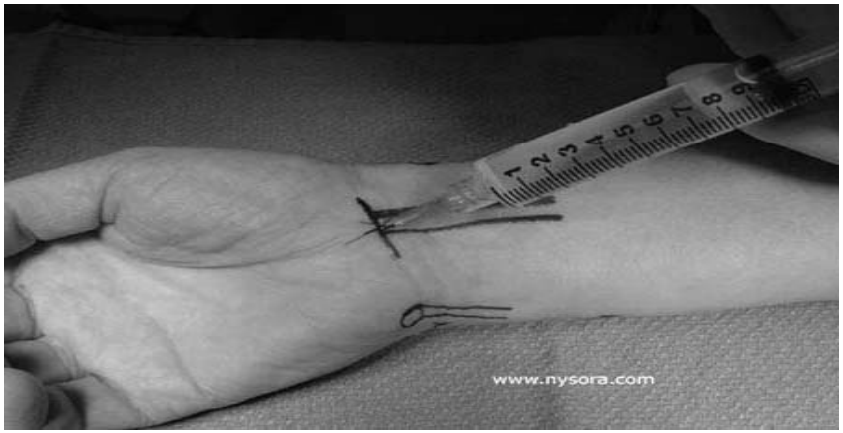


➢ MEDIAN NERVE

➢BETWEEN THE TENDONS OF THE PALMARIS LONGUS AND FLEXOR CARPI RADIALIS. PIERCING DEEP FASCIA. A FASCIAL "CLICK"

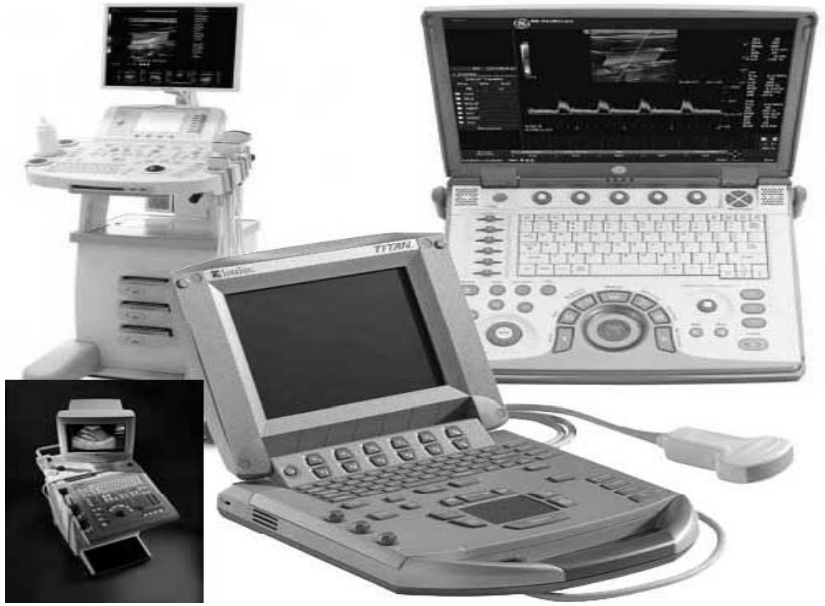


ULTRASOUND-ASSISTED NERVE BLOCKS

• GROWING INTEREST

• IMPROVING BLOCK SUCCESS AND DECREASING COMPLICATIONS.

• ALLOWS ONE TO VISUALIZE NEURAL STRUCTURES (PLEXUS AND PERIPHERAL NERVES) AND THE SURROUNDING STRUCTURES (E.G., BLOOD VESSELS AND PLEURA), NAVIGATE THE NEEDLE TOWARD THE TARGET NERVES, AND VISUALIZE THE PATTERN OF LOCAL ANESTHETIC SPREAD.



Ultrasound principles:

- An ultrasound probe emits and receives sound waves
- ultrasound waves are high-frequency sound waves (20,000 cycles/s, 20 kHz)
- not audible to the human ear.
- frequencies useful in clinical medicine are in the megahertz (MHz) range.(
- As the ultrasound waves move through body tissues they lose amplitude, reflected, and/or scattered.
- Waves reflected to the transducer are then transformed into an electrical signal that is then processed by the ultrasound machine to generate an image on the screen .

- Depending on the amount of wave returned, anatomic structures take on different degrees of echogenicity.
- high water content structures, eg blood vessels and cysts, appear hypoechoic (black or dark), because ultrasound waves are transmitted through the structures easily with little reflection.
- bone and tendons block ultrasound wave transmission and the strong signal returned to the transducer gives these structures a hyperechoic appearance (bright, white) on the screen.
- Structures of intermediate density and acoustic impedance, such as the liver parenchyma or the thyroid gland, appear gray on the screen.

- Knowing the speed of sound in tissue (1540 m/s on average) and the time of echo return, the distance between the probe and the target structure (depth) is calculated.

- Orientation is extremely important

Puncture site, Technique: Video 1

- Ant. Approach

Anatomical landmarks

Superior thyroid notch, sternocleidomastoid muscle (posterior scalene gap)

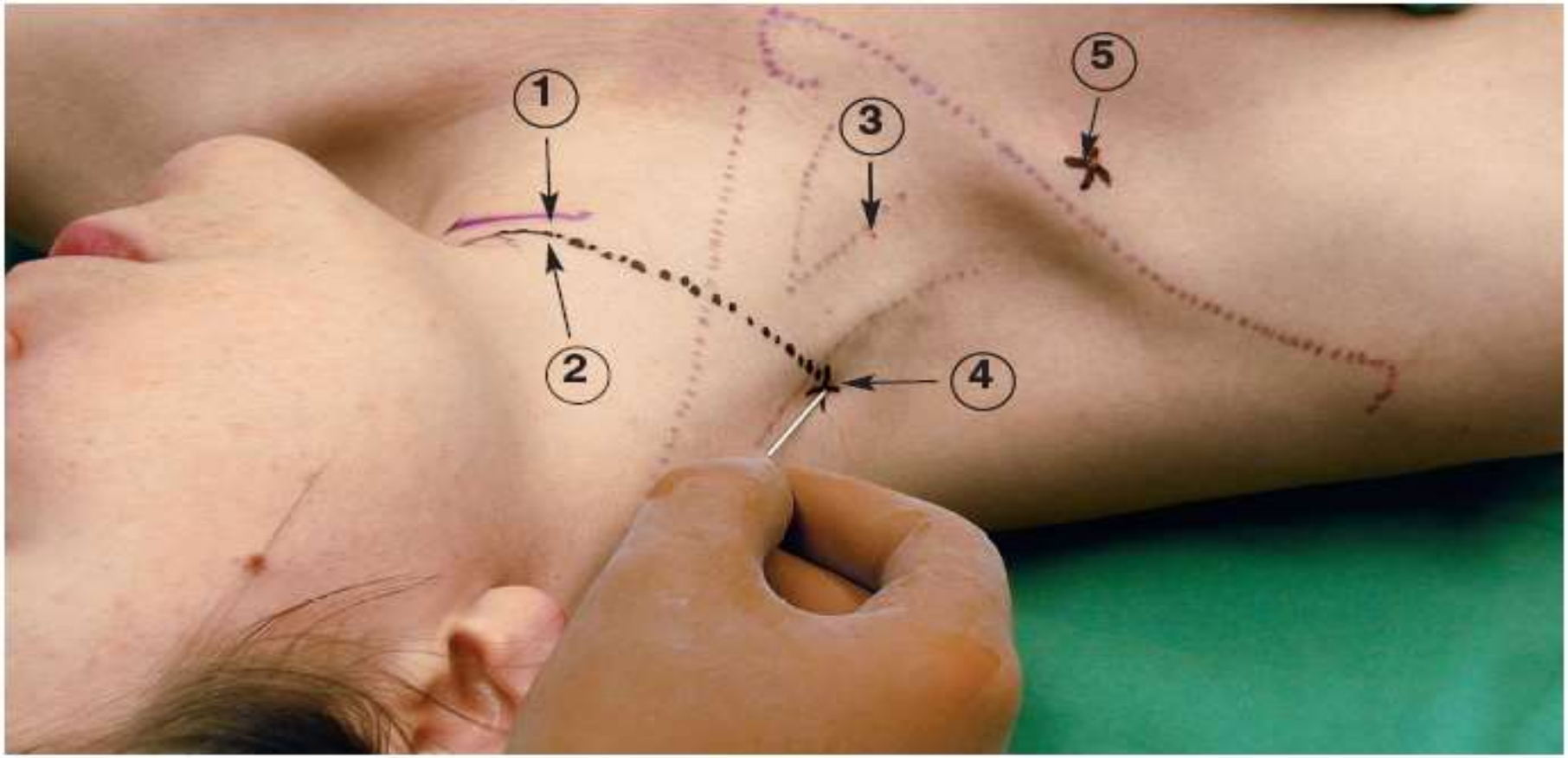


Fig. 11: Interscalene nerve block: Modification according to G. Meier

1. Cricoid
2. Superior thyroid notch
3. Sternocleidomastoid muscle
4. Puncture site for anterior access
5. Vertical, infraclavicular puncture site

Note:

- Pt supine (no pillow)
- Head tilted to other side
- External jugular vein
- Direction of puncture is caudad & dorsal to body axis
- Contractions of biceps indicates proximity to upper trunk
- Usually at 3-4 cm depth
- La injected at threshold current 0.2-0.3 mA
- Allow 15 minutes for adequate block

Post Approach: Video 2

Anatomical landmarks

Processus spinosus C7 (vertebra prominens), Hinterrand M. sternocleidomastoideus, Cricoid

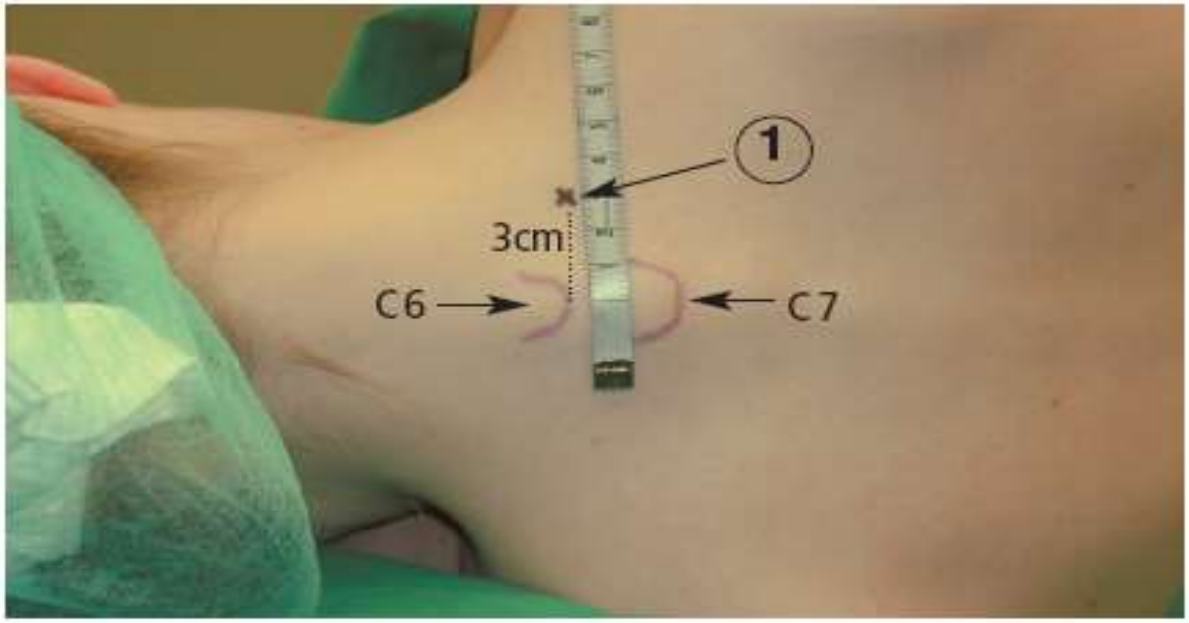


Fig. 13: Posterior access - Positioning

1. Puncture site

- Head placed on pillow
- Cervical spine flexed

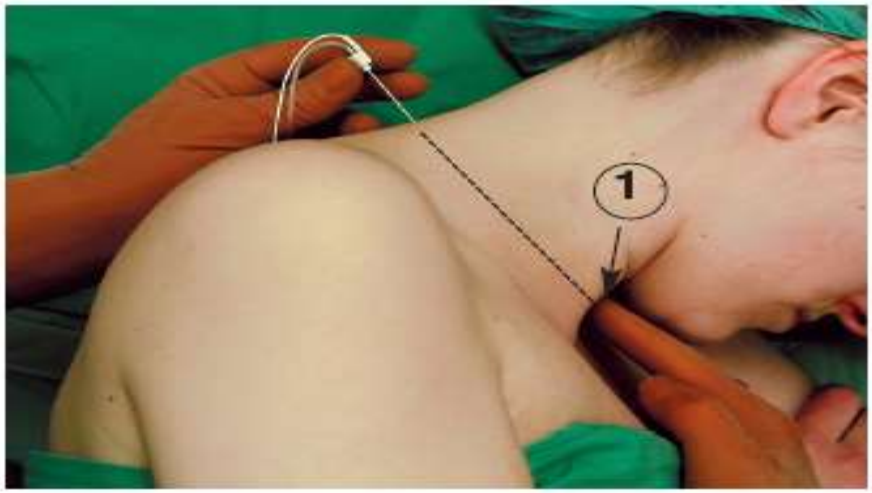


Fig. 14: Posterior access - Puncture technique

1. Cricoid

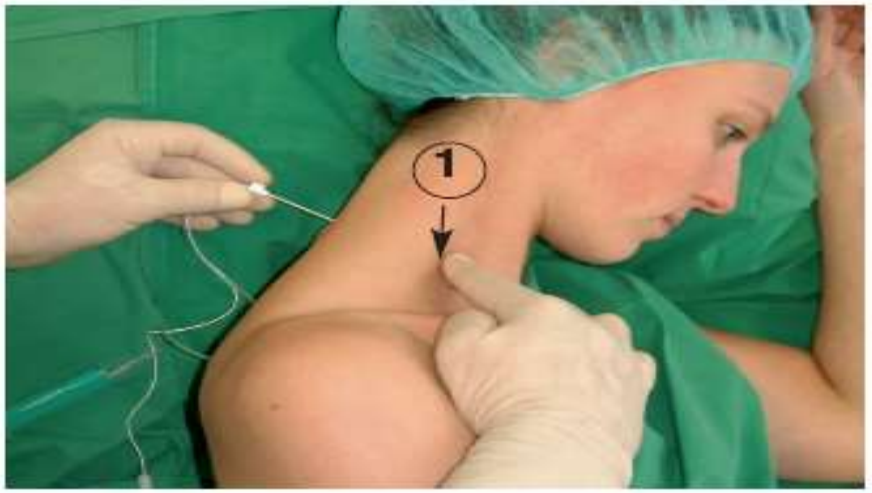


Fig. 15: Posterior access -Puncture technique

1. Posterior edge of the sternocleidomastoid muscle

- Puncture (10-15)° laterally towards post edge of SCM at level of cricoid
- Upon reaching transverse process of c7, adjust cranially
- Contraction of biceps indicates proximity to upper trunk

Infra clavicular brachial plexus block:

- Easy, safe, simple, low risk
- **Indications**

➢ procedures in the region of the distal upper arm, on the forearm and the hand.

- **Contraindications**

➢ Chest deformities

➢ dislocated fracture of the clavicle

➢ Uncertainity in puncture site identification

Side effects/complications

- Horner syndrome
- Vessel puncture (cephalic vein, subclavian artery and vein and their branches)
- Pneumothorax
- Phrenic nerve paresis (very rare)

Puncture site & technique:

Anatomical landmarks
Jugular fossa, ventral apophysis of the acromion, infraclavicular fossa

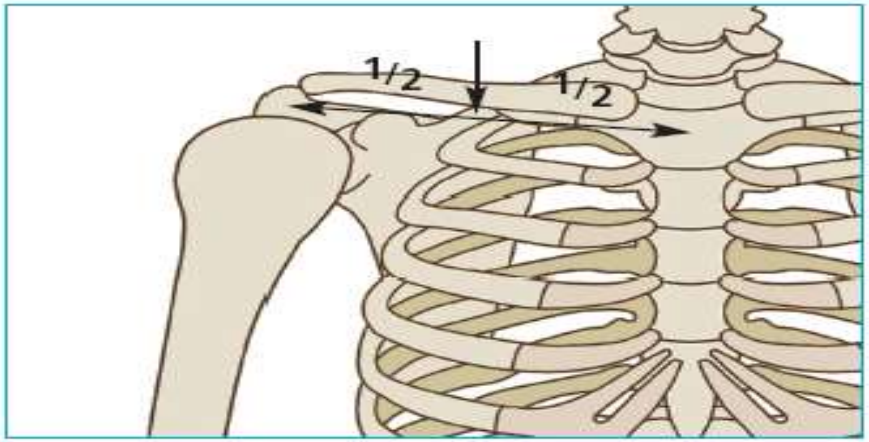


Fig. 16:
IVBP- Principal structures for orientation

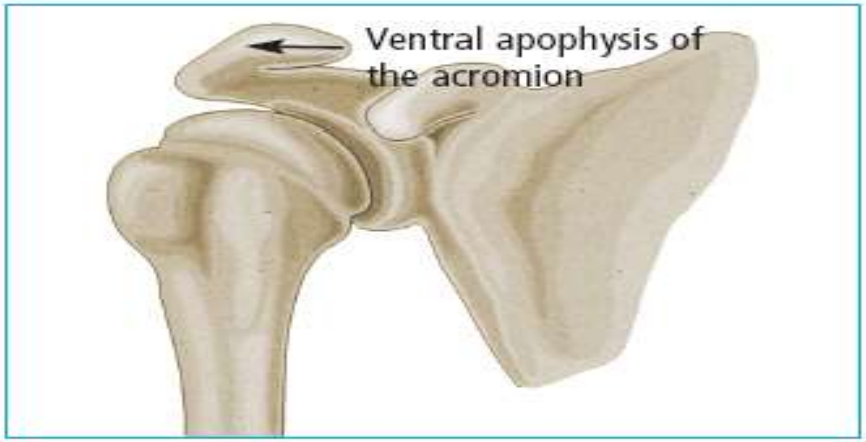


Fig. 17:
IVBP - Lateral limit

- Midway between sternal notch & acromion process not coracoid process
- 4 cm is the maximum depth of plexus
- Puncture should be vertical & immediately under clavicle
- Medial direction results in injury to axillary vessels

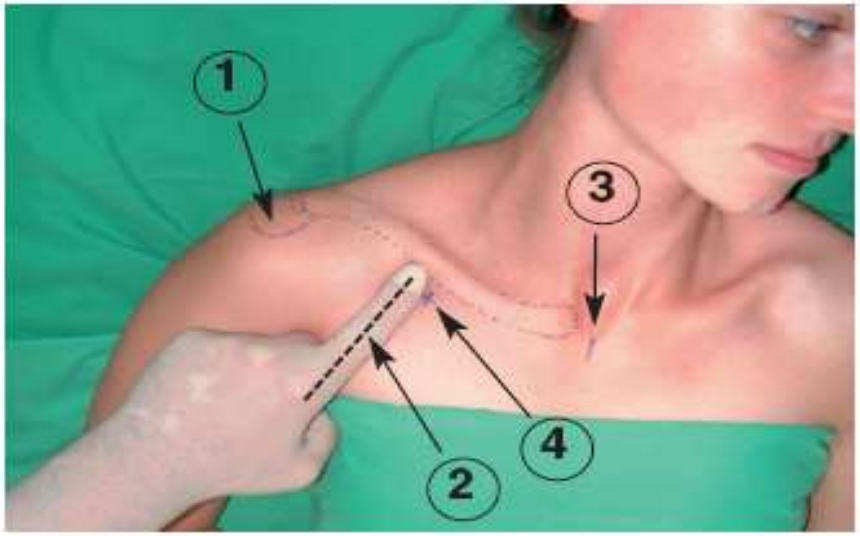


Fig. 20: VIP - Puncture site

1. Ventral apophysis of the acromion
2. Infraclavicular fossa
3. Jugular fossa
4. Puncture site

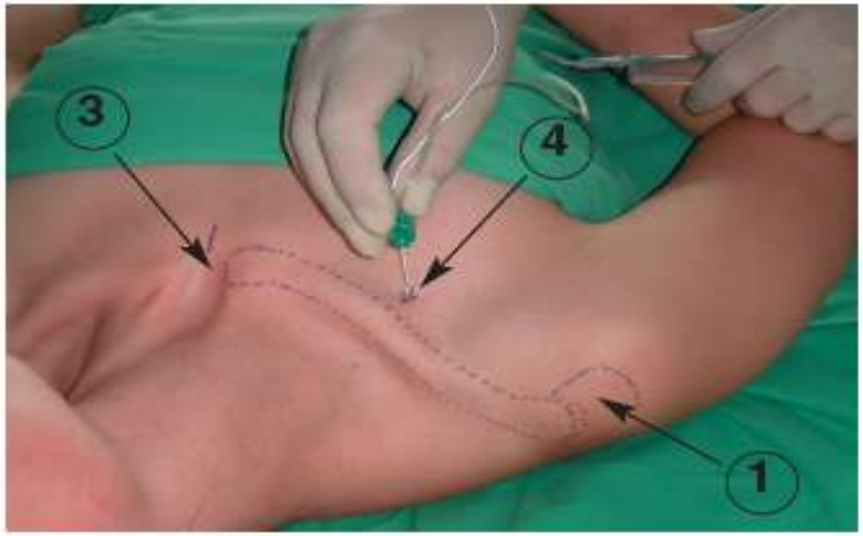
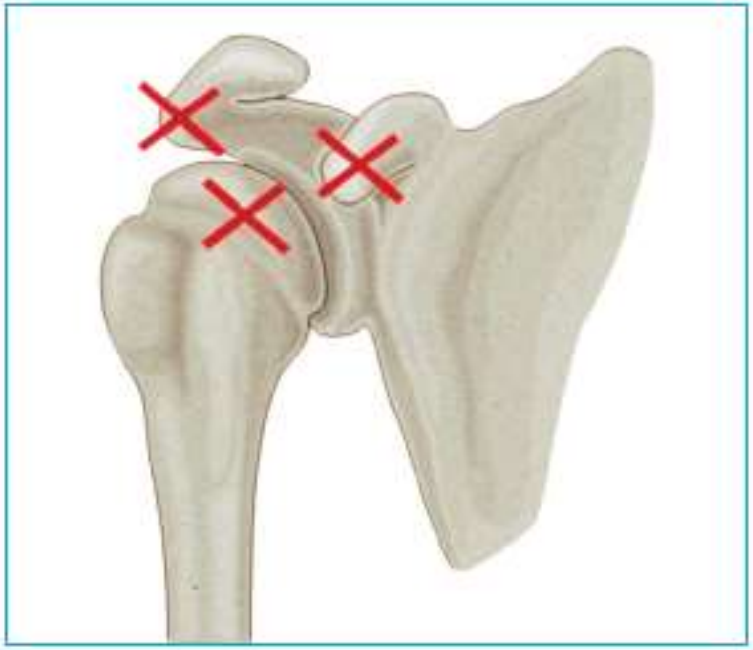


Fig. 21: VIP - Puncture technique

- Pt supine
- Ipsilateral hand relaxed, preferably on abdomen
- Thin pts are more at risk of pneumothorax
- Local injected when desired muscle group are stimulated (flexors or extensors of the fingers)
- Video 3

Sources of error:

- Precise & exact localisation of land marks is extremely important to avoid complications
- Puncture should be vertical to supporting surface (Bed) not to the pt.
- Stay close to clavicle
- If in doubt, re-localize ur landmarks



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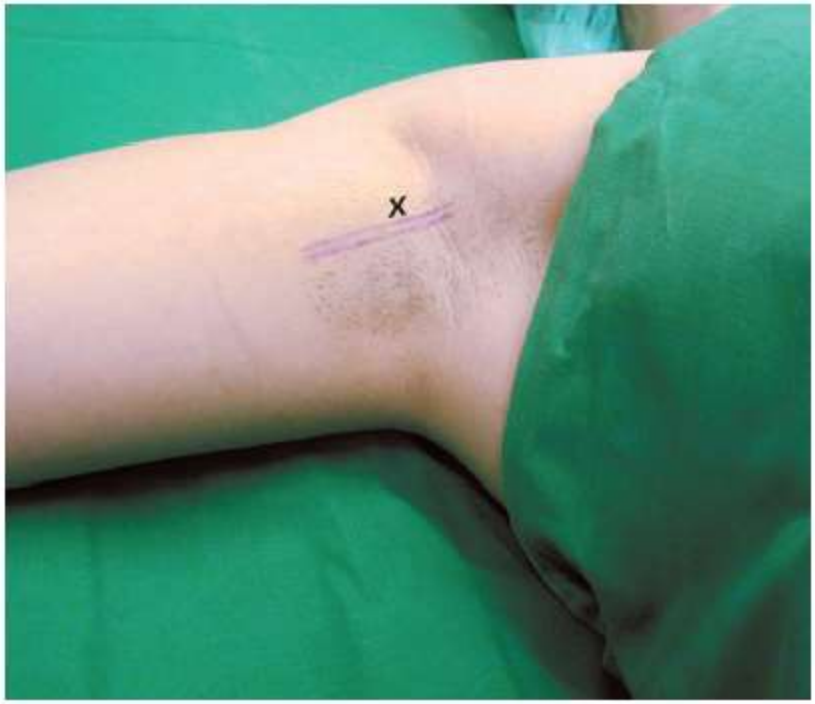
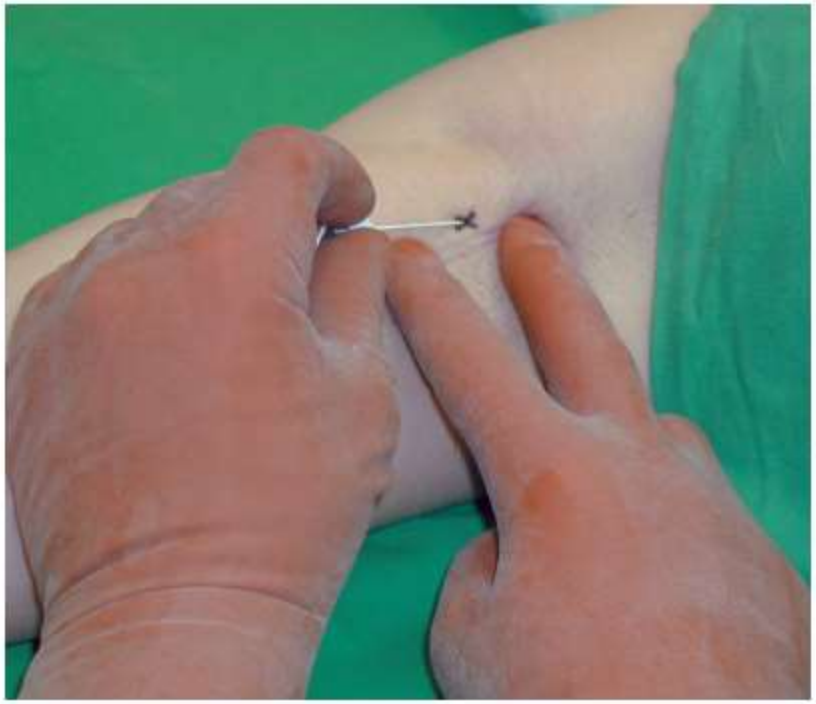


Fig. 23:
Axillary nerve block - Puncture site



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Axillary nerve block -Puncture technique

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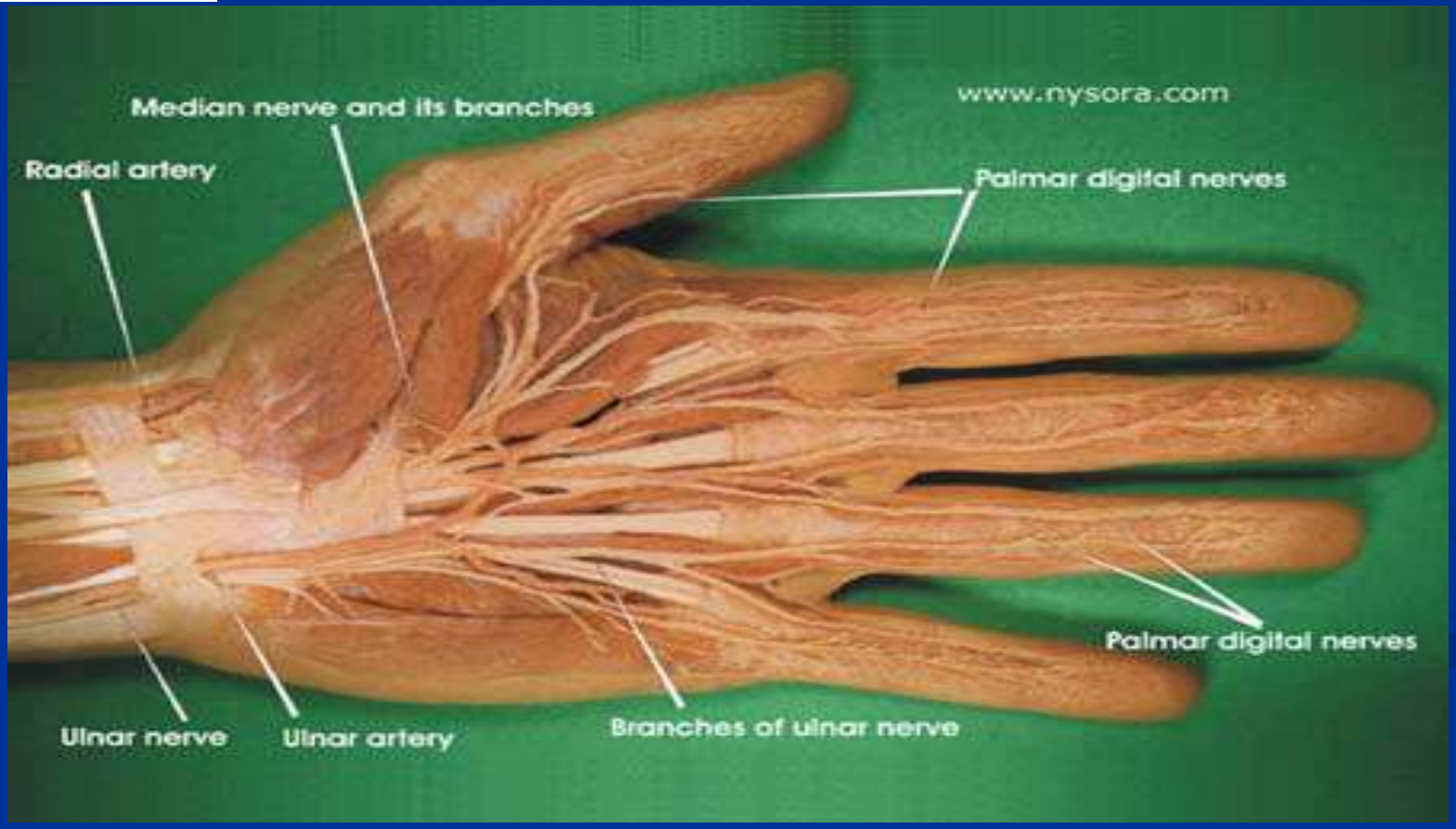
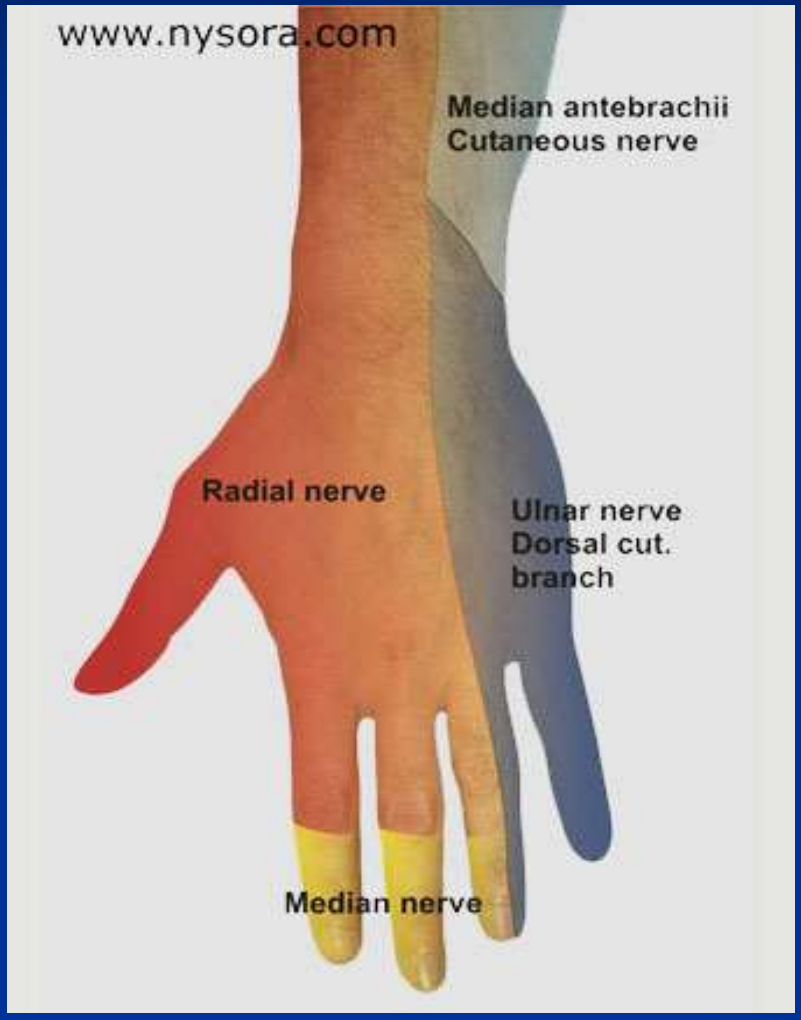
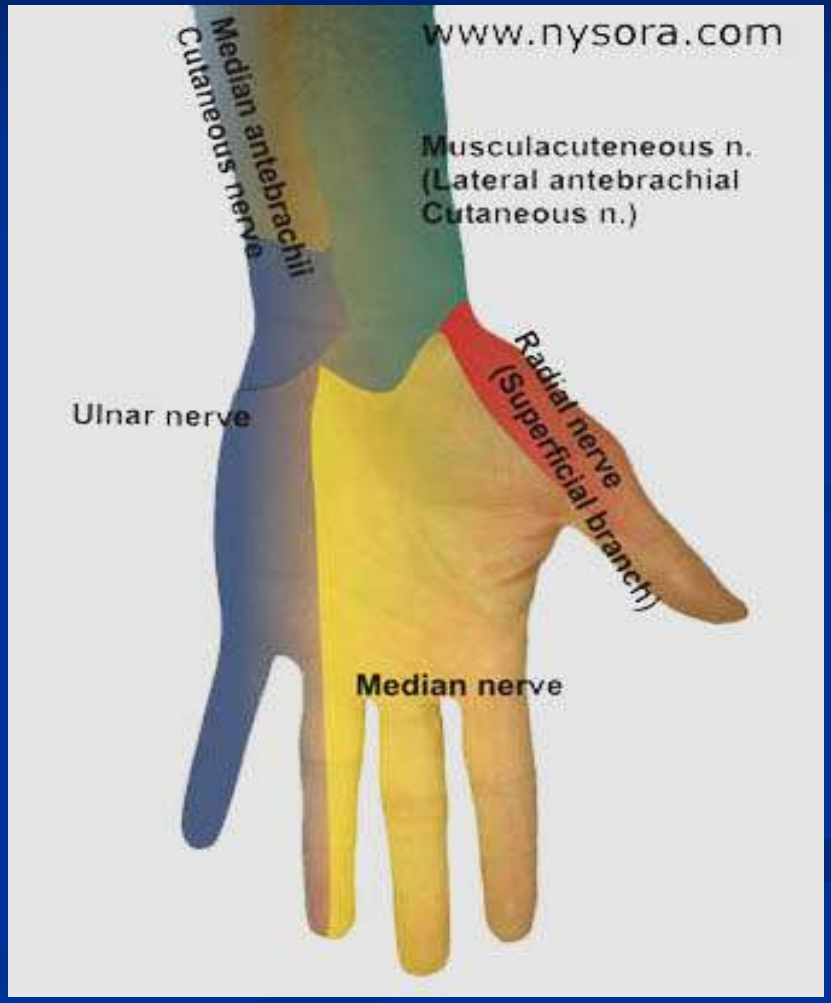
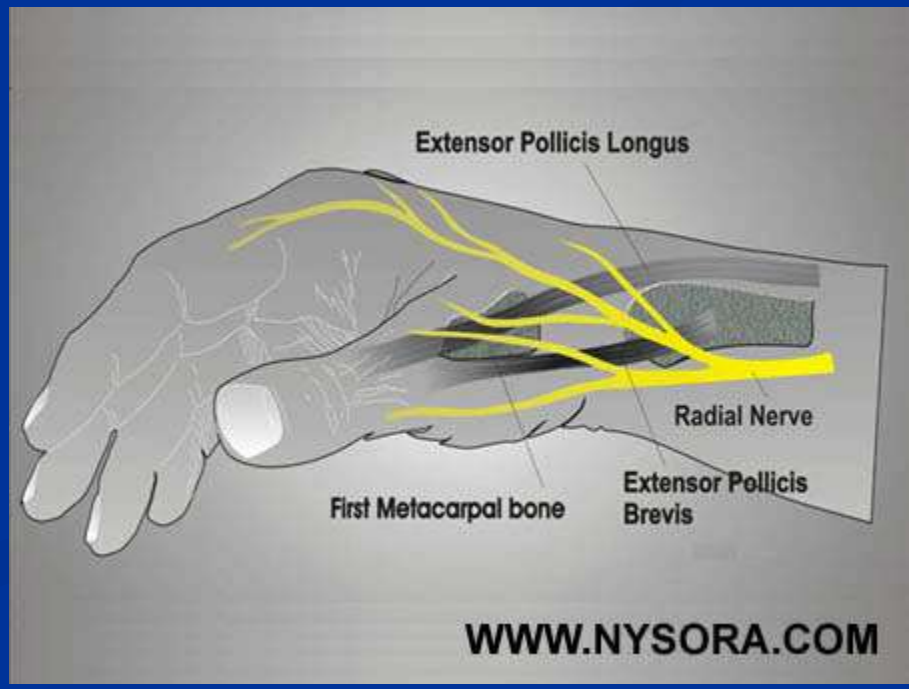


Distribution of Anesthesia





- **Landmarks:**
  - **Radial Nerve**



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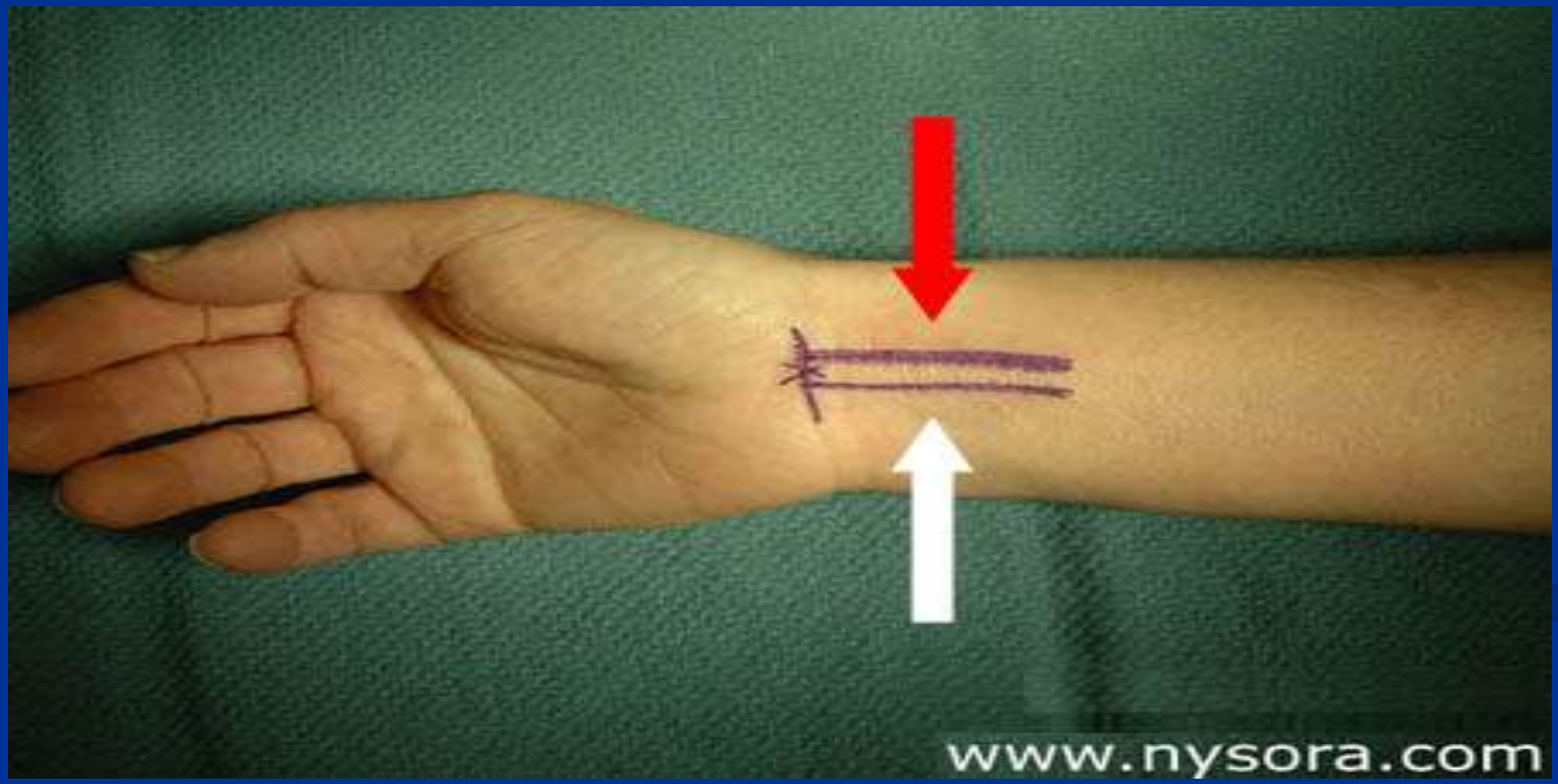

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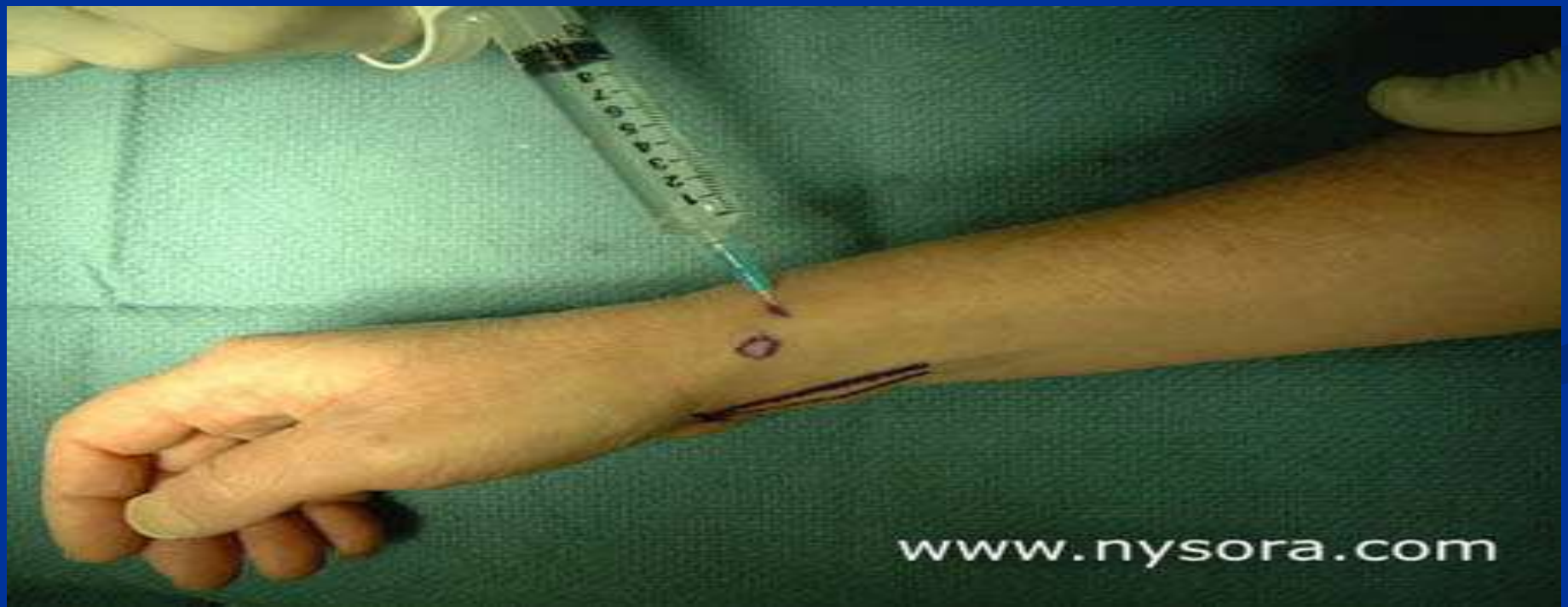

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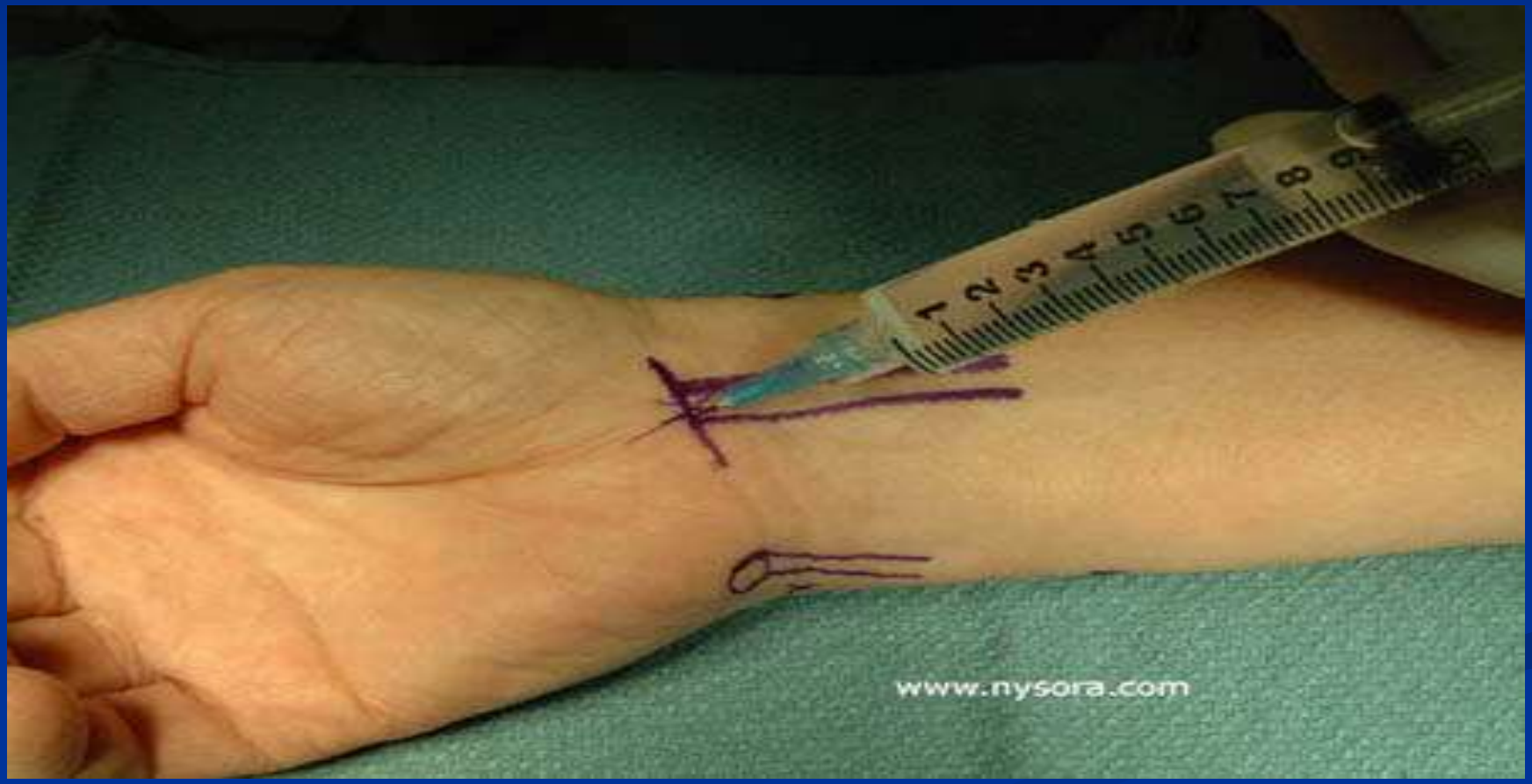

Ultrasound-Assisted Nerve Blocks

- growing interest

- improving block success and decreasing complications.
- allows one to visualize neural structures (plexus and peripheral nerves) and the surrounding structures (e.g., blood vessels and pleura), navigate the needle toward the target nerves, and visualize the pattern of local anesthetic spread.


TITAN

ND PRINCIPLES:

SOUND PROBE EMITS AND RECEIVES SOUND

UND WAVES ARE HIGH-FREQUENCY SOUND
,000 CYCLES/S, 20 KHZ)
BLE TO THE HUMAN EAR.

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- Orientation is extremely important

Part 2

Lecture topic :Airway Managment
Lecturer: Dr.Ibraheem Qudaisat
Date: 10/2012
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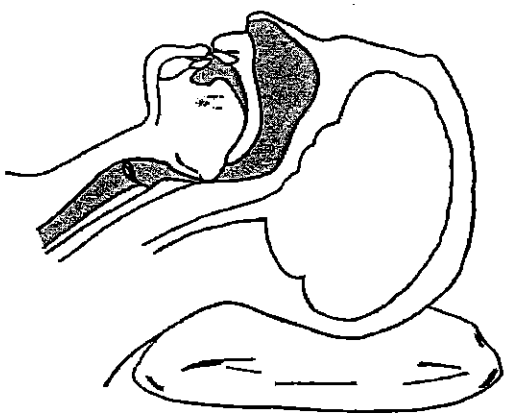
Airway Managment

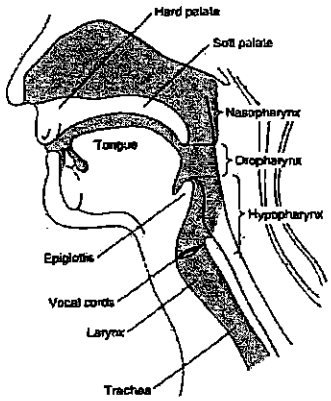
When a casualty becomes unconscious, all of his muscles may relax. This relaxation may cause the casualty's tongue to slip to the back of his mouth(against the posterior pharyngeal wall) and cover the opening to his trachea (windpipe).

If the casualty appears to be unconscious, check the casualty for responsiveness. Ask in a loud, but calm, voice: "Are you okay?" Also, gently shake
If the casualty does not respond, open his airway

If you see something in the casualty's mouth (foreign material, loose teeth, dentures, facial bone, vomitus, etc.) that could block his airway, use your fingers to remove the material as quickly as possible

Removing the obstruction and opening theairway may allow the casualty to resume breathing on his own. Two approved methods of opening the casualty's airway are the head-tilt/chin lift method and the jaw thrust method.
If you suspect that the casualty has suffered a neck or spinal injury, use the jaw thrust method. Otherwise, use the head-tilt/chin-lift method





Non-invasive (Non-definitive airway) (Above the vocal cord) (Supra glottic)

A. Head-Tilt/Chin-Lift Method.

Place one of your hands on the casualty's forehead and apply firm, backward pressure with the palm of your hand to tilt the head back.

Place the fingertips of your other hand under the tip of the bony part of the casualty's lower jaw and bring the chin forward.

CAUTION:

- Do not use this method if a spinal or neck injury is suspected

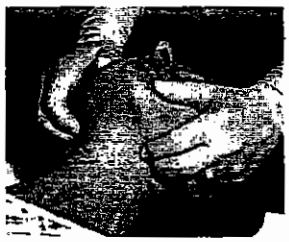
- Do not use the thumb to lift the lower jaw.
- Do not press deeply into the soft tissue under the chin with the fingers as this could close the casualty's airway.
- Do not allow the casualty's mouth to close. The mouth must remain open so the casualty can breath air in and out. the thumb may be used to depress the casualty's lower lip slightly to keep his mouth open

this method (chin left) will pull the hyoid bone so can pull the tongue away from the posterior pharyngeal wall

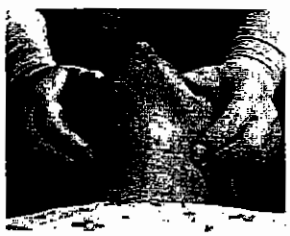
B. JAW THRUST METHOD.

Rest your elbows on the surface where casualty is lying (ground, etc.).

- Place one hand on each side of the casualty's lower jaw at the angle of
- the jaw, below the ears.
- Stabilize the casualty's head with your forearms.
- Use the index and middle fingers to push the angles of the patient's lower jaw forward.
- Use the thumb to retract the patient's lower lip to keep the casualty's
- mouth open, if necessary.

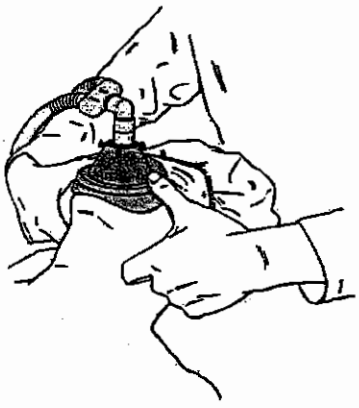


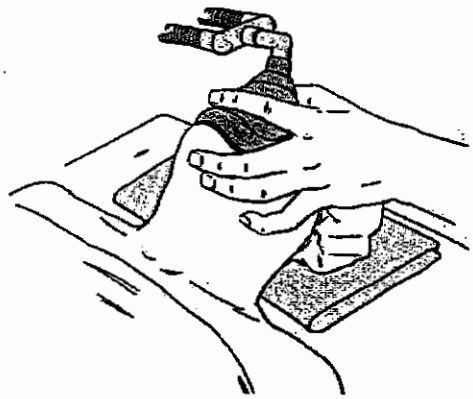
CAUTION:
Do not tilt or rotate the casualty's head if suspect the neck injury.
Do not allow the casualty's mouth to close. The mouth must remain open so the casualty can breath air in and out.



Applying face mask with jaw thrust

USING SINGLE HAND TECHNIQUE (c- clamp method)
USING TWO HAND TECHNIQUE





By tow above method I can not use my hands to another thing so if I need to do any task I can use the other methods for open the airway as that are given below

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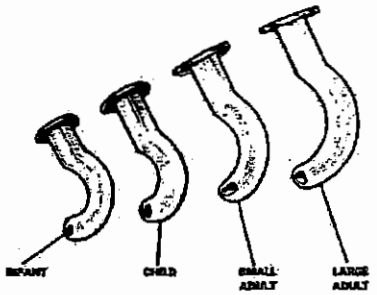
C. OROPHARYNGEAL AIRWAYS (J-TUBES)

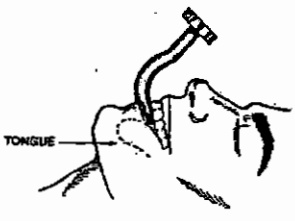


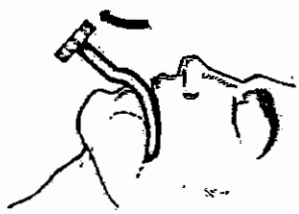
The oropharyngeal airway is
a semicircular apparatus of plastic. The apparatus is also called a J-tube because of its shape. It is curved to fit
over the back of the tongue and is inserted into the lower posterio r wall of the pharynx.
In this location, the apparatus will hold the tongue away from the posterior wall of the
pharynx and keep the patient's airway patent (open).
The insertion of this mechanical airway device is advised only when the patient is unconscious and does not have a gag reflex.

Select the Proper Size Oropharyngeal Airway.
To select the correct size of airway,select one of the J-tubes and hold it alongside the patient's jaw(jaw in the normal position with the mouth closed). The n measure from the corner of the patient's mouth to behind the angle of the mandible or anterior to the bottom tip of ear. Use the J-tube that best matches this Measurement.If the size is small the oral airway will push the tongue to the back.

Insert the Oropharyngeal Airway.
Remember, the oropharyngeal airway is
used for UNCONSCIOUS patients only. Do not try to insert the artificial airway in a conscious or semi conscious patient due to the patient's gag reflex. The presence of an airway in such patients may induce vomiting and cause aspiration of the stomach contents into the lungs.

- Place the tip of the airway into the patient's mouth
- Point the tip of the airway toward the roof of the patient's mouth to prevent the tongue from being pushed into the back of the throat.
- Slide the airway along the roof of the mouth, following the natural curvature of the tongue, past the soft palate.
- Rotate the airway 180 degrees as the tip reaches the back of the tongue
- Gently advance the airway and adjust it so the flange rests on the patient's lips. If the flange of the airway does not seat properly or if the patient begins to gag or vomit, the airway may be the wrong size. The tip of the airway should rest just above the epiglottis.





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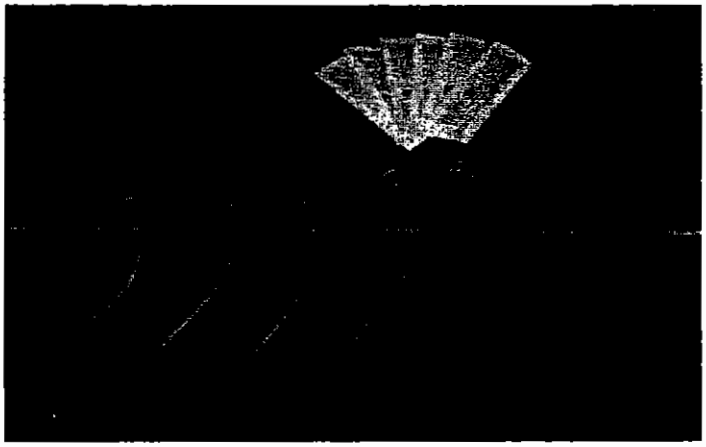


The nasopharyngeal airway is a flexible tube inserted through the external nasal passage and into the nasopharynx and oropharynx to elevate the tongue off of the back of the throat to maintain a patent airway. The insertion of this devise can be used with a conscious or unconscious patient; therefore it is the preferred airway during the tactical field care phase. It is also more effective than the oropharyngeal airway because it is less likely to be dislodged during movement of the patient during evacuation

PROCEDURE FOR INSERTING THE NASOPHARYNGEAL AIRWAY

- **Determine the Need for the Nasopharyngeal Airway.** Before you insert the nasopharyngeal airway (NPA), you must assess the patient. This assessment should include checking for signs of facial trauma; this is a contraindication for the use of the nasopharyngeal airway. The assessment should also include determination of the casualty's mental status. Any casualty with an altered mental status should have their airway protected with a nasopharyngeal airway. Use the following indications and contraindications to help with determining the need for the nasopharyngeal airway.

Indications:

- Casualty is conscious, semi-conscious or has an active gag reflex.
- Casualty has injuries to mouth (for example, broken teeth, massive
- oral tissue damage).
- Seizure casualties who may have clenched teeth due to active seizing.
- When vomiting is likely to occur.

Contraindications.

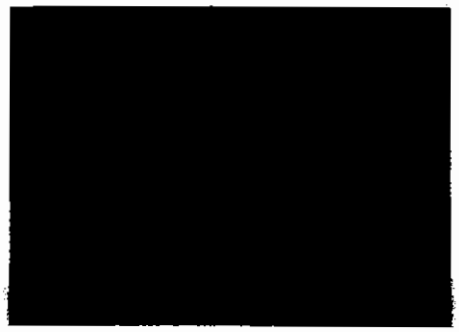
- Any evidence of a head injury or roof of mouth (cribriform) fracture;
- the airway may inadvertently enter the cranial vault with this type fracture.
- Exposed brain matter.
- Cerebrospinal fluid (CSF) draining from nose, mouth, or ears

Determine the Proper Size Nasopharyngeal Airway.

- **Diameter**: Select an airway with a diameter smaller than the casualty's

nostril or one that is approximately the diameter of the casualty's little finger.

- **Length**: Measure from the tip of the patient's nose to their ear lobe



NOTE:
Most nasal pharyngeal airways are made to fit the right nostril. If you have to insert it into the left nostril, turn the airway upside down so that bevel remains toward the septum, then insert it straight back until you reach the posterior pharynx. Turn the airway 180 degrees until it lies behind the tongue.

Nasopharyngeal Insertion Procedures (It is easy to use)

- Place the casualty on a firm surface in the supine position with the cervical spine stabilized.

- Lubricate the NPA with a water-soluble lubricant (or tap water if lubricant is not available)

- Position the tube so that the bevel (pointed end) of the airway faces toward the septum (the partition inside the nose that separates the nostrils) or the concave aspect toward the mouth or forward

- Push the tip of the patient's nose slightly upward to expose the opening in the nostril

- Keeping the head in a neutral position, insert the tip of the NPA through the nostril.
- Slowly advance tube along floor of nasal cavity with bevel pointing toward septum until flange rest firmly against casualty's nostril .
- If resistance is met during insertion, do not continue to insert.

- Stop, remove the adjunct, relubricate, and try the other nostril.

- If resistance is still met, check proper size or use alternate artificial airway method.

Complications.

- The most common complication is minor tissue trauma such as epistaxis (nosebleed); this however, is not sufficient indication to remove the airway.
- The airway may trigger a gag reflex with some patients

D. COMBI-TUBE AIRWAYS

a. The Combitube is an esophageal-tracheal double lumen(one end distally and another end at the midway of the tube) and tow cuff airway.
The Combitube airway is designed to provide a patent airway. The Combitube is a blind-insertion airway device(BIAD). It has been used successfully in patients with difficult airways secondary to severe facial burns, trauma, upper airway bleeding, and vomiting where there was an inability to directly visualize the vocal cords

The Combitube can be used in patients whose cervical spine has been immobilized with a rigid cervical collar,.
The double lumen design allows for effective ventilations to be provided regardless of whether the tube is placed in the trachea or the esophagus.

Indications:

- Ventilation in normal and abnormal airways
- Failed intubation
- Airway management in trapped patients

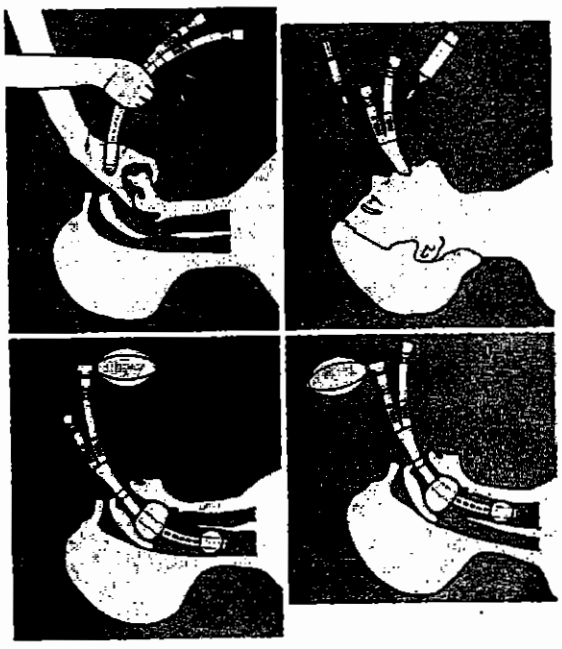
Combitube –2 lumens, 2 balloons:

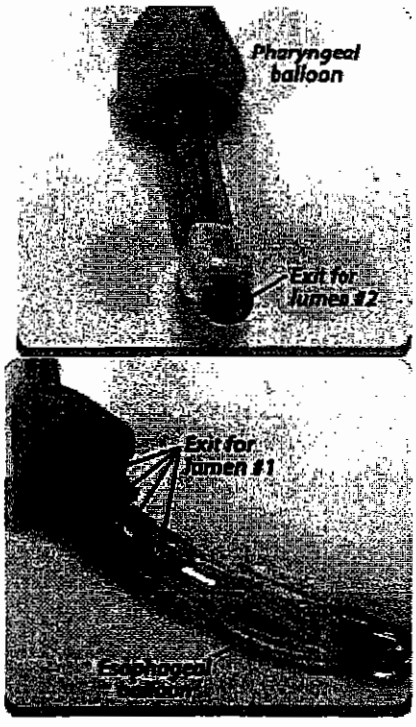
1) Twin-lumen device with two balloons: balloon #1 (big beige) seals pharynx or upper part of trachea(prevent the air to return),

balloon #2 (small white) seals esophagus(to separate between the trachea and esophagus).

2) Lumen #1 (blue proximal connector) goes to ventilation holes between balloons, while lumen #2 (clear proximal connector) runs completely through to distal tip.

3) Excellent seal pressures, trachea fully isolated from esophagus.





- Insert the Combitube in the same direction as the natural curvature of the pharynx.
- insert the Combitube gently, but firmly, into the pharynx until the black rings on the tube are positioned between the patient's teeth
- Inflate the Number 1 (blue) pilot balloon with 100 ml of air using a 100 ml syringe. Inflate the Number 2 (white) pilot balloon with 15 ml of air using a 20 ml Syringe
- Ventilate through the primary Number 1 (tall blue) tube. If auscultation of breath sounds is positive(or there is chest expansion) and auscultation of gastric sounds is negative, continue to ventilate (that mean the balloon #2 (small white) inside the esophagus) as in figure c.
- If auscultation of breath sounds is negative and gastric insufflation is positive(that mean the balloon #2 (small white) inside the trachea) as in figure d , immediately begin ventilations through the shorter (white) Number2 connecting tube. Confirm tracheal ventilation of breath sounds and absence of gastric insufflation

Advantages:

- Requires minimal training
- May be more useful in non-fasted patients
- Successful passage and ventilation in many patients via esophageal route
- Portable, useful in remote setting
- Functions in either the trachea or esophagus

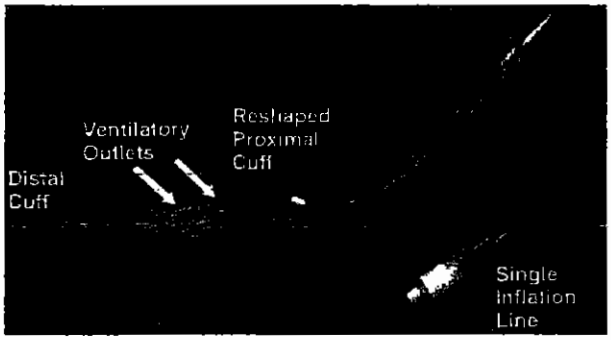
Contraindications:

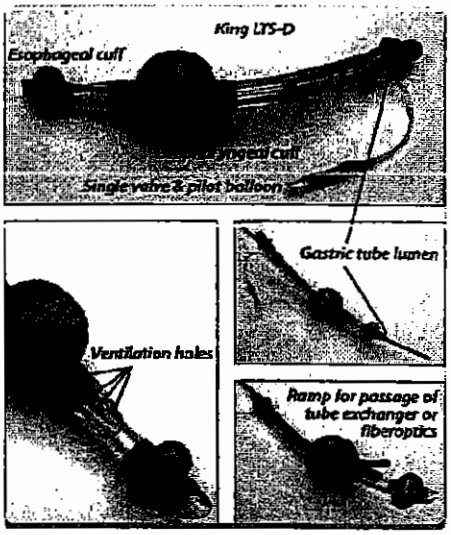
- Not practical for pediatric patients
- Patients with intact gag reflexes
- Patients with esophageal pathology
- Patients with caustic substance(acid or lye) ingestion if insert it may cause slough and cause bleeding.

Disadvantages:

- Mostly adult sizes>15 years
- Potential for esophageal trauma
- Expensive
- Can not be used to guid fiber-optic intubation

E. LARYNGEAL TUBE AIRWAYS





- A single lumen tube with both an esophageal and pharyngeal cuff.
- A single pilot balloon inflates both cuffs.
- Available in a variety of sizes.. available in pediatric sizes.
- Successful insertion by non-anesthetists.
- New versions have an open esophageal end allowing for drainage and suctioning.

The cuff for safety of aspiration if want to suction of stomach content with full stomach pt.

- Insert the tube in the same direction as the natural curvature of the pharynx. As combitube.
- Insert the gently, but firmly, into the pharynx until the black rings on the tube are positioned between the patient's teeth
- Inflate according to size of device (range 45-90 ml) and test for optimal ventilation while withdrawing device and gently bagging the patient.
- Ventilate through the primary Number 1 (tall blue) tube. If auscultation of breath sounds is positive(or there is chest expansion) and auscultation of gastric sounds is negative, continue to ventilate (that mean the small balloon inside the esophagus)
- If auscultation of breath sounds is negative and gastric insufflation is positive(that mean the balloon inside the trachea)..so remove it and reinsert it again

F. LARYNGEAL MASK AIRWAY:

An LMA is a curved, wide-bore tube with a spoon-shaped inflatable cuff. When the cuff is inflated, it seals the hypopharynx around the laryngeal opening.
Insertion of the laryngeal mask airway (LMA) is often smooth, atraumatic, and successful on the first attempt.and their is different sizes for neonate.

Size selection in lecture

Examination of of the LMA

- Visually inspect the LMA cuff for tears or other abnormalities
- Inspect the tube to ensure that it is free of blockage or loose particles or loose particles
- Deflate the cuff to ensure that it will maintain a vacuum
- Inflate the cuff to ensure that it does not leak

LMA Insertion

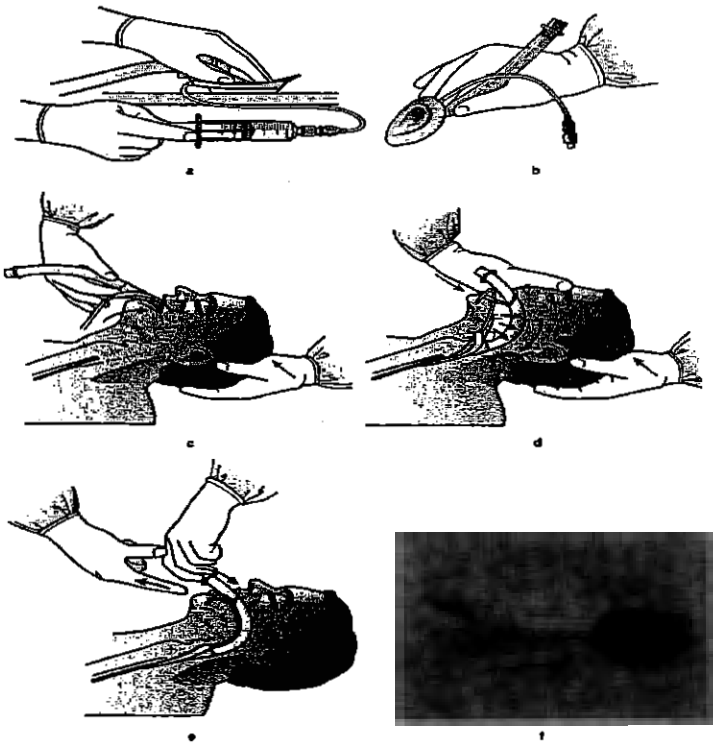
- Slowly deflate the cuff to form a smooth deflate the cuff to form a smooth

flat wedge shape which will pass easily around the back of the tongue and behind the epiglottis.

- Grasp the LMA by the tube, holding it like a pen as near as possible to the mask end.
- Place the tip of the LMA against the inner surface of the patient's upper teeth
- Press the mask tip upwards against the hard palate to flatten it out.
- Using the index finger, keep pressing upwards as you advance the mask into the pharynx to ensure the tip remains flattened and avoids the tongue.
- Press the mask into the posterior pharyngeal wall using the index finger.Continue pushing with your index finger
- Grasp the tube firmly with the other hand then withdraw your index finger from the pharynx.
- Press gently downward with your other hand to ensure the mask is fully inserted.

Inflate the mask with the recommended volume of air..as in the lecture.. Do not over-inflate the LMA.

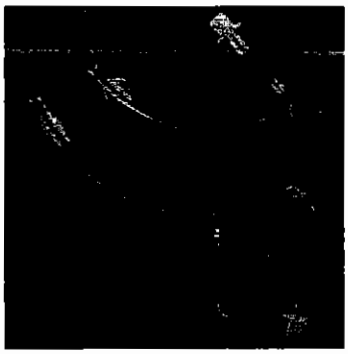
Advantages and disadvantages of LMA in the lecture

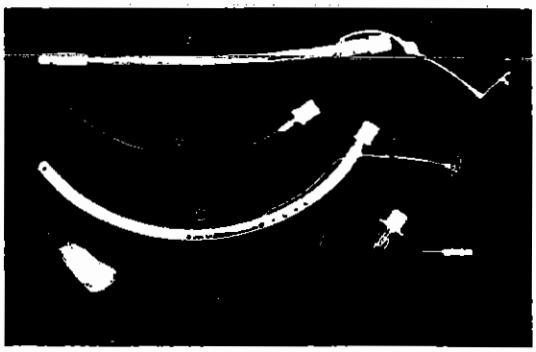


Invasive (definitive airway) (below the vocal cord) (ifra glottic)

i. ENDOTRACHEAL TUBE

1. Selecte the type that is needed

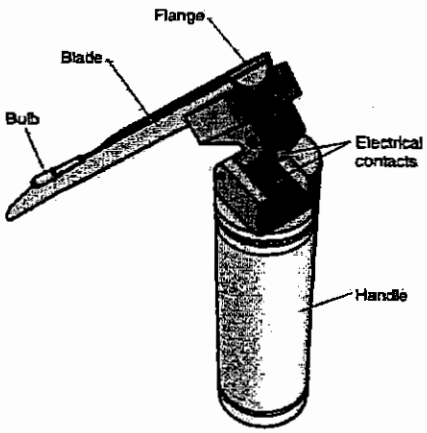




2. Selecte the Proper size of the tube and Laryngoscope Blade that is needed>>as in lecture

Oral endotracheal tube size guidelines:

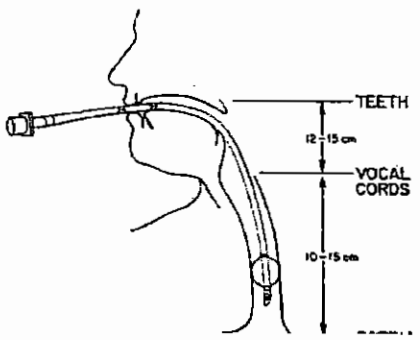
Age	Internal Diameter (mm)	Cut Length (cm)
Full-term infant	3.5	12
Child	$4+\frac{Age}{4}$	$14+\frac{Age}{2}$
Adult		
Female	7.0-7.5	24
Male	7.5-9.0	24



3.Airway assessment as in lecture

4. Ideal Patient Position:
The "sniffing" position, which requires flexion of the neck on the body and extension of the head on the neck, is widely recognized as the optimal position for direct laryngoscopy. This position is contraindicated with spinal injury>>كحركة الرأس عند شم الورد

5. To begin the procedure, opens the patient's mouth by separating the lips and pulling on the upper jaw with the index finger. Holding a laryngoscope in the left hand,
inserts it into the mouth of the patient with the blade directed to the right tonsil. keeping the tongue on the left to bring the epiglottis into view. The laryngoscope blade is then advanced until it reaches the angle between the base of the tongue and the epiglottis.
Next, the laryngoscope is lifted upwards forward (towards the chest and away from the nose) to bring the vocal cords into view. Often an assistant has to press on the trachea to provide a direct view of the larynx.
then takes the endotracheal tube(the concave aspect is forward) in the right hand and starts inserting it through the mouth



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opening. The tube is inserted through the cords to the point that the cuff rests just below the cords and between 22-24cm from the lip. Finally, the cuff is inflated to provide a minimal leak when the bag is squeezed. Using a stethoscope , listens for breathing sounds on both side to ensure correct placement of the tube.

If auscultation of breath sounds is positive and equally in both side (or there is chest expansion) and auscultation of gastric sounds is negative, that mean it inserted truly in the treachea continue to ventilate

If auscultation of breath sounds is positive and only in rt side (that mean it is in the rt bronchus because it more alignment and wide than the left) so deflate it and slightly withdraw it up then reassess by auscultation

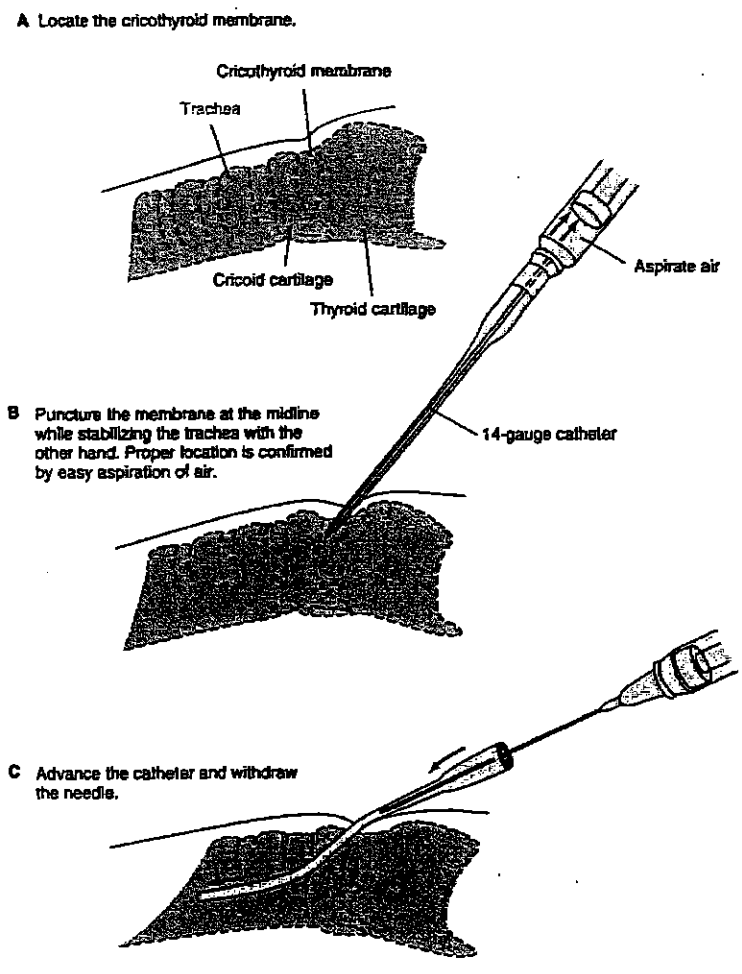
If auscultation of breath sounds is negative and gastric insufflation
is positive then remove it and reinsert it

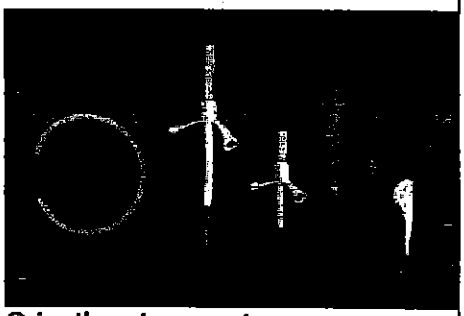
6.Read Principles of Direct Laryngoscopy and Principles of Intubation from the lecture

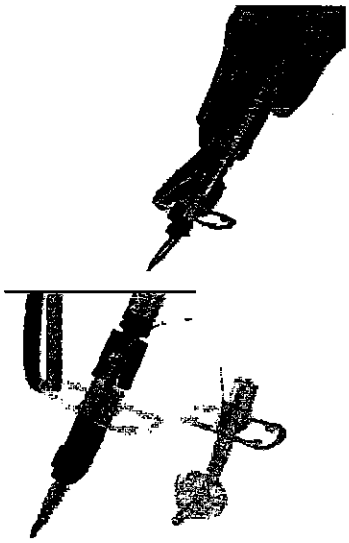
ii. CRICOTHYROTOMY

The establishment of an opening in the cricothyroid membrane (cricothyrotomy) or use the tracheostomy procedure is indicated for relief of life-threatening upper airway obstruction when:
(1) Manual maneuvers (head-tilt-chin-lift, modified jaw-thrust) and attempts at ventilation have failed.
(2) Endotracheal intubation is not feasable due to:
(a) Unable to remove an upper airway foreign body airway obstruction (FBAO). (b) Laryngeal or glottic edema (anaphylaxis, burns, etc.).
(c)Maxillofacial trauma (distortion of landmarks).
(d)Severe oropharyngeal hemorrhage. or can not raise the secretion (secretional obstruction)

Although creating a surgical opening in the cricothyroid membrane is a very invasive procedure, its use may be necessary due to life threatening conditions. Inability to establish an airway due to obstruction or other complication creates a situation that requires quick thinking and rapid action. The surgical airway may be the only viable option in this critical time




Cricothyrotomy set



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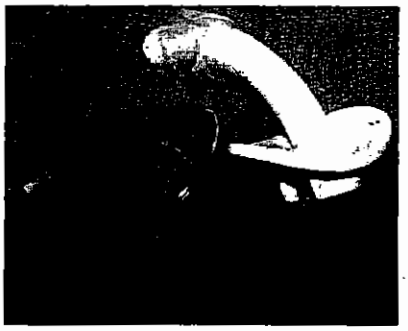
iii. TRACHEOSTOMY



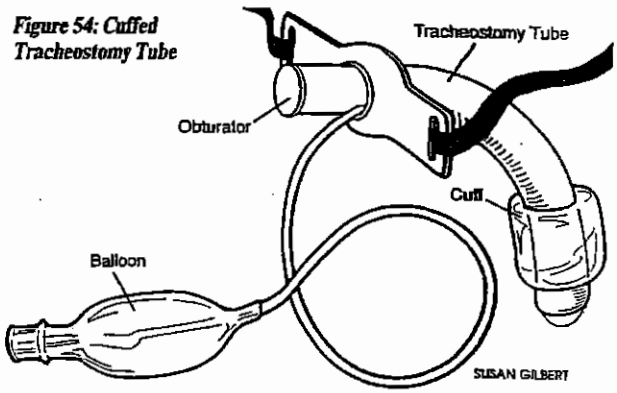
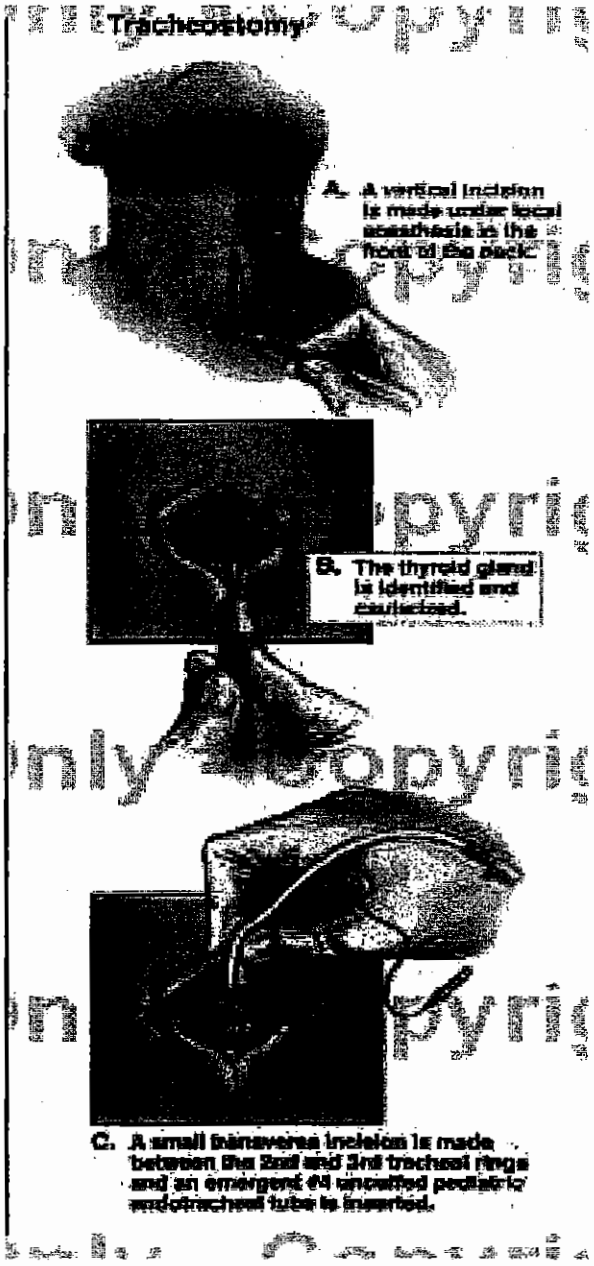
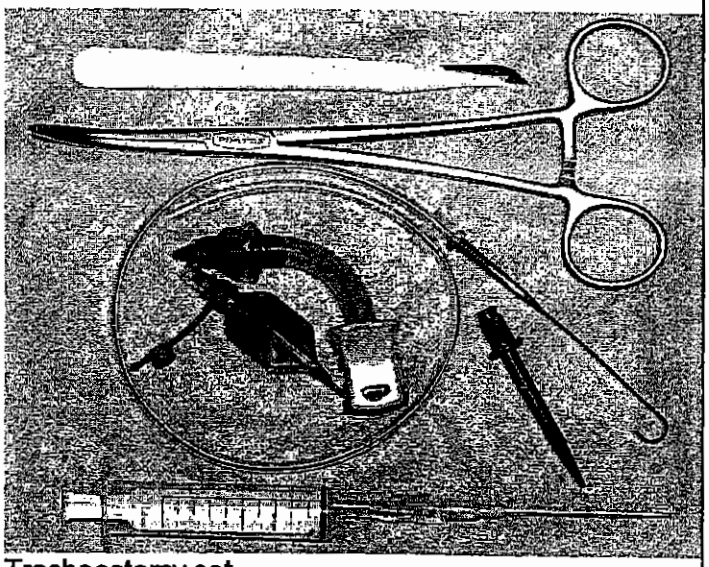
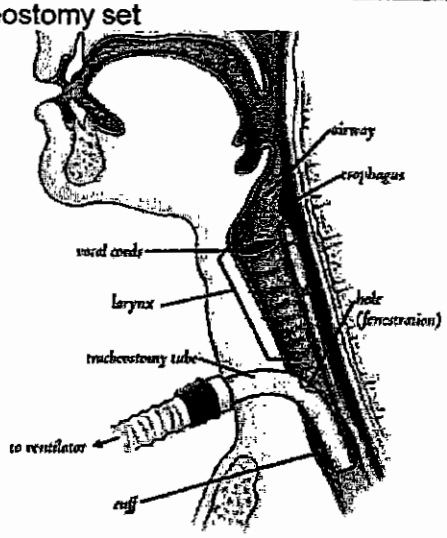


Figure 54: Cuffed Tracheostomy Tube





Tracheostomy set



2012/2013

Insertion of peripheral Intravenous canula

It means putting a catheter to the patient through a vein.
A canula has two parts: 1. An introducer, the pointed metal part.
2. Intravenous catheter,the flexible part that stays inside the vein After you remove the introducer.

IV canula sizes:

1. 24 gauge canula,yellow,used for neonates.
2. 22 gauge canula,blue,the most common canula used for children till 10-12 years of age.
3. 20 gauge,pink canula,most common canula for adults.
4. 18 gauge,green canula,used for patients bleeding and requiring blood transfusion,for dehydrated patients-hypovolemic patients- and for patients undergoing bloody operations.
5. 16 gauge,grey canula. The doctor said it has the largest diameter but there is a brown canula with 14g.

Note: the less the gauge→ the more the diameter and the more the flow rate through the catheter.

What can limit our choice of needles??

1. vein diameter.
2. vein length.

e.g: of the patient is shocked with vasoconstriction we go to more proximal veins such as veins in the antecubital fossa and the basilica vein.

Why do we insert canula's?
To give nutrients and electrolytes,to supply blood in case of bleeding or to supply separate blood products,to give drugs (in this case the bioavailability is high-100%- and the drugs action will be rapid),....etc.

Note: always insert the canula in the non-dominant hand,in the dorsum of the hand or in the forearm.

Preparation before canula insertion:

1. Applying a pressure cuff.
   Normally,veins are collapsed so we need to dilate them and make them congested to increase their diameter to allow for easier canula insertion,to achieve this we can do the following: A. applying a pressure cuff : this will decrease venous return causing congestion in veins and increase their diameter. B. lower the patients arm below the level of the heart-to make use of gravity to engorge the veins with blood-. C. vain tap : as this will cause release of mediators that cause vasodilatation. D. Make the patient open and close his hand.

2. Disinfectant
   To prevent thrombophlebitis.

3. Gloves
   To protect yourself against hepatitis and other diseases.

4. Patient must be supine or sitting.

Note: if a patient came with trauma and bleeing,even if this patient is a child,don't use the blue canula or any other smaller canulas,use the large ones.
Note: we prefer to use veins in areas such as the dorsum of the hand and the forearm because there are no arteries near,these areas are away from joints and they contain superficial veins,...etc

Procedure:
1.You should fix the vein and make sure its fixed in its place with your left hand (by either holding the forearm or hand depending on the site of injection).
2.With your right hand (using your lateral three fingers to hold the canula) insert the canula at an angle of 45° between the canula and skin surface,until some blood collects in the collecting chamber.
3.Then continue inserting the introducer at an angle more parallel to the skin surface.After that pull the introducer out (with your index finger) a little and insert the part of the catheter that you pulled the introducer out of, gradually keep pulling out the introducer while inserting the part of the catheter that you pulled the introducer out of till you take the whole introducer out and then insert the part of the catheter left in, but be careful not to advance too much with the introducer as you may puncture the vein.
4.Or you can pull the introducer out till its more proximal to you than the catheter and then insert the whole catheter in till the hub.
5.Insert the whole catheter in till the hub.
6.Before taking out the whole introducer apply some pressure proximally to prevent bleeding and also remove the cover from the introducer.
7.Remove the introducer.
8.Apply the cover at the part of the canula left.
9.Finally reomove the turnicate.
10.After you insert the canula infuse normal saline to make sure you inserted it correctly in the vein and there shouldn't be any resistance during infusion or pain and swelling caused by the infusion of fluid in the canula.

Note: when inserting a canula at the antecubital fossa area ,be careful not to insert it in the brachial artery which is located more medially.

Arterial catheters:

A. Arterial catheter is used in:
1. continuous blood pressure measurement.

Like in pheochromocytoma surgery in which there are dramatic blood pressure changes.

2. when repeated gas samples are needed.

B. are usually inserted in the radial artery.
C. arterial catheters are NOT used to give medication as this may cause ischemia.

Notes:
1.after you insert the canula,there are two ports through which you can infuse different kinds of fluids into the venous circulation, the first port is the one you covered with the screw you removed from the introducer and this is a 2-way port (in and out) for giving saline,withdrawing blood,...etc. The second one is located superiorly and it is a one way (in) port and is used for giving medication.

2.If you couldn't see the vein like in obese people you can palpate it.
3.the vein is normally compressible when palpated.

Withdrawing blood:

While withdrawing blood insert the needle at an angle of 45° and don't forget to apply negative pressure as you insert the needle in to know when you have reached the vein (when you start to withdraw blood as you apply negative pressure).

Central venous line insertion:

The needle used for central venous line insertion has the same components as the peripherally applied canula (a catheter and an introducer) but it is larger,it has different sizes for children and adults.
Triple lumen central venous catheter, its 16-18 gauge,multitask to give the patients more than one type of fluid(total parenteral nutrition , blood, fluids, ionotropes, medication, sedation,continuous infusion,...) and one of the ports is used to monitor fluid status and blood pressure.

Cenral venous line insertion and such advanced invasive procedures have complications such as the puncture of an artery,
Examples:
Femoral artery is near the femoral vein and can be punctured causing lower limb inchemia.
The subclavian artery can be punctured causing hemothorax.
The carotids are near the jugulars and can be punctured causing stroke.

A cetral venous line can be used for long-term intravenous antibiotic administration like in osteomyelitis.
To monitor central venous pressure the catheter should be located at the junction of the superior vena cava and the right atrium,so the femoral and the basilica veins are not for

monitoring the central venous pressure but for long term IV antibiotic administration and are called peripherally located central lines.

Done by : shaker haitham kakish

Wish you all the best ☺

Regional anesthesia 2012/2013

Extra notes

- Anesthesia is divided into:

1-general: we use it most of the time

2-regional

3-comibinathion between regional and general: for pain management (to deal with the patient intra-operatively and post-operatively)

- Regional anesthesia is used in the diagnosis or therapy topatients with chronic pain syndrome: we do CT scan and inject local anesthetic agent in facet joint, if pain disappears, this means that the pain is from facet joint, so we inject higher doses in the facet joint (for therapy). If pain persists, this means that the pain is not from the facet joint (so it is used in the diagnosis).

-Local anesthetic agent is a substance which reversibly inhibits conduction when applied directly to the tissues at non-toxic concentrations. Alcohol and phenol reversibly inhibits neve conduction.

- Determinants of systemic absorption:

1- site of injection(intercostal space is associated with more systemic toxicity as blood perfusion in this area is high, so the wash of the drug to the circulation is high and this will elevate plasma drug level immediately)

2-dose

3-physiochemical properties (lipid solubility, protein binding)

4-addition of epinephrine will decrease the wash of the drug to the circulation so we can give higher dose (safety margin)

- The most commonly used local anesthetic: bupivacaine (marcaine), lidocaine (xylocaine) and ropivacaine. In JUH, the most commonly are used bupivacaine and lidocaine.

- To differentiate between the amide and ester classes: the most commonly used local anesthetics in the amide class contain double "i", while the most commonly used local anesthetics in the ester class contain one "i",

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- What dose concentration 0.5% mean?

0.5%= 0.5 g/100cc =500mg/100cc = 5 mg/cc = 5mg/ml

[so by multiplying the percent by 10, we can know how many mg/ml we have]

***Bupivacaine:**

- Usage:

1- SC infiltration for surgeries that are done in SC tissues such as lipoma (the usual concentration that is used in infiltration is 0.5%)

2-spinal anesthesia (concentration is less than 0.5%)

3-epidural anesthesia (concentration is less than 0.5%)

4-peripheral nerve block

-contraindicated to be used in IV routes because of cardiotoxicity that may lead to arrhythmia and cardiac arrest.

-its max dose without adding epinephrine: 2mg/kg, and its max dose when we add epinephrine is 3 mg/kg.

-example(1):

if we want to do infiltration to a 100 kg patient and we want to use 0.5% bupivacaine without epinephrine .What is the max dose that is allowed to us to use and how many mls?

*concentration = 0.5% = 5 mg/ml

*max dose without epinephrine = 2mg/kg = 2*100 = 200 mg

* how many mls = 200mg/5(mg/cc) = 40cc = 40m

-example(2):

if we want to do infiltration to a 100 kg patient and we want to use 0.5% bupivacaine with epinephrine .What is the max dose that is allowed to us to use and how many mls?

*concentration = 0.5% = 5 mg/ml

*max dose= 3 mg/kg * 100kg= 300 mg

*how many mls= max dose with epinephrine(mg)/concentration(mg/cc)

= 300/5 =60 ml

***Lidocaine:**

- is an anti-arrhythmic drug class 2B.

- Usage:

1- infiltration

2-spinal anesthesia

3-epidural anesthesia

4-IV regional anesthesia: Not cardiotoxic

- Its usual concentration 1% or 2%

- its max dose without adding epinephrine: 5mg/kg , and its max dose when we add epinephrine is 7 mg/kg.

-example(1):

if we want to use 1% of lidocaine in 70 kg patient without epinephrine. what is the max dose that is allowed to us to use and how many mls?

*concentration = 1% = 10 mg/ml

*max dose= 5mg/kg *70 kg = 350 mg

*how many mls= max dose without epinephrine /concentration(mg/cc)

= 350/10 =35 ml

-example(2):

if we want to use 1% of lidocaine in 150 kg patient with epinephrine . how many mls we will use?

*how many mls= max dose with epinephrine (mg)/concentration(mg/ml) =7*150/10 =105 ml

---

***Rupivacaine:**

- It is cardiotoxic but less than bupivacaine, so it is contraindicated to be used in IV regional anesthesia.

-Toxicity related to local anesthesia is very rare.

- Tissue toxicity is usually related to preservation (such as alcohol) added to the solution.

-Systemic toxicity is related to blood level of the drug. The systems usually affected are:

1- CNS: lightheadedness, tinnitus, seizures

2- CVS: arrythmias that may lead to cardiac arrest

3- Respiratory system

-The toxicity of the CNS starts before CVS, so you must talk to the patient while you are giving the local anesthetic agent to insure that CNS effects haven't happened. So if the patient has tinnitus, stop the medication immediately because CVS effects (such as cardiac arrest and arrythmias) may happen (more serious).

- If any toxicity happens; stop the injection and deal with the patient as an emergency case according to ABCDE.

- If cardiac arrest happens, do CPR. If arrhythmias happen, deal with the patient according to cardiac arrhythmias ALS. If seizures happen, suppress seizure activity.

-Vasoconstrictor usage is an absolute contraindication in the following locations (fingers, toes, nose, ear lobes, and penis) because the arteries here are end-arteries so there is a higher possibility of ischemia.

-Degree of toxicity and dose determine if it is reversible/not.

-Regional anesthesia:

1- topical.....example: topical spray before bronchoscope

2-local..... example: lipoma, sebaceous cyst

3-peripheral nerve block..... example: digital nerve block

4-plexus blockade

5-central neuraxial blockade (spinal and epidural)

6-Bier block / IV regional anesthesia

*** Bier block / IV regional anesthesia**

-used for surgeries less than 60 mins duration because of increasing discomfort from the tourniquet

-used for surgeries below the level of the elbow and knee, but mainly used for surgeries of the upper limb (because mainly we use spinal anesthesia for lower limb surgeries)

- Rapid onset

-We insert 2 IV lines (one in the site of surgery to give LA through and the other one for emergency in the other hand)

- Technique:

1-we inflate the proximal cuff (so the area under it is anesthetized), then we give LA through the cannula.

2-Then after 30 minutes, there is pain from the proximal tourniquet (because the pressure is 100 mmHg higher than the systolic).....so we inflate the distal cuff then deflate the proximal.

-We use double-cuff tourniquet for:

1-pain relief (when the proximal one cause pain, we inflate the distal and deflate the proximal)

2-safety purposes (to prevent leak of medications to the systemic circulation)

-If the surgery last less than 30 mins after we inject local anesthetic agent, don't deflate the tourniquet before 30 mins have pass because we are afraid from toxicity.

***spinal anesthesia**

-When we do spinal anesthesia, we will penetrate these layers:

1- skin

2-subcutaneous fat

3-ligaments (supraspinatus then infraspinatus then ligamentum flavum)

4-epidural space

5-dura

6-CSF

- Spinal anesthesia indicated for surgeries below dermatome T10 (level of the umbilicus). For example any fracture in lower limb can be done by spinal anesthesia.

-Spinal anesthesia produce complete profound block of sympathetic, sensory and motor of the lower limb and abdomen surgeries (this means that we will achieve sympathetic, sensory and motor block at the same time once we inject the dose)

-spinal anesthesia works quickly after injection.

-most commonly we inject single dose in CSF dose but we may inject continuous infusion.

- SC ends at L1 in adults and at L3 in children.

-The recommendation is to insert the spinal needle between L3 and L4 or between L4 and L5. Some insert the needle between L2 and L3.

- Iliac crest is at the same level of the 4th vertebrae.

-Sometimes when we do high spinal we will have aggravated hypotension. So spinal anesthesia must be at the lumbar area not higher than this.

-Spinal anesthesia will cause hypotension due to spinal block, so to counter this we give IV fluid (ringer lactate) before spinal anesthesia and we prepare ephedrine (vasoconstrictor) for emergency.

-Post dural puncture headache occur when we use large hole needle as this will cause leakage of CSF into epidural space and that will decrease CSF volume and lead to traction on cranial nerves.

-The characteristics of post dural puncture:

1-fronto-occipital

2-positional (decrease when the patient sleeps)

-The diameter of spinal needle is less than the diameter of epidural needle.

-Spinal needles:

1- pencil point tip: separate fibers rather than cutting them. The incidence of post dural headache is less. Need introducer to penetrate the skin (the pencil tip spinal needle passes through the intoducer because it is not easy for it to penetrate the skin alone)

2-sharp tip

- There are different sizes of the spinal needle represented as different gauges. The available gauges are: 20,22,25,27. As the gauge of the needle increases, the diameter of the needle decreases, so the hole created by the needle will be smaller, so the incidence of post dural headache will be less.

-Old people have fibrous ligaments, so it is difficult to pass through the ligaments by large gauge needle (diameter small), so we use small gauge needle (20).

-Although we use small gauge needle in elderly, the incidence of post dural headache in elderly is rare.

-In young patients we use large gauge needle as post dural headache in them is common. We use gauge (25)/ (27)

-equipments of spinal anesthesia: needle, introducer (in cases of pencil needle), syringe and local anesthetic agent

-technique:

1- explain to the patient the procedure

2-proper IV access

3- monitoring of the patient (BP, ECG, oxygen saturation, heart rate)

4- approach: midline (most commonly used) or paramedian(if ligaments are fibrous) . Previously they thought that when they use sharp needles they must insert the needle in lateral position then make it straight to decrease the tear in the fibers, but nowadays they found that it is not effective.

5-position of the patient: it is better to be performed in sitting position because this opens the vertebrae and the anatomy is clear. But we can use lateral position especially if there is fracture or the patient cannot bend down (the anatomy will be disturbed).

6- under complete sterile technique: First we identify iliac crest which is at the same level of L4. If we use pencil needle we must use introducer. The curved tip of the needle must be directed upwards. We enter between the bones of L3 and L4. We usually give 2ml of local anesthetic infiltration in skin and subcutaneous tissue to prevent pain upon entry. Then we advance the spinal needle until we estimate that we are in CSF, and then we remove the stylet. If we are in CSF, there must be CSF

backflow when we remove the stylet (stylet prevent collection of fat). Some do aspiration of CSF to insure it is CSF.

-In spinal anesthesia we usually wear gloves only not gown. While in epidural anesthesia we wear gown.

-We usually use lidocaine in spinal anesthesia.

***epidural anesthesia**

-It is used in cases of continuous infusion of local anesthetic agent (not a single dose) in epidural space.

-Usually we give multiple doses through the catheter

-Epidural space: is a potential space not a real space, has a negative pressure same as pleural cavity.

-It is performed at any level, so it can be used to produce analgesia and anesthesia to the thorax, abdomen or lower limb (for example: pulmonary surgery, cardiac surgery, vascular surgery, abdominal surgery, gynecology, neurological surgery, orthopedic procedures). It is also used for acute pain relief (post-op pain, labour pain, trauma) and chronic pain relief (diagnostic and therapeutic)

-Epidural anesthesia takes about 10 mins to start working.

-Its effect is not profound. So according to the dose we give, we can determine the level of blockage (whether sympathetic block, sensory block or motor block). If we give small dose we will block the sympathetic, if we increase the dose we will block the sensory, if we increase the dose more we will block the motor. (we titrate the level of block)

-It can cause spinal headache if you advance the needle beyond the epidural space. The incidence of post dural headache is higher than spinal anesthesia if we puncture the dura.

-Epidural anesthesia effect at the same level of insertion of medication is more than the area below.

-Equipment:

1-The epidural needle is called tuohy needle, characterized by a curved tip (that is directed upward) and different number of marks (usually we use a needle with 8 or 9 marks, in obese patients we use 11 marks but the average number of marks are between 4-5marks) each mark measures approximately 1 cm. We usually use gauge 16 / 18 so it is larger than spinal needle where we usually use gauge 25(rarely we use gauge 20 in spinal anesthesia).

2-special catheter that is connected to the adapter and this adapter is connected to a filter (filter is bacteriostatic because we are giving medications around CSF so we should insure sterility)

3-low resistance syringe

4-local anesthetic agent

-In epidural anesthesia we wear gown.

-technique:

*1st one called loss of resistance technique:

We go with the needle 2-3 cm and pass through skin, subcutaneous tissue until we enter into the ligaments (here we will feel resistance). Here we will remove the stylet and insert the low resistance syringe that is usually filled with air but maybe filled with saline to dilate the space. If we inject here we will feel resistance that is higher when we reach ligamentum flavum(harder). Once we feel loss of resistance this means that we are in the epidural space. Now we remove syringe and calculate how many needle marks are inside (usually 5 cm to reach epidural space). Then we insert catheter(through the needle) that is also calibrated until we reach 20cm then we remove needle (we enter with the catheter and exit with the needle). Now we exit with the catheter until we reach 9 cm (we need 5cm from the skin to the epidural space and 4cm inside the epidural space). Now we connect the catheter with the adapter, connecter and filter.

-The calibration of the catheter: the first 5 cm aren't calibrated, the 1st mark indicate 6 cm then 7 cm,....etc, until we reach two marks which means 10 cm, then one mark which means 11 cm,....., until we reach 3 marks which means 15 cm, then one mark which means 16 cm,....., until we reach 4 marks which means 20.

*2nd technique called Hanging drop technique:

The same as the above technique, but we fill a syringe with a few drops of saline, when we enter the epidural space because of its negative pressure these drops will be absorbed so we know that we are in the epidural space. The success rate is less than the loss of resistance technique.

*Nowadays they use US to identify epidural space.

-If we want to do thoracic surgery and we want to use epidural anesthesia: we insert the needle in the high lumbar area then go with the catheter cephalic to enter into the space between the vertebrae but we don't advance the catheter too much because the chance to go to one side is high. We can insert the needle at the same level of thoracic surgery and advance the catheter 3-4 cm (but it is difficult to do thoracic anesthesia because the space and angle between the vertebrae is small).

***Notes about spinal and epidural anesthesia**

- A patient with severe COPD who has femur fracture (emergency case), we perform GA to him and the patient will go the ICU post-op where he will stay long time on ventilators.

- By performing regional anesthesia we protect mainly respiratory system, CVS. You keep your patient breathing spontaneously alone; you only apply oxygen and perform the procedure.

-By performing regional anesthesia (such as spinal anesthesia) we can discuss with the patient and take his permission to do things inside the

operation but the patient must not have taken midazolam(sedative drug).

-By performing GA we block the communication with the patient.

-Contraindication to use regional anesthesia are divided into relative and absolute.

-Patient refusal to do regional anesthesia is an absolute contraindication and he carry the risk of GA.

- When we advance the needle in dura we may touch a blood vessel and cause bleeding and hematoma that will cause compression on the nerves and this will lead to lower limb weakness and neurological deficit so in cases of coagulation problem, the risk will be higher.

- Some drugs affect coagulation status of the patient:

1- Aspirin: No contraindication to perform regional anesthesia in cases of aspirin intake (half life of aspirin is 7-10 days)

2- Warfarin
*absolute contraindication to be used in regional anesthesia
*duration of action: 3-5 days

*if we want to perform regional anesthesia to a patient taking warfarin regularly: we admit him to the hospital and stop warfarin for 3-5 days (to eliminate warfarin effect), we put the patient on continuous infusion of short-acting heparin (half life 6 hours). Before 6 hours of the surgery, we stop short-acting heparin. After the surgery we give the patient heparin and warfarin for 3 days until warfarin works. Then the patient continues wafarin.

*if we want to perform regional anesthesia to a patient taking warfatin irregularly: we measure INR (international normalised ratio), if it was >1.5 we perform general anesthesia and give vitamin k to counteract warfarin and decrease the risk of

bleeding (it is contraindicated to perform regional anesthesia). If it was <1.5 we perform regional anesthesia.

3- Plavex (clopidogrel)
*absolute contraindication to be used in regional anesthesia
*It works on platelets
*duration on action is 7 days

*Example: Before 6 mons, a patient underwent stent insertion (as he had chest pain) .The doctor advice him to take plavex for 1 year. Now the patient wants to do elective total knee replacement. What can we do? We postpone the surgery for another 6 mons as it is elective surgery and the patient can wait (the patient must take plavix for 1 year).

*Example: If the patient comes to ER with fractured femur (emergency) and he was on plavix. What can we do? We do GA

*Example: If the patient on plavix comes to ER with an emergency case and the surgeon says that he can postpone the surgery for 7 days (until the effect of plavix is eliminated), he can do regional anesthesia but the cardiologist must approve to stop plavix.

4- Heparin

-Infection in the skin is a risk factor for infection in CSF. Local anesthetic agent will not work properly if there is infection in that site.

-**Fixed cardiac output state eg: aortic stenosis is an absolute contraindication for regional anesthesia.** (in the slide it is written relative, this is wrong) . Because in normal cases, when spinal anesthesia cause hypotension the heart will compensate by increasing cardiac output.

-Skeletal abnormalities such as scoliosis and kyphosis are relative contraindication because it is techniquely difficult.

-Previous local surgery for example in the disc between L4 and L5 is a relative contraindication.

-In saddle anesthesia: same as spinal anesthesia but we use hyperbaric anesthetic agent that will go down to anal area.

-Caudal: same as epidural but performed in the sacral area, mainly in the pediatrics.

-hypobaric anesthetic agent will go up.

-Isobaric lidocaine/bupivacaine once injected in CSF, remains at the same level.

-We must insert IV line for ER in spinal and epidural anesthesia as they may cause hypotension in rare cases.

***Brachial plexus block**

-Brachial plexus consist from these roots: CS, C6, C7, C8, TI

-Block can be achieved at 4 levels

1- Interscalene level
2- Supraclavicular level
3- Infraclavicular level
4- Axillary level

-Each nerve consists of motor and sensory. We block the nerve that supplies the area we want to do surgery on (to block certain movements and certain dermatome).

-Interscalene level block:

-It is performed in a groove called interscalene groove that is found at the level of cricoids, posterior to the sternocledomastoid, above the midclaviclular point.

-Previously they depend on anatomy: they advance the needle, once they are near the nerve, the patient will feel paresthesia (electricity), then they inject the local anesthetic agent.

-Currently they use either single shout technique or catheter technique (which also depends on anatomy).

**Single shout technique:

* They use electrical nerve stimulation machine (nerve stimulator) this machine is connected to special needles called stimulation needles.

*Stimulation needles are insulated needles consist of 2 limbs:

1-One connected to the nerve stimulator

2-The other limb we give medications through

*Electricity comes from the machine and just exits through the tip (only the tip contains electricity)

*Once you are near the nerve there will be stimulation and contraction of the muscle

*Usually you start with the current 0.8 -1mA and then you go down with the current until you reach 0.3mA. If there is still contraction in the muscle at 0.3mA this means you are near enough to the nerve so you can give your medications (Once you go down with the nerve stimulation and still there is contraction this means you are near enough from the nerve and you can give your medications).

*Disadvantages

1-you are near the nerve from one side. The other side is far away so you need to give larger volumes of local anesthetic agents (approximately 40ml)

2-Nerve injury: rare because the tip of the needle is not that sharp to penetrate the nerve, also if you touch the nerve it will be very painful (may lead to neural deficit if you inject) and once you inject the needle in the nerve there will be high resistance (you can't inject LA).

*We can use this technique if the operation is not that painful such as orthoscopy to the shoulder, so single shout of medication is enough (covers for 24 hours)

**Catheter technique:

*The same technique, the differences:

1-The needle differs (2 pieces above each other only the tip is visible)

2-cathter: Once you are near the nerve, you remove the needle and insert a catheter through the stylet and advance little bit then you remove the stylet, now you can give to the patient continuous medication for 24-48 hours.

*So if the surgery is very painful and the patient is in need for long term pain management, we use this technique.

*We use this technique for patients who undergo total shoulder replacement; here we need good analgesia for long time and for rehabilitation (physiotherapy).

-Infraclavicular level block

-It is performed below the clavicle, at the midway between acromion process and suprasternal notch.

-We go perpendicular with the needle at this point.

-We use it if we want to perform surgeries on elbow (duration of the operation 6 hours)

-Axillary level block

-First identify the axillary artery in the biciptal groove. Just around axillary artery we go toward the tip of the humerous superficially (above the artery).

-The problem in this technique is that ulnar, median and radial are far away from each other. So according to the site of surgery we manipulate

the needle until we have a response in the muscle supplied by the desired nerve.

-If we want to block ulnar side >>>>we block ulnar nerve

-If we block median nerve >>we will have carpel tunnel syndrome

-There is a possibility of nerve injury and failure of the technique

-Nowadays, they start to use ultrasound technique for identification of the nerve and do block under vision (you see spreading of local anesthetic agent around the nerve). This technique raises the success rate and decreases the complications.

-Previously, before electrical nerve stimulation machine, they use transarterial technique (they go trans the artery by the needle....enter the artery .....aspirate.....there is blood....they exit....if there is no blood ....they inject the local anesthetic agent). This technique increases the possibility of hematoma formation.

***Femoral nerve block**

-We depend on anatomy. The best landmark is the femoral artery.

-From medial to lateral: vein, artery, nerve

- So we feel the femoral artery, then we check for quadriceps contraction by stimulating the nerve (we look for dancing of patella), then we insert local anesthetic agent just lateral to the artery.

-We may see Sartorius muscle contraction.

-We can block femoral/sciatic nerve by ultrasound. Here we see femoral nerve as a triangle (honeycomb appearance) which has black dots inside it. The femoral artery is pulsatile (we can distinguish it also by Doppler>>>flow is red). We will inject local anesthetic agent posterior to the nerve. Here we see the spreading of local anesthesia posterior to the nerve. Later on, we will move the needle to block the anterior part of the nerve.

***Sciatic nerve block**

-We depend on anatomy.

-We draw a line between the superior posterior iliac spine and greater trochanter. On the midpoint of this line we draw a perpendicular line measuring 5cm. The end of this line is the puncture site.

-We check for plantarflexion/dorsiflexion of the foot.

- When we inject local anesthetic agent we go deep (the depth of sciatic nerve is 7-8cm so we use a long needle).

-Example: In total knee replacement we block sciatic and femoral nerve.

Done by: Noor Saleh

2012/2013

بسم الله الرحمن الرحيم

Basic Life Support October 30, 2012

➢ What is the definition of:

- Cardiopulmonary arrest?
  It is the sudden cessation of the spontaneous function of both cardiovascular and respiratory systems.

- Cardiopulmonary resuscitation (**CPR**)?
  It is the artificial delivery of the oxygenated blood through the vascular beds of the vital organs to maintain their function.

In CPR, our main job is to keep doing external cardiac massage *without interruption* for more than 10 seconds at anytime, even for checking or doing anything else!

❖ The only 2 evidence based skills "maneuvers" that were 100% proven to be beneficial to the arrested patient are:
1) External cardiac massage
2) Defibrillation (if the rhythm is *shockable*)

Other things such as ventilation and drugs are still not having evidence based studies that proved their beneficial effect to the patients.

❖ In CPR, it is a matter of minutes! where the (**Golden Minutes** of the CPR) are the first 3-4 minutes, if you reached the patient within this period and started your action, then the patient will have a very good chance to survive.
But in case you didn't reach him/her at that "proper time", then for each minute delay in starting the CPR after those Golden Minutes, the patient will lose 7% from his chance to survive ☹

The Golden Minutes of the CPR:

To justify the value and the importance of the these minutes, we'll go briefly through the following clarification (*you can skip it!*) :

- The idea is mainly about the **"Oxygen flux"** which is:
  the total amount of oxygen carried in the blood, including bound and dissolved O2, per minute.

Oxygen bound to Hb:
= Cardiac output x [Hb] x SO2 x k

where:

- CO = cardiac output (L/min)
- [Hb] (g/L)
- SO2 = saturation (as fraction)
- k is **Hufner's** number;
  * Amount of O2 that can bind with 1g of Hb when fully saturated, it's normal value = 1.34mLs O2/g of Hb

Dissolved O2:
= CO x pO2 x 0.03

- PO2 = partial pressure (mmHg)
- 0.03mL O2 per mmHg per L of blood can be dissolved.
- CO = cardiac output (L/min)

Thus, ***Total oxygen flux*** =
Oxygen bound to Hb + Dissolved O2
= 5 x 150 x 0.98 x 1.34 + 5 x 100 x 0.03
= 984.9 + 15
≈ **1000mL O2 per min**

Assuming:

- cardiac output of 5L/min
- [Hb] = 150g/L (one of the main factors in this calculation)
- SaO2 = 98% and PO2 = 100mmHg
- pH 7.4, temp = 37

✓ **So 1000mL of O2 is available in our blood at any moment.**

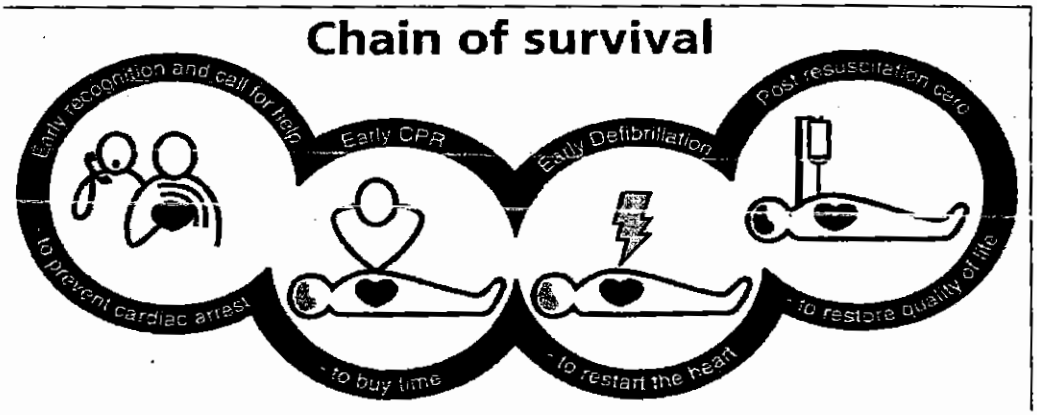
- The amount of O2 consumption in the body **per minute** is about 200 – 250 mL (Consumption by the *mitochondria,* and this depends on the metabolic state of the body).
  So we can conclude that this 1000 mL of O2 are sufficient only for 4 – 5 minutes only; as every minute about 200 to 250 mL are consumed.

- To complicate this more ☺; at the moment you arrived the arrested patient and you began to check him, there are 250 mL of O2 in use ((now)), so the remaining reserve is only about 750ml and which is sufficient for **3 - 4 minutes** only !!

- So that's why these minutes are called the *Golden minutes, as the patient is still having some oxygen in his blood*...And if you started your CPR during them, you will move around this oxygenated blood to the vital organs and prevent their permanent damage (mainly to the CNS).

Outcome to achieve from this subject:

1. To know the importance of the early recognition of the deteriorating patients; (patients who are critically ill and at high risk), and how you should try your best to prevent cardiac arrest for those patients.
2. To know the causes of the cardiorespiratory arrest
3. To identify and treat patients at risk of, or having cardiorespiratory arrest using the ABCD system.



Chain of Survival:

This very important diagram (seen a lot in exams"OSCEs") emphasizes the sequence of interventions (events) that should be taken by you as a medical professional when you deal with patients during their deteriorating period and /or cardiac arrest.

Note:
the above written sentences in each ring of the image represent the step to be taken, below is the goal from it.

- We will go through a brief description of each step in this very important chain:
  1. The 1st ring indicates the early recognition of deteriorating patients who are under you care (you should be able to identify such patients, in order to try to prevent cardiac arrest from happening).
  2. The 2nd ring shows the importance of the early start of the CPR during the golden minutes (the actions of the basic life support algorithm). And the importance is to buy time until the CPR team comes.
  3. The 3rd ring shows the importance of the *early defibrillation* for patients *who are in need* for it (only those having shockable rhythm, so it is not used in all types of cardiac arrest). It should be done early; as each minute delay decreases the chance of survival of the patient.
  4. The 4th ring shows the importance of the post resuscitations "special" interventions in the ICU (intensive care unit) for those successfully resuscitated patients. And this is to prevent complications such as (hypotension, pneumothorax...etc) and to restore a good quality of life.

Notes:

- Prevention of cardiac arrest is much more beneficial than leaving the patient to have a cardiac arrest and then doing CPR!

- Most of the cardiac arrests which happen in the hospital ward are ***predictable***. So don't be **"ignorant"** watching the patient deteriorating (literally; hypoxic and hypotensive) and in a high risk to develop cardiac arrest *without intervening to prevent this!!*

Be at the level you are in when you are caring for patients!

- Up to 80% of patients who develop cardiac arrest have a deterioration period before it and only *minority* of patients are healthy before developing cardiac arrest.
- ***Never ever*** delay asking for help if you don't know how to take an action in this regard.

The ABCD System:

- How are we going to approach those deteriorating patients or who already developed cardiac arrest?
  By the universal **ABCDE** (Airway, Breathing, Circulation, Disability, and Exposure) system. (Whatever the patient condition is, you have to approach him/her in a **systematic way**)

- When you want to ***evaluate*** the patient, you have to go through the ABCDE system (orderly). ***But*** at intervention, i.e. when the patient is having cardiac arrest and you want to ***act***, you should start with the external cardiac massage "circulation" before the airways (i.e. start with the C before the A).
  - ✓ This is an update in the new guide lines after 2005.

- If you have been called to assess a patient who is deteriorating, you should do a complete quick initial evaluation for him/her by using this ABCD system and see what is the main problem and its cause (upper airway obstruction, bleeding somewhere, diarrhea, bleeding per rectum, gunshot...etc).
- Always in critical conditions **call for help,** never try doing everything by your own; you will need other staff members to help you in the management of such conditions.

Further clarification on ABC is discussed later on in this sheet, but here we'll mention briefly the D&E (disability & Exposure):

- In Checking for **disability**, you must check for the following:
  1- Conscious level
  2- Pupil size
  3- Blood glucose

- **Exposure:**
  you should expose every patient who is in a critical condition in order to be evaluated, as through this we can know whether the patient has bleeding, injuries, burns, melena...etc!

  - ✓ Two important things must be taken in consideration in this regard:
    1- you should try your best to maintain the dignity of the patient you are exposing.
    2- Avoid hypothermia to the patient (cold environment)

Causes of Cardiorespiratory Arrest:

(Airway, Breathing & Circulatory causes)
Please refer to the slides for more elaboration on these causes.

Notes:

- ***Airways***:
  - Any blood, secretions or gastric contents in the airways with depressed level of consciousness may cause death by asphyxia, so you should be able to suck them and clean the airways or to put the patient in a position that can drain the blood or these secretions away.
  - Airway problems can cause death by severe hypoxia to the patient.
  - You can know if the patient is not having airway obstruction by simply being able to talk to you!

- ***Breathing***:
  - When we want to treat the underlying *breathing problem* we almost always start our management by the airways.

- ***Circulation***:
  -Acute coronary syndrome is the most important circulatory cause that could lead to cardiac arrest.
  - When patients with this syndrome present to the ER, they are given " MONA" which

stands for (morphine, oxygen, nitroglycerin & aspirin).
-The most common cause of arrhythmias in hospitalized patients is electrolyte disturbances.

Steps of the BLS:

The steps that are to be performed in an OSCE station☺ or in any real condition one might encounter.

(In the OSCE exam, you have to be like an *actor!* Consider as if the case is in the real life)

Literally, you have to say and do the following:

1. First of all, you will start by ***"I should check the surrounding of the patient if it's safe for me to approach him/her."***

2. ***"I will check the responsiveness of the patient by shaking him gently from his shoulders, calling him by his name"*** (if you know it) or else by saying ***"Hello there!"***
   If he is unable to answer, then this means that he is unconscious. So at this point you should say: ***"because he is unresponsive, he is unconscious. He may have cardiac arrest. And because he may have cardiac arrest, I can't deal with this patient on my own; I should call somebody to help me".***
   You should shout loudly: ***"Help please!"***
   The instructor is supposed to tell you: ***"the help (the CPR team) is on the way"***

3. Now say: ***"now, I'm going to deal with the patient in a systematic way. I'm going to follow the ABC system"***

4. Say: ***"I'm going to start with the airways, so I'm going to open the airways by doing 3 maneuvers: head tilt, chin left and jaw thrust "***. You should do them properly!

5. Say: ***"now, I'm going to check the breathing and circulation at the same time";***

   - **To check the breathing and circulation:**
     The fingers of one hand must feel the carotid pulse, And at the same time you are opening the airways, bring your face near the airways of the patient and check for breathing by (look, listen & feel):
     - **Look** for chest movement;
     - **Listen** for any sound arising from the upper airways and;

- Feel for any expiration coming into your face

✓ Why do we check both breathing and circulation at the same time?
To save time for the external cardiac massage..

Then, the instructor will most probably tell you: ***"There's no breathing, no circulation"!***

6. Say: ***" because there's no breathing and no circulation, then the patient is having cardiac arrest, so I should start doing external cardiac massage for this patient". "I should start pressing the sternum down".***

Remember:
In *intervention,* where we have confirmed that the patient is having cardiac arrest, we should start with the external cardiac massage before the airways (start with the **C** before the **A**), opposite to the evaluation of the patient's status (where we start with the **A** before **C**).

❖ **To perform external cardiac massage:**
- Intermingle your fingers; the heel of your hand should be on the sternum at the middle of the chest (for males, between the nipples. For females, it's obvious; just position your hand at the center of the chest).
- At least, you have to go deep 2 inches (5 cm). Regarding the frequency, you should do 30 continuous compressions.
- Note that while doing the cardiac massage, your elbows should be extended, and the pressure should arise from your shoulders and you have to count while you are pressing down (1, 2, 3, 4.....30)!

For sake of time, the instructor will often tell you:***" 30 compressions are done!".***

7. After this you should do mouth to mouth breathing;
Say:***" now, I'm going to do mouth to mouth breathing".***
Most probably, the instructor will not let you do it (u know!☺)

*Nevertheless, we have to **master** every step in the CPR, as it's not merely an OSCE exam, we may encounter such case in our real life!*

❖ **Mouth to mouth breathing:**
With the airways open, close the nose of the patient. Your mouth should seal the patient mouth (your mouth is firmly placed around the patient's mouth). Then Give *two nice short breaths*, with only one second for each.

8. Then, quickly, you have to continue doing the external cardiac massage; (1, 2, 3,.... 30), followed at the end by another 2 breaths.

   *You should repeat this for an additional 3 times, so that we have done the whole process (30 compressions & 2 breaths) **5 times.***

9. At the end of this you should revaluate (**recheck**) the patient. ***Say: "I should check the airways and circulation"***
   (as mentioned above; carotid pulse for **circulation** and, look, listen and feel for **breathing**.)

10. The instructor will most probably tell you that ***"there's no pulse"*** (no return of the spontaneous circulation).
    so you should say:***" I should continue doing the external cardiac massage"***

11. So you are going to do the same thing mentioned above, (30 compressions followed by 2 breaths; **5 times**).

> ***Note:***
> Doing 30 compressions followed by 2 breaths **five times** will take approximately 2 minutes, and this is called a **CPR cycle**.
> So each CPR cycle lasts for about 2 minutes and consists of 30 compressions followed by 2 breaths done 5 times.

12. After each CPR cycle you have to (recheck) the patient for **Return Of Spontaneous Circulation (ROSC)**

❖ *Important to Know:*

- If there were 2 doctors, and both were trained for doing BLS (CPR), then one should do the external cardiac massage and the other will keep the airways patent and do breathing.

But in such case, the one who do the cardiac massage will do **100 compressions/min** and the other **8-10 breaths/min.** Note that in this case it's difficult to do breathing simultaneously with the cardiac massage.

- But suppose that the patient is in the floor (of a hospital) and is placed on an endotracheal tube with an ambu bag available, and also there are 2 staff members to do the CPR, then one will do 100 compressions while the other will do breathing **simultaneously** (at the same time) via the Ambu bag (8-10 breaths/min).

✓ **Note that in such cases, the cycle (2 min) is composed of 200 compressions.**

When can we stop the CPR?

We will discuss two instances:

A. You are at home☺, and you (alone) have been called by your neighbors to offer help in an emergency case of cardiac arrest!..
You did external cardiac massage for (20 – 30 min) and you then you got exhausted, and there's nobody to help you...!
Then you can stop the CPR.. ☹

B. In case there was a CPR team (many persons) in a hospital, then the decision when to stop CPR depends on the **team leader.**

✓ You should know that **age** of the patient & **co-morbidities** play an important role in making such decision.

Complications related to CPR:

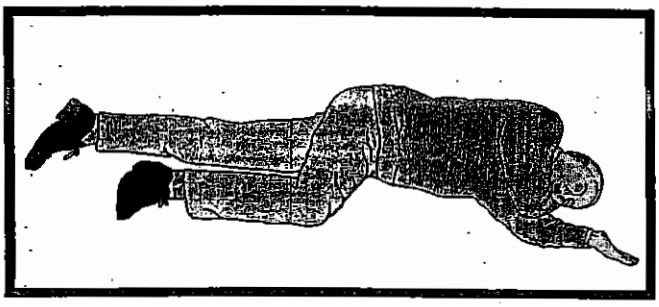
1. **Fracture ribs:** due to improper positioning of the hands during external cardiac massage which may result in:
(Damage to the lung tissue, pneumothorax, haemothorax, hemopericardium, cardiac tamponade, damage to the liver, perforation of a viscus,...etc)

II. And in case you placed your hands **over the stomach** and compressed! This might result in regurgitation and aspiration of stomach contents!!

<u>During the external cardiac massage</u>, you should be at a good level regarding the chest of the patient. For example, if you are short, you can stand on steps, or to get up over the bed of the patient and then flex your knees. And in case the patient is initially on the ground, then you have - of course ☺- to kneel.

- *After few cycles of CPR, if you have identified* **return of spontaneous circulation (ROSC)** and **breathing**, then you must put the patient in a position known as the *<u>Recovery position</u>* which is:

  A lateral position, with one leg flexed and the other extended. One of the upper limbs beneath the face and the other in a comfortable position. And this is because the patient will remain unconscious for a prolonged time.



  - If he is unconscious and lying supine (on his back) then there's a risk of regurgitation and aspiration of stomach contents!!

- In case **only circulation** has returned *but without breathing*, then we stop external cardiac massage, and we keep doing mouth to mouth breathing at a rate of 8 – 10 / min.

  - Mouth to mask breathing: in some drug stores, they sell a ***pocket*** face mask that has a "safe guard" valve which prevents transmission of infections from you to the patient and vice versa.

The End

Basic Life Support - 1

Causes and Prevention of Cardiorespiratory Arrest

Objectives

- The importance of early recognition of the critically ill patient
- The causes of cardiorespiratory arrest in adults
- Identify and treat patients at risk of cardiorespiratory arrest using the ABCDE approach

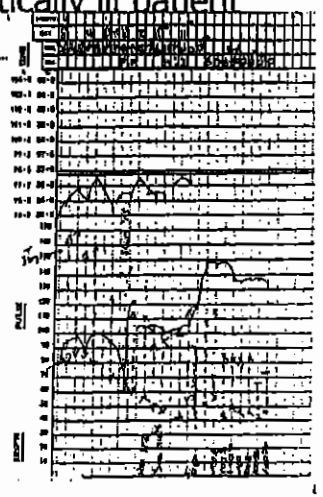
Chain of survival

Early recognition prevents:

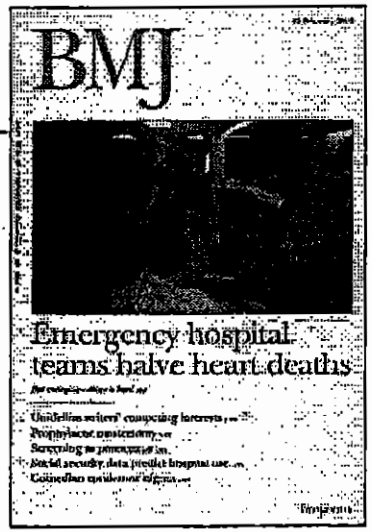
- Cardiac arrests and deaths
- Admissions to ICU
- Inappropriate resuscitation attempts

Early recognition of the critically ill patient

- Most arrests are predictable
- Deterioration prior to 50 - 80% of cardiac arrests
- Hypoxia and hypotension are common antecedents
- Delays in referral to higher levels of care



Medical Emergency Teams


BMJ

Emergency hospital teams halve heart deaths

Outcome after cardiac arrest in adults in UK hospitals

	VF/VT	PEA/Asystole
Number	422 (32%)	903 (68%)
ROSC	298 (71%)	344 (38%)
Survive discharge (overall 17.6%)	179 (42%)	58 (6%)

Recognition of critically ill patients

Modified Early Warning System (MEWS)

	3	2	1	0	1	2	3
Pulse		< 40	41-50	51-100	101-110	111-130	> 130
Systolic BP mmHg	< 70	71-80	81-100	101-199		> 200	
Respiratory Rate		< 8		9 -14	15-20	21-29	> 30
Temp °C		< 35	35.1-36.5	36.6-37.4	> 37.5		
CNS				A	V	P	U

Causes of cardiorespiratory arrest

Most are caused by problems with:

- Airway
- Breathing
- Circulation

Causes of cardiorespiratory arrest
Airway problems

Obstruction caused by:
- CNS depression
- Blood
- Vomit
- Foreign body
- Trauma
- Infection
- Inflammation
- Laryngospasm
- Bronchospasm

Causes of cardiorespiratory arrest
Breathing problems

- **Decreased respiratory drive**
  - CNS depression
- **Decreased respiratory effort**
  - muscle weakness
  - nerve damage
  - restrictive chest defect
  - pain from fractured ribs
- **Lung disorders**
  - pneumothorax
  - haemothorax
  - infection
  - acute exacerbation COPD
  - asthma
  - pulmonary embolus
  - ARDS

Causes of cardiorespiratory arrest
Circulation problems

Primary
- Acute coronary syndromes
- Dysrhythmias
- Hypertensive heart disease
- Valve disease
- Drugs
- Hereditary cardiac diseases
- Electrolyte / acid base abnormalities
- Electrocution

Secondary
- Asphyxia
- Hypoxaemia
- Blood loss
- Hypothermia
- Septic shock

The ABCDE approach to the critically ill patient

A... airway
B... breathing
C... circulation
D... disability
E... exposure

ABCDE approach

Underlying principles

- Complete initial assessment
- Treat life-threatening problems
- Reassessment
- Assess effects of treatment/ interventions
- Call for help early
  - e.g. Medical Emergency Team

ABCDE Approach

- Personal safety
- Patient responsiveness
- Vital signs
  - pulse, respiratory rate, BP, SpO_2, ECG, temperature

ABCDE approach

Airway

Recognition of airway obstruction

- Talking
- Difficulty breathing, distressed, choking
- Shortness of breath
- Noisy breathing
  - stridor, wheeze, gurgling
- See-saw respiratory pattern, accessory muscles

ABCDE approach

Airway

Treatment of airway obstruction

- Airway opening
  - i.e. head tilt, chin lift, jaw thrust
- Simple adjuncts
- Advanced techniques
  - e.g. LMA, tracheal tube
- Oxygen

ABCDE approach

Breathing

Recognition of breathing problems

- Look
  - respiratory distress, accessory muscles, cyanosis, respiratory rate, chest deformity, conscious level
- Listen
  - noisy breathing, breath sounds
- Feel
  - expansion, percussion, tracheal position

ABCDE approach

Breathing

Treatment of breathing problems

- Airway
- Oxygen
- Treat underlying cause
  - e.g. drain pneumothorax
- Support breathing if inadequate
  - e.g. ventilate with bag mask

ABCDE approach

Circulation

Recognition of circulation problems

- Look at the patient
- Pulse – tachycardia, bradycardia
- Peripheral perfusion - capillary refill time
- Pulse – tachycardia, bradycardia
- Blood pressure
- Organ perfusion
  - chest pain, mental state, urine output
- Bleeding, fluid losses

ABCDE approach

Circulation

Treatment of problems

- Airway, Breathing
- Oxygen
- IV access, take bloods
- Treat cause
- Fluid challenge
- Haemodynamic monitoring
- Inotropes/vasopressors
- Oxygen/Aspirin/Nitrates/ Morphine for ACS

ABCDE approach
Disability

- Recognition – AVPU or GCS, and pupils
- Treatment - ABC
- Treat underlying cause
- Blood glucose
  - if < 3 mmol l^{-1} give glucose
- Consider lateral position
- Check drug chart

ABCDE approach
Exposure

- Remove clothes to enable examination
  - e.g. injuries, bleeding, rashes
- Avoid heat loss
- Maintain dignity

Summary

- Early recognition of patients at risk may prevent cardiorespiratory arrest
- Most patients have warning symptoms and signs before cardiorespiratory arrest
- Airway, breathing or circulation problems can cause cardiorespiratory arrest
- ABCDE approach to recognise and treat patients at risk of cardiorespiratory arrest

Respiration Rate	≤8		9 - 11	12 - 20		21 - 24	≥25
Oxygen Saturations	≤91	92 - 93	94 - 95	≥96			
Any Supplemental Oxygen		Yes		No			
Temperature	≤35.0		35.1 - 36.0	36.1 - 38.0	38.1 - 39.0	≥39.1	
Systolic BP	≤90	91 - 100	101 - 110	111 - 219			≥220
Heart Rate	≤40		41 - 50	51 - 90	91 - 110	111 - 130	≥131
Level of Consciousness				A			V, P, or U

*The NEWS initiative flowed from the Royal College of Physicians' NEWSDIG, and was jointly developed and funded in collaboration with the Royal College of Physicians, Royal College of Nursing, National Outreach Forum and NHS Training for Innovation.

ALS lec 5 Thursday morning

Advanced cardiac life support ACLS

European resuscitation council 2010

BLS for all patients is the same But ACLS depends on the type of cardiac arrest.

We start ACLS when the qualified person, the equipments & the drugs are available.

When you connect the pt. to ECG monitor the rhythm is one of the following;

I. **Non-shockable rhythm**: only 7% of these patients will survive & get out of the hospital without significant morbidity.
   a. Asystole: flat ECG

   b. PEA (pulsless electrical activity): also known as electromechanical dissociation, clinical condition characterized by absence of palpable pulse with the presence of recordable electrical activity.

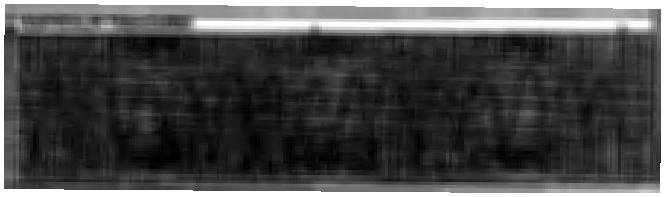
II. **Shockable rhythm:** nearly half of these patients will survive & get out of the hospital without significant morbidity. So you have to do an enormous effort with them.
   a. V Fib. (Ventricular fibrillation):
      - Coarse; Bizarre & irregular ECG, No recognized QRS complex & random in frequency & amplitude.

- Fine; difficult to be differentiated from asystole.

Note; before saying that your pt. Has V fib you have to exclude two things:

- The pt. is not hypothermic since shivering can produce ECG changes that mimic V Fib.
- There is no electrical interference e.g. coutery during surgical procedures.

b. PVT (Pulsless ventricular tachycardia):
   - Monomorphic; broad complex tachycardia with absent pulse.



- Polymorphic; torsade de pointes (forget about it !)

Note;

- Broad complex tachycardia; QRS complex > .12 seconds (>3 small squares)
- Narrow complex tachycardia; QRS complex < /= .12 seconds.
- The normal QRS complex is the NARROW one

Defibrillator;

It is a machine that is connected to an ECG & gives an electrical shock in order to:

- Arrest an abnormal rhythm to allow the heart to restart in a new normal rhythm as in arrhythmias.
- Depolarize the heart i.e. to stimulate the heart as in cardiac arrest.

Two types;

I. Monopahsic
   - Gives an electrical current that flows in one direction.
   - Charge it to 360 Jules from the 1st to the last shock.
   - 75% success rate from the 1st shock.

II. Biphasic
   - Gives an electrical current that flows in one direction & then back in the opposite direction.
   - Charge it to 150-200 Jules from the 1st to the last shock.

- 85% success rate from the 1st shock , >90% success rate from the 2nd shock

The defibrillator is connected to pedals or adhesive pads.

One of the pedals (or pads) is **apical** (on the LT. ant. axillary line between 4th & 5th intercostal spaces) & the other is **sternal** (lateral to the Rt. Border of the sternum just below the right clavicle).

Note; these locations are not evidence based but we think that the current will flow from SA node to the ventricles.

AED; automated external defibrillator is a simple-to-use device which is designed to analyze the heart rhythm itself, and then advise the user whether a shock is required. It is supposed to be part of BLS but in our country it is considered as ACLS.

Reversible causes of cardiac arrest (4 H & 4 T) ;

Hypothermia

Hypovolemia e.g. ruptured spleen.

Hypoxemia e.g. COPD.

Hyperkalemia e.g. chronic kidney failure.

Thrombosis e.g. PE (give anticoagulant patients with PE are very resistant to CPR)

Tension pneumothorax → convert to simple pneumothorax

Temponade → evacuate the temponade.

Toxins → stomach lavage, give antidote.

N.B; correcting the reversible causes of cardiac arrest goes hand in hand with CPR.

Evidence based medicine in CPR:

Two evidence based medicine in CPR to have good outcome:

1. **Effective continuous NON-interrupted chest compression.**
2. Early defibrillation if the pt. has a shockable rhythm.

N.B: effective means: chest compressions at least 5 cm deep and at a rate of at least 100 per minute.

At any time you can stop to assess, re assess or do any procedure but if you think that procedure will interrupt chest compression for more than 10 seconds DO NOT do it !! remember chest compression is more important than intubation.

NOTE ; wherever you see CPR in this sheet it means (30 compression:2 breaths OR 100 compression/minute with 8-10 breaths/ minute)

Asystole/ PEA algorithm;

1. Continue chest compression.
2. Give 1 mg adrenaline **as soon as** you have an IV access; repeat every 4-5 minutes.
3. Outcome , one of three :
    1. Recovery of the pt. → send the pt. for post resuscitation care & correct the reversible causes.
    2. Persistent asystole → continue CPR with 1 mg adrenaline IV every 4-5 minutes.
    3. Shift to a shockable rhythm → change the algorithm.

V Fib. / PVT algorithm;

1. Once you see a shockable rhythm continue chest compression & ask for the defibrillator to be charged IMMDIATELY & give the shock as soon as it is charged.
2. Resume CPR immediately & repeat the shock every two minutes.
3. After the **third** shock give 1 mg adrenaline & 300 mg amidarone.
4. No response after the third shock and the drugs : continue the shocks every 2 mins & give 1 mg adrenaline every other shock.
5. Outcome:
    1. Recovery of the pt. → send the pt. for post resuscitation care & correct the reversible causes.
    2. Persistent V fib. / PVT→ continue CPR with a shock every 2 minutes & 1 mg adrenaline every other shock (4-5 minutes).
    3. Shift to a non-shockable rhythm → change the algorithm.

NOTES:

- In the shockable rhythm the cycle is **2** minutes while in the non-shockable rhythm it is **4** minutes.
- In the non-shockable rhythm you give adrenaline as soon as you have an IV access while in the shockable rhythm you don't give any drug before the third shock.
- Keep your eye on the clock in order not to give more than **1** mg adrenaline in less than **4** minutes.
- Do chest compression **while** charging the defibrillator.
- When the leader of the CPR team says : "Clear !! " ; the person with Oxygen source must go to the nearest wall.
- Always have a plan in mind, do the step & think about the next e.g. ask for an IV access because you may give drugs after the 3rd shock.
- Don't forget to put your pt. in a flat position before you start CPR.
- When your CPR is done don't forget to say that you want to correct the reversible causes & send your pt. for post resuscitation care.

Now study the following clinical scenario, note that things in italic are said by the examiner.

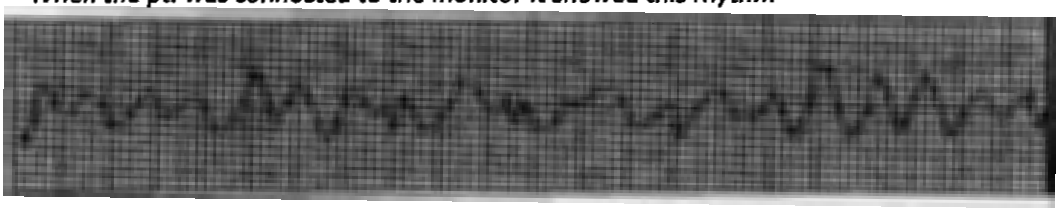
Scenario 1: A nurse came to you saying that a pt. in the floor became unresponsive she shacked his shoulder & called his name but he didn't reply, then she did the ABCD approach & thought that the pt. has cardiac arrest, when you went there you found a nurse doing chest compression. What should you do?

Quickly perform the ABCD approach to make sure the pt. has cardiac arrest.

There is no pulse & the pt. isn't breathing

Ask the nurse to continue chest compression, order an emergency trolley & a defibrillator.

When the pt. was connected to the monitor it showed this rhythm



This is a ventricular fibrillation (V fib), it is a shockable rhythm Charge the defibrillator to 180 Jules immediately,,

It is charged sir.

I'm clear, everybody clear, you give the first shock.

You reassess the pt.

The ECG still shows the same rhythm

Continue for another CPR cycle (2 minutes duration), during this ask for the defibrillator to be charged & for an IV access

Two minutes passed, it is charged sir,,

I'm clear, everybody clear, you give the second shock.

You reassess the pt.

The ECG still shows the same rhythm

Continue for another CPR cycle (2 minutes duration), during this you ask for the defibrillator to be charged & to prepare 1 mg of adrenaline & 300 mg of Amidarone.

Two minutes passed, it is charged sir,,

I'm clear, everybody clear, you give the third shock.

The ECG still shows the same rhythm

Give the dugs

You reassess the pt.

The ECG shifts to this new rhythm

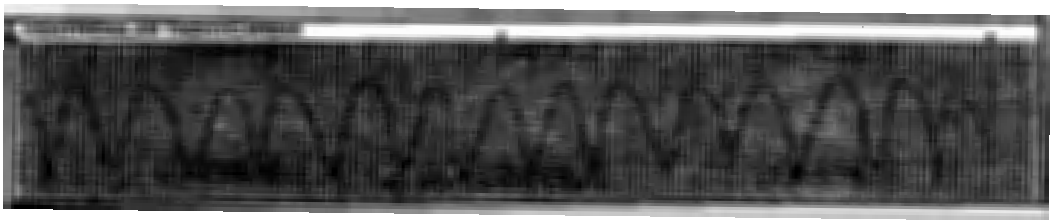
This is asystole so you have to change non-shockable algorithm, perform CPR for 4-5 minutes,

You reassess the pt.

The ECG still shows the same rhythm

Give 1 mg of Adrenaline,

The pt. shifts to this new rhythm



This ventricular tachycardia so you have to check the pulse

The pulse is absent

Pulsless ventricular tachycardia, so you should shift the algorithm to shockable cardiac arrest, your eye on the clock to know the time when you gave adrenaline (DON'T ever give more than 1 mg adrenaline in less than 4 minutes interval), ask for the defibrillator to be charged IMMEDIETLY & continue chest compression

It is charged sir

I'm clear, everybody clear, you give the shock.

The pt. returned to life, your CPR was successful.

You send him to the post resuscitation care & correct the reversible cause.

Scenario 2: you are the doctor in charge in the ER, a 44 year-old male arrived complaining of chest pain that started after his last meal you asked him about the character of the pain but he didn't reply & became unresponsive. What should you do?

You shake the pt. & call his name,

The pt. is still unresponsive

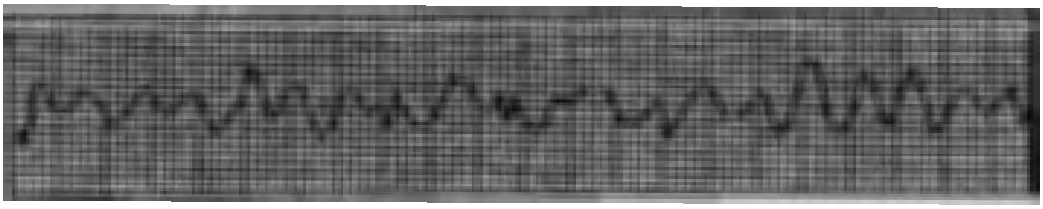
Order an emergency trolley, a defibrillator & ask for the CPR team.

Perform the ABCD approach

The pt. isn't breathing & pulsless

Start chest compression & ask the nurse to connect him to a monitor

It showed this rhythm



This is ventricular fibrillation so ask the nurse to charge the defibrillator IMMEDIATELY to 180 Jules.

It is charged sir

I'm clear, everybody clear, you give the first shock.

The ECG still shows the same rhythm

You perform a CPR cycle (2 minutes duration) & while doing this ask for the defibrillator to be charged again & ask for an IV access

2 minutes passed & the defibrillator is charged

I'm clear, everybody clear, you give the second shock.

The ECG still shows the same rhythm

You perform another CPR cycle (2 minutes) & while doing this ask the nurse to charge the defibrillator & to prepare 1 mg Adrenaline & 300 mg Amidarone.

The defibrillator is charged sir,,

I'm clear, everybody clear, you give the third shock.

The ECG still shows the same rhythm

Continue chest compression & ask the nurse to give the drugs.

The ECG still shows the same rhythm

You perform another CPR cycle (2 minutes) & while doing this ask the nurse to charge the defibrillator

You give the forth shock & you don't give any drug.

You perform another CPR cycle (2 minutes) & while doing this ask the nurse to charge the defibrillator & to prepare 1 mg of adrenaline only.

You give the fifth shock & you give the drug.

You go on like this until the pt. shows signs of coming back, shifts to another algorithm or you decide to stop, but a pt. who presents with a shockable rhythm has an excellent prognosis & you should do all what you can to bring him back to life. Don't forget about correcting the revisable causes of cardiac arrest in this case MI so you should give an anticoagulant.

Scenario 3: A 32-year old lady was in labor when she became so ill & you've been called as the head of the CPR team when you arrived there everyone was screaming & the pt. was unresponsive. What should you do?

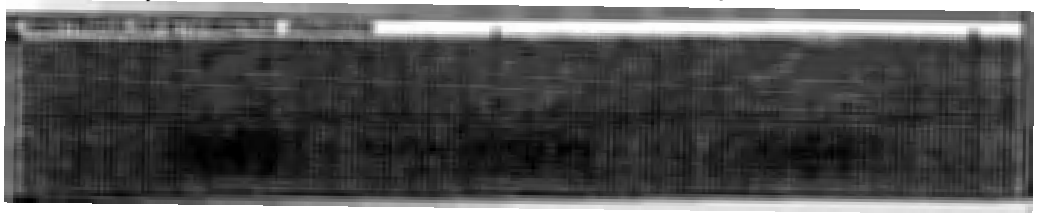
First you have to calm down & forget about everyone around in order to do the best for the pt.

Do the ABCD approach,

The pt. isn't breathing & there is no pulse,

Ask for help, emergency trolley & a defibrillator & start BLS

When the pt. was connected to the monitor it showed this rhythm:

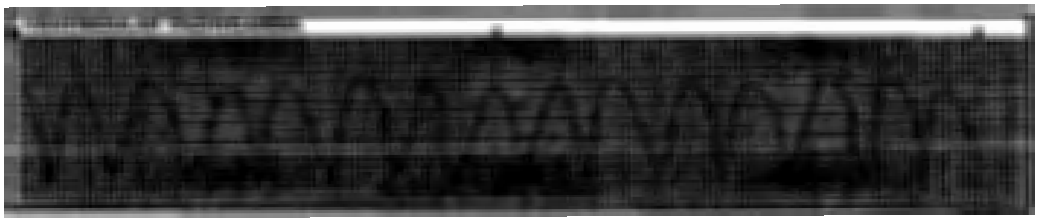


This is an asystole, a nonshockable rhythm, so continue chest compression, and ask for an IV access

The IV access is ready sir,

Give 1 mg adrenaline & look at the clock.

The ECG showed this rhythm:



This is ventricular tachycardia (V tach.), so check the pulse,

There is no pulse

This is pulseless ventricular tachycardia (PVT), a shockable rhythm, ask for the defibrillator to be charged to 180 Jules **immediately** & continue chest compression

It is charged sir,

I'm clear, everybody clear, you give the first shock.

The ECG is still the same,

You perform a CPR cycle (2 minutes) & while doing this ask the nurse to charge the defibrillator.

Two minutes passed, it is charged sir,,

I'm clear, everybody clear, you give the second shock.

The ECG is still the same,

You perform another CPR cycle (2 minutes) & while doing this ask the nurse to charge the defibrillator & to prepare 1 mg of adrenaline & 300 mg of Amidarone.

I'm clear, everybody clear, you give the third shock.

The ECG is still the same,

Give the drugs

The pt. came back to life, it was an effective CPR because chest compression wasn't interrupted & the doctor had a plan in mind before each step.

Now you should find why the pt. had cardiac arrest (in this scenario most probably hypovolemia), correct this & send the pt. for the post resuscitation care.

Scenario 4: You are the resident in charge in the ICU, the nurse came to you saying that the pt. who was admitted as a case of CVA yesterday became unresponsive & the ECG shows that he has V Fib. What should you do?

N.B: the pt. has an IV access & on a face mask.

You shake the pt. & call his name,

The pt. is still unresponsive

Order an emergency trolley, a defibrillator & ask for the CPR team.

Perform the ABCD approach

The pt. isn't breathing & pulsless

Check the ECG to see if the patient really has V Fib., make sure he is not hypothermic (shivering can lead to changes in the ECG that mimic V Fib.).

The pt. temperature is 37.4 C

Start chest compression & ask for the defibrillator to be charged to 180 Jules **IMMEDIATELY.**

It is charged sir,,

I'm clear, everybody clear, you give the first shock.

The ECG still shows the same rhythm

You perform a CPR cycle (2 minutes duration) & while doing this ask for the defibrillator to be charged again.

2 minutes passed & the defibrillator is charged

I'm clear, everybody clear, you give the second shock.

The ECG still shows the same rhythm

You perform another CPR cycle (2 minutes) & while doing this ask the nurse to charge the defibrillator & to prepare 1 mg Adrenaline & 300 mg Amidarone.

The defibrillator is charged sir,,

I'm clear, everybody clear, you give the third shock.

The ECG still shows the same rhythm

Continue chest compression & ask the nurse to give the drugs.

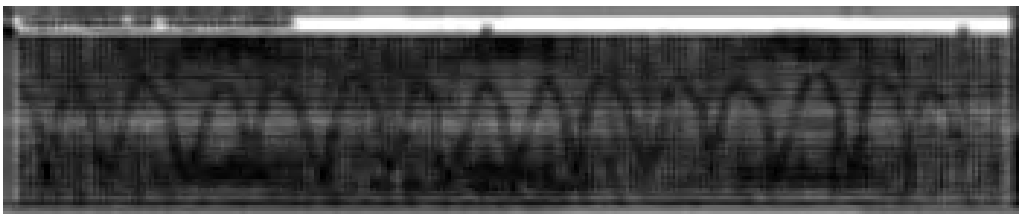
After giving the drugs, the pt. had flat ECG.

This is asystole; a non-shockable rhythm so you perfume CPR for 4-5 minutes, ask the nurse to prepare 1 mg adrenaline.

4 minutes passed,,

Give adrenaline

The pt. shifts to this rhythm:



This is ventricular tachycardia (V tach.), so check the pulse,

There is no pulse

This is pulseless ventricular tachycardia (PVT), a shockable rhythm, ask for the defibrillator to be charged **immediately** & continue chest compression

It is charged sir,

I'm clear, everybody clear, you give the shock.

The pt. reverted, your CPR was successful.

Done by : Aya Ghassan

Peri-arrest conditions

Peri-arrest conditions are conditions that may lead to cardiac arrest if not managed properly, happen during the CPR or after it leading to arrest again.

Bradycardia algorithm:

RULE NO.1: Not all patients with heart rate less than 60 bpm should be treated.

When should you treat bradycardia?

I. When the heart rate is < 40 bpm.
II. If the heart rate is 40-60 & the pt. has one of the following (signs of instability):
    1. Heart failure (HF); because the cardiac output in this pt. is dependent on his heart rate (cardiac output= heart rate × stroke volume).
    2. Unstable vital signs especially if systolic blood pressure is < 90 mmHg.
    3. If the pt. has frequent PVCs; >7/min (premature ventricular contractions /extrasystoles).
III. If the pt. is at risk of asystole;
    1. Recent history of asystole.
    2. 2nd or 3rd degree heart block.
    3. Pause on ECG for > 3 seconds.

Bradycardia algorithm:

1. Assess using the ABCD approach.
2. Put an oxygen mask to compensate for the decrease in cardiac output & obtain an IV access.
3. Obtain a 12-lead ECG; to find the cause of bradycardia & to look for PVCs or pauses on the ECG.
4. Assess the pulse & blood pressure.
5. If any of the above conditions is present you should manage as indicated below, otherwise keep your pt. under observation.
6. Manegment:
    a. Start with Atropine .5 mg (500 µg) & repeat according to the response & with a maximum dose of 3 mg.
    b. If didn't work shift to isoprenaline : a *Sympathomimetic*, the ampoule contains 200 µg , its diluted in IV fluid and given according to time as infusion 2-5 µg/ min

c. No response → shift to adrenalin : 2-10 µg/ min
d. If didn't work seek a cardiologist consultation to implant a pace maker.

((((Atropine → Isoprenaline → Adrenaline → pacemaker))))

Notes;

- People with autonomic neuropathy e.g. diabetic patients or people with desensitization of the heart i.e. cardiac transplant; do not respond to atropine.
- Isoprenaline is a sympathomimetic drug that acts mainly on β-1 receptors.

Tachycardia algorithm;

Tachycardia is a heart rate > 100 bpm, BUT we start management when the heart rate exceeds 140bpm.

Types of tachycardia;

I. Wide (broad) complex tachycardia;
   - Regular→ V tach. With pulse.
   - Irregular→ A fib. With bundle branch block.

II. Narrow complex tachycardia;
   - Regular→ SVT.
   - Irregular→ A fib. With rapid ventricular response.

In all the previous types , the pt. may be stable or unstable

Signs of instability in a pt. with tachycardia:

1. **Systolic blood pressure is < 90 mmHg.**
2. **HF;** pt. will have ineffective ventricular contraction since there is no time for ventricular filling.
3. Ineffective cardiac output that presents as hypoperfusion of vital organs;
   - Brain; leads to **decreased level of consciousness.**
   - Heart; hypoperfusion of coronary arteries in addition to increased demand leads to **angina pain.**

Tachycardia algorithm;

1. Assess using the ABCD approach.
2. Put an oxygen mask to compensate for the decrease in cardiac output & obtain an IV access.
3. Obtain a 12-lead ECG; to find the cause of tachycardia.

4. Assess the pulse & blood pressure.
5. You should always manage tachycardia wether the pt. is stable or not.

Management of unstable pt.;

1. Synchronized dc shock; the shock will synchronize with the T-wave of SA node to arrest it to give the heart the chance to start in a new normal sinus rhythm. You can repeat this up to **three** sequential times.
2. Amidarone ;

   Loading dose: 300 mg over 20 minutes.

   Maintenance dose: 900-1000 mg over 24 hours.

N.B;

- You should use sedatives because it is very difficult to give the pt. a shock while awake.
- Amidarone is available in 150-mg ampoules.
- If the pt. is still unstable you can repeat the 3 shocks every hour.

Management of stable pt.:

Chemical pacing;

- **SVT;**

  Adenosine 6 mg rapid IV bolus, if unsuccessful give 12mg, if unsuccessful give further 12mg.

  *** adenosine is metabolized quickly in the blood , that's why flush of saline must be given after it , or give it in a central line

- **For all other types of tachycardia;**

  Amidarone ;

  Loading dose: 300 mg over 20 minutes.

  Maintenance dose: 900 mg over 24 hours.

Done by : Aya Ghassan

Mechanical ventilators

The first mechanical ventilators found used to work by negative pressure ventilation, called the iron lung, which is a metallic chamber surrounding a patient's chest, work by pistons to generate an intrapleural pressure (IPP) of -2 to -10 cm H2O.

Following the epidemic of polio, in 1945, a new scheme of ventilation has been introduced. That is a face – bag system that forces air inside the lung founding the positive pressure ventilation scheme.

|| Types of mechanical ventilators:

1. Invasive
2. Non-invasive

#Anything above the larynx (which marks the beginning of the definitive air way) is non-invasive, while anything below the level of the larynx is invasive.

|| Non-invasive mechanical ventilators:

1. CPAP (continuous positive airway pressure):

2. BPAP (biphasic positive airway pressure):
   It delivers a high pressure in inspiration and a low pressure in expiration

|| Invasive mechanical ventilators:

<u>Indications of use:</u>

1. For adequate gas exchange in patients with a metabolic disturbance. Whether the disturbance is due to;
   pulmonary cause; in the airways (like tracheal stenosis), airspace (like atelectasis), interstitium (like thickening of pulmonary interstitium), vascular (like sepsis), and pleural (like pneumothorax).
   Extrapulmonary cause; muscular (like myasthenia gravis), neuromuscular (like polio), shock (all types).

2. To control $PaCO_2$ at a certain level,
   in cases of;
   > elevated intracranial pressure; as elevation of $PaCO_2$ causes vasodilatation in the brain vesseis which resuit in increased cerebral perfusion and thereby ICP adding the risk of herniation and conning.
   > malignant hyperthermia
   > central anticholinergic syndrome
   > septic patients
3. To decrease the work load of the heart in pts with heart failure; as 12 % of total body oxygen consumption goes for ventilation.

Criteria of pt put on invasive mechanical ventilator:

1. Respiratory rate > 35; which causes a danger of exhaustion
2. Tidal volume < 5 ml/Kg; due to decreased alveolar ventilation
   normal Tv is 7ml/Kg, it's measured by spirometer
3. Vital capacity < 15 ml/Kg
   vital capacity = tidal volume + end inspiratory reservoir+ end expiratory reservoir
   it's normally 65-75 ml/Kg
4. PaO_2 < 60 mmHg while the pt is on 40 % FiO_2
5. $PaCO_2$ > 60 mmHg
6. A-a gradient > 350

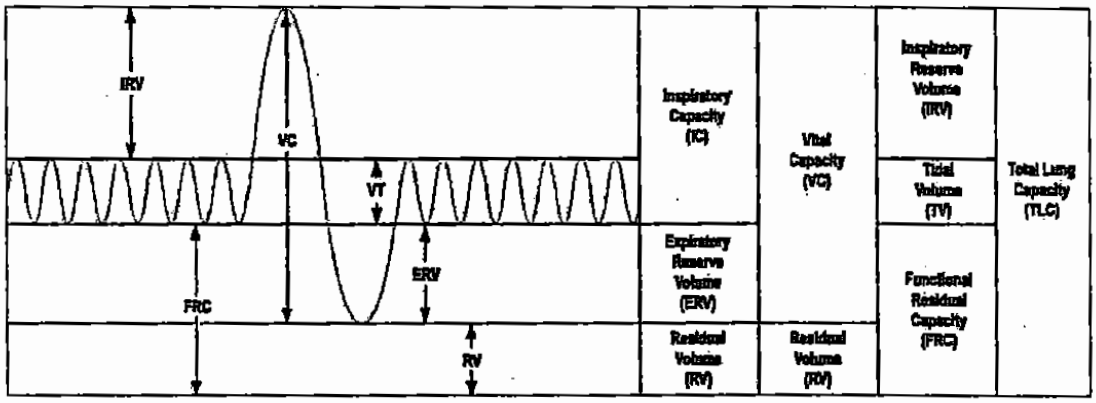
Modes of invasive ventilation:

They are mechanisms by which the ventilator changes from inspiration to expiration. It can by; volume cycle, pressure cycle, or time cycle

1. CMV (conventional mechanical ventilation):
   for pts;
   > deeply unconscious (GCS < 8)
   > no breathing effort
   > high airway pressure
   ## indicated in pts with elevated ICP to control the levels of $PaCO_2$
   it can be either pressure or volume controlled
   in this mode the machine does all the respiratory mechanics (RR, Tv, I:E); as the pt is passive either by disease, muscle relaxant, or heavy sedation

2. SIMV (synchronized intermittent mandatory ventilation):
   a weaning mode of ventilation
   can be used for pts whom we want their spontaneous breathing effort to stay, as the machine synchronize the pt own breath
   >> full spontaneous breathing without help from the ventilator, the ventilator helps in few breaths initiated by the pt
3. ASV (assisted-controlled ventilation):
   another weaning mode from the ventilator
   can be used to ventilate pts whom we want the ventilator to help them in every single breath initiated by the pt himself
   we determine Tv only, while the RR is controlled by the pt

<<PEEP>>
positive end expiratory pressure
> can be added to any type of ventilation
> indicated;
1- if you can't maintain adequate oxygenation on FiO_2 50 %
2- to keep alveoli opened to enhance passage of gasses from alveoli to pulmonary capillary blood in cases of atelectasis
> this mode increase FRC (functional residual capacity) of the resting lung which is normally approximate to 2400 ml in a 70 kg



Brain death

Coma divided into three types :

1- simple reversible like metabolic coma or mild head injury

2- vegetative irreversible in which there is cortical damage but the vital centers are working

3- brain death which is the damage of the neurons in the brainstem

Brainstem death = brain death = clinical death

Functions of the brainstem :

1- centre of vital organs ex: vasomotor and respiratory sys

2- origin of cranial nerves

3- pathway bet upper and lower centers

4- contains RAS

5- coordination of movement bet trunk and limbs

The criteria of medical people who are allowed to assess the brain death :

1- Two teams are required to confirm the brain death

2- each team contains two doctors :-

- senior medical resident

- consultant , neurologist , admission consultant or intensivist

3- they should've been registered in GMC (general medical council) for at least five years

Timing :

1- the first team should test the pt after six hours of the incidence of the event that led to the extensive irreversible damage of the brain

2- the second team should test the pt after 12 hours

*** the second team is the one who write the certification of death

... ... do brain death assessment :

1- for organ donation (most important)

2- those pt cost large amount of money

3- we can't decrease the level of care without having ethical reasons

4- to decrease the suffer and disturbance of their families

before doing the brain death assessment three preconditions are required :

1- pt in deep coma and the GCS = 3 /15 , this should not be due to drugs or hypothermia

*** u should wait for five half lives of the drug

2- the pt should be apneic

3- the metabolic process should be normal and there is no any electrolytes disturbance

Reflexes we test :-

1- pupils and its reactivity to light (3rd cranial nerve function)

2- corneal reflex (5th cranial nerve)

3- stimulate the supraorbital region (5th and 7th cranial nerves)

4- caloric reflex test (vestibule-ocular reflex) >>>> producing an eye movement in the direction opposite to head movement

*** make sure that the external meatus is open and the tympanic membrane is intact

5- gag reflex (9th and 10th cranial nerves)

6- apnea test

Normally if the $PaCO_2$ in our body exceeded 60 mmHg , the respiratory centre should work

This procedure is based on disconnection of the ventilator and bring the pt to hypercarbia in order to stimulate the respiratory center and induce spontaneous breathing .

How to do the apnea test :-

1- Preoxygenate the pt at 100 % o2 so the pt will not become hypoxic (prolong the period of apnea)

2- put an arterial line and take the gases baseline from it

4- test the ABG every 2 minutes and at the same time note the pt chest and abdominal movement (signs of breathing)

5- now in the ABG if the co2 increases more than 3-5 ml and no spontaneous breathing is observed then the test is positive and u have to confirm the brain death

*** if the o2 decreases to less than 90 stop the test and ventilate the pt

*** in brain death cases the organs start failing in 1-30 days without lowering the level of care

بسم الله الرحمن الرحيم

Lecture topic: Pain and Narcotics
Lecturer : Dr. Bashir Attyat
Date : 2008
Written by : Fadi Riad Sunna'

Pain and Narcotics

Pain is not only as physical suffering or anxiety, it is human suffering
The policy for JUH that patients should not suffer! (we are the one who only suffer ☹)

*Categories of Pain:

Acute: It is the pain that appears suddenly as headache and may disappear by simple medication. For example, a patient who have headache can be resolved by paracetamols.

Chronic: It is an existing and continuous type of pain.
We have to find the underlying cause of this type of chronic pain and treat it.

Source of Pain:

a. **External:** The billion nerve endings that are distributed in our skin serve as a guard (Think of burn) and protect us. Also, they try to transfer all normal and abnormal sensations regardless of what they are to our brain to be translated.

b. **Internal:** The nerve endings are different in quantity (one tenth of the external nerve endings) and quality. These are distributed mainly in our GIT.

   If the colon was obstructed due to any reason (tumor, adhesions, infection...) distention will occur and will reach to a maximum limit above which we start feeling of pain in that area.

So, we can conclude that internal sensation depends upon the following 3 factors:

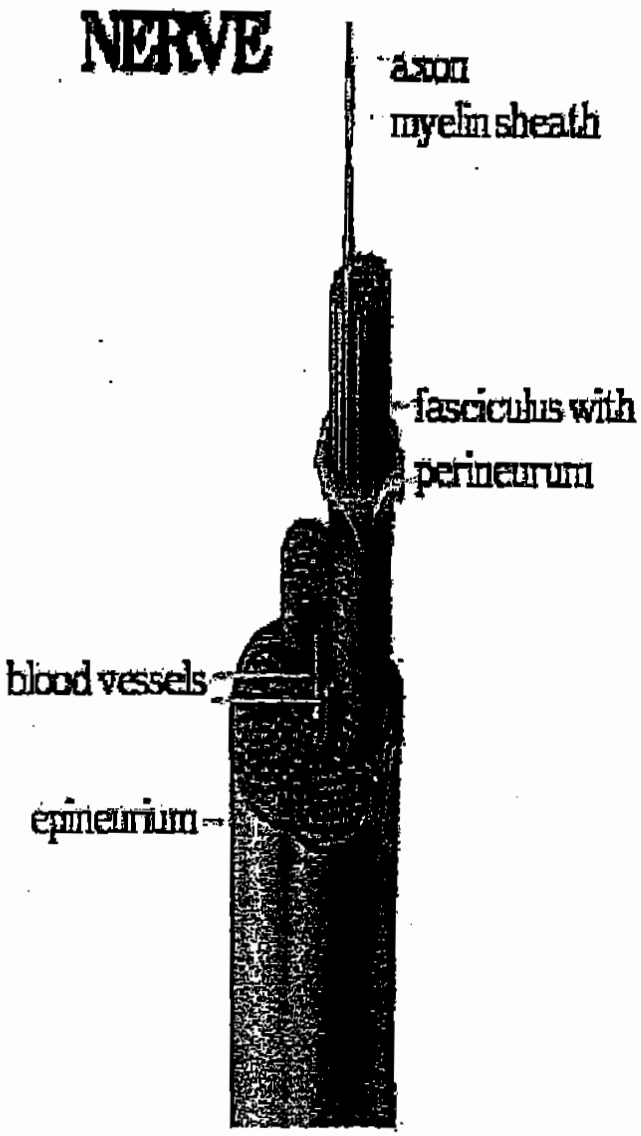
- Tolerance of the nerve ending: The pressure and dilatations will increase up to a limit above which the nerve endings will be stimulated.
- Ischemia beside pressure: For example, thrombosis of the mesenteric artery in an old patient will lead to ischemia that will give certain chemical substances and alarm the nerve endings
- Invasion: invasion of a tumor till it reaches the internal nerves.

To make things clearer we will present our following ideas from this example:

An abscess was present in a certain area, the body will produce prostaglandins, potassium ions and bicarbonate ions at the site of abnormal sensation that serve as alarming factors to the nerves.

The nerves will conduct the message to next stations in 2 ways:

- Through myelinated nerves: conduction here is fast through salutatory conduction across the nodes of Ranvier (exposed area of the nerve)
- Thinner and non-myelinated nerve: slower conduction



This picture is just to refresh your memory.

"Gate-Control Theory":

The afferent sensory nerves will enter the spinal cord from the dorsal horn. Of course, some of the nerves will reach sooner than the other due to the way of conduction we explained simply above.
And efferent motor nerves will get out of the anterior horn.

What we want to say that at the level of the spinal cord will protect and filtrate the information it receives. The 12 laminae and especially the "Substantia Gelatinosa" will filter the incoming information according to the

sharpness duration of the incoming information (alarm).Also, it will block some of the sensations and protects us.

We will give a simple example: by pricking the tip of a finger with a sharp pin or staple we will feel pain for a very short duration because the spinal cord blocked the sensation as the sharpness and duration of the alarm is very short. Protection occurs when we have reflex mechanism as touching a hot iron.

So, some of the main functions of the spinal cord are:

1. Filtration
2. Blocking
3. Reflex-defense mechanism (protection).

Additional information about "Gate-Control Theory":

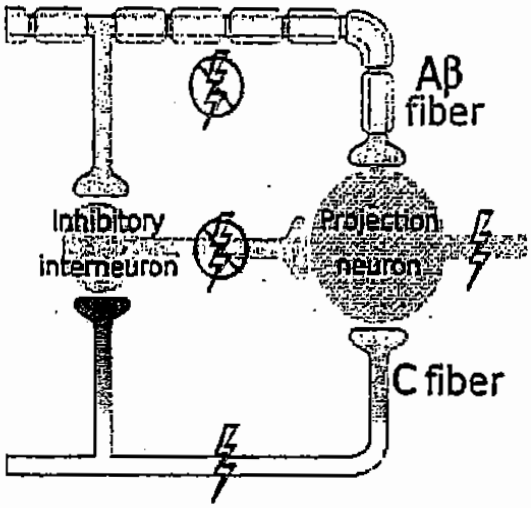
Gate control theory asserts that activation of nerves which do not transmit pain signals, called nonnociceptive fibers, can interfere with signals from pain fibers, thereby inhibiting pain.Afferent pain-receptive nerves, those that bring signals to the brain, comprise at least two kinds of fibers - a fast, relatively thick, myelinated "Aδ" fiber that carries messages quickly with intense pain, and a small, unmyelinated, slow "C" fiber that carries the longer-term throbbing and chronic pain. Large-diameter Aβ fibers are nonnociceptive **(do not transmit pain stimuli) and inhibit the effects of firing by Aδ and C fibers.**

The peripheral nervous system has centers at which pain stimuli can be regulated. Some areas in the dorsal horn of the spinal cord that are involved in receiving pain stimuli from Aδ and C fibers, called laminae, also receive input from Aβ fibers.The nonnociceptive fibers indirectly inhibit the effects of the pain fibers, 'closing a gate' to the transmission of their stimuli.In other parts of the laminae, pain fibers also inhibit the effects of nonnociceptive fibers, 'opening the gate'.

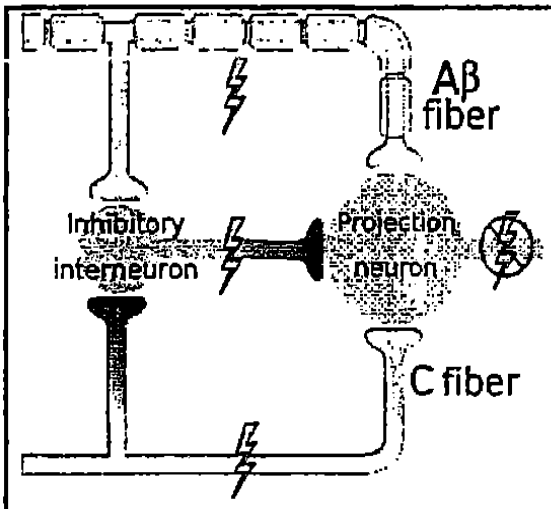
An inhibitory connection may exist with Aβ and C fibers, which may form a synapse on the same projection neuron. The same neurons may also form synapses with an inhibitory interneuron that also synapses on the projection neuron, reducing the chance that the latter will fire and transmit pain stimuli to the brain.The inhibitory interneuron fires spontaneously.The C fiber's synapse would inhibit the inhibitory interneuron, indirectly increasing the

projection neuron's chance of firing. The Aβ fiber, on the other hand, forms an **excitatory** connection with the **inhibitory** interneuron, thus decreasing the projection neuron's chance of firing (like the C fiber, the Aβ fiber also has an excitatory connection on the projection neuron itself). Thus, depending on the relative rates of firing of C and Aβ fibers, the firing of the nonnociceptive fiber may inhibit the firing of the projection neuron and the transmission of pain stimuli.

The following pictures may make things clearer:



The firing of the projection neuron determines pain.The inhibitory interneuron decreases the chances that the projection neuron will fire.Firing of C fibers inhibits the inhibitory interneuron (indirectly), increasing the chances that the projection neuron will fire.A lightning bolt signifies increased neuron activation, while a crossed-out bolt signifies weakened or reduced activation.



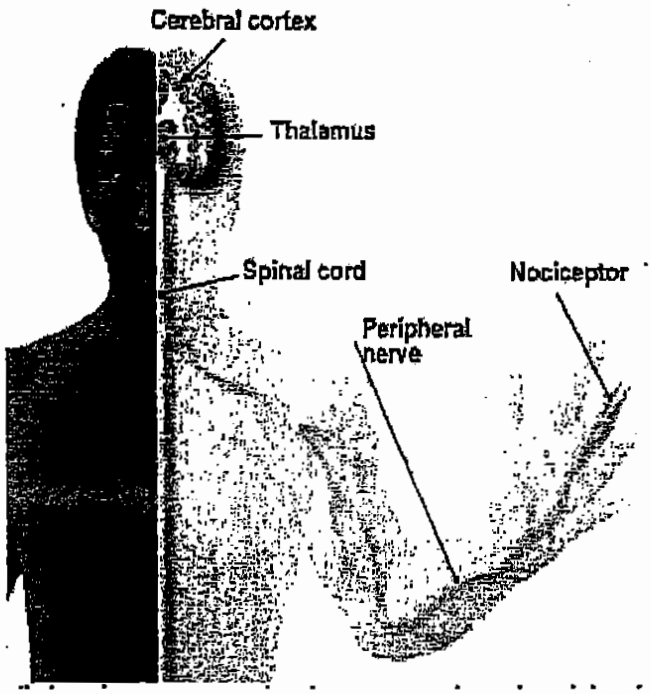
Firing of the Aβ fibers activates the inhibitory interneuron, reducing the chances that the projection neuron will fire, even in the presence of a firing nociceptive fiber.

*Identification of Pain Pathway:

In the past they used to think that for each type of pain there is a selective nerve. But, what is true is our recent "Gate-Control Theory". Since the discovery of this theory our understanding of the pain pathway has improved dramatically. And what is important that the success rate in treating chronic pain reached up to 85%

Now, let us summarize what we have just said:
We revised the categories and source of pain. Moreover, we said how impulses are conducted across the nerves (myelinated and unmyelinated or more correctly thinly myelinated) till reaching the SC. Also, we discussed the "Gate-Control Theory"

The SC will receive, filter the information and is responsible for some direct reflexes. Other information will be sent to the higher centres (those of high duration). So, **part** of the nerves from the periphery will decussate across the laminae to the other side to get high up to the medulla oblongata → to thalamus and finally to cortex.
The crossing tract is called the dorsal horn. While there is another part that did not decussate and is called spinothalamic tract (STT).



Some nerves pass through the thalamus because it is the centre of **emotion and mood.**
Which means that according to our situation or pain will be reflected in our mood. Not only that, the education and experience in the past will determine the degree of mood.

On the other hand, the cortex is responsible for organization, severity & localization of pain and it provides information about the pain (sharp sensation in our burned hand).
The cortex also has its special pharmacy providing endorphins that are 6 times more potent than morphine!!! Endorphins will protect our body for a certain period that differs from one person to another. Soldiers in the war can have a gunshot and still fighting without feeling of a severe pain.

"لما تبرد الضربة بتشعر بالوجع"

Narcotics:

We have three stations for the pain impulses that narcotics can act upon:

I. Site of action
II. Spinal Cord (SC)
III. Brain

The type of medication should be given according to the type, severity and duration of pain. We don't give pethidine for a simple and normal headache.

Our Map of Treatment (These are only example):

I. Peripheral source of pain: prevent the pain sensations from passing to next stations by using non-inflammatory drugs that overcome the prostaglandins, potassium and bicarbonate ions.
II. Spinal Anaesthesia in intrathecal space according to the level can paralyze the limbs in about 5 minutes and I can use that when we have a patient with a broken leg.
III. At level of the brain I can give analgesics in the ventricles in cases of tumor.

*Summary:

According to 1.Site of pain, 2.Severity of pain, 3.Degree of pain and 4.Duration we treat our patients.

What is important in management is the 3 step leader of the WHO:

"Three Step Leaders" States that:

1. All mild pain: paracetamol and non-steroidal

2. Moderate type of pain (appendicitis, cholecystitis, piles, colic pain…) we will have no effect by using paracetamols or nonsteroidal alone. So, we mix mild narcotics as codeine(Tramal) with non-steroidal. **OR** mild narcotics **alone** are enough.

3. Severe pain of long duration that we can not control it immediately we give narcotics **with or without** non-steroidal/codeine(mild narcotics) and sometime cortisone is even given.

"تمت بحمد الله"