وزارة التعليم العالي والبحث العلمي
هيئة التعليم التقني
المعهد التقني / الموصل

قســـــم ألتخـديــر

مـــــادة

ألـعنـــــــايـة ألمـــــركزة

أعداد .....

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OVERVIEW

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intensive care unit :

Is A Specialized Area Concentrating Care For The Sickest Patients In The Hospital In One Place & Is Staffed By A Multidisciplinary Team Of Doctors, Nurses, Physiotherapists And Dieticians, Among Others, Who Combine Treatment With Constant Patient Observation, Monitoring And Support , Has The Equipment And Medication Required To Provide Multi-Organ Support , Needs To Be Physically Close To Operating Theatres, Emergency Departments And Radiology Services

Different Units :

- Verity Names Depends On Specific Purpose And The Degree Of Dependency Of The Patient
- Many Different Hospitals Have Many Different Terms , Frequently Seen Are

MICU = Medical ICU
SICU = Surgical ICU
TICU = Trauma ICU or Transplant ICU
NICU = Neuro ICU or Neonatal ICU
PICU = Pediatric ICU
CVICU = Cardiovascular ICU
CCU = Coronary Care Unit
CICU = Cardiac ICU
BICU = Burn ICU
RCU = Renal care unit

- ITU-(Intensive Treatment Unit)Highest Level Of Patient Dependency ,Most Aggressive Treatment And Monitoring Protocols ...
- CSU-Cardiac Surgery Units Are Best Example SCBU-(Special Care Baby Unit)Neonatal Problems Often Requiring IPPV And Invasive Monitoring Techniques
- HDU-(High Dependency Unit)Recovering Area Of An Operating Theatre)With Low Level Of Monitoring And High Level Of Nursing Care .

The Main Functions Of Any ICU Is ……

((To Provide Optimum Life Support & Provide Adequate Monitoring Of Vital Functions)) .

Prepration Of The Unit :

The Unit Should Be Kept Ready All The Time Which Should Include The Following

1.Special Bed Having The Following Facilities :

- Head Board Should Be Detachable To Facilitate Intubation (In Case Of Cardio Pulmonary Arrest)
- Bed Should Be Firm And Non Yielding To Facilitate Cardiac Massage
- Should Have A Tilting Mechanism (To Keep Position Of Patient)
- Should Have Side Rails To Prevent Falling (Psychiatric And Anxious Patient)
- There Should Be A Bed Side Locker An Over Bed Table And A Foot Stool Kept Adjacent To The Bed

2.Cardiac Monitor System With Alarm That May Be Connected To The Central Console

3.Oxygen And Suction Apparatus (Preferably Pipeline Model)

4.Resuscitation Unit Containing The Following

- Syringes,Needles, IV Cath, Intravenous Administration Sets, Blood Sets, Scalp Vein Sets And Intra Venous Fluid
- Spirit, Swabs, Adhesive Plaster (Micropore/ Transpore), , Torniquets And Arm Board
- Airways, Endotracheal Tubes And Laryngoscopes Of Different Sizes
- Ambu Bag And Suction Catheters
- Oxygen Cylenders Special Trays Such As Tracheostomy Tray, And Catheterization Tray
- Drugs Such As (Antiarrhythmics,Antianginals, Antihypertensive ,Diuretics ,Anticoagulents ,Antibiotics,Anticonvulsants Etc...
- Infusion Pump

Following Equipments Be Easily Available :

- Defibrillator In Working Mode With Electrodes And Jell
- Cardiac Pacemaker With Pacing Catheters In The Sterile Tray
- Mechanical Ventilators (To Ventilate The Lungs In Case Of Resp:Arrest)
- Facility For Invasive And Non Invasive Procedure Like (Cvp Line, Intra Arterial Pressure Monitor)
- Portable X-Ray Machine
- Ecg Machine
- Oxygen Therapy

Indications For Admission :

- Pre And Post-Operative Patients And Who Underwent Major Surgeries.
- Craniotomy Patients.
- Thoracotomy Patients.
- Ultra Major Surgeries.
- Unstable Multiple Trauma Patients.
- Patients With Head Or Spine Trauma Requiring Mechanical Ventilation.
- Any Surgical Patient Who Requires Continuous Monitoring Or Continuous Life Support

Monitoring system ….. The Monitor Screen Above The Patient Will Display Blood Pressure, Central Venous Pressure CVP, Heart Rate , Pulmonary Artery Pressure PAP Oxygen Saturation , Patient Temperature , Intracerebral Pressure ICP & ECG .
A Policy Should Be In Place For Accommodating Admissions When Unit Capacity Is Reached

Options May Include:

- Limiting Elective Surgery
- Re-Routing Critical Care Admissions From The Emergency Department
- Increasing The Functional Capacity Of The ICU By Boarding Patients In Other Advanced Care Areas

The ICU Admission Decision May Be Based On Several Models:

- Prioritization Model
- Diagnosis Model
- Objective Parameters Model

Admission, Discharge and Triage

Introduction :

Appropriate Utilisation Of ICU Bed Is Essential As Intensive Care Resources Are Limited And Expensive. Demand For Intensive Care Will Continue To Exceed Supply, Hence Clear And Rationale Decision-Making Regarding Admission And Discharge Is Required.

Principles

1. The Decision To Admit A Patient To ICU Should Be Based On The Concept Of Potential Benefit.
2. Critically Ill Patients With A Reversible Medical Condition, Having A Reasonable Prospect Of Meaningful Recovery Should Be Admitted.
3. A Combination Of Criteria Should Be Used To Determine ICU Admission Or Discharge.
4. ICU Triaging Is Necessary To Ensure Optimal And Equitable Use Of Limited Intensive Care Resources.

Approach to the critically ill Patient by **"ABC, MOVIE"** stands for:

Airway, Breathing, Circulation , Monitor, Oxygen, Vital Signs, IV, Exposure protecting their away, assess for airway obstruction (foreign body, signs of stridor), noisy breathing, grunting, cyanosis remove foreign body , Perform maneuvers as head tilt and chin lift or jaw thrust , use adjunct as oropharyngeal airway , LMA level of consciousness (GCS – "less than 8=intubate)

Classically >90% diagnosis made on history In critically ill, patient may not give history! so take it from Collateral: nurses, care aides, family, friends ,referral notes----.

Rapid History: **(SAMPLE)** Symptoms Allergies Medications Past history Last meal Events surrounding

(High risk patients).. **Emergency** admission- limited info , **Infants** and young children , **Pregnant** ladies , **Advanced age** – comorbidities, limited reserve **Severe coexisting** illnesses – mixed problems, limited reserve **Recent major** surgery **Severe bleed**, need for massive transfusion **Deterioration on** repeat assessment/fail to respond to treatment **Immunodeficiency Combinations** of above .

Basic Investigations CBC , Blood Sugar , Electrolytes ,Urea , Creatinine , Cardiac Markers Coagulation Profile , Lactate , Cxr , Ecg , Other Investigations Are Guided By History And Physical Exam

ICU Admission Criteria

To Optimise ICU Resources And Improve Outcomes, ICU Admissions Should Be Guided On The Basis Of A Combination Of Factors:

A. Prioritisation According To The Patient's Severity Of Illness
B. Specific Patient Needs Such As Life-Supportive Therapies
C. Diagnosis
D. Prognosis
E. Potential Benefit From Interventions
F. Objective Parameters At The Time Of Referral
G. Available Clinical Expertise
H. Bed Availability

Icu Admission Based On Priority

The Priority Should Be Based On The Needs Of The Patient And The Likelihood Of Benefitting From Admission. This Prioritisation Defines Those Who Will Benefit Most From Icu (Priority 1) To Those Who Will Not Benefit At All (Priority 3).

1. Priority 1

A. Critically Ill, Unstable

B. Require Life Support For Organ Failure, Intensive Monitoring And Therapies That Cannot Be Provided Elsewhere. This Includes Invasive Ventilation, Renal Replacement Therapy, Invasive Haemodynamic Monitoring And Other Interventions

C. Do Not Have Limitations Of Treatment

D. High Likelihood Of Benefit

2. Priority 2

Priority 2a

A. Acutely Ill, Relatively Stable

B. Requires Intensive Monitoring And/Or Therapies For Organ Dysfunction, That Can Be Managed In An Intermediate Care Facility (High Dependency Unit Or Post Anaesthetic Care Unit)

C. Admit To Icu, If Early Management Fails To Prevent Deterioration Or There Is No Intermediate Care Facility In The Hospital

D. Examples Include:

I. Post-Operative Patients Who Require Close Monitoring

Ii. Respiratory Insufficiency On Intermittent Non-Invasive Ventilation

Priority 2B

A. Critically Ill, Unstable

B. Require Life Support For Organ Failure

C. With Significantly Lower Probability Of Recovery Because Of Advanced Underlying Disease

D. May Have Specific Limitations Of Care E.G. No Cardiopulmonary Resuscitation

E. Lower Likelihood Of Potential Benefit

F. Examples Include:

I. Metastatic Cancer In Septic Shock Secondary To Hospital Acquired Pneumonia But With Some Limitations Of Therapy E.G. No CPR

Ii. Decompensated Heart Failure With Deteriorating Functional Status And Multiple Hospital Admissions

3. Priority 3

A. Terminally Ill Or Moribund Patients With No Possibility Of Recovery

B. Not Appropriate For ICU Admission

C. May Benefit From Palliative Care Rather Than Intensive Care

D. Examples Include:

I. Severe Irreversible Brain Pathology Impairing Cognition And Consciousness Or In A Persistent Vegetative State

Ii. Metastatic Cancer Unresponsive To Chemotherapy And/Or Radiotherapy

Iii. End-Stage Cardiac, Respiratory Or Liver Disease With No Options For Transplant

Iv. Severe Disability With Poor Quality Of Life

V. Advanced Disease Of A Progressive Life-Limiting Condition E.G.

- Motor Neuron Disease With Rapid Decline In Physical Status,

- Severe Parkinson's Disease With Reduced Independence And Needs Assistance For Activities Of Daily Living

Vi. Poor Response To Current Treatment E.G.

- Bowel Leak Despite Multiple Laparotomies,
- Recurrent Soft Tissue Or Musculoskeletal Infections Despite Multiple Surgical Intervention,
- Chronic Medical Conditions That Fail To Respond To Treatment Such As SLE Or HIV

Vii. End-Stage Renal Disease With No Option Or Refusal For Renal Replacement Therapy

Viii. Those Who Have Explicitly Stated Their Wish Not To Receive Life-Support Therapy

Triage

Triage Is The Process Of Placing Patients At Their Most Appropriate Level Of Care. It Is Often Needed As The Number Of Potential ICU Patients Exceeds The Availability Of ICU Beds. Appropriate Triaging Allows Effective Bed Utilisation And Resource Management. Factors To Consider When Triaging Include:

A. Likelihood Of Benefit
B. Prognosis
C. Life Expectancy Due To Disease
D. Anticipated Quality Of Life

Discharge Policy

1. The Decision To Discharge A Patient Shall Be Made By The ICU Specialist.
2. Prior To Discharge:
   A. The Primary Team Shall Be Informed Of The Management Plan Including Any Limitation Of Treatment.
   B. A Discharge Summary Shall Be Completed.
   C. Any Limitation Of Treatment Shall Be Clearly Documented Including Why And Amongst Whom These Decisions Are Made.
   D. Family Shall Be Informed.
   E. It Is The Responsibility Of The Primary Team To Receive And Review Patients Promptly In The Ward.
   F. Patients Who Require A Higher Level Of Nursing Care May Benefit From Admission To A Step Down Unit, If Available.

ICU Discharge Criteria

In Order To Maximise The Efficient Use Of ICU Resources, Patients Should Be Assessed Continuously To Identify Those Who May No Longer Need ICU Care. This Includes Patient With:

A. Stable Physiological Status And No Longer Needing ICU Monitoring And Treatment.
B. Stable Haemodynamic Parameters On Low Dose Inotropic Support.
C. Stable Respiratory Status With Oxygen Requirement Not More Than 60%.
D. Neurological Stability.
E. Tracheostomy, Not Requiring Frequent Suctioning.
F. Chronic Mechanical Ventilation (E.G. Motor Neuron Disease, Cervical Spine Injury) And The Acute Critical Problem Is Resolved.
G. Deteriorating Or Irreversible Physiological Status Where Active Interventions Are No Longer Beneficial. Withdrawal Of Therapy Should Be Initiated, However, Patient May Be Discharged To The Ward If ICU Bed Is Required.

Patient care

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multidisciplinary approach is essential for optimal patient care , This includes close collaboration between intensivists, other medical specialities (surgeons, physicians, microbiologists and radiologists), nursing staff, physiotherapists, dieticians, medical technologists and radiographers.

Asepsis with routine hand washing and the use of alcohol handrubs before and after working with each patient, is essential to minimise the spread of infection and cross contamination in the ICU.

Part of global patient care includes:

Tracheal suction
Washing
Mobilisation
Turning and skin pressure care to prevent bedsores
Eye, mouth and bowel care
Psychological support

Following on basic nursing care is basic ICU care, *summarised by the mnemonic* ***FASTHUG :***

F- FEEDING …. During a period of total starvation, the glycogen stores will be depleted within 24 -48 hr. In these situations energy must be obtained utilising triglycerides from fat stores and amino acids derived from protein. In critically ill patients, these mechanisms are impaired. This together with an increased metabolic rate, e.g. 30 - 50 % increase with sepsis; results in a negative nitrogen balance. There is an increased risk of complications in patients with protein /energy malnutrition , This is presumed to be associated with an impaired immune response. ***Nutritional support decreases incidence of complications such as sepsis and wound breakdown.***

Methods of feeding 1) Enteral feeding (preferable)

Usually via nasogastric or nasojejunal tubes, but occasionally via a gastrostomy or jejunostomy

2) Total parenteral nutrition (TPN)

when EF is contra-indicated or fails due to gastric stasis, intractable diarrhoea, malabsorption or where it is impossible to provide sufficient energy via the EF . TPN is administered via a central line, and strict asepsis is observed. Risks of TPN – Infection, septicaemia and hyperbilirubinaemia.

COMPLICATIONS OF FEEDING

ENTERAL FEEDING

Blockage of tube ,Patient discomfort , Skin and mucosal damage
cholestasis , Inadequate feeding due to interruptions , Aspiration of feed into lung causing pneumonia Displacement of tube: feeding into lung , Intolerance – diarrhoea, vomiting , Gastroparesis: optimize absorption with prokinetics (e.g. metoclopramide, erythromycin)

PARENTERAL FEEDING

Complications related to central venous catheter insertion (bleeding, pneumothorax, vessel damage) Catheter-related bloodstream infection Deranged liver function tests, Gut atrophy if no enteral feed given , Overfeeding (uncommon) hyperglycaemia, hyperuricaemia , increased triglycerides, fatty liver .

A – ANALGESIA

Inadequate analgesia can seriously inhibit the patient's ability to cough and expand the lungs. A multi-modal approach is used combining different methods and routes of administering analgesics. Options include: Simple analgesics, e.g. paracetamol; NSAIDS, and opiates (often morphine).Regional anaesthetic techniques such as epidural infusions provide excellent analgesia.

S- SEDATION

Sedation is often used to facilitate mechanical ventilation, decrease O2 demand and impose day night cycles. Sedation decreases O2 consumption significantly and thereby favourably influences the O2 supply : Demand relationship. Agents used in ICU include: Benzodiazepines - Midazolam / diazepam / lorazepam as IV/PO bolus or midazolam infusion , Propofol infusion. Providing adequate sedation and analgesia are essential to minimize anxiety and pain, and to enable tolerance of essential ,interventions such as mechanical ventilation, physiotherapy and therapeutic procedures, Both under- and oversedation result in worse patient outcomes

Consequences of inadequate sedation :

Agitation , Accidental extubation, removal of lines and tubes , Lack of synchronization with mechanical Ventilation Hypertension; tachycardia Myocardial ischaemia , Increased oxygen demands

Consequences of excessive sedation :

Hypotension and increased vasopressor use ,Delayed recovery of consciousness, delayed weaning from mechanical ventilation ,Ventilator associated pneumonia ,Prolonged intensive care stay Immunosuppression .

T- THROMBOPROPHYLAXIS

Critically ill patients are at high risk for the development of deep vein thromboses (DVTS) and venous thromboembolism. Unless contra-indicated, ALL patients routinely receive pharmacological prophylaxis in the form of unfractionated heparin or low molecular weight heparin subcutaneously , Dose : Unfractionated heparin 5 000 iu SC 3 x dly. If these drugs are contraindicated, mechanical devices such as TED stockings and calf compression devices are used.

H- HEAD UP

Nursing patients at least 15 degrees head up, decreases aspiration of feeds due to passive regurgitation in intubated patients. This also decreases the incidence of ventilator associated pneumonia (VAP)

U- ULCER PROPHYLAXIS

sucrulfate 1 g 6-hrly is administered via the nasogastric tube. Patients at higher risk of gastric bleeding should receive an H2 receptor antagonist and / or a proton pump inhibitor.

G- GLUCOSE CONTROL

Maintaining blood glucose within normal limits (4 -8 mmol l') decreases septic and cardiac complications, and improves wound healing. To limit hypo- or hyper- glycaemic episodes in ICU, blood glucose is measured 2-hourly. Most patients will require an insulin infusion to control their blood glucose, as hyperglycaemia is common due to the stress response in critical illness.

ORGAN SYSTEM SUPPORT :

Critically ill patients often have multiple organ system dysfunction or failure. Each organ system requires individual support. The most common organ system failures are the respiratory, cardiovascular, renal and coagulation systems. In terms of the central nervous system (CNS), even in patients with no specific intracranial pathology, neurological functioning will be impaired in critical illness and normally resolves once the patient improves. Psychological support of both the patient and family members during the ICU stay is crucial, and should not be neglected. Patients who could be considered for organ donation are supported in the ICU until organ harvesting occurs, with permission from the family.

A)Respiratory support

All critically ill patients should require additional **inspired oxygen**. Many will need ventilatory support. This is one of the main reasons patients are admitted to ICU.

Oxygen is a colorless, odorless, tasteless gas that is essential for the body to function properly and to survive ,**Oxygen therapy** is the administration of oxygen at a concentration of pressure greater than that found in the environmental atmosphere The air that we breath contain approximately 21% oxygen , Oxygen therapy is a key treatment in respiratory care. **Purpose ,** The body is constantly taking in oxygen and releasing carbon dioxide , If this process is inadequate, oxygen levels in the blood decrease, and the patient may need supplemental oxygen. The purpose is to increase oxygen saturation in tissues where the saturation levels are too low due to illness or injury.

INDICATIONS: Acute Respiratory Failure , Acute Myocardial Infarction , Cardiac Failure Shock , Hypermetabolic State Induced By Trauma,Burns Or Sepsis , Anaemia Cyanide Poisoning , During CPR During Anaesthesia For Surgery.

OXYGEN – WHEN A PRESCRIBED , MUST BE WRITTEN & DATED LEGIBLY BY THE DOCTOR WITH THE (DURATION , CONC. & FLOW RATE) OF O2 THERAPY .

Usually The Sources Of Oxygen (Oxygen Cylinder & Oxygen Wall Outlet), The oxygen cylinder is delivered with a protective cap to prevent accidental force against the cylinder outlet , Oxygen is moistened by passing it through a humidifier to prevent the mucous membranes of the respiratory tree from becoming dry.

Hypoxia: low level of oxygen at tissue level

Hypoxemia: low levels of oxygen in blood

What are the signs that a person needs oxygen?

Shortness of breath , Headache , Confusion restlessness and dizziness , Rapid breathing , Chest pain , High blood pressure

Oxygen delivery systems : The oxygen delivery devices are grouped into two:

Low flow oxygen delivery system & **High** flow oxygen delivery system

LFODS ... In Which (FiO2) will be based on the patient's anatomic reservoir and minute ventilation. They are: Nasal Cannula , Simple Mask , Partial Rebreather ,Nonrebreather

HFODS .. deliver a prescribed gas mixture (either high or low) at flow rates that exceed patient demand , **Venturi Mask , Aerosol masks**, **tracheostomy collars**, , and **face tents** can be used with high-flow supplemental oxygen systems but not all aerosol generators can deliver high oxygen concentration at the needed flows rate

Nasal cannula (prongs): It is a disposable, plastic devise with two protruding prongs for insertion into the nostrils, connected to an oxygen source, Used for low-medium concentrations of Oxygen (24-44%), Amount Delivered Fio2 (Fraction Inspired Oxygen) Low flow- 24-44

Advantages: …. patient able to talk and eat with oxygen in place , Easily used in home setting , Safe and simple , Easily tolerated , Delivers low concentrations.

Disadvantages: .... Unable to use with nasal obstruction , Drying to mucous membranes, so flow greater than 4 L/min needs to be humidified , Can dislodge from nares easily , Causes skin irritation or breakdown over ears or at nares, Not good for mouth breathers

Face Mask: The Simple Oxygen Mask , The Partial Re-Breather Mask, The Non Re- Breather Mask **&** The Venturi Mask

The simple Oxygen mask Simple mask is made of clear, flexible, plastic or rubber that can be molded to fit the face, It is held to the head with elastic bands, Some have a metal clip that can be bent over the bridge of the nose for a comfortable fit, The simple Oxygen mask delivers 35% to 60% oxygen. A flow rate of 6 to 10 liters per minute. It has vents on its sides which allow room air to leak in at many places, thereby diluting the source oxygen. Often it is used when an increased delivery of oxygen is needed for short periods (i.e., less than 12 hours).

Advantages: Can provide increased delivery of oxygen for short period of time

Disadvantages: Difficult to keep mask in position over nose and mouth , Potential for skin breakdown (pressure, moisture, Uncomfortable for patient t while eating or talking

The Partial Re Breather Mask: The mask is with a reservoir bag that must remain inflated during both inspiration & expiration, It collects part of the patients' exhaled air , It is used to deliver oxygen concentrations up to 80%, The oxygen flow rate must be maintained at a minimum of 6 L/min to ensure that the patient does not re-breathe large amounts of exhaled air. The remaining exhaled air exits through vents.

Advantages:

Patient can inhale room air through openings in mask if oxygen supply is briefly interrupted

Disadvantages:

Requires tight seal (eating and talking difficult, uncomfortable)

The non rebreather mask: This mask provides the highest concentration of oxygen (95-100%) at a flow rate 6-15L/min. It is similar to the partial rebreather mask except two: one-way valves prevent conservation of exhaled air & The bag is an oxygen reservoir

Advantages: Delivers the highest possible oxygen concentration , Suitable for patient breathing spontaneously with sever hypoxemia

Disadvantages: Impractical for long term Therapy , Malfunction can cause CO2 buildup Suffocation , Expensive and uncomfortable , Venturi Mask , It is high flow oxygen delivery device. Oxygen from 40 - 50% at liters flow of 4 to 15 L/min , The mask is constructed so that there is a constant flow of room air blended with a fixed concentration of oxygen

Oxygen toxicity: is lung damage that happens from breathing in too much extra (supplemental) oxygen. It's also called oxygen poisoning. It can cause coughing and trouble breathing. In severe cases it can even cause death , Risks of Oxygen Therapy (O2 toxicity) Determining factors of O2 toxicity … PO2 & Time of exposure i.e., higher the PO2 & exposure time the greater the toxicity , CNS effects occur with Hyperbaric Pressures , High PO2 damages capillary endothelium Followed by interstitial edema & alveolar-capillary membrane thickening

Depression of ventilation: It is seen in COPD patients with chronic carbon dioxide (CO2) retention who have hypoxic respiratory drive to
Hyperbaric oxygen toxicity: Long term hyperbaric O2 therapy can lead to pulmonary, optic and central nervous system toxicity ,
Absorption atelectasis: Given only pure oxygen results in the collapse of the dependent part of the lungs as it quickly taken up from the alveoli. It is also a risk in general anaesthesia induction
Retinopathy of prematurity (ROP): It usually occurs in low birth weight, very premature infant.
Pulmonary toxicity: Patients exposed to high oxygen levels for a prolonged period of time have lung damage. The extent of lung damage is dependent on FiO2 and duration of exposure.
Symptoms of oxygen toxicity .... Coughing , Mild throat irritation , Chest pain ,Trouble breathing , Muscle twitching in face and hands ,Blurred vision ,Nausea.
A drop in oxygen saturation to less than 86% to 90% during activity indicates that the patient needs supplemental oxygen
What are the effects of oxygen toxicity? Central nervous system oxygen toxicity can cause seizures; brief periods of rigidity followed by convulsions and unconsciousness. Pulmonary oxygen toxicity results in damage to the lungs, causing pain and difficulty in breathing.
At what level does oxygen become toxic? Oxygen is toxic to the lungs when high FIO2 (>0.60) is administered over extended exposure time (≥24 hours) at normal barometric pressure (1 atmospheres absolute (ATA)). This type of exposure is referred to as low pressure O2 poisoning, pulmonary toxicity, or the Lorraine Smith effect.
What are examples of oxygen toxicity? Oxygen toxicity occurs in three major forms: neurologic, pulmonary, and ocular. Central nervous system oxygen toxicity is the most common manifestation of oxygen toxicity and manifests itself as a generalized tonic-clonic seizure ("grand mal" type).

Method of delivery	FiO2 achieved	Type of patient
▪ Nasal cannula (1–2 L/min)	0.24–0.30	Stable patients
▪ Venturi mask	0.24–0.50	Type II respiratory failure and COPD
▪ Partial rebreathing mask	0.60 to 0.80	Acute Type I respiratory failure, e.g pneumonia, asthma and acute pulmonary oedema
▪ Non-rebreathing reservoir Mask	Up to 0.90	Severely hypoxaemic patients
▪ Tight-fitting mask or helmet (e.g. CaStar hood) used for NIV	Up to 1.0	Severely hypoxaemic patients
▪ Anaesthetic Face Mask Or Endotracheal Tube	Up to 1.0	Patients requiring intubation

Contraindications to NIV (adapted from the British Thoracic Society NIV guidelines)
· facial burns/trauma/recent facial or upper airway surgery · vomiting · fixed upper airway obstruction · undrained pneumothorax · upper gastrointestinal surgery · inability to protect the airway · copious respiratory secretions · life-threatening hypoxaemia
· haemodynamically unstable requiring inotropes/pressors (unless in a critical care unit)
· severe comorbidity · confusion/agitation · bowel obstruction

Categories of patients requiring ventilation

Acute ventilatory insufficiency (rising Paco, and respiratory acidosis - pH < 7,2)
Oxygenation failure (Pao, < 11 kPa on F,0, 2 0,4)
Raised intracranial pressure (ICP) and cerebral ischaemia (1 O2 supply & Ị demand)
Reduce the work of breathing (decrease O2 demand in cardiac failure or pulmonary oedema)

Clinical indicators for ventilation ..... Respiratory rate (RR) > 30 breaths min" , Exhaustion , Vital capacity (VC) < 10- 15 ml , Confusion , P202 < 8 kPa on room air or < 11 kPa if FO2 2 0,4 , Severe shock High Paco, with respiratory acidosis (pH < 7,2)

Types of ventilatory support Intermittent positive pressure ventilation (IPPV) is divided broadly into volume control and pressure control ventilation. The most common means of ventilation used in adults is volume control. One mode of volume control is intermittent mandatory ventilation (IMV), where a set number of fixed

volume breaths are delivered. SIMV mode is where the mandatory breaths from the ventilator are synchronised with patients' own breaths to prevent "stacking" and hyperinflating the lungs.

Basic ventilator settings Tidal Volume (VT) 6-10 ml kg , Respiratory Rate 10 - 15 breaths min , PEEP 3-5 hPa (cm“H,O") T:E ratio (Inspired : Expired time) 1:2 to 1:3- Preferably set inspired time in seconds, e.g. 1,5 s Fi02 0,4 - 0,6 (40 - 60 %) depending on degree of hypoxia & on disease process, 100% causes oxygen toxicity and atelectasis in less than 24 hours , 40% of O2 is safe , Goal is to achieve a PaO2 >60 Obstructive disease requires longer expirations &Restrictive disease requires longer inspirations .

Ventilator Complications

1- Pulmonary barotraumas , pneumothorax , Avoid high-pressure settings for high-risk patients (COPD). 2-Mechanical malfunction: Keep all alarms activated at all times .If malfunction occurs, disconnect ventilator and ventilate manually
3-Airway malfunction: Suction patient as needed, Auscultate chest , End tidal CO2 monitoring
4-Hemodynamic alterations; Decreased cardiac output, decreased venous return, Observe for :
□Decreased BP □Restlessness, □Decreased urine output □Decreased peripheral pulses
□Slow capillary refill □Pallor □Increasing Tachycardia
5-other complication include : Renal malfunction , Stress ulcer and gastric hemorrhage ,Pulmonary atelectasis , Infection ,Oxygen toxicity & Loss of respiratory muscle tone

Causes of Ventilator Alarms:

A- High-Pressure Alarm

Increased secretions in the airway, Bronchospasm causes decreased airway size , The endotracheal tube is displaced , The ventilator tube is obstructed because of water or a kink in the tubing. ,Patient coughs, or bites on the oral endotracheal tube , patient is anxious or fights .

B- Low-Pressure Alarm ... Disconnection or leak in the ventilator or in the airway cuff , The patient stops spontaneous breathing

Basic Ventilatory modes

Based on the types of respiratory cycles that are offered to the patient, three basic Ventilatory modes can be considered. These are: (A/C) V , (PS) V , (SIM) V

1- A/C Modes: (Assist/Control ventilation) :

The A/C mode is characterized by offering controlled and/or assisted cycles, depending on the settings programmed , There are 2 type of A/C mode : A/C-VCV and A/C-PCV modes..

The A/C-VCV mode: …. is normally chosen immediately after tracheal intubation when the patient is sedated or under a neuromuscular blocker. Acute Respiratory Distress Syndrome (ARDS), can be done more easily and safely in A/C-VCV mode. Special attention to airway pressure variation

The A/C-PCV mode: … Can also be an excellent option for predominantly assisted ventilation when the patient exhibits respiratory muscle effort, for example, during a transition phase in the process of weaning from mechanical ventilation. Special attention, however, should be given to monitoring the VT in this Ventilatory mode. The alarm for VT minimum and maximum should be carefully adjusted.

2- PSV Mode (pressure support ventilation)

For patients that exhibit good recovery from an underlying disease and sedation is reversed, the PSV mode is commonly employed, where there are only triggers by the patient. In this example all cycles are assisted with the addition of pressure support, The PSV mode is typically used for weaning, where Pressure Support is reduced gradually while evaluating the patient's ability to breath.

3- SIMV with PS Mode (Synchronized Intermittent Mandatory Ventilation)

The SIMV mode with Pressure Support (PS) is a hybrid of A/C and PSV modes. This mode allow for spontaneous cycles, only with PEEP, interspersed with cycles of VCV or PCV. Currently, PS is routinely used in this mode. A minimum RR is programmed with cycles in VCV or PCV. For example, setting a RR of 6, the ventilator divides a minute into 6 time windows of 10 seconds each. Within each of these windows, the ventilator should offer a respiratory cycle that may be assisted if the patient makes an effort, or controlled at the end of the time window if the patient does not trigger the ventilator.

Positive End-Expiratory Pressure (PEEP)

The application of positive pressure during expiration as an adjunct to a mechanical breath Allows underventilated lung units to expand and increase the functional respiratory capacity (FRC) , PEEP improves pulmonary compliance, ventilation perfusion abnormalities, and oxygenation(by enhancing gas exchange and preventing atelectasis), Higher amounts of PEEP (more than 15) increase the chance of complications, such as barotrauma and tension pneumothorax .

Continuous Positive Airway Pressure (CPAP)

positive pressure applied during spontaneous ventilation to the expiratory side of the breathing circuit. Usually applied via a tight-fitting facemask. This will require the patient to be alert and co-operative. To prevent aspiration, airway reflexes should be intact , The baseline pressure in the upper airways is then set above zero (relative to atmospheric pressure). This will prevent alveolar collapse and atelectasis, and recruit collapsed alveoli , CPAP improves oxygenation and reduces work of breathing. It can be used in the ward, so the patient does not need an 1CU bed. A pressure of 5- 10 hPa (cm“H,O") is used.

Weaning From Mechanical Ventilation

Ease of weaning is inversely related to duration of mechanical ventilation. Before attempting to wean, ensure: Disease that necessitated IPPV is reversed or under control , Patient is awake with sufficient muscle strength to maintain airway and clear secretions with an effective cough or vital capacity (VC) > (15 ml kg) in adults , weaning is Process of going from ventilator dependence to spontaneous breathing & can be done by:

1. **SIMV …** The patient breathes between the preset breaths per minute rate of the ventilator ,The SIMV rate is decreased gradually until the patient is breathing on his or her own without the use of the ventilator.

2. T-piece … The patient is taken off the ventilator and the ventilator is replaced with a T-piece or continuous positive airway pressure, which delivers humidified oxygen, The patient is taken off the ventilator for short periods initially and allowed to breathe spontaneously, Weaning progresses as the patient is able to tolerate progressively longer periods off the ventilator.

3. Pressure support …. Pressure support is a predetermined pressure set on the ventilator to assist the patient in respiratory effort , As weaning continues, the amount of pressure is decreased gradually. , With pressure support, pressure may be maintained while the preset breaths per minute of the ventilator gradually are decreased.

B) Cardiovascular and circulatory support

Multiple CVS disorders may precipitate an ICU admission: Refractory hypotension, hypertensive crises, dysrhythmias (brady- and tachy-), myocardial ischaemia /infarction and cardiac failure , Myocardial contractility may be impaired either as the primary abnormality (cardiogenic shock) or as a secondary phenomenon as in severe hypovolaemic or septic shock, While instituting measures to improve myocardial contractility, one must also optimise preload and Afterload , Hypoxia, hypercarbia, hypovolaemia, severe acidosis and metabolic abnormalities, e.g. hypocalcaemia. Severe metabolic acidosis depresses myocardial contractility, and can also limit the response to vasopressor and inotropic agents ,. Rational selection of the appropriate inotrope requires thorough understanding of the cardiovascular effects of the available drugs along with accurate assessment of the individual's haemodynamic disturbance. This requires invasive blood pressure monitoring and central venous cannulation, as inotropes should be administered via a large central vein , Cardiovascular support is required not only for hypotension or shock but also to prevent complications in patients at risk of organ failure

Types of shock

- **Cardiogenic shock** : caused by 'pump failure', for example acute myocardial infarction
- **Obstructive shock:** caused by mechanical impediment to forward flow, for example, pulmonary embolus, cardiac tamponade, tension pneumothorax
- **Hypovolaemic shock:** caused by loss of circulating volume. These losses may be exogenous (haemorrhage, burns) or endogenous (through leaks in the microcirculation or into body cavities as occurs in intestinal obstruction)
- **Distributive shock:** caused by abnormalities of the peripheral circulation, for **example, sepsis** and anaphylaxis

Severe sepsis is predominantly caused by bacteria, but fungi and viruses are also occasionally implicated. Themost common sources of severe sepsis include:

· pneumonia (community or hospital acquired)

· intra-abdominal infection, for example appendicitis, bowel perforation and peritonitis, bowel infarction, abscess or cholangitis

· urinary tract infection, for example pyelonephritis
· skin and soft tissue infection
· catheter-related blood stream infection, for example vascular access device
· central nervous system infection, for example meningitis
· endocarditis.

Many cases of severe sepsis are community acquired, Hospital-acquired infections are more frequently associated with resistant organisms,

INOTROPES IN CURRENT USE

1. Adrenaline (Ino-constrictor) **……**Stimulates a and B receptors ,
B effect predominate at low doses a mediated vasoconstriction at higher doses

2. Noradrenaline (Ino-constrictor) **…** Predominantly a agonist

Useful in severe hypotension with a low peripheral vascular resistance, e.g. septic shock .

3. Dopamine (Ino-constrictor) **….** B stimulant with minimal tachycardia

Low dose - Decreases peripheral vascular resistance which results in a fall in splanchnic and renal vascular tone and improved splanchnic perfusion High dose - Potent vasoconstrictor with the potential for peripheral tissue loss with prolonged infusions This is an outdated inotrope traditionally used for renal protection, which has since been disproved to have any value in this regard

4. Dobutamine (Ino-dilator) **…..** B mediated increase in cardiac output

Ideal for primary cardiac causes of cardiac failure Decreases systemic vascular resistance

5. Milrinone (Ino-dilator) **…..** A phosphodiesterase 3 inhibitor that dilates the pulmonary vasculature and useful in patients with pulmonary hypertension It is only available in specialist centres

Vasodilator therapy In selected cases, afterload reduction may be used to increase stroke volume and to decrease myocardial oxygen requirements. Vasodilator therapy is most beneficial in patients with cardiac failure. Vasodilators can be used to control angina and decrease ischaemia following myocardial infarction. This type of therapy is potentially dangerous. Vasodilatation should be achieved cautiously with continuous haemodynamic monitoring. Nitroglycerine infusions are used most frequently. Available drugs: Direct acting vasodilators – Hydralazine, sodium nitroprusside (SNP), nitroglycerine (GTN) Alpha blockers - Phenoxybenzamine, phentolamine .

C) Renal support Acute kidney injury (AKI) is the new nomenclature for acute renal failure. This is a common problem in the ICU - Incidence 10- 25 %., **Aetiology in ICU : Include ..**

Pre Renal **…..** Hypovolaemia , Hypotension & Poor cardiac output .

Intrinsic …. Ischaemia , Nephrotoxins especially radiological contrast agents

Sepsis frequently underlies these causes acting through hypotension, inflammatory mediators or direct toxins. Presence of co-morbidities or pre- existing renal dysfunction significantly increases the risk of developing AKI. Renal replacement therapy should be started early if there is a definite indication as this may reduce mortality.

Renal replacement therapy (dialysis) :

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Indications

- ❑ K* >7 mmol l' (unresponsive to standard therapy)
- ❑ Acidaemia pH <7,2 (unresponsive to standard therapy)
- ❑ Fluid overload not responsive to diuretics
- ❑ Uraemic complications (serum urea usually > 35 mmol l')
  ((Uraemic encephalopathy , Uraemic pericarditis , Uraemic neuropathy/myopathy))
- ❑ Oliguria (urine output < 200 ml 12-hourly or < 0,5 ml kg' hr")
- ❑ Hyperthermia
- ❑ Overdose of drugs, e.g. aspirin, lithium, methanol, ethylene glycol

Methods used in ICU ... 1. Intermittent Haemodialysis **2.** Continuous Haemodialysis .

Patient Monitoring

Introduction :

Monitor is a Latin word "monere" which means "to warn" , monitoring is the observation of one or several medical parameters over time. It can be performed by continuously measuring certain parameters directly or by using a medical monitor.

Aim of Patient Monitoring The aim of patient monitoring is to give warning of early dangerous deterioration, so early treatment is given and complications are avoided.

Any monitor consists of following main parts:

Sensor , System for data collection and translation, Display system In addition to System for interpretation , Recording system , Alarm system , Wireless communication links

Invasiveness of monitoring devices

1.Non invasive e,g, ECG monitor , pulse oximeter
2.Invasive e,g, arterial line, centeral venous line
3.Highly invasive intracranial pressure monitoring.

Types of monitors according to parameters measured

1.Single parameter monitors
2.Multi parameters monitors

How to select monitor? ..... Depend on the following factors:

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

Main physiological parameters to be monitored in the ICU :

1- ECG
2- Respiration e.g. O2 saturation
3- blood pressure
4- temperature.

Limitation of monitoring :

•Delay. •Danger. •Decreased skill. •Doubt of results. •Distracting set up.

E.C.G monitors attached to patient

Is essential in the diagnosis of chest pain and abnormal heart rhythms .
Electrocardiograph – is the instrument that records the electrical activity of the heart .
It works on the principle of Galvanometer .

ECG MONITORING SYSTEMS

1.Three electrode monitoring system
2.Five electrode monitoring system
3.Ten electrode,twelve lead monitoring system.

ECG interpretation :step-by-step

•Rate •Rhythm •P – wave •PR - interval •QRS Complex •ST Segment •T wave

•Other ECG signs Rate lead II - rhythm strip. Look at number of large(squares) between 2 R waves This applied if the rhythm is **regular**
If **irregular** Count the number of R waves in a 6-second strip and multiply by 10. Not very accurate, used for a quick estimate.
RHYTHM 1.ECG rhythm -usual rate between 60-100 bpm,
2. Every P wave must be followed by a QRS & every QRS is preceded by P wave
3.P wave is upright in leads I and II
P Wave Depolarization of both atria , Relationship b/w P & QRS - distinguish various arrhythmias Shape & duration of P - indicate atrial disease .
PR INTERVAL Onset of P wave to onset of QRS
• Normal = 0.12 - 2.0 sec
• Represents Atria to Ventricles conduction time Prolonged PR interval indicate AV block
QRS COMPLEX Ventricular depolarization
•Normal duration = 0.08 - 0.12 sec
•Abnormality Indicate ventricular disease
ST Segment Connects QRS complex , T wave & should be on the iso - electrical line
T Wave "small to moderate" size +ve deflection wave after QRS complexIt , It is 1/3rd - 2/3rd that of corresponding R wave

A. Pulse Oximeter :

- measures SaO2 by red and infrared light absorption by Hb; oxygenated and deoxygenated Hb have different absorption characteristics
- non-invasive
- can show pulse waveforms on suitably equipped monitors
- if ventilation is accidentally terminated, the SaO2 may remain normal for several minutes in a well oxygenated patient due to the high partial pressure of O2 remaining in the lungs .

The Hb –Oxygen (Hb-O) Dissociation Curve

Pulse oximetry may be in accurate with :

- ✓ low perfusion : …… (lowers)
  (eg, low cardiac output, hypothermia, increased systemic vascular resistance ” pressors “ atherosclerosis) .
- ✓ venous pulsations in a dependent limb:… (lowers)
- ✓ Movement (motion) : …. (Raises)… malpositioned sensor
- ✓ Nail polish : …(lowers)
- ✓ methylene blue dye :
- ✓ Co ‘Carboxy Hb” : (Raises) ……CO exposure
- ✓ Methemoglobinemia…..

causes **a falsely low** saturation reading when SaO2 is actually greater than 85% and a falsely high reading if SaO2 is actually less than 85%.

- ✓ Hemoglobinopathy : ….(lowers)
- ✓ Anemis ‘sever ‘ : ….(Raises)
- ✓ Acidosis : (lowers)
- ✓ Alkalosis :…. (lowers)
- ✓ Cardiopulmonary bypass:… (lowers)
- ✓ excessive ambient light from the light-emitting diode to the photodiode
- ✓ bypassing the arterial bed (optical shunting).

❖ Oxygenation (PaO2 , O2 saturation). The PaO2 is the amount of oxygen dissolved in the blood and therefore provides initial information on the efficiency of oxygenation.

❖ Ventilation (PaCO2). The adequacy of ventilation is inversely proportional to the PaCO2, so that when ventilation increases, PaCO2 decreases, and when ventilation decreases, PaCO2 increases.

Pulse Oximetry is a non-invasive method of measuring pulse & oxygen saturation of hemoglobin (SpO2 rate on a continuous basis to detect hypoxia .) , The general principle is based on **Beer-Lambert Law** by measuring the transmission of the red (600-750 nm wavelength) and infrared (850-1000 nm) lights through the pulsatile tissue beds, , The variation in the absorbability of light during pulsatile flow as compared to baseline is displayed as arterial waveform of plethysmography,

End - Tidal Co2 (Etco2)

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(Etco2) Monitoring Is A Noninvasive Technique Which continuously Measures The Partial Pressure Or Maximal Concentration Of instantaneous Carbon Dioxide (CO2) at the end of an exhaled breath, which is expressed as a percentage of co2 or mmhg , the normal values are 5% to 6% co2, which is equivalent to 35-45 mmhg, co2 reflects cardiac output (co) and pulmonary blood flow as the gas is transported by the venous system to the right side of the heart and then pumped to the lungs by the right ventricles, when co2 diffuses out of the lungs into the exhaled air, a device called **Capnometer** measures the partial pressure or maximal concentration of co2 at the end of exhalation.

A capnogram can be considered as two segments, an inspiratory segment and an expiratory segment, and two angles, an alpha and a beta angle, Capnograms predominantly identify respiratory abnormalities. It also can forewarn major impending depression of cardiovascular system, such as hypotension associated with massive blood loss, impending circulatory arrest, or pulmonary embolism, This CO2 waveform (CO2 concentration as a function of time or volume) is also called **a capnogram,** and a device that generates the CO2 waveform is called a **capnograph**, () to measure CO2 concenteration in expired gases.) Use of capnography monitoring can reliably and quantitatively provide vital respiratory information in intubated patients. Alterations in cardiac output, distribution of pulmonary blood flow, and metabolic activity can also be reflected by the change of CO2 .

The basic elements of an infrared spectrometry include an infrared light source, a sample chamber, and an IR detector. The absorption of the IR light is also governed by the Beer-Lambert law, that is, the absorption is proportional to the concentration of the absorbing gas in the sample chamber (It is based on the fact that CO2 polyatomic gas that absorbs infrared (IR) radiation of a specific wavelength).

Indications for End Tidal CO2

used in the **hospital setting with critically ill patients** and in patients receiving **general anesthesia** , but has become a standard assessment during pediatric **sleep studies** and during adult sleep studies with special populations. In 2015, Capnography included in the fee schedule for **pediatric polysomnography** , used with a more complex population of **sleep disordered** patients, CO2 monitoring is recommended for the following patient populations where **hypoventilation** can have adverse effects on development, comorbid disease and mortality: pediatric ,neuromuscular disease; respiratory failure; hypoventilation.

Types of capnographs

Depending upon the **location of the CO2 sensor** , capnographs can be divided into two types : **Sidestream (Aspiration) And Mainstream (Flow-Through)** capnographs, In **sidestream capnograph**, the gas sample is continuously aspirated by a fine tube from the breathing circuit (usually located at the T-piece), A unique advantage of sidestream capnograph is that it allows monitoring of spontaneously breathing non-intubated patients, as sampling of the expired gas can be obtained from face mask or nasal cannula, In mainstream capnograph, a cuvette containing a CO2 sensor with IR source and detector is inserted between the breathing circuit and the endotracheal tube, and therefore, the CO2 measurement takes place within concentration in expired gases , Comparing with the sidestream capnography, the **advantages of the mainstream capnography include the faster response time, no gas being subtracted from the breathing circuit, no need for sampling pumps.** The ***disadvantages*** include the relatively heavy measuring adapter with electrical cord (causing endotracheal tube kink), added dead space, and the expense for the repair of damage, Needless to say that the newer generation of main stream sensors are remarkably lighter and smaller. Calibration Capnographs must be calibrated periodically, usually at least daily. Modern instruments use self calibration, They are fairly accurate even in the extended range up to 100 mmHg, which is useful in rare cases of hypoventilation or malignant hyperthermia , Normal capnogram 10 .

Sidestream (Aspiration) … **CO2 sensor external to breathing circuit , Gas is constantly aspirated from circuit via 6 feet sampling tube, Minimal dead space , Light weight adapter ,Sampling tube may clog ,Wave form is delayed (1-4 sec), Easily adopted to non intubated patients .** **Mainstream (Flow-Through) .. CO2 sensor between the circuit and ET tube , No gas removed , Increased mechanical space , Heavy sensor and might kink or disconnection of circuit , Sensor may damage , Waveform in real time ,Difficult to adapt in non intubated patients.**

A. Normal capnograph waveforms

Phase I—Inspiration; CO2 should be 0.

Phase II—Beginning of expiration; CO2 rises rapidly as dead space gas containing no CO2 is exhaled.

Phase III contains almost all alveolar gas, and, in a healthy individual, the plateau should be relatively flat.

Phase IV— Beginning of inhalation. The a angle, the angle between phase II and III, should be between 100 and 110 degrees; patients with obstructive lung disease will have an angle greater than 110 degrees. The b angle, the angle between phase III and IV, is less than 100 degrees but will increase if there is any rebreathing because the gas being inhaled will contain some of the previously exhaled CO2.

B. Normal capnogram tracing over time—The end-tidal CO2 should be relatively constant.

C. Capnogram of a patient with obstructive lung disease. Such patients require a longer expiratory time; the capnogram has a more rounded appearance during the initial phase of expiration, a more obtuse a angle, and an upward slope to the alveolar plateau.

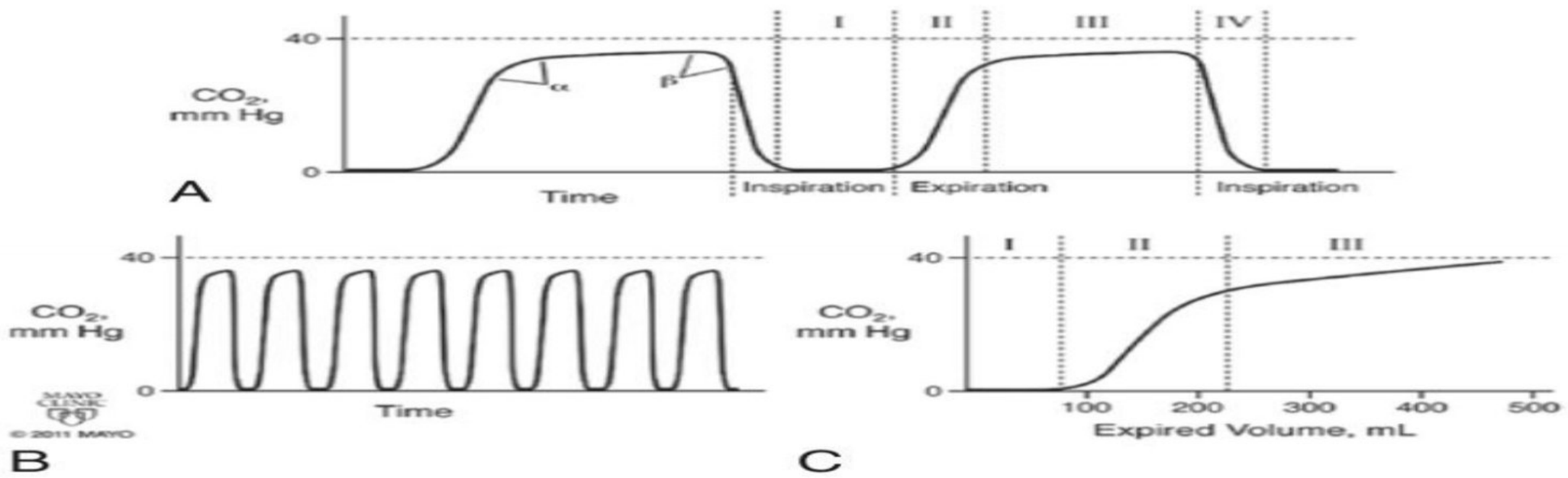


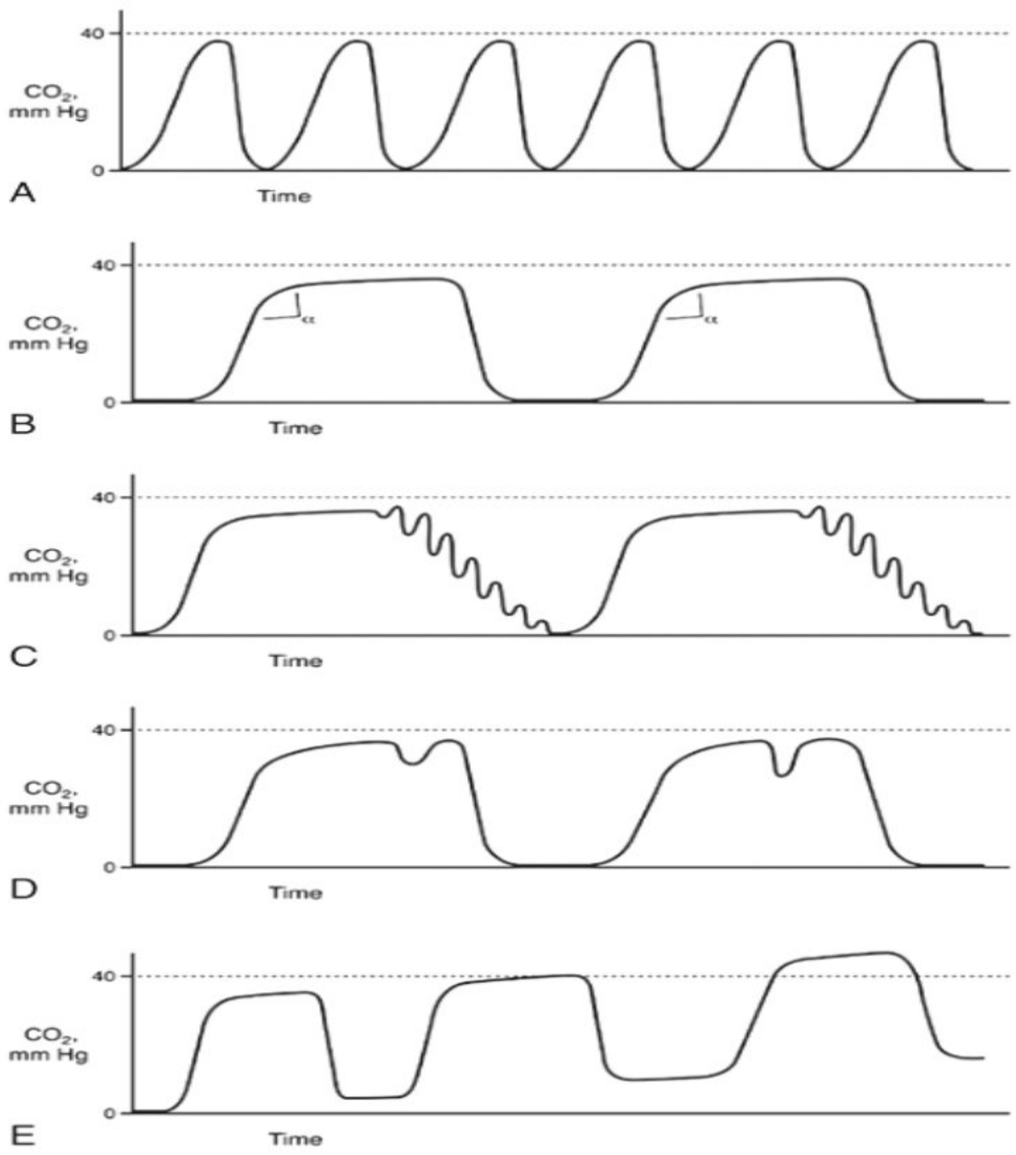
Table 9-1 Causes of Altered End-Tidal CO_2 during Anesthesia*

Cause	$P_{ET}CO_2$	$P_{ET}CO_2$-to-$PaCO_2$ Gradient
CO_2 insufflation	Increased	Normal
Increased CO_2 production†	Increased	Normal
Right-to-left shunt	Increased	Widened
Increased physiologic or anatomic dead space, or both	Decreased	Widened
Increased apparatus dead space	Increased	Normal
Hyperventilation	Decreased	Normal
Hypoventilation	Increased	Normal
Leak in sampling line	Decreased	Widened
Poor seal around tracheal tube	Decreased	Widened
High sampling rate	Decreased	Widened
Low sampling rate	Decreased	Widened
Rebreathing due to malfunctioning breathing valve	Increased	Decreased
Rebreathing with low fresh gas in the Mapleson system	Increased	Decreased
Rebreathing with circle system (absorbent problem)	Increased	Normal

*Normal pressure of end-tidal CO_2 ($P_{ET}CO_2$) is 38 mm Hg (5%). The $PaCO_2$-to-$P_{ET}CO_2$ gradient is normally <5 mm Hg.

†From hyperthermia, malignant hyperthermia, convulsions, pain, or bicarbonate administration.

Adapted, with permission, from Dorsch JA, Dorsch SE. Gas monitoring. In: Dorsch JA, Dorsch SE, eds. Understanding Anesthesia Equipment, 5th ed. Philadelphia: Lippincott, Williams & Wilkins; 2007:706-707.



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Examples of various capnograph waveforms and their interpretation.

A.Normal spontaneous breathing.

B. Normal mechanical ventilation.

C. Cardiogenic oscillations seen during the terminal portion of exhalation.

D. "Curare" cleft—seen normally in the last part of phase III caused by a lack of synchrony between diaphragm and intercostal muscles in a patient who has received NM -blocking agents and in whom muscle strength is returning.

E.Patient rebreathing CO2 due either to exhausted absorbent or incompetent inspiratory valve. The figure overdramatizes the increase in inspired CO2 that will occur over time if the underlying problem is not corrected.

F. Dual-plateau waveform because of a break in the sample line—early during exhalation, room air is entrained, lowering the CO2; toward the end of exhalation, as pressure in the tubing increases, less air is entrained—hence, a second "tail."

G. Patient with **severe obstructive lung disease** (forced expiratory volume at 1 second 20% or less of predicted.

H. Obstruction in the sampling line as less air is withdrawn.

I. Shift right (dotted line) to left (solid line) as would occur in a patient with obstructive lung disease whose disease worsened
(e.g., bronchospasm in a patient with chronic obstructive pulmonary disease).

Flat capnogram

When tracer does not find any CO, i.e., there is no CO, in exhaled air, (ETCO, is zero) capnography becomes flat.

1. Accidental extubation → Most common cause
2. Equipment failure
3. Disconnection (dissociation) of tube
4. Total occlusion of breating circuit (ET Tube etc.)
5. Mechanical ventilation failure
6. Severe Bronchospasm completely obstructing the airway
7. Total blokadge of pulmonary circulation due to large emboli.
8. Cardiac arrest.

CONDITIONS THAT AFFECT ET CO2

Decreased

o Hyperventilation

o Pulmonary embolism

o Hypoperfusion, hypotension, hypovalemia, shock

o Hypothermia

o Decreased skeletal muscle activity (relaxation)

o Hypometabolism

o Hypothyroidism

o Bronchospasm

Increased

o Hypoventilation

o Rebreathing

o Torniquet release

o Malignant hyperthermia41,MH II),

o Neuroleptic malignant syndromel-11)

o Increased skeletal muscle activity (shivering MIl-1)

o Hypermetabolism (41-11, MH-11)

o Hyperthyroidism (MH-I) & thyroid storm (Al-1I)

o Effective drug therapy for bronchospasm

o Sepsis

COMMONLY USED MONITORING DEVICES

A. Pulse Oximeter :
B. Capnometer :

C. ECG …….. Changes In Rate, Rhythm, ST Elevation/Depression .

D. BP cuff (manual/automatic): These instruments measure BP automatically at regular interval , The cuff size is very important & should cover two third of length of arm , Too large cuff will under-read and too small cuff will over-read the actual value

E.stethoscope (precordial, esophageal)

F. thermometer (surface or core)

G. peripheral nerve stimulators (when using neuromuscular blockade or blocking drugs)

- **deliver electrical stimulus to elicit muscle responses**
- **indicates degree of muscle relaxation**

H. machine function "monitors" : i.e. volume and pressure alarms and inspired O2 alarms **I. mass spectrometer/gas analyzer :** identifies and measures inhaled/exhaled gases

LESS FREQUENTLY USED MONITORS

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A. urinary catheter and urometer
B. central venous line

CVP is monitored most commonly in right internal jugular vein. It can also be measured in subclavian, basilica and femoral veins , Normal CVP is 3-10 cm of H,0 (2-8 mm Hg)
CVP is increased in fluid overload, CHF , Cardiac temponade, pulmonary embolism, IPPV with PEEP,pleural effusion, Hemothorax, Valsalva maneuver , coughing & straining ,
CVP is decreased in hypovolemia, shock, venodilators (nitroprusside) , spinal/epidural A, G.A

Complications : - Air embolism, Thromboembolism, Cardiac arrhythmias, pneumothorax, hemothorax , chylothorax, cardiac temponade, Sepsis , trauma to brachial plexus & cartoid artery (can cause pseudoaneurysm)

Uses (indications) for CVP monitoring

1) Estimation of intravascular volume and a guide to fluid therapy in patients with :-

a) Hemorrhage & shock b) Sepsis c) Trauma d) Cardiovascular dysfunction

2) Fluid resuscitation

3) Perioperative period

4) Blood transfusion Or plasma transfusion

5) Beside for monitoring, the same central venous line can be used for -

a) Parenteral nutrition b) Aspiration of air embolus c) Cardiac pacing

rapid fluid infusion, infusion of vasoactive drugs, measuring CVP

C. arterial line: invasive

continuous BP monitoring by putting a transducer in the artery, **easy access allowing for frequent ABGs , Blood samples :**
(Radial , Brachial , .Axillary , Dorsalis pedis((infants)) , **Femoral) . .Artery.**

Intra-Arterial Cannulation

o **Radial artery** at wrist is the most common site for insertion of an intravascular cannula, The advantages of this site are that the vessel is fairly superficial and easily palpable, it is of adequate diameter to accept standard-size catheter, and the area is easy to keep clean, Other arteries which can be used for cannulation are brachial, axillary, femoral and dorsal pedis arteries..

o Uses of arterial cannulation

1. Arterial blood sampling (e.g. ABG measurement)
2. Intra-arterial BP measurement
3. Drug injection
4. Expected large blood or fluid loss
5. To treat severe prolonged hypotension
6. Non pulsatile arterial flow (eg. cardiopulmonary bypass)
7. Blood transfusion

o Thrombosis of radial artery is the most common **complication** which can cause ischemic damage Aggressive management, e.g. brachial plexus block or stellate ganglion block may be required to avoid permanent damage. To prevent thrombosis a continuous flush with heparin is needed.

D. **Swan-Ganz catheter** is the insertion of a Catheter into pulmonary artery & is used for : -

1.Direct simultaneous measurement of pressure in right atrium, right ventricle, pulmonary artery

2. Indirect measurement of left atrial pressure through pulmonary capillary wedge pressure.

3. Cardiac output

4. Oxygen saturation of mixed venous blood , Pul. artery is considered best site for mixed venous blood sample.

5. Titration of fluid infusion.

6.core temperature

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E. ICP monitoring

F. EEG, brain and spinal cord evoked potentials

G. transcutaneous gas measurements

H. transesophageal echocardiography (TEE) :

Two dimensional TEE is the most sensitive method to detect myocardial ischemia and air embolism in peri-operative period. Detection of regional wall motion abnormality a rapid and more sensitive indicator of myocardial ischemia than is the ECG,Decreased systolic wall thickening may be more reliable index for ischemia than endocardial wall motion abnormality alone.

Blood gas analysis … Arterial oxygen is the better indicator of pulmonary function on the other hand mixed venous oxygen is the best indicator of cardiac out-put i.e., tissue oxygenation. For blood gas analysis, usually sample is talken from radial or femoral artery for arterial gas analysis and from right atrium or pulmonary artery for venous gas analysis , Blood gas analysis is particularly needed in thoracic surgeries, hypothermia and hypotensive anaesthesia.

Normal values

Arterial blood	Venous blood
pH=7.38-7.42	
pO,=96-98 mmHg	pO, =40 mm Hg
pCO, =35 - 45 mm Hg	pCO, = 46 mm Hg.
Oxygen saturation (spO,) 95 - 98%	

Defibrillators

Objective:

To provide basic understanding about the Defibrillator Machine , understand the concept of the Defibrillator applications & perform and identify basic problems, errors and basic troubleshooting solutions.

Introduction:

Cardiac arrest occurs in more than 500,000 people annually in the United States, Defibrillation is an important part of resuscitation that can change the outcome of this condition .

Defibrillation

is a process in which an electronic device sends an electric shock to the heart to stop an extremely rapid, irregular heartbeat, and restore the normal heart rhythm.

Defibrillator :

A device that corrects an abnormal heart rhythm by delivering electrical shocks to restore a normal heartbeat.

Types : **Manual & Automatic ((External,Internal ,Monophasic , Biphasic)) .**

CLASSIFICATION : …… **According to operation :**

1- Manual Defibrillator:

Clinical expertise is needed to interpret the heart rhythm and decide whether to charge the defibrillator and deliver the shock to patient. Energy selection and delivery is given to the patient manually.

2- Automated Defibrillator :

These defibrillators are small, safe, simple and lightweight with two pads that can be applied to the patient. The defibrillator guides the operator step-by-step through a programmed protocol. It records and analyses the rhythm and instructs the user to deliver the shock using clear voice prompts, reinforced by displayed messages.

According to site of application :

1- External Defibrillator

is the device which delivers the high energy shock to patients Heart externally on patient's chest by using a Defibrillator Paddle. The maximum energy deliver to the patient is about 360 Joules in Monophasic & 200 Joules in Biphasic Defibrillator.

2- Internal defibrillator

consist of sterilized internal Handle/Paddle through which shock is delivered directly to the heart.

3-Implantable Cardioverter Defibrillator (ICD)

If it detects an abnormally fast heart rhythm, it delivers a small electrical shock to the heart to convert the heart rhythm back to normal.

Joule : …. **It is the unit of energy delivered by the Defibrillator , It means - "The energy released in one second by a current of one ampere through a resistance of one ohm" , Also called as watt-second , The delivered energy is in the range of 50-360 joules and depends on: .. characteristics of patient , .. patient's disease , .. duration of arrhythmia , .. type of arrhythmia (more energy required for VF) , .. type of the machine used .**

Anatomy Heart

Located between the lungs in the middle of chest, behind and slightly to the left of breastbone (sternum) , Size of ones fist and shaped like a cone , An average heart pumps (70 milliliters) per heartbeat. An average heartbeat is 72 beats per minute , Therefore an average heart pumps 1.3 gallons (5 Liters) per minute.

Conductive system : …..Components of the Conduction System

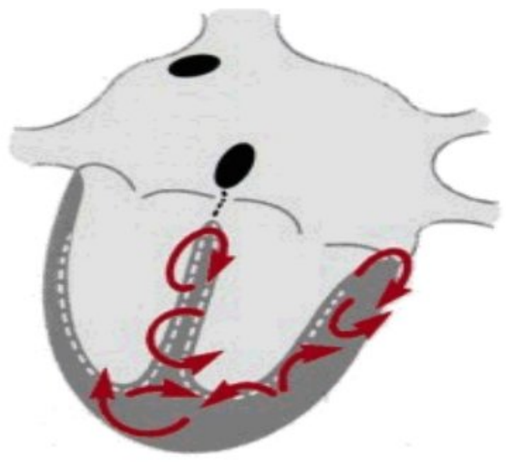
1-Sinoatrial Node … called the pacemaker of the heart **,** initiates impulses 70-80 times per minute without any nerve stimulation from brain **.** impulses move through atria causing the two atria to contract. **,** at the same time, impulses reach the second part of the conduction system

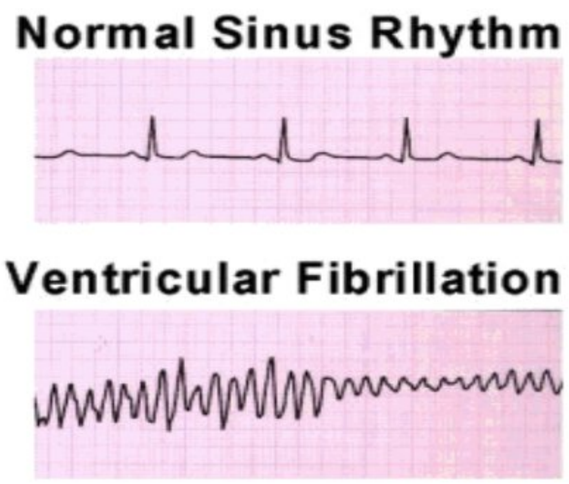
2-AtrioventricularNode (AV) node … cells in the AV node conduct impulses more slowly, so there is a delay as impulses travel through the node , this allows time for atria to finish contraction before ventricles begin contracting

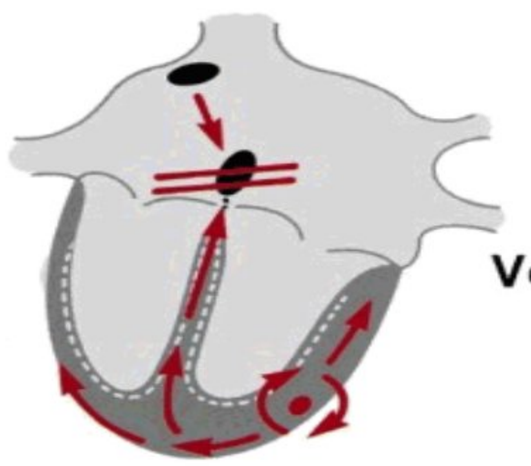
3-AtrioventricularBundle (bundle of His) …. From the AV node, impulses travel through to

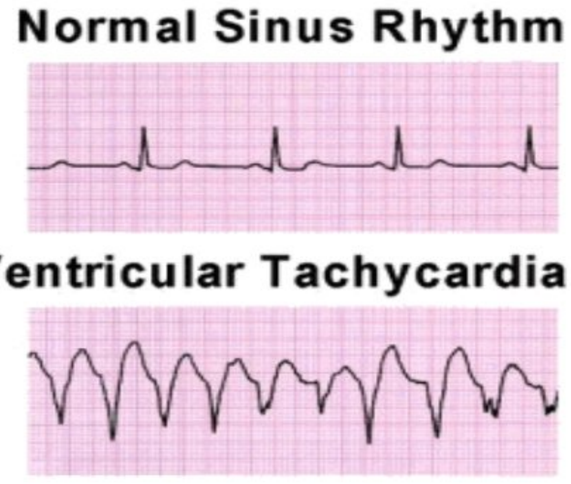
4-the right and left bundle branches … These branches extend to the right and left sides of the septum and bottom of the heart.

Cardiac arrest shockable rhythms :









Areas of frequent defibrillator application

Emergency department , Anesthesiology , Cardiology , Operation theatre, Intensive care areas &Ambulance services

STEPS OF USE AND APPLICATION :

1. Manual Defibrillation (incase of shockable cardiac arrest rhythm)

Switch 'ON' the Machine , Wait for initialization and self test, Make sure it is NOTin SYNC Mode, Apply gel to the paddles , Place them properly on the chest, Select 'ENERGY' to be delivered(energy in Joules) , Press 'CHARGE' button, Wait for Charging to complete. This is usually denoted by a continuous /long beep sound , Apply pressure to the paddles, Make sure that you and all the personnel are away from the patient ,Press both 'DISCHARGE' button simultaneously ,Observe patient and monitor ECG, Resume CPR, When finished, turn off and clean the paddles .

2. Synchronization Mode (manual cardioversionfor unstable tachyarrhythmia but with pulse)

Wait for initialization and self test , Connect ECG leads , Select 'SYNC / CARDIOVERSION' mode.. , Check for sync marker on the QRS waveform , If possible sedate the patient and maintain airway , Apply gel on the paddles and place it properly on chest , Select 'ENERGY' to be delivered(energy in Joules) , Press 'CHARGE' button , Wait for the Charge to be completed. This is usually denoted by a continuous /long beep sound , Check that everybody and you is away from the patient , Press both 'DISCHARGE' button simultaneously and hold till energy is delivered , Check patient condition and Heart rhythm , If required, cardiovertagain , Monitor the patient , Switch off and clean the paddles

Safety - General

The Defibrillator generates High voltage. It must be operated by trained, professional and qualified personnel only , Never use defibrillator with improper grounding or electrical leak socket. , Keep away the Defibrillator from any x-ray, Ultrasonic or other electronic instruments , Check the patient lead wire, cable and paddles for any damage or mishandling, otherwise replace it immediately. , Recommend using proper size and placement of recording paper. , Clean the print head regularly for clear printout

Safety - Monitoring

Use only the specified patient cable as recommended by manufacturer. , Place the patient cable in proper winding position or hang on to patient cable arm attached with Defibrillator trolley , Use good ECG electrodes to monitor ECG waveform , Don't use damaged patient cable , Confirm there is no ECG waveform because of electrical interference or defective patient cable. This may misinterpreted as QRS in synchronize mode.

Safety - Defibrillation

Excessive Gel can cause arcing of the current along the chest wall , Defibrillation in the absence of an ECG rhythm , ('blind defibrillation') to be avoided , A shock can be accidentally delivered to other rescuers if no clear protocol followed , If transthoracic impedance is high, a low energy shock (< 100 J) may fail to generate enough current to achieve successful defibrillation , Alcohol should never be used as conducting material for paddles because serious burns can result , Never discharge the Defibrillator in Air to check its performance , Never discharge with paddles shorted , Always clean the paddles after use

Precautions :

The paddles used in the procedure should not be placed ,, on a woman's breasts , over an internal pacemaker patients , Before the paddle is used, a gel must be applied to the patient's skin

Risks In Defibrillation

Skin burns from the defibrillator paddles are the most common complication of defibrillation. , Other risks include injury to the heart muscle, abnormal heart rhythms, and blood clots.

Cleaning the manual defibrillator

Wash your hands and wear gloves , Check the defibrillator for any damage , Clean and Disinfect all outside surfaces using isopropyl alcohol and be sure not to allow fluid into ports or battery connections. , Remove gloves and wash hands , Check that the readiness indicator (battery charge) is showing green Keep it in a clean, dry area.

Troubleshooting :

Attach The Paddles If The Monitor Reads, "No Paddles." , Check To Ensure That The Leads Are Securely Attached If The Monitor Reads, "No Leads.“ , Connect The Unit To AC Power If The Message Reads, "Low Battery." , Verify That The Energy Select Control Settings Are Correct If The Defibrillator Does Not Charge.Close The Recorder Door And The Paper Roll If The Monitor Message Reads, "Check Recorder”.

Cardiopulmonary Resuscitation (CPR)

(CPR) is a key part of emergency medical care designed to resuscitate individuals in cardiac arrest ,& is a technique of basic life support for oxygenating the brain and heart until appropriate, definitive medical treatment can restore normal heart and ventilator action, This is to stop the degenerative processes of ischemia and anoxia caused by inadequate circulation and inadequate oxygenation , Time to initiation of CPR is critical to improve likelihood of recovery; ideally, it should be started within 4 min

C .. Cardio (Heart) , P .. Pulmonary(Lung) , R .. Resuscitation (Recover)

Purposes :

1.To maintain an open and clear airway (A). 2.To maintain breathing by external ventilation (B). 3.To maintain Blood circulation by external cardiac massages (C). 4.To save life of the Patient.
5.To provide basic life support till medical and advanced life support arrives.
6- Restore cardiopulmonary functioning , 7- Prevent irreversible brain damage from anoxia

Basic life support (BLS): „ It is a life support without the use of special equipment .
CPR is a technique of basic life support for the purpose of oxygenation to the heart, lungs, and brain until the appropriate medical treatment come and restore the normal cardiopulmonary function.

Definition of Cardiac Arrest: .. It is loss of cardiac function, breathing and loss of consciousness
Causes of cardiac arrest (5 H and 4 T) :
(5-H) 1- Hypoxia , 2- Hypotension , 3- Hypothermia , 4- Hypoglycemia , 5- Hypo and Hyper kalemia
(4 T) 1- cardiac Tamponade , 2-Tension pneumothorax , 3- Thromboembolism, 4- Toxicity

Diagnosis of cardiac arrest: .. Loss of consciousness , N0 pulsation on the central arteries (carotid, femoral artery) , Apnea (no breathing)

What is advanced life support (ALS): „ It is a life support with the use of special equipment (eg. Airway, endotracheal tube, defibrillator).

How to do CPR : When you diagnose cardiac arrest start ***immediate {{ C- A- B }}*** ,

C ---- Compressions , A -----Airway , B -----Breathing .. services as an artificial heartbeat and an artificial respirator , CPR does not survive the victim even when performed properly , but if start within 4 minutes of cardiac arrest and defibrillation within 10 minutes ,the patient has 40% chance of survival .

(C) chest compressions : The human brain can not survive more than 3 minuts without circulation.
1.Place the heel of one hand in the centre of the chest 2.Place other hand on top 3.Interlock fingers
4.Compress the chest

 Rate 100 min Depth 3-5 cm(1.5 to 2 inches) Equal compression : relaxation
5.When possible change CPR operator every 2 min.

Chest compression in infants (0- 12 months) :

Place The Baby On His Back On A Firm Flat Surface , Image A Horizontal Line Between The Baby Nipples, Place 2 Fingers Of One Hand Just Below This Line, In The Center Of The Chest , Compress 1.5 Inch. (4cm.) At A Rate Of 100-120. 2 Breaths Every 15 Compression , Chest Compression Will Force Blood To Flow Either By Increase Intra-Thoracic Pressure (Thoracic Pump) Or By Directly Compressing The Heart (Cardiac Pump) , Chest Compression Must Be Continued For 2 Minutes Before Reassessment Of Cardiac Rhythm (2 Minutes = Equivalent To 5 Cycles 30:2) Be The Ratio Of Compression To Decompression (Recoil) Must 1:1

Complication of chest compression:

1- Ribs fracture
2- Fracture sternum
3- Pneumothorax and hemothorax
4- Injuries to the lung and liver

Common errors of chest compression:

1- Wrong Hand Position:

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Too high------- the heart is not compressed
Too low------- the stomach is compressed and risk of aspiration
Too laterally------ injury to underlying organs eg. Spleen, liver, bowel
2- Over Effort Lead To Cardiac Damage, Fracture Ribs And Damage To Lung And Liver
3- Inadequate Effort
4- Failure To Release Between Compression Prevent Venous Return And Filling Of The Heart
NOTE // CPR in patient 1-8 years is the same as adult ..

(CPR) , C ---- Compressions , A -----Airway , B -----Breathing

(A) Air way : Loss of consciousness often results in airway obstruction due to loss of tone in the muscles of the airway and falling back of the tongue **…** 1- Clear the airway 2- Do jaw thrust or head tilt…
(Helmlich manoeuvre) .. subdiaphragmatic abdominal thrust that elevate the diaphragm expelling ablast of air from the lung that displaces the foreign body.

(CPR) , C ---- Compressions , A -----Airway , B -----Breathing

(B)Breathing : Rescue breathing can be mouth to-mouth breathing, or mouth to-nose breathing (if the mouth is seriously injured or cannot be opened), To open the airway (by using head- tilt, chin lift maneuver) , pinch the nostrils shut for mouth to- mouth breathing .

Assessment of restoration of breathing and circulation:

1- Contraction of the pupil 2- Improved color of the skin 3- Free movement of the chest wall
4- Swallowing attempt 5- Struggling movement 6- Return of strong pulse and blood pressure

When to terminate BLS:

1- Pulse and respiration return 2- Emergency medical help arrive 3- When the rescue was exhausted

Advanced life support (ALS): includes ..

A-cardiac massage B- Airway management by equipment's
C- Breathing by advanced techniques D- Defibrillation E- Drugs

A- Chest Compression is the same as BLS
B- Airway management: by the following equipment's :

1- Face mask 2- Oropharyngeal airway 3- Laryngeal mask (LMA)
4- Endotracheal tube 5- Tracheostomy 6- cricothyroidotomy

C) Breathing … Give 100% O2 as soon as possible

Successful breathing is achieved by delivery of a tidal volume of 800-1200ml at a rate of 10-12 breath/min.

Advanced breathing techniques include:

1- Self-inflating resuscitation bag (ambu bag) 2- Mechanical ventilator

{{Expired air = 16 % o2 ,, ambu bag "room air" = 21 % o2 ,, ambu bag"o2 10-15 L"=85% o2 }}

D- Defibrillation :

consist of delivery of a therapeutic dose of electrical energy to the affected heart with a device called

defibrillator , In cardiac arrest the associated heart rhythms can be classified into two groups:

1) Shock able rhythm: VT (ventricular tachycardia) and VF (ventricular fibrillation)
2) Non shock able rhythm: asystole and PEA (pulseless electrical activity)

The basic difference in the treatment of these two groups of arrhythmia is the need for defibrillation in patients with VT and VF.
Position of paddles .. one is placed in the Rt. Infraclavicular region & the other is placed in the

Lt. 5-6 th I.C. space ant.axillary line

Alternatively Antero-posterior position may be used one paddle is placed in the
Lt.infrascapular region while the other in Lt. 5- 6 th I.C space ant.axillary line .

Drugs used in CPR

1- adrenaline:

Given as a vasopressor agent in a dose of 1 mg every 4 minutes (alternating cycles) while continuing CPR , It is given immediately in non-shockable rhythm

2- Amiodaron :

Given in a dose of 300 mg IV as a bolus dose (5 mg/kg) . it is given in shockable rhythm after the third shock. If not avaliable give lidocaine 100 mg IV

3- Calicium :

Dose 10 ml of 10% calicim chloride IV

Indication : PEA caused by hypekalmia and hypocalcaemia.

4- Sodium bicarbonate:

Used in sever metabolic acidosis (PH < 7.1) and life threating hyperkalemia

5- Atropine

Its routine use in PEA and asystole is not beneficial and only indicated in sinus bradycardia or AV block .

Dose: 0.5 mg IV. Repeated up to a maximum of 3 mg (full atropinization)

I.V FLUIDS :

infuse rapidly if hypovolemia suspected , usually use (Normal Saline or ringer solution),

Avoid Dextrose which is redistributed away from the intravascular space rapidly and causes

Hyperglycemia which may worsen neurological out come after cardiac arrest ,

Dextrose is indicated **only if there is documented Hypoglycemia** .

Deep Vein Thrombosis (DVT)

DVT is blood clotting that develops with deep veins, often in the leg or pelvis.
If the thrombus, or clot, breaks off, this will form an embolus , Emboli can make their way to the lung, causing pulmonary embolism (PE) , Clots may also form in the veins of the arm.

Symptoms ….. Some people may develop DVT without noticing symptoms. However, if symptoms develop, they may resemble the following:

1- Pain in the affected limb that begins in the calf
2- Swelling in the affected limb
3- A warm feeling in the swollen, painful region of the leg
4- Red or discolored skin

In most people, DVT only develops in one leg. However, on rare occasions, both legs may have DVT.
If a clot dislodges and travels to the lung, the following symptoms may **indicate PE :**

1- Rapid breaths
2- Chest pain, usually more severe while breathing deeply
3- A faster heart rate (tachycardia)

Complications

There are two possible complications:

1- Pulmonary embolism

PE is the most common complication of DVT and can be life threatening, It happens when a piece of a blood clot becomes dislodged and makes its way into the lungs, PE can lead to heart failure and can be fatal.

2- Post-thrombotic syndrome

This is more common among people with recurrent DVT.a person with post-thrombotic syndrome might experience the following symptoms :

1- Persistent swelling in the calf
2- Feeling of heaviness in the leg
3- Pulling sensation in the leg
4- An excessively tired leg
5- Redness of the skin
6- New varicose veins
7- Thickening skin around the area of the DVT
8- Leg ulcers for people with Sv. post-thrombotic syndrome

Risk factors for DVT:

1- Inactivity
If the human body is inactive for long periods, blood can build up in the lower limbs and pelvic area.

2- Injury or surgery
An injury or surgery that damages veins can slow the flow of blood. This increases the risk of blood clots. General anesthetics can also widen the veins, making it more likely that blood pools and clots may form. Knee and hip surgery, particularly, have a high risk of developing DVT.

3- Genetics: A person may have an inherited disorder that makes blood clots more likely,

4- Pregnancy: As a fetus develops , pressure against a woman's veins in the legs and pelvis increases.

5- Cancer: Some cancers have links to a higher risk of DVT, including colon, pancreatic, and breast cancers. Cancer therapies and procedures can also increase a person's risk of DVT, including chemotherapy,

6- Irritable bowel disease: People with irritable bowel disease (IBD) have a higher risk of DVT

7- Heart problems: that affect movement of blood in the body can cause problems with clots and bleeds.

8- Hormone medications: e.g. Estrogen hormone and contraceptive

9- Obesity: experience more pressure on their blood vessels, especially those in the pelvis and legs.

10- Smoking: People who smoke tobacco regularly are more likely to develop DVT

11- Varicose veins: varicose veins may lead to DVT unless a person receives treatment for them.

12- Age: Even though DVT may develop at any age, the risk increases as a person's age advances.

13- Sex: Females are more likely than males to experience DVT around childbearing age. However, females have a lower risk after menopause than men do at the same age.

Diagnosis of DVT: recommend tests, including:

1- Ultrasound: This type of scan can detect clots in veins.

2- Venogram: when ultrasound do not provide enough information, The doctor injects a dye into a vein in the foot, knee, or groin. X-ray images can track the dye as it moves to reveal the location of a blood clot.

3- Other imaging scans: MRI and CT scans may highlight the presence of a clot.

Treatment of DVT: The **Aims** of treatment are:

1- Stop the growth of a clot
2- Prevent a clot from becoming an embolism and moving into the lung
3- Reduce the risk that DVT might come back after treatment
4- Minimize the risk of other complications

Treatment of DVT includes :

1- Anticoagulant medications

drugs that prevent the clot from growing, as well as reducing the risk of embolism , Two types of anticoagulants support the treatment of DVT : (Heparin and warfarin).

Heparin has an immediate effect, For this reason, doctors usually administer it first through a brief course of injections lasting less than a week , With

warfarin, the doctor is likely to recommend a 3–6-month course of oral tablets to prevent recurrence of DVT.

2- Thrombolysis:

People with more severe DVT or PE require immediate medical attention, The doctor or emergency team administers drugs called thrombolytic, or clot busters, that break down clots, Tissue plasminogen activator (TPA) is an example of a thrombolytic drug, Excessive bleeding is a side effect of these drugs, As a result, medical teams only administer TPA or similar interventions in emergency situations.

Healthcare professionals administer TPA through a small catheter, or tube, directly into the site of the clot.

3- Inferior vena cava filter

A surgeon inserts a very small device, into the vena cava Th.e device catches blood clots and stops them moving into the lungs while allowing blood flow to continue.

4- Compression stocking

People wear these to help reduce pain, limit swelling, and prevent ulcers from developing , Stockings can also protect the individual from post-thrombotic syndrome.

Prevention :

1- Regular movement

2- Maintaining pressure on the at-risk area: This can prevent blood pooling and clotting. Medical professionals may recommend wearing compression stockings or a boot that fills with air to increase pressure.

3- Anticoagulant medication: A doctor may prescribe blood-thinning medication to reduce the risk of clotting before or after surgery.

4- As smoking and obesity are also key risk factors, it may be advisable to stop smoking and engage in regular physical activity.

Disseminated Intravascular Coagulation (Dic)

DIC ... is a disorder characterized by uncontrolled intravascular activation of coagulation and fibrinolysis with bleeding and thrombosis , Generalized intravascular thrombin generation and fibrin deposition in small blood vessels lead to the formation of microvascular thrombi causing tissue hypoxia.

Diagnosis :

- decreased platelet count
- decreased fibrinogen
- prolonged PT and PTT
- elevated fibrin degradation products or D-dimers

Both fibrin degradation products and D-dimers are elevated with trauma or surgery.

Treatment :

Patients with active bleeding should be transfused with platelet, plasma, cryoprecipitate, and red blood cells as needed. Heparin treatment has little role unless thrombosis is profound.

The Subcommittee on DIC of the **International Society on Thrombosis and Haemostasis** (ISTH) has developed a scoring card for disseminated intravascular coagulation (DIC) - each for "overt" and "non-overt" DIC. Following is the scoring for "overt" DIC:

1. Platelet count (more than 100 = 0; less than 100 = 1; less than 50 = 2)
2. Elevated fibrin degradation products (no increase = 0; moderate increase= 2; strong increase= 3)
3. PT upper limit of ref. range (less than 3 secs = 0; more than 3 secs = 1; more than 6 sec. = 2)
4. Fibrinogen level (more than 100 mg/dl = 0; less than 100 mg/dl = 1) Score of 5: compatible with overt DIC .

The Glasgow Coma Scale (GCS)

is the most common scoring system used to describe the level of consciousness in a person following a traumatic brain injury. It is used to determine the severity of an acute brain injury. The test is simple and reliable.

The GCS measures the following functions:

Eye Opening (E) • 4 = spontaneous • 3 = to sound • 2 = to pressure • 1 = none

Verbal Response(V) • 5 = orientated • 4 = confused • 3 = words, but not coherent • 2 = sounds, but no words • 1 = none

Motor Response (M) • 6 = obeys command • 5 = localizing • 4 = normal flexion • 3 = abnormal flexion • 2 = extension • 1 = none

Clinicians use this scale to rate the best eye opening response, the best verbal response, and the best motor response an individual makes, The final GCS score or grade is the sum of these numbers.

A patient's Glasgow Coma Score (GCS) should be documented on a coma scale chart, This allows for improvement or deterioration in a patient's condition to be quickly and clearly communicated.

Brain injury is classified as:

- **Severe :** GCS 8 or less
- **Moderate :** GCS 9-12
- **Mild :** GCS 13-15

Limitations of the Glasgow Coma Scale

Factors like drug use, alcohol intoxication, shock, or low blood oxygen can alter a patient's level of consciousness.

These factors could lead to an inaccurate score on the GCS.

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Children and the Glasgow Coma Scale

The GCS is usually not used with children, especially those too young to have reliable language skills. The Pediatric Glasgow Coma Scale, or PGCS, a modification of the scale used on adults, is used instead. The PGCS still uses the three tests — eye, verbal, and motor responses —

Here is the slightly altered grading scale for the PGCS:

Eye Opening (E) • 4 = spontaneous • 3 = to voice • 2 = to pressure • 1 = none

Verbal Response (V) • 5 = smiles, oriented to sounds, follows objects, interacts • 4 = cries but consolable, inappropriate interactions • 3 = inconsistently inconsolable, moaning • 2 = inconsolable, agitated • 1 = none

Motor Response (M)

- 6 = moves spontaneously or purposefully
- 5 = localizing (withdraws from touch)
- 4 = normal flexion (withdraws to pain)
- 3 = abnormal flexion (decorticate response)
- 2 = extension (decerebrate response)
- 1 = none

Pediatric brain injuries are classified by severity using the same scoring levels as adults, i.e. 8 or lower reflecting the most severe, 9-12 being a moderate injury and 13-15 indicating a mild TBI. As in adults, moderate and severe injuries often result in significant long-term impairments.

Pneumothorax

when air leaks into the space between your lung and chest wall. This air pushes on the outside of your lung and makes it collapse. Pneumothorax can be a complete lung collapse or a collapse of only a portion of the lung.

Causes of pneumothorax

1- Chest injury. Any blunt or penetrating injury to your chest can cause lung collapse.

2- Lung disease. Damaged lung tissue is more likely to collapse. Lung damage can be caused by many types of underlying diseases, including chronic obstructive pulmonary disease (COPD), cystic fibrosis and pneumonia.

3- Ruptured air blisters. Small air blisters (bleb) can develop on the top of the lungs. These blebs sometimes burst — allowing air to leak into the space that surrounds the lungs.

4- Mechanical ventilation. A severe type of pneumothorax can occur in people who need mechanical assistance to breathe. The ventilator can create an imbalance of air pressure within the chest. The lung may collapse completely.

Risk factors for a pneumothorax:

1- Smoking **2- Genetics** **3- Lung disease: eg.** Chronic obstructive pulmonary disease (COPD)
4- Mechanical ventilation.. **5- Previous pneumothorax.**

Types of pneumothorax ... Two basic types of pneumothorax are

1.traumatic pneumothorax or Open pneumothorax
results from a penetrating thoracic injury that permits entry of air into the chest,

2.non traumatic pneumothorax or closed pneumothorax
is the accumulation of air originating from the respiratory system within the pleural space.
Either type can lead to a tension pneumothorax if the air surrounding the lung increases in pressure. A tension pneumothorax is common in cases of trauma and requires emergency medical treatment.

Symptoms: ..The main symptoms of a pneumothorax are sudden chest pain and shortness of breath.

Treatment: .. For a pneumothorax usually involves inserting a needle or chest tube between the ribs to remove the excess air. However, a small pneumothorax may heal on its own.

Complications: Many people who have had one pneumothorax can have another, within one to two years of the first , Sometimes air may continue to leak if the opening in the lung won't close. Surgery may be needed to close the air leak.

Acute respiratory distress syndrome (ARDS)

(ARDS) is a type of respiratory failure characterized by rapid onset of widespread inflammation in the lungs , occurs when fluid builds up in the air sacs (alveoli), The fluid the lungs from filling with enough air, which means less oxygen , ARDS impairs the lungs' ability to exchange oxygen and carbon dioxide , typically occurs in people who are already critically ill or who have significant injuries , Many people who develop ARDS don't survive, The risk of death increases with age and severity of illness, Of the people who do survive ARDS, some recover completely while others experience lasting damage to their lungs.

diagnosis: Chest x-Ray , PaO2/FiO2 ratio (ratio of partial pressure arterial oxygen and fraction of inspired oxygen) of less than 300 mm

Symptoms: The main symptom of ARDS — usually develops within a few hours to a few days after the precipitating injury or infection. The signs and symptoms of ARDS can vary in intensity, depending on its cause and severity, as well as the presence of underlying heart or lung disease. They include:

1- Severe shortness of breath
2- Labored and unusually rapid breathing
3- Low blood pressure
4- Confusion and extreme tiredness

Causes of ARDS: .. The mechanical cause of ARDS is fluid leaked from the smallest blood vessels in the lungs into the tiny air sacs where blood is oxygenated. Normally, a protective membrane keeps this fluid in the vessels. Severe illness or injury, however, can cause damage to the membrane, leading to the fluid leakage . The underlying causes of ARDS include:

1- **Sepsis.** The most common cause of ARDS is sepsis, a serious and widespread infection of the bloodstream.

2- **Inhalation of harmful substances.** Breathing high concentrations of smoke or chemical fumes can result in ARDS, as can inhaling (aspirating) vomit or near-drowning

3- **Severe pneumonia.**

4- **Head, chest or other major injury.** Accidents, such as falls or car crashes, can directly damage the lungs or the portion of the brain that controls breathing.

5- **Corona virus disease (COVID-19**)

Treatment of ARDS There is no cure for ARDS at this time. Treatment focuses on supporting the patient while the lungs heal. The goal of supportive care is getting enough oxygen into the blood and delivered to your body to prevent damage and removing the injury that caused ARDS to develop.

1- Ventilator support … All patients with ARDS will require extra oxygen. Oxygen alone is usually not enough, and high levels of oxygen can also injure the lung.

2- Prone positioning … ARDS patients are typically in bed on their back. When oxygen and ventilator therapies are at high levels and blood oxygen is still low, ARDS patients are sometimes turned over on their stomach (prone position) to get more oxygen into the blood.

3- Sedation and medications to prevent movement

To relieve shortness of breath and prevent agitation, the ARDS patient usually needs sedation

4- **Fluid management** … Diuretic drugs are given to increase urination in hopes of removing excess fluid from the body to help prevent fluid from building up in the lungs.

5- **Extracorporeal membrane oxygenation (ECMO)**

ECMO is a very complicated treatment that takes blood outside of your body and pumps it through a membrane that adds oxygen, removes carbon dioxide and then returns the blood to your body. This is a high-risk therapy with many potential complications. It is not suitable for every ARDS patient.

Risk factors of ARDS: .. Most people who develop ARDS are already hospitalized for another condition, and many are critically ill. You're especially at risk if you have a widespread infection in your bloodstream (sepsis).People who have a history of chronic alcoholism are at higher risk of developing ARDS. They're also more likely to die of ARDS.

Complications:

1-Deep venous thrombosis

2-**Collapsed lung (pneumothorax)**. In most ARDS cases ventilator is used to increase oxygen in the body, the pressure and air volume of the ventilator can force gas to go through a small hole in the very outside of a lung and cause that lung to collapse.

3- Infections. Because the ventilator is attached directly to a tube inserted in your trachea, this makes it much easier for germs to infect and further injure your lungs.

4- Scarring (pulmonary fibrosis). Scarring and thickening of the tissue between the air sacs can occur within a few weeks of the onset of ARDS. This stiffens your lungs, making it even more difficult for oxygen to flow from the air sacs into your bloodstream.

Recovering from ARDS More people are surviving ARDS. However, many survivors end up with potentially serious and sometimes lasting effects:

1-Breathing problems. Many people with ARDS recover most of their lung function within several months to two years, but others may have breathing problems for the rest of their lives. Even people who do well usually have shortness of breath and fatigue and may need supplemental oxygen at home for a few months.

2-Depression. Most ARDS survivors also report going through a period of depression, which is treatable.

3-Problems with memory and thinking clearly.

Sedatives and low levels of oxygen in the blood can lead to memory loss. In some cases, the effects may lessen over time, but in others, the damage may be permanent.

4-Tiredness and muscle weakness. Being in the hospital and on a ventilator can cause your muscles to weaken. You also may feel very tired following treatment.

STRESS ULCER

Stress ulcers are multiple, superficial erosions which occur mainly in the fundus and body of the stomach. They develop after shock, sepsis, and trauma and are often found in patients with peritonitis and other chronic medical illness , It is the acute changes confined to the gastric mucosa under the condition of physiologic or psychological stress, The incidence of stress ulcer ranges from 20% to 100% in ICU

Causes of stress ulcer:

1- Stress is thought to worsen ulcers by increasing the production of acid in the digestive tract. H. pylori break down the protective lining of the stomach and duodenum, making the delicate tissue even more vulnerable to the effects of the acid

2- Ulceration is caused by ischemic injury to the gastric mucosa (increase adrenalin due to stress), loss of cytoprotectants

Risk Factors for ulceration

1- Multiple traumas
2- Respiratory failure, Intubated >48hrs
3- Coagulopathy,
4- Sepsis,
5- Hypotension
6- Hepatic and renal failure.
7- Burns of >35% BSA

There is a good relationship between severity of illness and incidence of ulceration., the longer a patient in ICU, the more likely they are to have stress ulcer.

Diagnosis

Most patients have no symptoms. Abdominal pain and perforation are rare, Endoscopy: Commonly mucosal erosions are found in the fundus .

Treatment

Patients who do not have major risk factors do not require treatment. Patients in shock, sepsis, respiratory, hepatic or renal failure, or who have a coagulopathy, who are admitted to intensive care, should all be given stress ulcer prophylaxis.

Principle of Treatment:

1-General measures

a) Maintenance of hemodynamic stability
b) Good sedation, analgesia,

2-Medical therapies: include

. Antacids
.. H2-receptor antagonists. (Cimetidine)
... Proton pump inhibitors. Indicated for the treatment of bleeding or the prevention of re-bleeding
.... Cyto-protective, Sucralfate

3-Surgery therapy

Prophylaxis of stress ulcer

1- Enteral feedings:

Enteral feeding refers to intake of food via the gastrointestinal (GI) tract. Enteral feeding may mean nutrition taken through the mouth or through a tube that goes directly to the stomach or small intestine

Principle of enteral feeding:

a) To neutralize gastric pH (to dilute the relatively acid environment)
b) To provide the cells of the gastric mucosa with a nutrient and to produce the protective surface lining. .

2- Control of the gastric PH
3- Cytoprotection
4- Hemodynamic management
5- Oxygen supplement

Prophylaxis for SUS should be confined to carefully selected at-risk patients .Stress ulcers is often present within hours of admission to an intensive care unit. So, it is more important to prophylaxis stress ulcers than to treat them.

PULMONARY EDEMA

Dr. DAWOOD

Definition :

Is an abnormal buildup of fluid (accumulation and swelling) in the lungs, lead to shortness of breath, by impairment of gas exchange & may cause respiratory failure, It is also known as pulmonary congestion & lung water.

Pathophysiology:

sever left ventricle failure , Back pressure of fluid volume into pulmonary veins, serum forced into alveoli, Left atrial pressure rises causing pulmonary edema & Gas exchange ability decreased .

Causes :

1.heart attack ,or any disease of the heart that weakens or stiffens the heart muscle , eg. Dilated and/ or hypertrophied cardiomyopathy with tachy- or Brady-arrhythmias , massive MI .

2. leaking or narrowed heart valves (mitral or aortic valves), eg. Critical MS (precipitated by arrhythmia or pregnancy),Critical AS , VSD and/ or acute MR complicating MI. , Prosthetic valve dysfunction.

3. sudden sever high blood pressure (HTN)

4. Fluid overload with oliguria or anuria .

5. major injury like Lung damage caused by poisonous gas , or sever infection.

6. Prolapsing LA myxoma obstructing LV inflow.

7. other causes include high altitude exposure , certain medicines ,renal failure ,narrow arteries that bring blood to the kidneys.

Signs and symptoms:

- Severe respiratory distress, orthopnea
- Pink, frothy sputum
- Severe apprehension, agitation, Hypoxia and smothering feeling , as hypoxia worsens.
- Cyanosis : a. Inadequate gas exchange b. Pao2 fails, PaCO2 rises
- Diaphoresis (Sympathetic stimulation)
- Adventitious lung sounds
  .. Rales from fluid in aiveoli .. Rhonchi from fluid in larger airways .. Wheezing from airway spasm ("cardiac asthma")
- Jugular Venous Distention (Right heart backs up from pulmonary congestion)
- Vital signs (.. BP : hypo or hypertensio .. Tachycardia .. Dyspnea/Tachypnea)
- Level of consciousness (May vary according to hypoxia , Agitation , confusion & unconsciousness)
- Chest pain (Present with acute MI , acute PE & tension pneumothorax.

Prevention :

in patients with known diseases that can lead to pulmonary edema, strict compliance with taking medications in a timely manner and following an appropriate diet (usually, low in salt) can significantly decrease one's risk.

Management of pulmonary edema :

Admit to ICU ## Monitor cardiac rhythm. ## Obtain ABG immediately. & ## Insert two IV lines.

LINES OF TREATMENT :

I -Non specific measures : some or all of these are applied to all patients regardless the etiology.

II - Identification & treatment of the precipitating factors.

III - Correction of underlying cause.

I - NON SPECIFIC TREATMENT

Dr. DAWOOD

1- O2 inhalation (60 - 100%).
2- Place the patient in the semi - sitting position.

3- Morphine : 3-5 mg diluted, injected IV over 3 minutes , Don't give if:

a. Bronchial asthma & COPD. , b. Intracranial hemorrhage. , c. Disturbed consciousness.

d. Marked respiratory depression. , e. Liver failure

4- Furosemide : 20-80 mg IV , over 2 minutes.

onset : 5 mn. , Peak at : 30 mn. , Duration : 2 hours , Can be repeated once more after 20- 30 mn.

5- Nitroglycerin: start with (0.4 to 0.6 sublingual) repeated every 5 minutes if needed , If systolic BP > 100mmHg nitroglycerine is administered IV (20 ug\min & may be titrated up to 100 ug/min) according to clinical response & arterial blood pressure till pulmonary edema is relieved or systolic BP decreases below 100mmHg.

6. Na Nitroprusside : starting dose (0.1 ug/kg/min) may be givin to patients whose pulmonary edema is due to acute mitral or aortic valvular incompetence or marked systemic hypertension, till pulmonary edema is relieved or systolic BP decreases below 100mmHg , Combination of Morphine, Diuretic and NTG, Will decrease the preload sufficiently in most of the patients.

7- Digoxin:

- ***If Patient Is Not On Digitalis*** , digitalis is particularly helpful if pulmonary edema is associated with:

  a- Rapid AF. , b- SVT. , c- If pulmonary édema is secondary to aortic valve disease or hypertension.

  Dose: 1.0 mg (2 amp.) slowly Iv, a second dose of 0.5 mg (1 amp.) can be given after 15-20 minutes.

- ***Patient was on digitalis :*** Don't give if toxicity is suspected , ***Any of the following suggests toxicity :***

  a- History of digitalis toxicity . , b- Nausea & vomiting . , c- Paroxysmal SVT with AV block.

  d- Frequent PVCS . , e - VT. , f- Hypokalemia

In case of rapid AF or SVT is precipitating pulmonary edema with no evidence of digitalis toxicity, give: Lanoxine 0.5 mg IV slowly

8-Amiophylline : Especially helpful if bronchospasm complicates pulmonary edema .

Dose : initial dose 4-5 mg/kgm, IV /hour for first 12 hours, then maintenance 0.1 mg/kg/hour

Effect: 1. Direct myocardial stimulant effect. , 2. Mild vasodilatation. ,3.CNS stimulation & mild diuretic effect.

N.B. give half of the above doses if the patient has: a. Renal insufficiency. , b. Hepatic insufficiency. , C. PVCS.

9- Inotropic infusion: If systolic blood pressure is < 100 mmHg.

A. **Dopamine :** ***effect is dose dependent*** , .. If pulmonary edema is associated with ***normal systolic BP*** give dopamine , at dose < 3 ugm/kgm/min. This will increase renal, mesenteric & coronary circulations which results in better diuresis, cardiac perfusion & contraction, .. If the ***patient is in a shock state*** give a dose around > 5 ug/kgm/min, because the intense inotropic & vasoconstrictor effect are desirable ,If a higher dose 10 ugm/kgm/min is required, NTG infusion may be added to antagonize spasm.
B. **Dobutamine:** Usual dose 2.5-15 ugm/kgm/min , It increases contractility with little or no effect on BP , Not a renal vasodilator ; even it redistributes cardiac output with increase to coronary & decrease of renal & mesenteric flow.

10- Thrombolytic therapy or urgent revascularization for acute myocardial! infarction.

11- Intra aortic balloon counter pulsation is of value if the patient does not respond to previous measures and is candidate to undergo urgent revascularization.

12- Correction of underlying cause when feasible.

II - IDENTIFICATION & TREATMENT OF PRECIPITATING FACTORS

Common precipitating factors are:

1. Infection in a patient with established heart disease ((lower the high temperature & proper antibiotic)) .
2. Tachyarrhythmia : If not induced by digitalis , not responding to drugs then DC shock should be considered.
3. Brady arrhythmia : Atropine 0.6 mg IV, repeat If not responding temporary pacemaker should be considered..
4. Correction of anemia. 5. Pulmonary embolism → proper treatment. 6. Fluid overioad → Avoid.
7. Thyrotoxicosis. 8. Rheumatic activity and / or SBE in RHD.
9. Hypertensive crisis → rapidly acting antihypertensive arugs e.g. Na Nitroprusside.

III - RECOGNITION & TREATMENT OF UNDERLYING CAUSE

After emergency therapeutic measures have been instituted and correction of precipitating factors dealt with, the underlying cause should be defined and dealt with properly..

COMMON CAUSES :

1. MI →((ECG & Enzymes))
2. Critical silent MS (precipitated by arrhythmia or pregnancy) .
3. Critical AS.
4. VSD and/ or acute MR complicating MI.
5. Dilated and/ or hypertrophied cardiomyopathy with tachy- or Brady- arrhythmias.
6. Prolapsing LA myxoma obstructing LV inflow. 7. Prosthetic valve dysfunction.

Artificial Ventilation

In patients with pulmonary edema , especially if rapid improvement is not achieved, ABG should be repeated every 20-30 minutes, disturbance in blood gases usually starts with hypoxemia followed later on by hypercapnea (Co2 retention).
If Hypoxemia is present give 100% O2 inhalation by nasal prongs, (Goal to keep PO2 => 60mmHg.}

Start ventilation if any of the following is present

((1. PO2 is 60mmHg despite inhalation of 100%O; at 20 liters /min. & 2. Progressive hypercapnea)) .

INDICATION FOR MECHANICAL VENTILATION :

The decision to intubate and initiate mechanical ventilation has always seemed more complicated than it should be; The following simple rules should be remembered

1. The indication for intubation and mechanical ventilation is thinking of it. (mean if the patient needs this , do it)
2- Intubation is not an act of personal weakness. (it doesn't need to apologize about .)
3- Initiating mechanical ventilation is not the kiss of death (& doesn't mean it creates ventilator dependence)

A new strategy for mechanical ventilation :

Always remember when decide to put patient an ventilator the following facts;

1. Minimal duration (There must a plan for early weaning as much as it can be)
2. Minimal effective TV (avoid large TV)
3.minimal accepted pressure (through out the respiratory cycle)

All these to avoid ventilator – induced lung injury , but still there are ***side effects for this strategy*** :

1-low TV ; Promote lung collapse (to avoid this apply low PEEP)
2- Low TV; cause slight hypercapnia which can be accepted as known permissive hypercapnia .

Final word ; *Since mechanical ventilation is a support measure and not a treatment ,*Modality ,nothing that is done with a ventilator will have a favorable impact on the outcome of primary illness, *On other hand ,mechanical ventilation can have negative impact on outcome by creating adverse effect,*This mean that the best mod of mechanical ventilation is the one with the least adverse effects , it also means that if we really wont to improve outcomes in ventilator – dependent patients , less attention should be directed to the knobs on ventilators and more attention should be directed at the diseases that create ventilator dependency

TRACHEOSTOMY

Dr. DAWOOD

Definition : It is an invasive medical procedure ,is done to bypass the larynx for maintaining the patency and security of the airway.

Types :

1. **Urgent** tracheostomy (is done as a life saving measure).
2. **Elective** tracheostomy

   A. **Short term** ; as preoperative precaution for certain kind of surgery for the purpose of safety and easier early post operative care example; surgery for large goiter, laryngeal tumors facio-maxillary operation.

   B. **Long term ;Permanent** ,is done for certain comatose patient in order to make the care easier and the transfer from the I. C. U. more possible practically.

Level Of Tracheostomy

1- Urgent ; at crico- thyroid membrane[crico- thyrotomy]

2- Elective ; at 3rd-4th tracheal rings

Types of the tube:

Either portex or silicon cuffed tubes [large volume low pressure cuff]or metal tube with inner tube to be easily cared by the patient himself or by one member of his family permanent T. , Elective T. , either done by a surgeon through the classical surgical procedure or by the intensivist through what is called Percutaneous Dilation Tracheotomy.

Tracheostomy Tubes Care :

1. Periodic inflation and deflation of the cuff [minimal occlusion pressure is 20 mmHg. (The P. which will occlude the blood supply of the tracheal mucosa is 30 mmHg) .

2. Suction is done as fallow,

a. if the patient has spontaneous breathing and preserve cough reflex , do the suction during chest physiotherapy which provoke cough reflex.

b. if the patient has spontaneous breathing, but has absent cough reflex ;suction is done during chest physiotherapy by introduce the suction catheter inside the tracheostomy tube and timing the suction during the expiratory phase only.

C . if the patient is unconscious and on I.P.P.V. the suction can be done freely because it possible to re-expand collapsed lungs which is develop by the suction process by the action of the ventilator.

Change the tracheostomy tube; every 7-10 days according to policy in that unit,

3. Stoma's care by washing with half conc. of hydrogen peroxide and saline solution

4.Fixation of the tracheostomy tube; tight to allow 2 fingers between the band and the skin of the patient.

Complications :

1- Early complications:

a- Bleeding b- Suffocation C- reflex ; hypoxia ,bronchospasm , arrhythmia or hypotension or even cardiac arrest due to a strong vagal stimulation.

2- Late complications

a- infection for many reasons lack of humidification ,lack of air conditioning, low blood supply, or aspiration

b- tracheostomy sinus. c- Tracheo- esophageal fistula

d- the presence of tracheotomy tube will interfere with the normal swallowing reflex, which makes oral feeding of that patient very difficult.

RESPIRATORY SYSTEM (PATIENT MANAGEMENT IN ICU)

Respiration is necessary to sustaining life , and the nurse plays an important role is helping the critically ill patient breathe , the nurse must be knowledgeable and skilled in assessing patient needs providing quick and efficient care

Bronchial Hygiene;

Is helpful in preventing and treating pulmonary complications , the aim is to improve ventilation and diffusion , these are accomplished through the therapeutic goals at mobilization and removal of secretions and improved gas exchange .

Methods Of Bronchial Hygiene :

1. Effective coughing and deep breathing : The objectives of deep coughing are to promote lung expansion , mobilize secretions and prevent side effect of retained secretion (in atelectasis & pneumonia.) & breathing exercise with PEEP. The efficacy of these techniques is limited to patients who able to cooperate , Ideally the patient is positional upright on the edge of the bed or chair with the feet supported , he or she is instructed to take a slow, deep breath , hold it for ot least 3 seconds and exhale slowly , if secretions are auscultate then a cough is initiated on maximum inspiration

2- Chest physiotherapy:

A - Postural drainage , positions facilitate gravitational drainage of pulmonary secretions into main bronchi and trachea based on anatomy of the lung segments , this is not tolerated in all positions in critically ill patients and there are many contraindications, so now a days are not so useful (such as increased intracranial pressure , after or during tube feeding , inability to cough respiratory instability ,hemodynamic instability , obesity , decreased mental status)

B - Chest percussion and vibration:

Are used to dislodge secretions , neither method has been shown to superior

Contraindication : *Fracture ribs , *Chest / abdominal trauma , *Pulmonary hemorrhage or,*embolus *Pneumothorax / subcutaneous emphysema, *Cervical cord trauma, *Tuberculosis, *Pleural effusions/ empyema, *Asthma .

c- Bronchodilator aerosol therapy : The goals are to relax the airways and mobilize secretions and reduce mucosal edema , delivered either by inhalers , or by mobilization .

Assessment before and after treatment includes :

Breath sound , Pulse rate & Respiratory rate , The last two commonly increase during bronchodilator therapy .

Artificial Airway :

Good bronchial hygiene and carefully monitored oxygen therapy may eliminate the need for an artificial airway or ventilator support, An artificial airway and ventilator support become mandatory if these measures fail to provide adequate oxygenation and removal of Co2

Artificial airway have a fourfold purpose :

1- Establishment of an airway
2- Protection of the airway ,with cuff inflate
3- provision of continuous ventilator assistance
4- Facilitation of airway clearance

Knowledgeable , and aggressive nursing care is required to maintain airway patency , maximize therapeutic effects and minimize damage to patients natural airway

SUCTIONING :

The presence of an artificial tube prevents glottis closure as a result , the patient is unable to use the normal clearing mechanism (effective coughing), Additionally , the foreign object (tube) increases production of secretion So suction become necessary for removing secretion and maintaining patency

Indication for suctioning;

1. Visual observation of secretion
2- Determination of presence of secretions by chest auscultation , coughing
3. increase in peak airway pressure ,and decrease in tidal volume during pressure ventilation
4. Deterioration of the patient's oxygenation

Suction of the secretion through ET tube is not allowed (for bidden) in: **Dr. DAWOOD**

1.recovery from anesthesia (during weaning from ventilator & extubating)

2.pulmonary edema & ARDS(adult respiratory syndrome)

Procedure :

1- Pre oxygenation

2- Gently insert the catheter as far as possible into the artificial airway without application of suction

3. Withdraw the catheter slowly and apply intermittent suction while rotating and removing the catheter

4- Aspiration should not exceed 10 -15 seconds , prolonged aspiration can lead to severe hypoxia , hemodynamic instability and sometimes cardiac arrest

5- Hyper oxygenation between each subsequent pass of the catheter and at the end of the procedure before reconnection to the ventilator

6- The nurse performs suctioning as a sterile procedure

Complications of suctioning :

1- Hypoxemia	2- Dysrhythmia	3- Vagal stimulation (bradycardia and hypotension)
4- Bronchospasm	5- Elevated intracranial pressures	6- Atelectasis
7- Tracheal mucosal trauma	8- Bleeding	9- Nosocomial infection.

Bronchial wash (sanitation) :

The routine instillation of normal saline has become increasingly questionable because ; (can be forbidden now)

1-in a test tube , saline and sputum act like oil and water they don't form a mixture , so saline instillation unlikely liquefies or increases the amount of sputum obtained during suction

2- It causes decrease in oxygenation Airways

Pharmacological agents :

1- Bronchodilators ; principally to dilute the airways by relaxing bronchial smooth muscles , divided into 3 groups;

a- Beta2 adrenergic agonists (Ventolin); stimulate bronchodilation , in the bronchial smooth muscles

b- Anticholinergic agents (atropine) ; Produce bronchodilation by reducing intrinsic vagal tone to the airways also block reflex bronchoconstriction caused by inhaled irritants ,

c- Theophylline agents: (aminophylline , intravenous form); nowadays the using of such drug as a bronchodilation is controversial it has a narrow therapeutic index.

2- Anti – inflammatory agents;

They interrupt the development of bronchia inflammation and have a prophylactic or preventive action

a- Corticosteroids ; most effective anti inflammatory agents for the treatment of reversible airflow obstruction

b- Mast cell stabilization , (cromolyn , nedo-cromil); prevent release of mediators from mast cell

3- Antibiotics ;

4- Sedation; , in critical illness ; analgesia and sedation to control anxiety and facilitation of mechanical ventilation

5- Neuromuscular blocking agents; is to maximize oxygenation and prevent complication such as barotraumas that can be caused by high ventilator pressures .

PHYSIOTHERAPY FOR CRITICAL ILL PATIENT IN ICU

1. Chest physiotherapy;

- *For conscious patient*, do Breathing exercise , encourage cough to get rid of respiratory tract secretion.
- *For unconscious patient* :(when the patient is breathing spontaneously) do Chest taping (ant. & posterior) to help drainage of secretion from peripheral lung tissue to the bronchial tree so that to be cleared by coughing & suction .so by this procedure ,encouraging breathing exercise & coughing for proper clearance of the airway.
- *For unconscious patient :(when the patient is intubated & on Assisted ventilation)*do chest physiotherapy with suction of the Secretion through the Endo Tracheal Tube & re inflation of the lungs by AMBO bag to prevent atelectasis .

2. Change patient's position in his bed at cretin interval :
every 4h , 6hs & so on to prevent bed sores (pressures ulcers specially at sites of bony prominent)
3. Skin care; daily cleansing with sprit & soothing by skin powder
4. Passive ***joints movement*** for(unconscious patient), Active joint movement (conscious patient) ,to prevent joints stiffness .
5. Muscles massage ; to prevent muscles spasm & to enhance venous return from the muscles of the extremities specially calf muscles to improve cardiac output &to prevent vein thrombosis ,Early mobilization of critically ill patient is very important , Which is either active movement or passive one.

Transportation of critically ill patient

It carry's high risk due to following physical factors :
1- It is well known that the effect of the gravity on a moving load is greater than that on static load , so effect of gravity on volume of blood in patient's body is greater during transportation than when the patient lying in bed statically. so homodynamic stability of the patient become unstable during transportation & this may threatening the life of critically ill patient .

2- It is proved that transportation induce stress response which has harmful effect on patient's vital organs whom are already under the stress of his own critically ill condition , For this reason stability the patient's has when lying in bed as a compensated state may become uncompensated & then may be collapse at any time of transportation i.e. his stability is unstable.

3- The vital organs are present in bonny cage (brain in the skull , lung & heart in thoracic cage.) as it is physically known that the momentum of these organs are differ from that of bonny cage which they are present inside , *So during transportation & due to acceleration & de acceleration process These organs will have coup & counter trauma to bonny wall . This trauma will aggravate the primary pathology* which any one of these vital organs has to start with, It is good to remember that these factors are in general present physically regardless the type of vehicle used in transportation , technical level & the availability of medical devices in that concerning vehicle .

Transfer Guide lines ;
There are 5 phases applied to achieve safety transfer of critically ill patient from one hospital to another ;
1. Notification & acceptance by receiving hospital.
2.Preparation of the patient by transport team, including all the information about patient's condition with copy of all his medical records
3.Transport process; include : Vehicle. Medical equipment .Time of transport .
4. Turnover the patient in emergency department of receiving hospital by reception team.
5.Post transport assessment in receiving in I.C.U. The issue of patient - family communication in I.C.U.
6.***Visiting the patient in 1.C.U. is forbidden*** because , A. It will *break the aseptic barrier* of I.C.U. **,** B. It will *interfere with the freedom of decision & action* by medical personnel working in ICU. **,** C. *At any moment, critical ill patient in the I.C.U. may need urgent medical intervention*, presence of visitors in The I.C.U will *carry undesirable effects.*

There is exceptional occasion for :
1.medical reason when needs patients family contact which may play help for final medical assessment(level of consciousness)
2. Humanity reason , for example to have final look by his family member before death.
It is obvious now **That each unit in this system has its own** : 1. Supply . , 2. Staff.
3. Function to do (specific function which are within the general scope of primary functions of I.C. services)
but each unit; On one hand can work independently & on other hand it represent a ring in a chain . i.e. each ring is linking to another one in order that each unit Is supporting & completing the job of other units so that their work will be synchronized & integrated to complete the picture of modern intensive care services.
Finally: Putting this system on the ground can be considered as our finger prints in this new field of medicine .
The issue in every remarkable job is ; To believe **first** , **Second** have the will to act, **Third** the beginning of first action.Please remember_ This system can be done in wide rang scale of medical & technical facilities .i.e. from simple facilities to high complicated & sophisticated standard _Lack of advanced & sophisticated facilities is not an excuse for not putting the first step in this field of medicine which is Intensive Care Medicine.

LEC : 1 Mon. 28 / Feb. / 2022 **Brain Death** Dr.DAWOOD

Brain death, also known as death by neurologic criteria, involves the permanent and complete absence of All brain function, in both the **cerebrum** the "thinking" part of the brain, and the **brainstem**, the deep brain structure responsible for reflexes and breathing & and level of consciousness .

Causes of Brain Death

Brain death results from a devastating brain injury, commonly due to :
head trauma, bleeding in the brain, stroke, or loss of blood flow to the brain after the heart stops (cardiac arrest).

How is brain death diagnosed?

The following 3 conditions must be confirmed to declare brain death :

- **Patient Is In Persistent Coma**

No Eye, Verbal, Or Movement Response To Painful Stimulus, the patient should be in unresponsive coma with a known irreversible cause of brain injury.

Exclusions ……There should be an absence of :

1.Sedative and paralyzing drugs

2. Hypothermia (below 34°C)

3. Metabolic or endocrine disturbances

4.Circulatory disturbances (hypotension).

- **Patient Cannot Breath Without Assistance**

no breathing for 10 minutes without mechanical ventilation

- **Patient Has No Brainstem Reflexes >>>>**

no eye response to light or cough response to throat suction

Test (REFLEX) For Brainstem Death >>>>

Light Reflex
Comeal reflex
Stimulate the eye from the side: object may stimulate via the visual pathway causing them to blink if neurologically intact
Cold caloric reflex
For the conscious patient. this test would invoke nystagmus with the fast component away from the side stimulated.
For the unconscious brainstem-intact patient. this test would invoke slow tonic movement towards the side of stimulation (i.e. lacking the fast component of nystagmus).
Gag reflex … This is usually performed by moving the ETT against the posterior pharyngeal wall
Cough reflex … This is usually performed by stimulating the carina with a suction catheter
Reponse to pain … when pain administered to limb or trunk & when pain administered to face.
Breathing response to hypercapnia
To confirm the absence of respiratory movements in response to hypercapnia :
• Pre-ox/genate patient with 100% oxygen for 10 minutes. Allow arterial carbon dioxide presure to rise to above 6.0kPa by deceasing ventilation rate on ventilator (confirm with ABG sampling; pH sheuld be <7.4).

• Disconnect from the ventlator and insuflate axygen via the ETT to prevent hypoxia. Observe and confirm the absence of spontaneous respiration.

• Re-attach patient to ventilator if organ donation is being considered.

Controlled mechanical ventilation in a critically ill patient

Anaesthesia, intubation, and ventilation of critically ill patients can be a stressful and challenging time for the anaesthetist , Patients may require care in remote or low-dependency areas (eg, hospital wards, ABE. Or radiology suites), and they are usually unstable at the time of intubation,

The indications for Intubation can be summarized as follows :

1) Actual or threatened airway obstruction
2) Failure to oxygenate
3) Failure to ventilate
4) To reduce work of breathing, eg. in pulmonary oedema
5) Tissue hypoxia, e.g. carbon monoxide poisoning
6) Severe metabolic upset
7) Facilitation of IPPV and control of CO; (eg. raised ICP)
8) Sedation or reduced level of consciousness to protect airway
9) To assist with bronchial toilet.
10) A critically patient might require intubation and ventilation for a combination of reasons. For example, a patient with opiate intoxication may be unconsciousness, may hypo ventilate and may be hypoxic.
11) With worsening clinical conditions & If a patient demonstrates the following then support is indicated:

A. Patient exhaustion
B. Respiratory rate > 35 breaths/min
C. Increasing PaCO,
D. Respiratory acidaemia with pH < 7,30.

Post-induction hypotension : Hypotension is common post-Induction and is the result of

- Unresolved hypovolaemia.
- Positive pressure ventilation, which reduces venous return and therefore cardiac output
- The action of induction agents. These often cause vasodilatation, antagonizing compensatory vasoconstriction in shock states.
- The sick patient often has a loss of sympathetic tone. Vasodilatation is also induced by nitric oxide, produced as a result of sepsis.

Patients may require fuid boluses, vasopressors, and possibly small doses of adrenaline (10-100mcg) in order to prevent this.
Initiation of ventilation

Principle of mechanical ventilation

The ventilator is able to support respiratory function by.

- Improving oxygeration
- Controlling excretion of CO,
- Assisting the respiratory muscles, therefore reducing the work of breathing & further reduces oxygen consumption.

Ventilator Strategy

The pulmonary pathology of a critically ill patient influences gas exchange, Common patterns include:

- Pneumonia, consolidation, or lung segment collapse
- Acute lung injury/ARDS
- Chest wall oedema.
- Chronic obstructive airways disease
- Pulmonary oedema

The choice of mode of ventilation will vary according to the patient's problems ,There is no evidence that one mode of ventilation is better than all others, However, the common strategy is the avoidance of further lung damage (ventilator-induced lung injury) .

Early use of modes that allow spontaneous respiratory effort may confer some advantage, Critical care ventilators have sensitive flow triggers that respond to patient effort and adapt to patient requirements.

This is more comfortable. and less sedation is required for the patient, with fewer side effects, It is important to decide whether the ventilation strategy should be

- Control of oxygenation with permissive hyper capnia
- Aggressive pursuit of pH and PaCO, targets.

Using excessive pressures or volumes causes lung tissue trauma, worsening pre-existing lung pathology and causing further impairment of oxygenation and CO2 clearance. All patients need to be adequately oxygenated to surrvive, but for the vast majority, CO2 level will not influence outcome.

Permissive hypercarbia desuri'ves the practice of allowing PaCO2 to climb in an effort to limit ventilator pressures or volumes and ventlator-induced lung injury. For those with raised ICP, however, strict control of PaCO, may be impoitant Satisfactory levels of oxygen and CO, are specific for each patient, but generally a previous y fit and healthy man will tolerate oxygen saturations down to 8-90%. Pre-existing respiratory disease may mean that even lower saturations are acceptable.

Ventilator settings

The ventilator performs the work of the respiratory muscles However, with judicious use it can improve gas exchange.

How can we improve oxygenation?

- Increase FiO
- Increase mean inta-thoracic pressure
- Increase peak inspiratory pressure (PCV)
- Increase tidal volume (VCV)
- Increase PEEP
- Increase inspiratory time, change the l:E ratio

icreased mean airway pressure aids lung recruitment and thus oxygenation However, excessive use of any measure that increases lung volume or pressure may damage the lungs. There are also CVS effects to be considered: the increase in intra-thoracic pressure reduces venous relurn, adversely affecting cardiac output. In this way, although PaOz may be improved. tissue oxygen de very may ultimately be made Worse, Drainage of pleural effusions or pneumothoraces will also allow lung expansion and more effective ventilation.

How can we improve CO, clearance?

Increasing the minute volume will clear CO, but this depends on the mode of ventilation and the characteristics of the patien. For most patients 100 ml/kg/min (ideal body weight) is a good staring minute volume. A tidal volume of 5-7ml/kg is usually considered safe. Although even lower volumes may be appropriate in ARDS. Respiratory rates will usually be 14-20 breaths/min.

Positive end-expiratory pressure (PEEP)

This improves arterial oxygenation by increasing FRC and preventing collapse of small anways. It decreases the shunt fraction, The pressure-volume curve may guide the use of PEEP , Although PEEP recruits lung volume, It may also cause over-distension of other alveolar units, To protect against this. tidal volumes should be reduced when high levels of PEEP are used.

PEEP is assessed by its effect on oxygenation. It is usually set at 5-10cmH2O, although it may be set higher (up to 18-20cmH;O) in

patients with severe hypoxia receiving 100% oxygen, PEEP causes a further fall in venous return leading to a decreased cardiac

output and tissue oxygen delivery. 'Best' PEEP Is associated with maxi- mum tissue oxygen delivery and minimum reduction of cardiac output.

Weaning and extubation

Weaning is the reduction of ventilatory support and can be started after the first sign of ventilatory or gas exchange improvement. In some patients it takes weeks for completion, and generally the best improvements will be made when the followlng are satisfied

- The reason for mechanical ventilation has resolved
- Minimal pain and discomfort
- No acidaemia or metabolic disturbance
- Adequate neuromuscular function
- Respiratory drive minimally depressed by drugs
- Maintenance of adequate oxygenation and CO, clearance without excessive work of breathing.

Criteria For Extubation Are More Specific Patients Should :

- Need FiO2 <0.5
- Have adequate clearance of CO2 without support
- Be able to cough and cope with volumes of sputum produced
- Be conscious. cooperative. and responsive
- Be in control of their airway reflexes
- Be generally improving and not deteriorating.

Following extubation, they should be closely monitored for signs of deterioration, since some may fail and require re-intubation.

Guillain-Barré syndrome

GBS is considered the most common immune-mediated inflammatory polyneuropathies ,also called acute inflammatory demyelinating polyradiculo neuropathy (AIDP), is an inflammatory disorder affecting the peripheral nervous system that is characterized by acute or subacute onset and typically monophasic course of the disease. Some patients with this disorder need hospitalization in an intensive care unit (ICU).

GBS characterized by variable degree of motor weakness, often presenting with quadriparesis, respiratory failure requiring endotracheal intubation with mechanical ventilation is common, affecting approximately 30% of patients. In addi tion, they present with sensory features, cranial nerve involvement, and autonomic disturbance. The majority of GBS patients have preceding respiratory tract infection or gastroenteritis.

incidence rates increase with age, especially in those 60 years or older. There is also a gender difference; males are affected more than females (1.5:1 ,GBS is the most common cause of acute paralytic neuropathy,It usually presents with an acute and rapid progression of symmetric weakness of limbs, hyporeflexia or areflexia, and with or without involvement of respiratory muscles or cranial nerve, The majority of patients have an infection before the onset of GBS that is within 4 weeks, most commonly in the form of respiratory tract infection or gastroenteritis.

Common Subtypes Of Guillain-Barré Syndrome :

Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)
Acute motor axonal neuropathy (AMAN)
Acute motor sensory axonal neuropathy (AMSAN)
Miller–Fisher syndrome (MFS)

What Are The Symptoms Of GBS :

Unexplained sensations often occur first, such as tingling in the feet or hands, or even pain (especially in children), often starting in the legs or back. Children will also show symptoms with difficulty walking and may refuse to walk. These sensations tend to disappear before the major, longer-term symptoms appear. Weakness on both sides of the body is the major symptom that prompts most people to seek medical attention. The weakness may first appear as difficulty climbing stairs or with walking. Symptoms often affect the arms, breathing muscles, and even the face, reflecting more widespread nerve damage, Most people reach the greatest stage of weakness within the first two weeks after symptoms appear; by the third week 90 percent of affected individuals are at their weakest.

In addition to muscle weakness, symptoms may include:

- Difficulty with eye muscles and vision
- Difficulty swallowing, speaking, or chewing
- Pricking or pins and needles sensations in the hands and feet
- Pain that can be severe, particularly at night
- Coordination problems and unsteadiness
- Abnormal heart beat/rate or blood pressure
- Problems with digestion and/or bladder control.

These symptoms can increase in intensity over a period of hours, days, or weeks until certain muscles cannot be used at all and, when severe, the person is almost totally paralyzed. In these cases, the disorder is life-threatening—potentially interfering with breathing and, at times, with blood pressure or heart rate.

How Is Guillain-Barré Syndrome Diagnosed?

based on clinical features criteria supported by cerebrospinal fluid (CSF) findings and nerve conduction studies, The common subtypes of GBS based on clinical features, pathological, and electrophysiological findings , In GBS, Deep Tendon Reflexes In The Legs, Such As Knee Jerks, Are Usually Lost. Reflexes may also be absent in the arms. Because the signals traveling along the nerve are slow, a nerve conduction velocity test (NCV, which measures the nerve's ability to send a signal) can provide clues to aid the diagnosis. There is a change in the cerebrospinal fluid that bathes the spinal cord and brain in people with GBS. Researchers have found the fluid contains more protein than usual but very few immune cells (measured by white blood cells). Therefore, a physician may decide to perform a spinal tap or lumbar puncture to obtain a sample of spinal fluid to analyze. In this procedure, a needle is inserted into the person's lower back and a small amount of cerebrospinal fluid is withdrawn from the spinal cord. This procedure is usually safe, with rare complications.

((Key diagnostic findings include)) :

1. Recent onset, within days to at most four weeks of symmetric weakness, usually starting in the legs
2. Abnormal sensations such as pain, numbness, and tingling in the feet that accompany or even occur before weakness
3. Absent or diminished deep tendon reflexes in weak limbs
4. Elevated CSF protein without elevated cell count.This may take up to 10 days from onset of symptoms to develop.
5. Abnormal nerve conduction velocity findings, such as slow signal conduction
6. Sometimes, a recent viral infection or diarrhea.

Predictors of intubation and ventilatory assistance in Guillain-Barré syndrome

1.Clinical evidence of inability to cough, unable to clear secretions, and neck weakness
2. Signs of impending respiratory failure such as tachy pnea, use of accessory muscles, inadequate chest rises, and paradoxical breathing with inadequate effort
3. Simple bedside assessment tool Single breath count (SBC) < 20 Unable to complete the sentences
4.Pulmonary function Vital capacity (VC) < 15–20 mL/kg , Maximum expiratory pressure (P max) < 40 cm H O , Maximum inspiratory pressure (P max) < 30 cm H 2 O , > 30% reduction in baseline VC, P max, and P max
5. Radiographic abnormalities include pulmonary infil trates or atelectasis
6. Arterial blood gas showing hypoxemia or hypercarbia or both

Criteria for diagnosis of Guillain–Barré syndrome

Features required for GBS diagnosis ....

Progressive motor weakness in the lower limbs and upper limbs & Areflexia

Clinical features strongly supporting GBS diagnosis

Symptoms and signs progression over days, lasting up to 4 wks

Relative symmetry of symptoms and signs

Mild sensory symptoms or signs

Cranial nerve involvement (bilateral facial weakness or other cranial nerves) Autonomic dysfunction

Absence of fever at onset

Recovery beginning 2–4 wks after progression ceases

CSF features strongly supportive of GBS diagnosis

Elevated CSF protein with cell count < 10 cells/µL

Electrodiagnostic features strongly supportive of GBS diagnosis

Electrodiagnostic features of nerve conduction slowing or block Findings suggestive of an axonal neuropathy.

The diagnosis of GBS is based on clinical features supported by cere brospinal fluid (CSF) examination and nerve conduction studies, Early course of the GBS requires meticulous monitoring and early initiation of: immunotherapy ,Plasma exchange (PE) and intravenous immunoglobulin (IVIg) are the proven therapies, and both have been shown to be equally effective , General supportive care is an import ant part of management of GBS , A multidisciplinary approach to prevent and manage potential complications in rapidly progressing GBS is important to reduce morbidity and mortality.

Myasthenia Gravis

Dr.DAWOOD

Myasthenia Gravis (MG) is a postsynaptic disorder of the motor plate (nicotinic) receptors of the skeletal muscle. It is purely an autoimmune disorder in which there are circulating acetylcholine receptor (Ach-R) or muscle specific tyrosine receptor (MuSk-R) **antibodies**, which result in skeletal muscle fatigue,It may be associated with other autoimmune diseases e.g. rheumatoid arthritis, thyroiditis, SLE.

Lambert-Eaton Myasthenic Syndrome (LEMS) is a disorder affecting presynaptic voltage gated calcium channels at the motor end plate. It is most commonly associated with para-neoplastic diseases e.g. small cell lung carcinoma.

Considerations

- Bulbar/skeletal muscle weakness resulting in ↑ risk of:
  - Aspiration
  - Perioperative respiratory failure
- Potential systemic complications:
  - Thymoma & possible anterior mediastinal mass
  - Myocarditis causing cardiomyopathy, atrial fibrillation, heart block
- Altered response to NMB (neuromuscular blocking) medications:
  - Very sensitive to NdMR (nondepolarizing muscle relaxants): avoid or use 1/10 normal dose with continuous monitoring
  - Resistant to succinylcholine (ED95 2.6X normal)
- Treatment: steroids, immunosuppressants, anticholinesterases
- Risk of perioperative myasthenic or cholinergic crises

Goals

- Minimize risk of aspiration (prophylaxis, RSI)
- Minimize risk of perioperative respiratory failure (judicious NMBs & opioids) & need for post-op ventilation
- Minimize risk of myasthenic or cholinergic crisis
- Optimize neuromuscular function

Conflicts

- RSI vs altered response to neuromuscular blockers
- RSI vs cardiac involvement
- RSI vs anterior mediastinal mass
- Magnesium for pre-eclampsia in pregnancy vs contraindicated due to muscle weakness

Myasthenic Crisis vs Cholinergic Crisis

❖ **Myasthenic Crisis:**

- Weakness exacerbated by infections, electrolyte abnormalities, pregnancy, surgery, emotional stress, drugs (aminoglycosides), or interruption of immunosuppressants
- Improvement with edrophonium (tensilon test):
  Tensilon test: 1.5 mg increments of edrophonium to 10 mg total (should get better in about 2 minutes)
- Consider elective intubation if vital capacity < 20cc/kg or max. inspiratory force worse than -30 cmH2O
- Consider PO or IV dose of pyridostigmine:
  - PO: 30-120 mg/day, onset 15-30 min, peak 2 hrs, duration 4 hrs
  - IV dose is 1/30 of PO dose
- Alternative treatment is neostigmine 0.5-2.5 mg IV/SC q1-3 hr. titrated to response (max = 10mg/24hr)
- Neurology consult for management (steroids)

❖ **Cholinergic Crisis:**

- Due to excessive cholinesterase inhibitors
- Symptoms of acetylcholine excess (SLUDGEBBB) : **s**alivation, **l**acrimation, **u**rination, **d**iarrhea, **G**I symptoms , **e**mesis, **b**radycardia, **b**ronchorrhea, **b**ronchospasm
- Distinguish by giving edrophonium (tensilon test) which improves symptoms if myasthenic crisis & worsens symptoms if cholinergic crisis
- Treatment includes ETT, atropine & cessation of cholinesterase inhibitors until the crisis is over.

Preparation for Surgery Requiring the Use of Neuromuscular Blockers (NNB)

Where possible for planned / elective procedures advice / involvement of a neurologist is desirable. Patients should be operated on in the morning, preferably first on the list (as their muscle function is at its strongest), Where possible, any elective procedure can be deferred until treatment is optimised by the neurologist and drugs associated with exacerbation of MG stopped or modified.

Patients Listed for Surgery Should Receive the Following:

Ongoing anticholinesterase therapy upon arrival in the anaesthetic room. If unable to take internal medication (e.g. for emergency surgery or for elective oesophageal or stomach surgery), then pyridostigmine can be given IV as 1 / 30th of current oral dose if required). Be aware, however, that pyridostigmine can make some types of myasthenia worse. Alternatives are IV neostigmine. Steroids need to be supplemented as IV hydrocortisone or dexamethasone if the patient is currently on >20mg / day for 3 weeks or more. Ongoing immunosuppressant treatments can be stopped the night before surgery (e.g. azathioprine, mycophenolate, cyclosporine, tracolimus).

Where intubation of the airway is required in the emergency setting, an awake fiberoptic intubation under local anaesthesia is preferable. NMB use for emergency intubation is associated with need for postoperative ventilation (e.g. suxamethonium and rocuronium in LEMS, rocuronium in MG).

Intra-operative management of NMB agents can be difficult in terms of predicting the dose, duration and the use of reversal agents (which are anticholinesterases e.g neostigmine). The latter can precipitate a cholinergic crisis. A new reversal agent, Sugammadex, appears to be safe and effective for some NMB agents (rocuronium and vecuronium).

It is advisable to use neuromuscular monitoring (relaxagraph or train of four-TOF) to assess muscle function before and after reversal agent is given (where a TOF <82% of maximum - it is unlikely the patient will be adequately reversed and therefore will need postoperative ventilation).

Concomitant intra-operative drugs can lessen the success of reversal.

Antiobiotics (erythromycin, clarithromycin, gentamicin, vancomycin, ciprofloxacin, clindamycin)

Cardiovascular Drugs (atenolol, metoprolol, labetalol, propranolol, magnesium, verapamil)

Anaesthetic Agents (Lignocaine, prilocaine, bupivacaine, isoflurane, halothane)

Antiepileptic / Antipsychotic Drugs (haloperidol, phenytoin)

Factors Associated with Post-Operative Ventilation / Need for Intensive Care / Post-Operative Complications Are:

- Co-existing respiratory disease (chronic obstructive airways disease)
- Previous myasthenic crisis
- A low forced vital capacity (FVC) < 2.1 litres
- Existing features of bulbar symptoms (dysphagia, poor phonation)
- Concomitant active infection
- Post-operative thymectomy patients
- Predicted intra-operative blood loss > 1000mls
- Concomitant administration of intra-operative drugs listed above
- TOF count<82% of maximum

Post-Operative Care

If any factors above are met, then it is not advised to extubate (remove the endotracheal tube) in recovery - and the patient should be transferred to ITU for ongoing ventilation.

If the patient is to be extubated then it is advisable to perform thorough suctioning and lavage of the tracheal-bronchial tree prior to removal of the endotracheal tube. The patient should be nursed in recovery (post anaesthetic care unit) for > 2 hours to ensure all residual anaesthetic agents are clear, and reversal agents are still effective. It is particularly important that the use of reversal agents does not lead to a cholinergic crisis within the first 30 minutes. Patients should then be nursed on a high dependency unit (HDU) preferably attached to the ITU.

Obstetric Patients

These can be a very high risk group for complications, but in essence:

- Those in labour who need an epidural, should receive a very low concentration local anaesthetic (preferably 0.1% bupivacaine or 0.2% ropivacaine with a liquid soluble opiate (fentanyl or diamorphine) as a low volume infusion
- Those in need of an instrumental delivery can receive a top up of 0.25% bupivacaine, but special attention is required to ensure the block height doesn't go above T10 and affect the respiratory muscles
- Those in need of Caesarean section cannot safely tolerate the high block levels required and should have a general anaesthetic and post-operative HDU or ITU care

Post-Operative ITU Care

It is important to continue anticholinesterase drugs (pryidostigmine and normal pre-operative doses. Also immunosuppressant agents should be restarted with neurology advice. Drugs associated with potential exacerbations must be stopped. Vigilance to high incidence of super imposed infections and appropriate antibiotic therapy and use of antithrombotic drugs (enoxaparin) to reduce the risk of deep vein thrombosis and coronary events).

Patients must receive frequent tracheo-bronchial lavage prior to extubation. Criteria for extubation are a successful spontaneous breathing trial of > 30 minuted associated with tidal volumes of > 7mls / kg and vital capacity of > 15mls / kg plus a peak inspiratory pressure of > than -25cm H2O (i.e. -26cm to -35cm).

Myasthenic Crisis

It occurs in 2-3% of all MG patients but is the first presentation in 20% of cases. In a first presentation, it is important to differentiate it from other peripheral neurological emergencies below:

- Cholinergic crisis (normally existing MG patients)
- Guilliain-Barré syndrome
- Severe depressive psychosis
- Syringomvelia
- Acute botulism
- Polio
- Acute hypothyroid
- Mitochondrial myopathies

In those with MG / LEMS, precipitating factors can be:

- Stress (post-operative surgery)
- Hypothermia (extreme physical activity, bright sunlight)
- Menstruation
- Infection
- Drug induced (some of which have been listed but also eye drops including timolol, tropicamide and betaxolol

ITU Management

Any patient in which lung funtion testing shows a vital capacity of < 20mls / kg (ideal body weight) with a reduction in peak inspiratory pressure of less than - 25cm H2O should be referred to ITU.

Criteria for intubation and ventilation include:

- PaCO2 > 6KPa associated with a respiratory rate > 20 / min
- Bulbar weakness with poor cough and aspiration on chest X-ray
- Any evidence of cholinergic crisis (often due to overdosing of anticholinesterase or inappropriate reversal following anaesthesia with NMBs) - weakness and excessive SLUDGE (salivation, lacrimation, urination, defecation, gastrointestinal distress and emesis)

Patient needs emergency intubation and ventilation. Exclude precipitants including known:

- Stop pyridostigmine or other anticholinesterase drugs
- Send following tests - complement and immunoglobulin levels, AChR and MuSK antibody levels, creatinine kinase and thyroid function tests

If clear evidence of myasthenia crisis, then start the following:

- Plasma exchange of IgC selective plasmapheresis (3-5 litre exchanges per day for 10-14 days) - or pooled human immunoglobulin (except in IgA disease) at 400mg / kg / day for 5 days
- Steroids at 60-80mg / day of prednisolone for 2 weeks (note that this may worsen MG within the first 5 days)
- Liaise with neurologist regarding long-term immunosuppression
- Liaise with thoracic surgeons if thymic mass is present

If clear evidence of cholinergic crisis then start the following:

- Glycopyrollate - 1mg 4 hourly
- Hyoscine
- Loperamide

For patients with MG or LEMS, the treatment options are similar but patients with LEMS are less likely to respond.

Criteria for extubation are a successful spontaneous breathing trial of > 30 minutes associated with tidal volumes of > 7mls / kg and vital capacity of > 15mls / kg plus a peak inspiratory pressure of > than -25cm H2O (i.e. -26cm to -35cm).

However, there is a need for re-intubation in 27% of patients, so careful consideration needs to be taken early in the ITU stay as to timing of tracheostomy (especially in the presence of a thymic mass).

For all these patients despite treatment above there is 5% mortality, even when managed on ITU (higher in LEMS) with an average of 2 weeks need for intubation and ventilation. Such patients are at high risk of developing super-imposed infection (lungs / kidney / colon) as well as thrombo-embolic phenomena (pulmonary emboli) and heart failure (takotsuba cardiomyopathy).