# FUNDAMENTALS OF ICU CARE


STATE INSTITUTE OF HEALTH AND FAMILY WELFARE
UTTAR PRADESH

# ACKNOWLEDGEMENTS


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# MESSAGE

**Shri Brajesh Pathak**
**Hon'ble Deputy Chief Minister**
**Minister of Medical Health and Family Welfare**
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Teamwork in the intensive care unit (ICU) refers to the leadership, decision-making, communication, and coordination behaviors used by multidisciplinary team members to provide patient care. Patient safety research has demonstrated the importance of effective teamwork for ensuring positive patient outcomes in the ICU. Poor communication during rounds and handovers (or handoffs) is frequently cited as a cause of medical error, and units with high levels of nurse-doctor collaboration have improved patient mortality rates and reduced average patient length of stay.

All this is dependent upon the level of exposure the Medical Officers have in managing intensive care Units settings. ICU is a complex, high-risk, and stressful setting, and a well trained team can manage better results.

In Uttar Pradesh state, we have to work holistically not only in treating cases but also finding evidence-based solutions to the cases in various stages of the disease. The Critical Care Medicine and ICU services have to contribute to each other to improve the health care services of the state. It is desirable that state develops tailored made Continuing Medical Education modules to caret the medical needs specific to its inhabitants.

Considering the above stated facts, Continuing Medical Education (CME) on Fundamentals of ICU Care is a minimum standard of critical care to be offered to all. Through this module on ICU Care, Medical Officers in Provincial Health & Medical Services in Uttar Pradesh, will be exposed to much needed training and quality care, thus ensuring that the end product i.e. intensive care is available at the remotest of location. It is high time that Medical Officers start working holistically on Critical and Intensive Care to improve the health care outcomes.

I wish the team of State Institute of Health & Family Welfare, Uttar Pradesh and subject matter experts to continue developing such module on CME for the benefit of medical officers in Provincial Health & Medical Services in Uttar Pradesh that ultimately benefit their patients too.

**(Brajesh Pathak)**

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# MESSAGE

**Shri Mayankeshwar Sharan Singh**

**Hon'ble State Minister**
**Medical Health and Family Welfare**
**Department Government of Uttar Pradesh**

The demand for critical care services in developing countries is high due to the increased burden of illnesses which culminate in critical illness and require critical care. Critical care is a specialty which serves diverse needs of patients with actual or potential life-threatening organ dysfunction. Patients requiring critical care have complex needs and receive multiple therapies which require technical and/or artificial life support. The complex environment and inability to operate the equipment triggers stress among team who provide advanced care. Therefore, critical care staff is expected to possess appropriate knowledge and skills to cope with the challenges of critical illness in an environment that is fast evolving in terms of science and technology.

In the absence of pre-hospital care, critically ill patients die or their conditions worsen before they reach the hospital. These circumstances create enormous challenges for health professionals who are at the frontline of critical care service delivery.

In the light of above mentioned facts, it becomes important that the population of Uttar Pradesh is provided with all the possible medical intervention at the PHC/CHC level. Continuing Medical Education (CME) on on Fundamentals of ICU Care, for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh is one of the several useful interventions in the right direction.

I am happy that the team at State Institute of Health & Family Welfare, Uttar Pradesh along with the experts from the field, have come up with such an intensified and detailed CME for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh.

I wish team at SIHFW success in their endeavors of aiding an improved health service delivery system through such CME on Fundamentals of ICU Care.

**(Mayankeshwar Sharan Singh)**

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# FOREWARD

**Shri Partha Sarthi Sen Sharma**

**Principal Secretary**
**Department of Medical, Health and Family Welfare**
**Government of Uttar Pradesh**

ICU teams manage risk, complex technologies, changeable workloads, and uncertainty. Fatigue and stress are known to negatively influence performance in the ICU, and non-technical factors such as team communication, situation awareness, and decision making frequently underlie error. Teams in acute medicine frequently encounter emergency situations. They must tolerate high levels of risk and develop an ongoing understanding of the complex interactions between medical treatments and patient physiology.

However, unlike other work environments, ICUs consist of large medical and nursing teams that care for numerous patients simultaneously. Patients usually enter the ICU in an already critical state. Problem solving is key, and teams must diagnose poorly understood patient illnesses, stabilize the condition of patients, and stimulate recovery. Team members have minimal prior knowledge of patient histories, and patient populations are diverse in terms of demographic background, risk factors, and underlying pathology.

Having considered the various dimension dealing with the critical care situation of the population, it becomes extremely important to provide early diagnosis and treatment at the ground level for the patients.

This Continuing Medical Education (CME) on Fundamentals of ICU Care, for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh in an important tool for not only improving health care practice, skill up-gradation and knowledge enhancement of Medical Officers in Provincial Health & Medical Services in Uttar Pradesh but also reducing the screening and intervention time for the patients.

I wish the team of State Institute of Health & Family Welfare, Uttar Pradesh and subject matter experts from King George's Medical University (KGMU), Lucknow, Uttar Pradesh, to continue developing such module on CME for the benefit of Medical Officers in Provincial Health & Medical Services in Uttar Pradesh that will ultimately benefit their patients too.

**(Partha Sarthi Sen Sharma)**

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# MESSAGE

**Dr. Deepa Tyagi**

**Director General**
**Medical and Health Services**
**Uttar Pradesh**

In India, to accommodate for the rapid fluctuations in the number of patients with COVID-19, alongside Department of Health & Family Welfare other healthcare organizations have been forced to optimize resource and staff allocation procedures. Global pandemics and other unprecedented events have increase in the demand for intensive care services. Building competence and confidence is an essential principle for providing quality healthcare.

Pandemic and other health care challenges have posed multiple challenges, including devising new ways of working and rapid development and delivery of training. In countries with the limited health-care budget, we have to work holistically not only in handling emergency cases but also finding evidence-based solutions to the cases in various stages of natural history of the disease.

Continuing Medical Education (CME) on Fundamentals of ICU Care is an effort in improving the minimum standard of intensive care to all who are need and to alleviate the current state of ICU services in Provincial Health & Medical Services.

Considering the above stated facts, this module on Continuing Medical Education (CME) on Fundamentals of ICU Care, for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh, State Institute of Health & Family Welfare, Uttar Pradesh with the help of Subject Matter Experts has provided a comprehensive, coherent and insightful module for Medical Officers to deal with intensive care and emergency.

I wish the team of State Institute of Health & Family Welfare, Uttar Pradesh and subject matter experts for such a commendable job.

**(Dr. Deepa Tyagi)**

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# MESSAGE

**Dr. Brijesh Rathor**

**Director General**
**Medical Health and Family Welfare**
**Uttar Pradesh**

The intensive care unit (ICU) is a complex and rapidly evolving environment with many challenges for its health care providers. Although ample evidence indicates that care offered by subspecialists trained in critical care medicine (CCM) improves patient outcomes at a lesser cost, most ICUs in the country are staffed by non-CCM physicians. This situation is likely to persist or increase due to the projected shortage in the number of intensivists in critical care.

Despite this growing need, CCM is extremely challenging. The severity and unpredictability of patients' illnesses, focus on patient safety and quality of care are some factors that may compromise intensive care.

These stresses on the current health care system may generate adverse impact in providing optimal patient care. Therefore, an efficient, effective, and standardized system of continued medical education is the need of the hour to achieve the dual goals of providing high-quality patient care and excellent skill enhancement.

Considering the above stated facts, this module on Continuing Medical Education (CME) on Fundamentals of ICU Care, State Institute of Health & Family Welfare, Uttar Pradesh with the help of Subject Matter Experts has provided a comprehensive, coherent and deep insight for intensive care for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh.

I congratulate the faculties at State Institute of Health & Family Welfare, Uttar Pradesh in developing this module. This module addresses the need to have a holistic view on public health.

**(Dr. Brijesh Rathor)**

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# MESSAGE

**Dr. Shailesh Kumar Srivastava**

**Director General (Training)**
**Medical Health and Family Welfare**
**Uttar Pradesh**

The intensive care unit (ICU) is a complex and stressful environment posing many challenges for healthcare providers. Although ample evidence indicates that care offered by subspecialists trained in critical care medicine improves patient outcomes, the scarcity of workforce and lack of trained critical care staff posed a significant challenge while handling the disease outbreak and pandemics. This was evident even in high-income countries with high-quality medical care and large economies.

Reflecting upon the fact that there is an urgent need to train non-intensivists in critical care, which could have been of utmost utility during pandemics like COVID-19, Continuing Medical Education (CME) on Fundamentals of ICU Care is meant to enhance competencies of Medical Officers working in the Provincial Health & Medical Services peripheral units by exposing them to various treatment interventions in great detail.

This Continuing Medical Education (CME) lays emphasis on the physical presence of the Medical Officer and the Subject Matter Experts rather than "virtual". More so, hands-on training may also allow the Medical Officers to interact with the trainer on an "as and when" required basis and clear their doubts. It may also help to better understand the ICU paraphernalia with which they are not well versed.

In addition to improving technical competencies this Continuing Medical Education (CME) on Fundamentals of ICU Care also highlight the aspects of quality care, thus assisting patients in understanding their choices for medical treatment.

Through this module on Continuing Medical Education (CME) on Fundamentals of ICU Care, for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh, State Institute of Health & Family Welfare, Uttar Pradesh with the help of Subject Matter Experts has provided a comprehensive, coherent and deep insight on Intensive care. I appreciate their efforts and wish them success for future.

**(Dr. Shailesh Kumar Srivastava)**

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# ACKNOWLEDGEMENT

**Dr. Rajaganapathy R.**

**Director**
**State Institute of Health and Family Welfare**
**Uttar Pradesh**

There is growing evidence that intensive care units (ICUs) have evolved from observation wards into units that comprehensively care for critically ill patients with complex multisystem illnesses. There is a current need for Medical Officers with exposure in Critical Care Medicine (CCM) with the aim of better understanding and delivering optimal patient care.

Specialist, generally have unique insight into the importance of additional critical care training and its relevance to ICU patient care in their practice but non-intensivists are not exposed to critical care training regularly. Skills that are most fundamental to patient care in contemporary ICUs like ventilator management, multisystem organ failure management, airway management, and end of life care are not adequately emphasized in general practice.

As the situation stands, along with improving health infrastructure, skill up-gradation and knowledge enhancement of Medical Officers in Provincial Health & Medical Services in Uttar Pradesh is equally important, and hence this Continuing Medical Education (CME) on Continuing Medical Education (CME) on Fundamentals of ICU Care will allow Medical Officers in Provincial Health & Medical Services in Uttar Pradesh, to update their knowledge base and expose them to the concurrent practices in intensive care.

This module on Fundamentals of ICU Care is a result of great efforts taken by the faculty of State Institute of Health and Family Welfare and the team of experts which has developed this very useful module. I am especially thankful to Dr. Zia Arshad, Additional Professor (Critical Care), Department of Anaesthesiology and Critical Care, King George's Medical University (KGMU), Lucknow for making this module an exceptional tool for Medical Officers in the state.

**(Dr. Rajaganapathy R.)**

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Section

1

# Fundamentals of ICU Care

# 1. Introduction

ICU is the area where professional excellence is required in each and every aspect of patient care Intensive care unit (ICU) is also known as Critical care unit (CCU), intensive therapy unit or intensive treatment unit (ITU). Intensive care unit is well organized and well-equipped health care facility where trained health care personnel are committed to provide intensive care medicine.

Intensive care medicine also termed as critical care medicine (CCM) is special medical facility, related with care of critically ill patients. These patients have life threatening illness which needed constant care, close supervision by life support equipment and medication in order to stabilize the patients for normal body functioning.

**ICU staff**: Team of ICU comprises of trained physician, intensivist, nursing, technician and physiotherapist. There is more staff to patient's ratio and easier access to advanced medical resources in comparison to general hospital ward. Two types of ICU are in practice open and closed model ICU.

**Infrastructure of ICU:** The infrastructure of ICU includes all medical equipment and resources for support of different organ and system.

A multipara monitors for continuous monitoring and recording of all Vitals.

Mechanical ventilator for assisting the breath through endotracheal tube or tracheostomy tube

Difficult airway cart has all necessary instrument including video laryngoscope, flexible fibreoptic bronchoscope, emergency surgical airway access kit etc for airway management in ICU patients

Dialysis unit: Special machine for haemodialysis, hemofiltration and continuous renal replacement therapy

Defibrillator for management during cardiopulmonary resuscitation

Imaging device like ultrasonography and portable x ray machine

Besides this another necessary item includes ABG analyser, thromboelastography (TEG) machine

**Types of intensive care units**: Hospital or any institute having common ICU according to their speciality like:

- Coronary intensive care unit (CCU or sometimes CICU) for heart disease like myocardial infarction, heart failure and cardiac arrest
- Medical intensive care unit (MICU) for life threatening medical emergency
- surgical intensive care unit (SICU) for surgical and trauma case
- Paediatric intensive care unit (PICU) for all child of one month or older for serious condition like acute bronchial asthma, diabetic ketoacidosis, respiratory failure, post-surgical and trauma patients
- Neonatal intensive care unit (NICU) for newborn up to 28 days (Neonatal period) for premature baby with complication, congenital cardiac, neurological, respiratory abnormality etc.
- Neuroscience critical care unit (NCCU) for post neurosurgical patients, brain stroke, brain tumour and post neuro trauma patients.
- Geriatric intensive-care unit, (GICU) a special unit for management of critically ill elderly patients.

  High dependency unit (HDU): it is an intermediate ward between general ward and ICU. Patients are kept here for close observation if not possible in general ward and usually attached to any ICU.

# 2. General Working in ICU

- Any critically ill patient should not be left unattended by nursing staff.
- All critically ill patients should have continuous ECG monitoring.
- All alarms must be on, and the alarm limit be appropriately selected.
- Any change in rate or rhythm should be informed immediately.
- In hemodynamically stable patients' vitals should be recorded at least once every hour.
- Core temperature (rectally, tympanic, pulmonary artery, esophageal, Foley) recorded at least every 6 hours.
- In neurological patient frequent neurological assessment required and recorded at least 4 hourly using GCS.
- Frequent posture change (every 2 hours) required if not contraindicated.
- Skin examinations are also recorded every 4 hours and if turning is contraindicated pressure point will be relieved.
- Chest physiotherapy to be ensured on an 8 hourly basis.
- ICU Patients should have minimum 3 sessions of limb physiotherapy and passive exercises to avoid the muscle contracture and reduce the incidence deep vein thrombosis.
- Intubated patients have increased risk for developing oral ulcer, so every intubated patient should have oral care 2-3 times a day with brushing of teeth 12 hourly.
- Compression stockings and intermittent pneumatic compression devices should be removed for 30 minutes every 8 hour.
- The patient who does not have bowel movement should be checked for impaction and recorded.
- Procedures should be explained to the patient.
- Maintain normal sleep wake pattern.
- Emotional support and necessary information will be provided to the family members.
- The ICU area should be regularly cleaned and properly maintained. Equipment used in ICU should be properly serviced and record should be maintained. Equipment should be routinely checked and cleaned.
- Infection control protocol should be followed for isolation area.
- Waste disposal should be done as per BMW (Bio Medical Waste) management guidelines.
- All medications, indwelling, drains, infusions should be adequately labeled. Previous records including drugs and investigations should properly reviewed at the time of admission.
- Advice by visiting doctors should duly informed to the intensivist in charge and if any changed has to be done, should be approved by the intensivist before implementation.
- It is better to make a register for the entry of narcotic medications and drugs should be kept safely.
- Proper and detailed handover is the key for successful transfer of responsibility to the next shift.
- Visiting hours should be specified and always try to limit the traffic in ICU, allow not more than two visitors at a time.
- One of the family members is designated and should be briefed about the patient's condition at regular interval and any significant development in the patient's medical condition.
- Children visiting ICU should be discouraged and if necessary should be with proper care.
- Any bad news should be broken with empathy and care.
- It is necessary to record the patient weight daily as it required for the calculation of drug dosage, fluids and nutrition.
- Universal precaution and infection control protocol should be followed while attending the patients.

# 3. Admission, Discharge and Triage Criteria

Requirement of ICU admission are exceeding the ICU facility and resources. Admission in the ICU always should be based on resources availability, severity of illness and potential benefit of intervention and medication. Principle of admission and discharge is based on and rationale decision making.

## ICU Admission criteria

Patients in ICU should be admitted on concept of better utilization of ICU resources with favourable outcomes. Following condition of different system warrants for admission in ICU:

**(a) Neurological:**
- Any neurological disorder with low Glassgow coma scale.
- Acute status epilepticus with altered sensorium with risk of airway obstruction.
- Some neuromuscular disorder with respiratory failure e.g. Guillen barre syndrome, Myasthenia gravis.
- Brain dead patient planned for organ retrieval.

**(b) Cardiovascular:**
- Cardiogenic or obstructive shock
- Any life threatening brady or tachyarrhythmia
- Any hypertensive emergency with fatal complication
- Post surgical of cardiac surgery or noncardiac surgery of cardiac patients.

**(c) Respiratory:**
- Acute respiratory failure (type I or II)
- Acute massive haemoptysis
- Acute pulmonary embolism

**(d) Renal:**
- Acute or chronic renal failure presenting with intoxication or electrolyte imbalance and need renal replacement therapy

**(e) Endocrine:**
- Diabetic patients with diabetic ketoacidosis or hyperosmolar hyperglycaemic state
- Endocrinal disorder with hemodynamic instability.

**(f) Gastrointestinal:**
- Gastrointestinal bleeding with hypovolemic shock
- Acute or chronic liver failure with encephalopathy

**(g) Haematology:**
- Severe bleeding diathesis resulting in hemodynamic instability
- Haematological malignancy with multiorgan failure

**(h) Obstetric:**
- Haemorrhagic complication (antepartum and postpartum)
- Hypertensive disorder of pregnancy
- Amniotic fluid embolism

**(i) Miscellaneous:**
- Any types of shock with multi-organ dysfunction syndrome (MODS)
- Polytrauma patient with vital injury
- Environmental injury (Severe poisoning, lightning, hyper/hypothermia, electrocution injury etc.)
- Post surgical patients requiring elective ventilation and monitoring

**Patients generally not appropriate for ICU admission:** Patients with end stage cardiac, respiratory or liver disease with no option for transplant

- Irreversible brain injury or patients with coma in vegetative state

- Metastic cancer with unresponsive to chemotherapy or radiotherapy
- End stage renal disease with not possibility for renal transplant.

## Discharge policy:

- Decision of discharge of ICU admitted patients will be taken by treating physician after considering all discharge criteria.
- Primary team/parent team should be informed and proper summary should be prepared before shifting the patients
- Concerned family person and attendant should be informed and counselled further care and follow-up

**Discharge criteria:**

- Patients who are hemodynamically stable with no or minimal vasopressor support
- Better physiological condition and no longer ICU care and monitoring is needed.
- Neurological status is stable enough to maintain airway reflexes intact.
- Mechanical ventilation is not required
- Oxygen support requirement is less than 60% for maintain SpO2 >90
- Patient on permanent tracheostomy need minimum care for tracheostomy care

## Triage for ICU admission

In view of limited resources and bed availability ICU admission should always be based on priority basis and following factor is considered in triaging

1. Priority determination based on severity of illness
2. Requirement of life supportive therapy
3. Potential benefit from intervention
4. Patient's diagnosis and prognosis
5. Availability of clinical expertise
6. Availability of ICU bed and resources

**Priority in admission:** The priority is based on two factors, first is requirement of ICU care that depends on severity of illness and second is possibility of benefiting from ICU admission.

Priority is categorized from priority 1 (those who will benefited maximum) to priority 3 (those who will benefited least)

**Priority 1:** Those patients who are critically ill, with multiorgan failure and unstable but high probability of benefit from admission.

**Priority 2A:** Those patients who are acutely ill but relatively stable. This patient may have organ dysfunction, need intensive monitoring but can be managed in intermediate ward, HDU and postoperative care unit.

**Priority 2B:** Those patients who are critically ill, with multiorgan failure and unstable but low probability of benefit from admission.

**Priority 3:** Terminally ill patients with no possibility of recovery from admission

# 4. Investigations and Microbiological Surveillance

All ICU patients must be evaluated after admission and investigation is one of the integral parts of assessment. Investigation is laboratory parameter of blood, urine and important body fluid and imaging study of like ultrasonography, CT scan, MRI etc. Investigation is done on day of admission and then daily throughout ICU stay.

**At the time of admission:**

- Complete blood count (hemoglobin, total & differential leucocyte count, platelet count).
- Kidney function test (blood urea, serum creatinine and s. electrolytes-Na, K, Ca)
- Liver function test.
- Prothrombin time (PT)/ international normalized ratio (INR).
- Blood glucose (random).
- Viral marker.
- ABG (arterial blood gas).
- Electrocardiogram (ECG).
- Almost all of the tests are repeated in daily routine test for daily to see treatment response and for decision making of any new plan.

**Additional investigation (on the day of admission or further):**

- Microbiological screening: Culture & sensitivity of blood, urine, ET aspirate (if on mechanical ventilation) and other body fluid (like Pus, vaginal if indicated)
- X-ray chest (indicated only after placement of central line in neck, patient on mechanical ventilation or history of lung pathology present)
- Some special investigation like Trop-t, Pro-BNP, S. LDH, fever profile, iron profile, CSF, pleural fluid, ascitic fluid, USG, CT, MRI etc.is also done in special case for diagnosis and treatment purpose.

**Microbiological Surveillance:**

In one study the most frequently isolated microorganism was methicillin-resistant Staphylococcus aureus (MRSA) (24.1%) followed by P. aeruginosa (17.0%) and Acinetobacter spp. (17%). [(1)]

MRSA screening is indicated in the following conditions:

- History of hospital stay of more than 5 days.
- Patients with previous positive cultures for MRSA either in the blood, tracheal aspirate, or urine.
- Patients transferred from other hospitals.
- Patients have a history of prolonged history of hospital admission and care.
- CKD patients on regular dialysis
- Sample for tracheal aspirate should be sent for C&S once a week in intubated or tracheostomized patients.

References:

1. Akcam FZ, Karaaslan D, Dogan M et al Microbiological surveillance in the Intensive Care Unit: A tertiary hospital experience. Med Sci Monit, 2006; 12(2): CR81-85

# 5. Monitoring in ICU

Monitoring is continuous assessment of patient's physiological condition and Vitals sign. As ICU patients' status are rapidly changing so a continuous monitoring is always recommended.

The purpose of monitoring to find a change in status and promptly intervention for stabilization. Monitoring is always done in following points:

**General monitoring:**

Watch the patient from head end to foot end of bed and observe: -

- General appearance of patient.
- Position of patient and covering.
- Surrounding of patients including all monitor, ventilator machine and other necessary equipment.
- Endotracheal tube, tracheostomy tube, CVP line and other indwelling catheter.
- Any discharge, bleeding or oedema.

**Cardiovascular system:**

- **Heart rate and pulse rate**: It should be checked at same time, any discrimination in both rates suggest any arrythmia. Look for tachycardia (HR>100) or bradycardia (HR<60) and correlated with patient condition.
- **Blood pressure (BP) –**
  It can be measured by invasive and non-invasive method. Invasive method prefers for hemodynamic unstable patient to know beat to beat variation of BP.
  Commonly radial, ulnar, dorsalis pedis artery and less commonly femoral or brachial artery used for invasive purpose.
  Systolic, diastolic and mean blood pressure is measured. Normal range of BP are systolic 90-140 mm hg, diastolic BP 60-1100 mm hg and mean 65-105 mm hg.
- **Central venous pressure:**
  Pressure in central venous compartment of the body with normal range 8-12 cm $H_2O$. Prefer central vein like internal jugular, subclavian vein (femoral vein is an alternative when unable to access central vein). Initially CVP used as guide for intravenous fluid therapy but obsolete now. Raised CVP is seen in many clinical conditions like heart failure, hypervolemia, pulmonary embolism, valve abnormality, pulmonary arterial hypertension, pleural effusion, tension pneumothorax and mechanical ventilation with high PEEP and during forced exhalation. Low CVP is found in hypovolemia, distributive shock and during deep inhalation.

**Nervous system:**

Neurological monitoring is done with following parts:

- **Consciousness:** Basic two clinical tools are used for evaluation of nervous system (1) Glassgow coma scale (GCS) (2) AVPU

(1) **Glassgow coma scale [(1)] (GCS)-**
Three response eye, verbal and motor response are assessed by voice and painful stimuli. (Figure No. 1) Maximin GCS score is 15 and minimum is 3. GCS less than 12 indicate neurological involvement and GCS ≤ 8 indicate unprotected airway due to loss of reflex and patient airway must be secured.

(2) **AVPU [(2)]-** It is acronym of four-word Alert, Verbal, Pain and unresponsive. (Figure No. 2) It is usually assessed in emergency protocol not suitable for ICU patients.

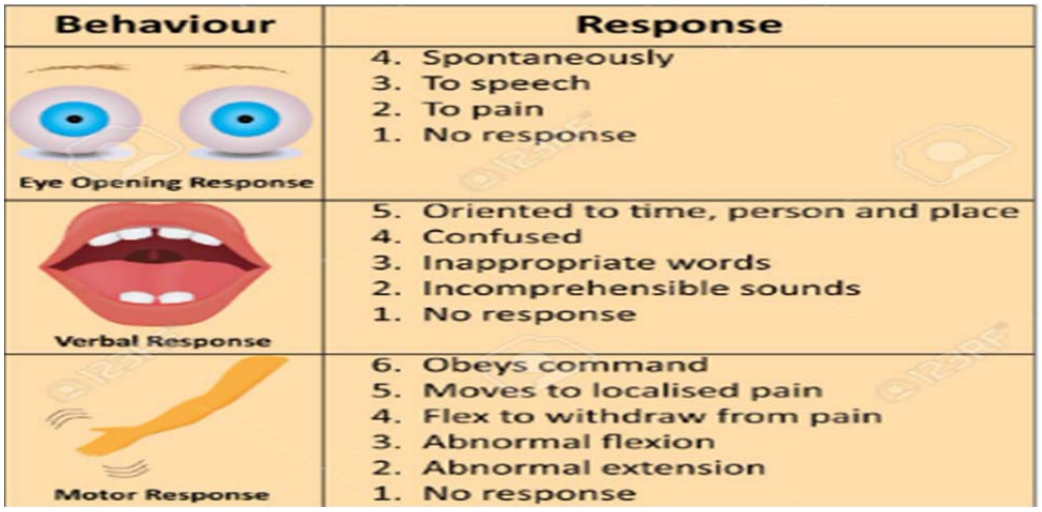


Figure No. 1: Glasgow coma scale

- **Pupil assessment-**

Bilateral assessment of Pupil diameter and reaction to light done to determine brain functional status.
Normal size and react- normal.
Constricted pupil- seen in miotic drug, opioid and pontine haemorrhage.
Unequal size- unilateral brain lesion.
Fixed and dilated pupil- in irreversible brain injury and use of cycloplegic drugs.

- **Doll's eye test** [3] **(**oculocephalic reflex**)-** done to see lesion brain stem (mid brain or pons). Exclude any cervical spine injury before performing test. Rotate the head one side and eyes moves to opposite side indicate positive dolls eye test means intact brain stem. (Figure No.3)
- **Abnormal posture and movement-** Muscle stiffness and rigidity indicate some neurological disease. Abnormal movement like seizure may be due to some stroke (haemorrhage or infarct), hypoxic insult or electrolyte imbalance.

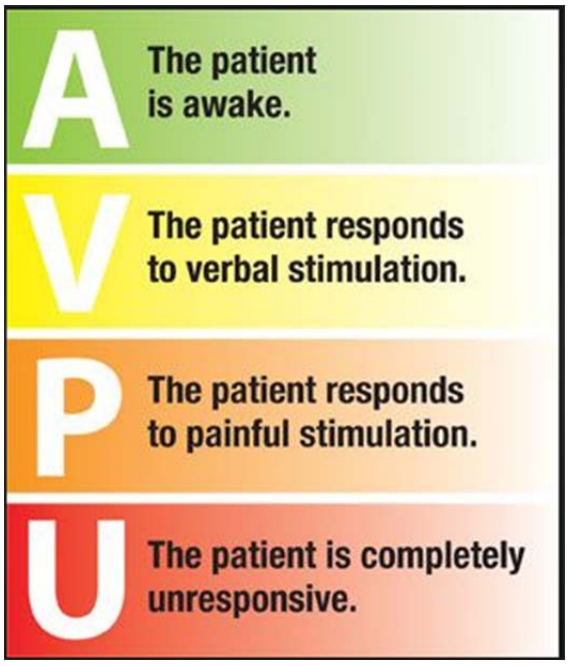


Figure No. 2: AVPU assessment

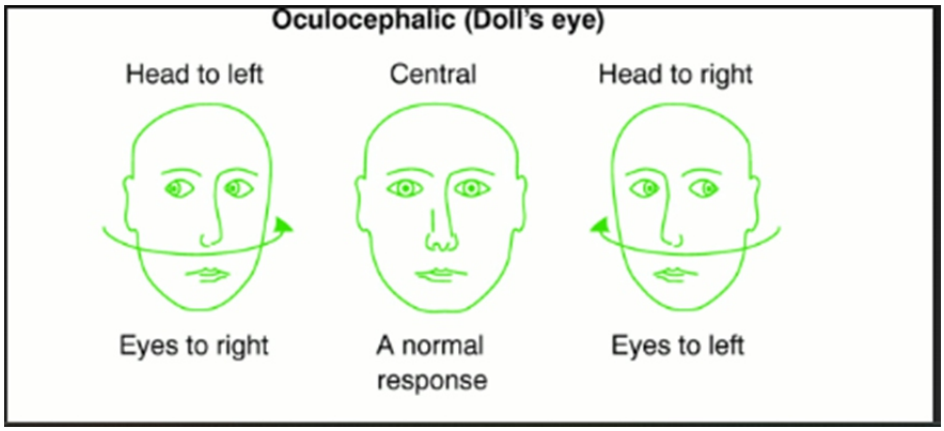


Figure No. 3: Oculocephalic reflex

**Respiratory system:**

It is assessed by respiratory rate and saturation.

- **Respiratory rate:** It is very good indicative of lungs pathology, cardiac disease, and other medical condition. Normal RR is 14-18/ minute. Increased RR is found in ICU patients is usually due to fever, anaemia, pain, hypoxia, hypercarbia, metabolic acidosis, setting of low tidal volume on ventilator. It is decreased in some neurological disease, medication (CNS suppressant drugs)
- **Saturation (SpO2):** It is measured by pulse oximeter and normal range is 95-100%. SpO2 < 94 indicates some respiratory or cardiovascular disease. Spurious low value in patients having nail paints, shivering and cold extremity. Flow-Volume loop and pressure volume loop on ventilator helps in estimating patient's lung compliance, obstructive, restrictive pathology.

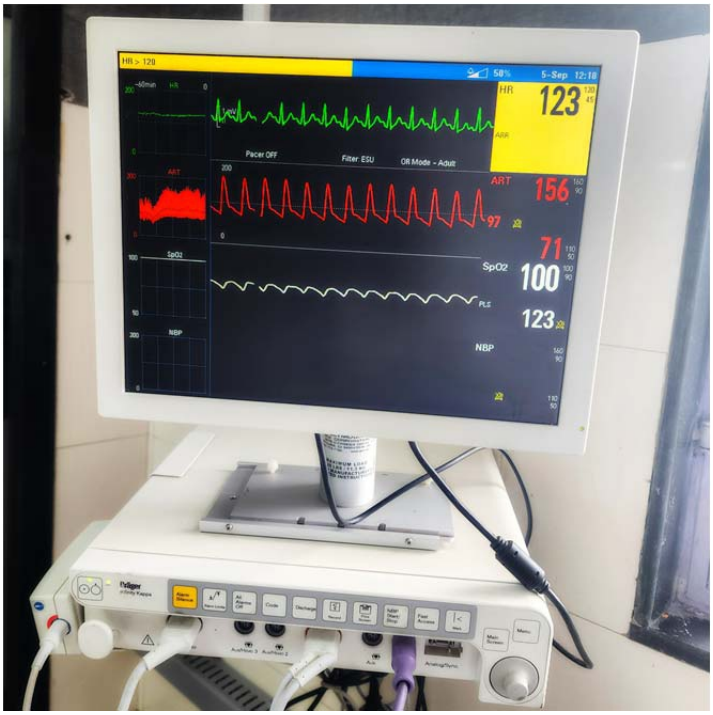


Figure No. 4: A multi-parameter monitor.

## Renal system:

Urine output should be measured in 24 hours to estimate renal function. Urine output >0.5 ml/kg is suggestive of adequate renal perfusion and normal kidney function. UOP < 500 ml in 24 hours is indicative of poor renal function and at risk of renal failure. Besides volume also monitor for colour and any cast in urine. Red colour may be due haematuria or medication like rifampicin. Dark yellow or brown colour indicate dehydration (fluid deficit). Mustard yellow colour means hyper bilirubinuria

## Abdomen:

Abdominal girth and distension should be measured daily. Abnormal increase in girth may be due to fluid, faeces, flatus and any haemorrhagic complication in ICU patient.

Drain placed in abdomen should be monitored for volume and colour.

## Temperature:

Temperature is important vital sign in ICU patients and a temp of >100$^{o}$ F is considered as significant. Common site for measuring temp. is oral, axilla, groin and core temp is measured by rectum, oesophagus, nasopharynx, tympanic membrane.

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2. Romanelli, D., & Farrell, M. W. (2023). AVPU Score. In StatPearls. StatPearls Publishing.
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# 6. Stress Ulcer and DVT Prophylaxis in ICU

## Stress Ulcer Prophylaxis

Stress ulcer is defined as ulceration of gastric mucosa of upper gastrointestinal (GI) tract due to hospitalization. Also called stress related mucosal disease (SRMD). It ranges from common superficial mucosal injury, occult GI bleed, hematemesis, to severe upper GI bleeding causing hemodynamic instability. There are two mechanisms responsible for stress ulcers. First, there is **increased acid secretion** by the parietal due to excess gastrin. This is more common in patients with traumatic brain injury. Second, is the **disruption of the glycoprotein mucous layer**, which is a physical barrier protecting the gastric mucosa from gastric acids.

### Risk Factors:

1. **Prolonged mechanical ventilation for more than 48 hours.**
2. **Coagulation disturbance.**
3. Shock.
4. Severe traumatic brain injury.
5. Burn > 30%.
6. Multi organ dysfunction syndrome (MODS).
7. Acute kidney injury.
8. Liver Failure.
9. History of gastrointestinal ulcers.
10. Glucocorticoid therapy.

Patients with two or more risk factors are at greater risk.

### Drugs used for prophylaxis:

Proton pump inhibitors (PPI) e.g., Intravenous pantoprazole 40 mg IV OD.

$H_2$ antagonists e.g., Injection ranitidine 50 mg every 8 hours.

Oral sucralfate 1 gram every 6 hours.

### Withdrawing SRMD prophylaxis

1. Once a patient is on full feeds stop prophylactic therapy if the patient does not have upper GI bleeding, and with no risk factors mentioned earlier.
2. Switch over to oral medication as soon as patients start accepting oral feeding.
3. Those patients who develop clinically significant bleeding continue proton pump inhibitors for at least 2 weeks.
4. Enteral feeding is considered to have a protective effect on gastric mucosa and is used along with the pharmacological measure for prophylaxis. Combination of prophylactic therapy along with enteral nutrition has been shown to reduce SRMD incidence.

## Deep Venous Thrombosis Prophylaxis

Deep vein thrombosis (DVT) is formation of blood clot in deep vein of body. Most commonly involved veins are pelvic, thigh and legs but less commonly in arms. DVT is most prevalent in critically ill patients admitted in ICU.

**Sign and symptoms:**

The common symptoms are swelling, erythema, pain in affect part of body and in long term it causes ulceration. The most fatal complication is pulmonary embolism when clot dislodges from its origin and reaches in pulmonary artery via systemic vein and heart.

**Pathophysiology of DVT:**

Virchow's triad (Figure No. 1): The three involved mechanisms are-

1. Decreased blood flow (venous stasis)
2. Endothelial injury
3. Hypercoagulability of blood

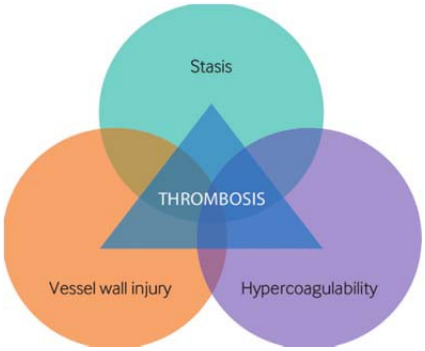


Figure No. 1: Virchow's triad

**Risk factors for DVT:**

- Old age
- Prologed immobilization
- Major surgery
- Previous DVT
- Malignancy
- Hormonal replacement therapy
- Trauma and long bone fracture
- Pregnancy and postpartum
- Obesity
- Elevated CVP due to heart failure
- Coagulation disorders e.g., Polycythemia, thrombocytosis

**DVT prophylaxis: General guidelines**

- Every patient in ICU should be assessed for requirement of DVT prophylaxis.
- Non-pharmacological (mechanical) and pharmacological methods used as prophylactic.
- Early ambulation is most crucial non pharmacologic method for prevention of VTE.
- There is no advantage of combined pharmacologic and mechanical prophylaxis over pharmacological prophylaxis alone.
- Thromboprophylaxis should be reviewed daily and changed accordingly.
- Thromboprophylaxis should be continued even after transfer from ICU till the risk of DVT is over.

## Methods for Prophylaxis

1. **Mechanical prophylaxis:**
   a. Graduated compression stockings.
   b. Intermittent pneumatic compression.
2. **Pharmacological prophylaxis:**
   a. Unfractionated heparin.
   b. Low Molecular Weight Heparin (LMWH)
   c. Fondaparinux.

1. **Mechanical prophylaxis:**
    - It should be used in patients of high risk of bleeding such as post-surgical patients, neurosurgical, hemorrhagic stroke and patients having bleeding disorder.

a. **Graduated compression stocking (anti-embolic stockings):**
    - It exerts the greatest degree of pressure at the ankle and gradually decreases when goes upwards (graduated compression)
    - It ensures blood flow upwards and prevents backflow (Figure No. 2).
    - Avoid in peripheral neuropathy, allergy to material, local soft tissue infection, improper size.

Figure No. 2: Graduated compression stockings.

b. **Intermittent pneumatic compression:**
    - Intermittent pneumatic compression device (Figure No. 3) is made of inflatable sleeves attach through a air pump
    - Inflate every 20-60 seconds then deflate, act as leg massage
    - It is more effective than graduated compression stocking

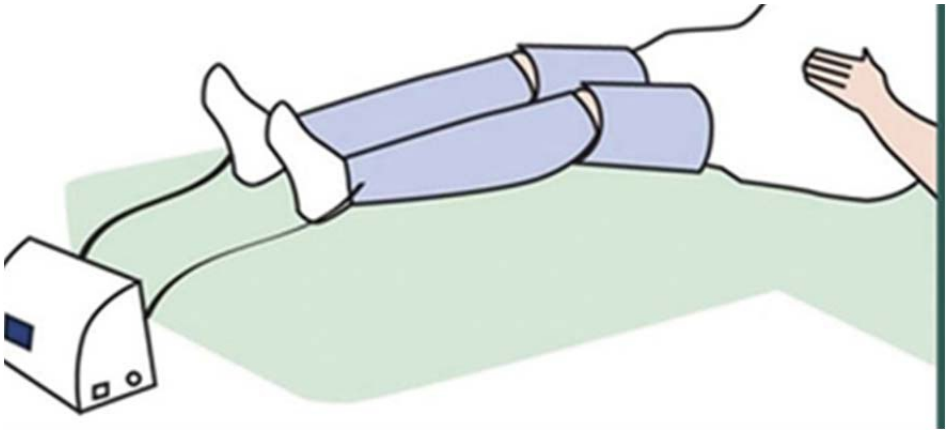

Figure No. 3: Intermittent pneumatic compression device

2. **Pharmacological prophylaxis:**
    - It should be started as soon as possible if not contraindicated.
    - Post-surgical patients' pharmacological prophylaxis can be started once risk of bleeding is excluded after 24-72 hours depending upon the surgery and hemostasis achieved.
    - Unfractionated or Low Molecular Weight Heparin (LMWH) should be avoided in patients with platelet counts less than 1,00,000/L or INR >1.5.
    - LMWH should be stopped 12 hours before removal of epidural catheter and can be restarted only after 2 hours after removing it.

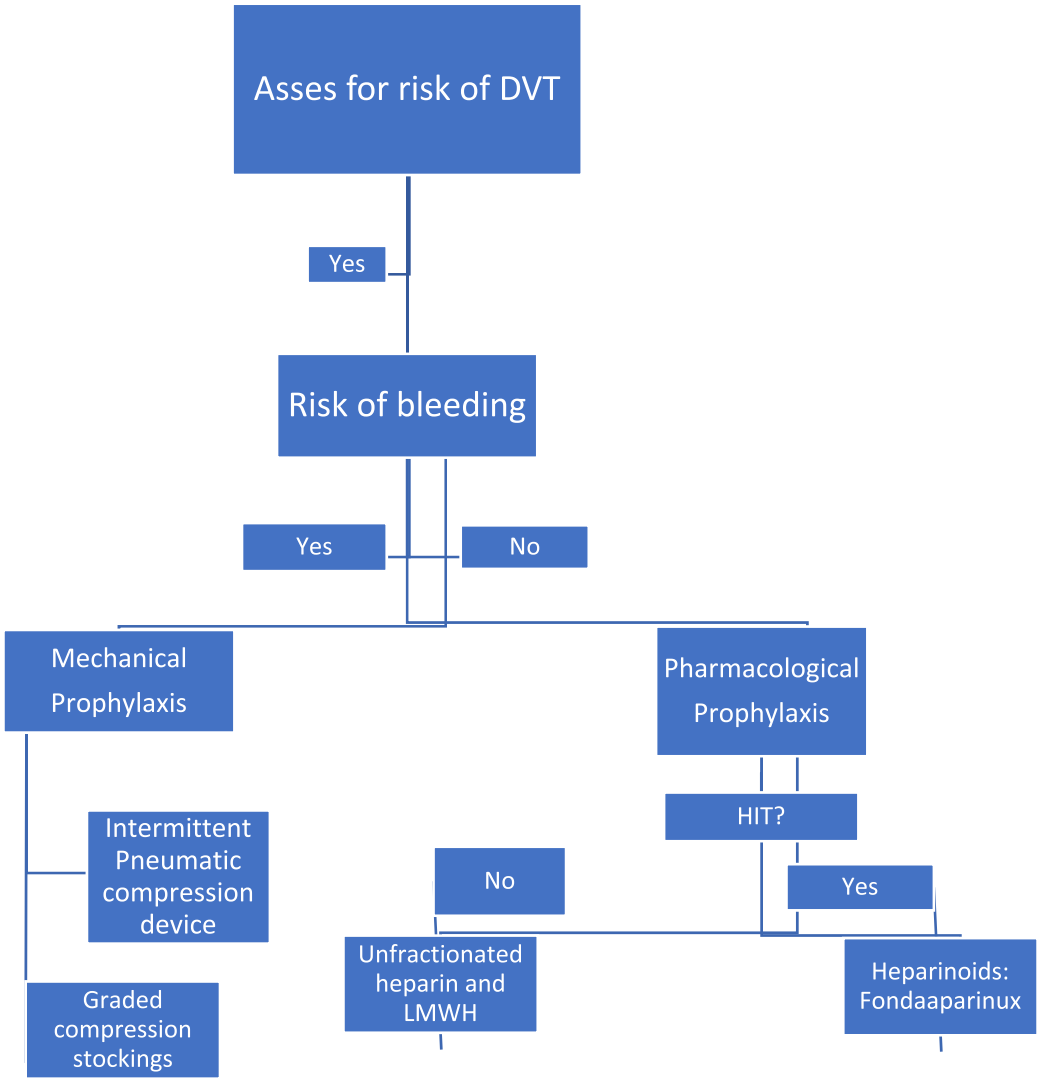


Figure No 4: Decision making and options for DVT prophylaxis

**Advantages of LMWH over UFH:**

Once daily dosing,
Enhanced bioavailability,
Minimal incidence of heparin-induced thrombocytopenia
Cost benefit
No requirement for laboratory monitoring.

**Recommended dose of anticoagulant as prophylaxis:**

UFH- 5000 units SC 8-12 hourly
Enoxaparin- 30 mg 12-24 hourly or 40 mg once a day SC
Daltaparin – 5000-10000 units 12 hourly SC
Fondaparinux – 5-10 mg daily SC

# 7. Delirium in ICU

Delirium is an acute confusion state that is caused by direct physiological effects of some medical disease conditions, use of any psychoactive substances that develop over the course of hours and day.

**Sign and symptoms:**

It is clinical syndrome which usually has attention disorder, lack of awareness, cognitive impairment, and other less common features of altered psychomotor activity, disturbed circadian rhythm, emotional disturbance and perception disorder like hallucination and delusion.

**Delirium Subtypes:**

Delirium can be divided depending on psychomotor activity.

1) Hyperactive Delirium: Patients present with increased agitation and sympathetic activity. They can present with hallucinations, delusions, and occasionally combative or uncooperative behavior.

2) Hypoactive Delirium: Patients have increased somnolence and decreased arousal. Hypoactive delirium is dangerous as it is often unrecognized or mistaken for fatigue or depression. It is associated with higher rates of morbidity and mortality.

3) Mixed Presentation: Patients can fluctuate between Hyperactive and Hypoactive presentations

**Causes:** It is multifactorial in nature and there are several predisposing factors and precipitating factors and interaction these two-cause delirium

**Predisposing factor:** These are non-modifiable [1]

- Elderly patients (> 65 years)
- Preexisting Cognitive impairment/dementia
- Associated comorbidity like hearts disease, cerebrovascular disease, and cancer
- Psychiatric morbidity (e.g., depression, bipolar disorder)
- Sensory impairment (i.e., vision and hearing)
- Substance use disorder, including alcohol abuse.

**Precipitating factor:** These factors are modifiable[2]

- Medication (benzodiazepine, opioids, anticholinergic steroid)
- Infection (e.g., URTI, UTI)
- Hospital environment (dim light, noise in ward, unfamiliar person)
- Hypoxia, hypercarbia and anemia
- Dehydration/malnutrition
- Inadequate analgesia
- Stroke, meningitis
- Sleep deprivation
- Emotional stress
- Constipation /urinary retention
- Alcohol withdrawal
- Major surgery (cardiac, vascular surgery)

**Screening of delirium:** two validated tools are used for screening:

- **Intensive care delirium screening checklist (ICDSC)** [3]
- **Confusion assessment method for ICU (CAM-ICU)** [4]

**Intensive care delirium screening checklist (ICDSC):**

It is assessed by using 8 parameters (Figure No. 1)

**(1)** Altered level of consciousness
**(2)** Inattention
**(3)** Disorientation
**(4)** Hallucination, delusion or psychosis
**(5)** Inappropriate mood or speech
**(6)** Psychomotor agitation or retardation
**(7)** Sleep wake cycle disturbance
**(8)** Fluctuations

- Each parameter is scored 0 to 1. Maximum score is 8 and minimum is zero.
- Score of 4 is considered having delirium.
- Sensitivity of 99% and specificity of 64%.
- Not a good tool for stupor or comatose patient.

**Intensive Care Delirium Screening Checklist (ICDSC)**

| | | | | |
|---|---|---|---|---|
| **1. Altered level of consciousness**<br>Deep sedation/coma over entire shift [SAS = 1,2; RASS = –4,–5]<br>Agitation [SAS = 5,6 or 7; RASS = 1–4] at any point<br>Normal wakefulness [SAS = 4;RASS = 0] over the entire shift<br>Light sedation [SAS = 3; RASS = –1,–2,–3] | = Not assessable<br>= 1point<br>= 0 point<br>= 1 point (if no recent sedatives)<br>= 0 points (if recent sedatives) | No | 0 | 1 Yes |
| **2. Inattention**<br>Difficulty following instructions or conversation; esily distracted by external stimuli<br>Will not reliably squeeze hands to spoken letter "A":S A V E A H A A R T | | No | 0 | 1 Yes |
| **3. Disorientation**<br>In addition to name, place, and date, dose the patient recognize ICU caregivers?<br>Does patient know what kind of place they are in? (list examples such as dentist's office, home,work,hospital.) | | No | 0 | 1 Yes |
| **4. Hallucination, delusion,or psychosis**<br>Ask the patient if they are having hallucinations or delusions (e.g., trying to catch an object that isn't there).<br>Are they afraid of the people or things around them? | | No | 0 | 1 Yes |
| **5. Psychomotor agitation or retardation**<br>EITHER: Hyperactivity requiring the use of sedative drugs or restraints to control potentially dangerous behavior (e.g., pulling IV lines out or hitting staff).<br>OR: Hypoactive or clinically noticeable psychomotor slowing or retardation. | | No | 0 | 1 Yes |
| **6. Inappropriate speech or mood**<br>Patient displays inappropriate emotion, disorganized or incoherent speech, sexual or inappropriate interactions, or is apathetic or overly demanding. | | No | 0 | 1 Yes |
| **7. Sleep-wake cycle disturbance**<br>EITHER: frequent awakening /<4 hours sleep at night.<br>OR: Sleeping during much of the day | | No | 0 | 1 Yes |
| **8. Symptom fluctuation**<br>Fluctuation of any of the above symptoms over a 24-hours period. | | No | 0 | 1 Yes |
| **Total shift score (Min 0 - Max 8)** | | | | |

Figure No. 1: Intensive care delirium screening checklist. (RSAS= Riker sedation agitation scale, RASS= Richmond agitation sedation scale)

**Confusion assessment method for ICU (CAM-ICU):** (Figure No. 2)

Most common and reliable method in ICU

4 Important features are key points for diagnosis of delirium.

- Feature 1: Acute Onset or Fluctuating Course
- Feature 2: Inattention
- Feature 3: Altered Level of Consciousness
- Feature 4: Disorganized Thinking

**CAM-ICU Worksheet**

| **Feature 1: Acute Onset or Fluctuating Course** | **Score** | **Check here if Present** |
|---|---|---|
| Is the pt different than his/her baseline mental status?<br>OR<br>Has the patient had any fluctuation in mental status in the past 24 hours as evidenced by fluctuation on a sedation scale (i.e., RASS), GCS, or previous delirium assessment? | Either question Yes → | ☐ |
| **Feature 2: Inattention** | | |
| Letters Attention Test (See training manual for alternate **Pictures**)<br>Directions: Say to the patient, *"I am going to read you a series of 10 letters. Whenever you hear the letter 'A,' indicate by squeezing my hand."* Read letters from the following letter list in a normal tone 3 seconds apart.<br>**S A V E A H A A R T**<br>**Errors are counted when patient fails to squeeze on the letter "A" and when the patient squeezes on any letter other than "A."** | Number of Errors >2 → | ☐ |
| **Feature 3: Altered Level of Consciousness** | | |
| Present if the Actual RASS score is anything other than alert and calm (zero) | RASS anything other than zero → | ☐ |
| **Feature 4:Disorganized Thinking** | | |
| Yes/No Questions (See training manual for alternate set of questions)<br>1. Will a stone float on water?<br>2. Are there fish in the sea?<br>3. Does one pound weigh more than two pounds?<br>4. Can you use a hammer to pound a nail?<br>**Errors are counted when the patient incorrectly answers a question.**<br>Command<br>Say to patient: "Hold up this many fingers" (Hold 2 fingers in front of patient) "Now do the same thing with the other hand" (Do not repeat number of fingers) *If pt is unable to move both arms, for 2nd part of command ask patient to "Add one more finger"<br>**An error is counted if patient is unable to complete the entire command.** | Combined number of errors >1→ | ☐ |

| **Overall CAM-ICU**<br>**Feature 1 plus 2 and either 3 or 4 present = CAM-ICU positive** | **Criteria Met →** | ☐ **CAM-ICU Positive** (Delirium Present) |
|---|---|---|
| | **Criteria Not Met →** | ☐ **CAM-ICU Negative** (No Delirium) |

Figure No. 2: Confusion assessment method for ICU

## Treatment:

### Stop and THINK

Stop all medication if patient having that can precipitate like anticholinergic, corticosteroid, benzodiazepine, opioids etc.

THINK:

T= treat the toxic situation like CHF, Shock, dehydration,

H= treat for hypoxia, hypercarbia

I= treat infection and avoid immobilize (early mobilization)

N= non-pharmacological intervention

K= k+ or electrolyte correction

**Non-Pharmacologic:**

- Noise Reduction by use of earplugs for patients at night, reduce alarm volume.
- Adequate light in ICU ward.
- Communicate with patients and covey date, place and reason for hospitalization.
- Avoid any type of stress.
- Adequate analgesia.
- Family interaction with patients.
- Familiar belongings near the patient.
- Improving sleep by sedation.
- Reduce interruption at night.
- Optimize bladder and bowel function.

**Pharmacological:**

- Haloperidol: short term use of low dose haloperidol at least for a week is
- Atypical antipsychotics: use of this medication may reduce the duration of delirium (eg. risperidone, olanzapine, ziprasidone, and quetiapine )
- The antidepressant trazodone is sometimes used for the same but effect should be weighed against its sedation side effect.
- Dexmedetomidine should be practiced for sedation in place of benzodiazepine to reduce delirium.

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# 8. Sedation and Analgesia in ICU

Sedation is commonly required in ICU patients to decrease anxiety, anxiety, and facilitate mechanical ventilation. Light levels of sedation are preferred over deeper sedation. Using the Richmond Agitation Sedation Scale (Table No. 1), the goal is -1 (drowsy) to -2 (light sedation).

1. Non benzodiazepine sedatives are preferred over benzodiazepine sedation to improve clinical outcomes of ICU patient on mechanical ventilation.
2. Assess Patients at regular interval (every 4 hours) for sedation and agitation based on the Sedation and Agitation scale. Worst score to be recorded within the last 4 hours.
3. Titrate the infusion rate of sedative medication with the aim of keeping the patient calm and co-operative.
4. In patients with head injury in view of cerebral protection deep sedation is required.
5. Routinely in ICU the drugs used for sedation are midazolam and fentanyl.
6. Propofol is preferable for patients where neurological status is of concern, and patient is for early weaning.
7. Postoperative patients who require overnight ventilation may be give sedation and analgesia using:
    a. Fentanyl + propofol
    b. dexmedetomidine
8. Daily sedation vacation is given at a fixed time every morning.
9. If patients are agitated, look for alternative cause of agitation. Communicate with patients, assure and increase the sedative dose if required.
10. Titrate the infusion rate according to sedation score at frequent interval by assessing the patient's sedation score regularly.
11. Opioids and sedatives have a synergistic action. Lower doses of sedatives should be used if opioids or other sedatives are used.

| Score | Term | Description | |
|---|---|---|---|
| +4 | Combative | Overtly combative, violent, immediate danger to staff | |
| +3 | Very agitated | Pulls or removes tube(s) or catheter(s); aggressive | |
| +2 | Agitated | Frequent non-purposeful movement, fights ventilator | |
| +1 | Restless | Anxious but movements not aggressive vigorous | |
| 0 | Alert and calm | | |
| -1 | Drowsy | Not fully alert, but has sustained awakening (eye-opening/eye contact) to *voice* **(≥10 seconds)** | Verbal Stimulation |
| -2 | Light sedation | Briefly awakens with eye contact to *voice* **(<10 seconds)** | Verbal Stimulation |
| -3 | Moderate sedation | Movement or eye opening to *voice* **(but no eye contact)** | Verbal Stimulation |
| -4 | Deep sedation | No response to voice, but movement or eye opening to *physical* stimulation | Physical Stimulation |
| -5 | Unarousable | No response to *voice or physical* stimulation | Physical Stimulation |

**Procedure for RASS Assessment**

1. Observe patient
   a. Patient is alert, restless, or agitated. **(score 0 to +4)**
2. If not alert, state patient's name and *say* to open eyes and look at speaker.
   b. Patient awakens with sustained eye opening and eye contact. **(score –1)**
   c. Patient awakens with eye opening and eye contact, but not sustained. **(score –2)**
   d. Patient has any movement in response to voice but no eye contact. **(score –3)**
3. When no response to verbal stimulation, physically stimulate patient by shaking shoulder and/or rubbing sternum.
   e. Patient has any movement to physical stimulation. **(score –4)**
   f. Patient has no response to any stimulation. **(score –5)**

Table No. 1: Richmond Agitation Sedation Scale

**Goals:** The patients should be:

- Calm
- Comfortable
- Cooperative

**Sedation Options:**

- Benzodiazepines
- Propofol
- ketamine
- fentanyl
- Dexmedetomidine – restricted access

**Propofol vs Benzodiazepines:**

Multiple studies show that **Propofol** has the following benefits compared to Benzodiazepines:

- Better sedation quality.
- Better Ventilator synchrony.
- Lesser awakening time.
- Lesser time to extubation after stopping sedation.
- Less Ventilator days.
- Less ICU length of stay among survivors.
- Decreased cost of sedation.

**Dexmedetomidine:** Dexmedetomidine is a highly selective $\alpha_2$-adrenergic receptor agonist. This produces a normal sleep-like and cooperative sedation. The characteristic feature of Dexmedetomidine sedation is its opioid sparing effect that may reduce the number of days of mechanical ventilation as well as length of stay in ICU. Prevalence and duration of delirium is also reduced. Dexmedetomidine has an excellent safety profile. Dexmedetomidine is considered as a promising agent for sedation in ICU.

Table No. 2: Commonly used sedatives and analgesics with their doses

| Drug | Loading dose | Maintenance dose |
|---|---|---|
| **Propofol** | Not commonly used in ICU | 5 to 50 mcg/kg/minute |
| **Dexmedetomidine** | 1 mcg/kg over 10 minutes | 0.2 to 1.5 mcg/kg/hour |
| **Midazolam** | 0.5 to 4 mg | 0.02 to 0.1 mg/kg/hour infusion |
| **Lorazepam** | 1 to 2 mg | 0.01 to 0.1 mg/kg/hour infusion |
| **Ketamine** | 0.25 to 0.5 mg/kg bolus IV | 0.05 to 0.4 mg/kg/hour |
| **Fentanyl** | 25–50 µg | 12.5–200 µg/h |
| **Morphine** | 2–4 mg | 2-30 mg/h |
| **Paracetamol** | 15 mg/kg IV every 6 hours, Maximum ≤4 g/day | |

Section 2

# Mechanical Ventilation

# 1. Mechanical Ventilation: Introduction

Mechanical ventilation may be required:

1. In cases of respiratory failure not responding to medical therapy.
2. In Patient intubated for reasons other than respiratory failure to reduce work of breathing.

For mechanical ventilation we require a conduit in the form of orotracheal intubation or tracheostomy tube in situ. The patient must be adequately sedated so that the tube can be tolerated, and the chances of dislodgment are minimal.

The primary aim of mechanical ventilation is to insure adequate oxygenation and ventilation, prevention of deterioration and improvement in lung function. Inappropriate ventilatory setting can cause lung damage, hemodynamic compromise, morbidity, and mortality. Thus, frequent reassessment of ventilatory parameters and clinical condition of the patient is essential.

## Initiation of mechanical ventilation

The initial ventilatory setting should be aimed to:

1. Limit the tidal volume to 6-8 ml/kg.
2. Limit the alveolar distending pressure to <30 cm of $H_2O$.
3. Maintenance of FRC with the use of appropriate positive end expiratory pressure (PEEP).
4. Prevention of $O_2$ toxicity by limiting $FiO_2$ to < 60% if possible.
5. Prevention of hyperoxia ($PaO_2$ >150 mmHg).

In patients with severe lung disease, sometimes it is necessary to ventilate them on controlled mode, but aim is to switch over to assisted mode as soon as feasible, to avoid ventilator dependence and respiratory muscle weakness.

Any change in the ventilatory setting can be interpreted by ABG analysis after 20 minutes of change.

## Ventilation of patients without lung pathology

These patients have normal or near normal lungs without any obvious lung pathology but have inadequate ventilator drive.

In these patients ventilation should be started initially, with synchronized intermittent mandatory ventilation (SIMV) mode using pressure regulated volume control (PRVC) with a tidal volume (Vt) of 6 – 8 ml/kg and a respiratory frequency (F) of 10-12 /min, with an inspiratory/expiratory time ratio (I:E) of 1:2. Initial PEEP should be set at 5cm $H_2O$. Inspiratory time (Tinsp) should be at least 1.5s. Pressure support ventilation (PSV) should additionally be set, initially at 10 cm $H_2O$ to support respiratory efforts made outside the SIMV trigger window period.

Patient should be allowed to be ventilated with these initial settings, stabilize for 15 – 20 min, when an arterial blood gas (ABG) sample should be drawn to check appropriate oxygenation and $CO_2$ clearance.

Alternative controlled modes include volume control (VC) and pressure control (PC), which may also be used with SIMV and PSV or fully controlled. VC guarantees VT but generates a higher corresponding peak pressure (Ppeak). If we select VC, Ppeak varies with patient resistance and compliance factors while Palv is affected primarily by compliance, such that vigilance is required to prevent excessive Palv if compliance falls.

PC requires the setting of inspiratory pressure (Pinsp) and Tinsp/I: E and delivers a square pressure waveform and decelerating flow, as with PRVC. Vt is however a resultant of settings and patient resistance and compliance factors. Constant vigilance is mandatory to ensure an appropriate Vt.

In a patient who is felt to have adequate ventilatory drive, PSV alone may be used, titrating Pinsp to achieve a Vt of – 5ml/kg, while ensuring a comfortable respiratory pattern.

## Ventilation of patients with lung pathology

Patients with alveolar lung disease, {for example pneumonia, pulmonary edema or hemorrhage, acute lung injury (ALI), acute respiratory distress syndrome (ARDS)}there may be impaired oxygenation and ventilation. It may be diffuse, patchy or lobar, consisting of collapse, consolidation or a combination of both. Diffuse or patchy disease predominantly affects dependent parts of the lungs. All these in turn lead to reduced compliance, but there are always alveoli of relatively normal compliance which must be protected from over distension.

In such a disease with impaired oxygenation requires ventilation with prolonged I:E of 1:1 to increase mean airway pressure (Pmean), allowing more time for oxygen diffusion during inspiration and increased PEEP to maintain maximal alveolar recruitment (opening of closed alveoli) throughout the respiratory cycle.

As compliance is decreased, Ppeak for a given tidal volume Vt will be increased. Vt may need to be reduced to prevent excessive Palv. Where compliance is severely limited, use of PC or PSV allows safe limitation of Palv, while a low Vt is accepted.

Where compliance is poor but airway resistance less so, the time taken to fill alveoli is reduced. In this situation, higher F may be used to augment alveolar minute ventilation (MValv) without worsening dynamic hyperinflation or “air trapping”.

Methods of alveolar recruitment of closed alveoli lead to improved FRC, oxygenation, $CO_2$ clearance and compliance.

## Ventilation of patients with obstructive airways disease (asthma, COPD)

The following points should be considered when putting a COPD patient on ventilator:

- Minimizing Tinsp
- Maximizing expiratory time by minimizing F
- Minimal or low PEEP and
- Keeping Palv below 30 cm $H_2O$.

Default settings should consist of Vt 6ml/kg IBW, F 8-10/min and Tinsp 1s. If after stabilization on these settings, it appears that full expiration is occurring before the next inspiration, F may be increased if $PaCO_2$ is high. Hypercarbia is tolerated as long as oxygenation is maintained. Use of a volume preset mode such as PRVC or VC protects against excessive Vt being delivered if airway resistance improves rapidly, as it may well do in severe acute asthma. High Ppeak is tolerated, as this pressure is only transmitted to the large, relatively rigid, proximal airways.

## Heart – Lung interactions:

1. Spontaneous ventilation is equivalent to exercise, and it has following implications:
    - Weaning failure can lead to cardiovascular insufficiency.
    - Weaning is a stressor to cardiovascular system and may unmask its deficiency.
    - Breathing is stressful to both heart and lung.
2. Changes in lung volume after change in autonomic tone, pulmonary vascular resistance and at high lung volume compresses the heart in the cardiac fossa similar to cardiac tamponade.

- Low lung volume increases pulmonary vascular resistance by stimulating hypoxic pulmonary vasoconstriction.
- High lung volume increases pulmonary vascular resistance by increasing trans pulmonary pressure gradient.

3. Spontaneous inspiratory efforts decrease intrathoracic pressure.
   - Increase venous return.
   - Increases left ventricular afterload.
4. Positive pressure ventilation increases intrathoracic pressure.
   - Decreases venous return.
   - This decrease in venous return is attenuated by the simultaneous rise in intraabdominal pressure.
   - Decreases left ventricular afterload.
   - Eliminating negative swings in intrathoracic pressure selectively reduces left ventricular (LV) afterload without reducing venous return.

## Indications for mechanical ventilation:

- Acute respiratory failure ($PaO_2$<50mm of Hg or $PaCO_2$>50mm of Hg).
- Acute ventilation failure.
- **Other indications:**
  - Elective ventilation in the postoperative period.
  - Head trauma – to avoid hypoxia, hypercarbia and raised ICP.
  - Chest Trauma – chest wall and lung contusion.
  - Severe LVF with pulmonary edema.
  - Coma with breathing difficulties (e.g., following drug overdose)**.**

## Initiation:

Follow basic parameters to put a patient on ventilator:

1. Airway access: endotracheal intubation or tracheostomy
2. Sedation with or without muscle relaxation
3. Choosing the appropriate ventilator setting

## Ventilatory strategies:

- Tidal volume (TV) usually kept 4-8 ml/kg with the goal of end expiratory plateau pressure of < 35 cm $H_2O$.
- Respiratory rate: usually 12-20 per minute
- I:E ratio: generally, 1:2
- $FiO_2$ and PEEP: initially $FiO_2$ kept 100% but brought down subsequently and PEEP added to avoid oxygen related acute lung injury. $FiO_2$ requirement must be below 50% to initiate weaning.
- Inspiratory flow rate: it is common practice to begin with an inspiratory flow rate of 60 liters per minute. High flow rates are needed for patients with air hunger to improve ventilator synchrony.
- Sensitivity: to initiate ventilator support, either pressure or flow trigger are set. Flow trigger is more sensitive than the pressure trigger.

## What is a mode?

The method or the way in which a breath is delivered by alerting or changing the available variables is called mode of ventilation.

Majority of cases are managed using one of the four modes of mechanical ventilation:

- Volume Controlled Mandatory Ventilation (CMV).
- Pressure Controlled Mandatory Ventilation (PCV).
- Synchronized Intermittent Mandatory Ventilation (SIMV).
- Pressure Support Ventilation (PSV).

**Components of a mode:**

- Type of Breath – Mandatory/Spontaneous/Assisted.
- Control Variables – Pressure/Volume/Flow.
- Phase Variables – Trigger Variable/Limiting Variable/Cycling Variable/Baseline Variable.
- Condition Variables.

Broadly the modes of MV are either:

a) **Volume pre-set**
   - Assist control/CMV.
   - Synchronized intermittent mandatory Ventilation (SIMV).

❖ The clinicians set the rate and tidal volume, but airway pressure is not controlled.

b) **Pressure pre-set**
   - Pressure Controlled Mandatory Ventilation (PCV)
   - Pressure Support Ventilation (PSV)

❖ The clinicians set a maximal inspiratory pressure but delivered tidal volume is not controlled.

## Patient – Ventilator Asynchrony:

**Could be due to various reasons:**

- Airway issue?
- Respiratory problem?
- Cardiac disease?
- CNS disease?
- Are mode and setting appropriate for the patient and his disease?

❖ Consider pharmacotherapy only if no cause has been found for the asynchrony.

Step 1 – Reassurance to the patient if he is awake.

Step 2 – Provide adequate pain relief.

Step 3 – provide adequate sedation.

Step 4 – consider muscle paralysis.

## Tracheostomy

Tracheostomy should be planned when patients are supposed to require mechanical ventilatory support for prolonged periods. The timing of tracheostomy is still controversial. Early tracheostomy is done within 72 hours of intubation if patient is likely to be ventilated for > 7 days or only airway protection is required without ventilation support. Late tracheostomy is usually done after 7 days.

There are several advantages of tracheostomy:

- Decreased requirement for sedation,
- More secure airway,
- Greater patient mobility
- Improved efficiency of airway suctioning,
- Faster weaning from mechanical ventilation and
- Reduced length of stay in the ICU.

Unless there is evidence for clearly irreversible disease (e.g. high spinal cord injury, advanced amyotrophic lateral sclerosis), a patient requiring prolonged mechanical ventilatory support for respiratory failure should not be considered permanently ventilator dependent until 3 months of failed weaning attempts.


Figure No. 1: ICU ventilator.

# 2. Non-Invasive Ventilation

Non-invasive ventilation (NIV) refers to the provision of ventilatory support using techniques that do not bypass the upper airway. The advantages of NIV include avoiding the complications associated with endotracheal intubation, improving patient comfort, and preserving airway defense mechanisms.

## Equipment and Techniques

**Interfaces:** Nasal masks, nasal pillows, oronasal masks (Figure No.1), helmet, mouthpieces

**Ventilator Modes:** Continuous Positive Airway Pressure, Pressure Support Ventilation, Bi-Level Positive Airway Ventilation, Controlled Mechanical Ventilation, Assist/Control Ventilation, And Proportional Assist Ventilation.

## Practical application

NIV should be considered as soon as patients first develop signs of incipient respiratory failure suggesting need for ventilatory assistance. It should be identified who will benefit from NIV and those in whom NIV would be unsafe. To initiate NIV, an interface and ventilatory mode must be chosen. Initial steps include fitting the interface, familiarizing the patient with the apparatus, and explaining the purpose of each piece of equipment. Patients should be motivated and reassured by the clinician, instructed to coordinate their breathing with the ventilator, and encouraged to communicate any discomfort or fears.

### Patient Selection

The criteria for patient selection for NIV includes clinical indicators of acute respiratory distress, such a moderate to severe dyspnea, tachypnoea, use of accessory muscle, paradoxical abdominal breathing, and impaired gas exchange. Blood gas analysis helps in identifying patients' respiratory problems. A conscious and cooperative patient is crucial for initiating NIV.

**Indications**:

- Acute exacerbations of chronic obstructive pulmonary disease.
- Asthma.
- Hypoxemic respiratory failure.
- Cardiogenic pulmonary edema.
- Pneumonia.
- Immunocompromised patients.
- Facilitation of weaning and extubation.
- Postoperative sleep apnea.
- Trauma.

### Contraindications to Non-Invasive Ventilation

- Unconsciousness or mental obtundation (chronic obstructive pulmonary disease [COPD] may be an exception?)
- Inability to protect the airway.
- Inability to clear respiratory secretions.
- Severe upper gastrointestinal bleeding.
- Life-threatening hypoxemia.
- Unstable hemodynamic conditions (blood pressure or rhythm instability).
- Recent gastroesophageal surgery.
- Fixed obstruction of the upper airway.
- Vomiting.
- Recent facial surgery, trauma, burns or deformity.
- Undrained pneumothorax.

### Criteria for NIV Discontinuation and Endotracheal Intubation:

- Patients not tolerating NIV (pain, discomfort, or claustrophobia)
- Inability to improve patient condition.
- Hemodynamic instability or shock, cardiac ischemia, or ventricular dysrhythmia
- No improvement in mental status within 30 min after the application of NIV in hypercapnic, lethargic COPD patients or agitated hypoxemic patients.

### Machine Settings

Commonly used CPAP values in patients with acute respiratory failure, ranges from 5 to 12 cm $H_2O$. For pressure-cycled ventilation, it is prudent to start at low pressures to facilitate patient tolerance (CPAP of 3 to 5cm $H_2O$ and inspiratory pressure of 8 to 12cm $H_2O$) and, if necessary, gradually increase pressure settings as tolerated to relieve dyspnea. Decrease respiratory rate, allow adequate exhaled tidal volume and encourage good patient-ventilator interaction.

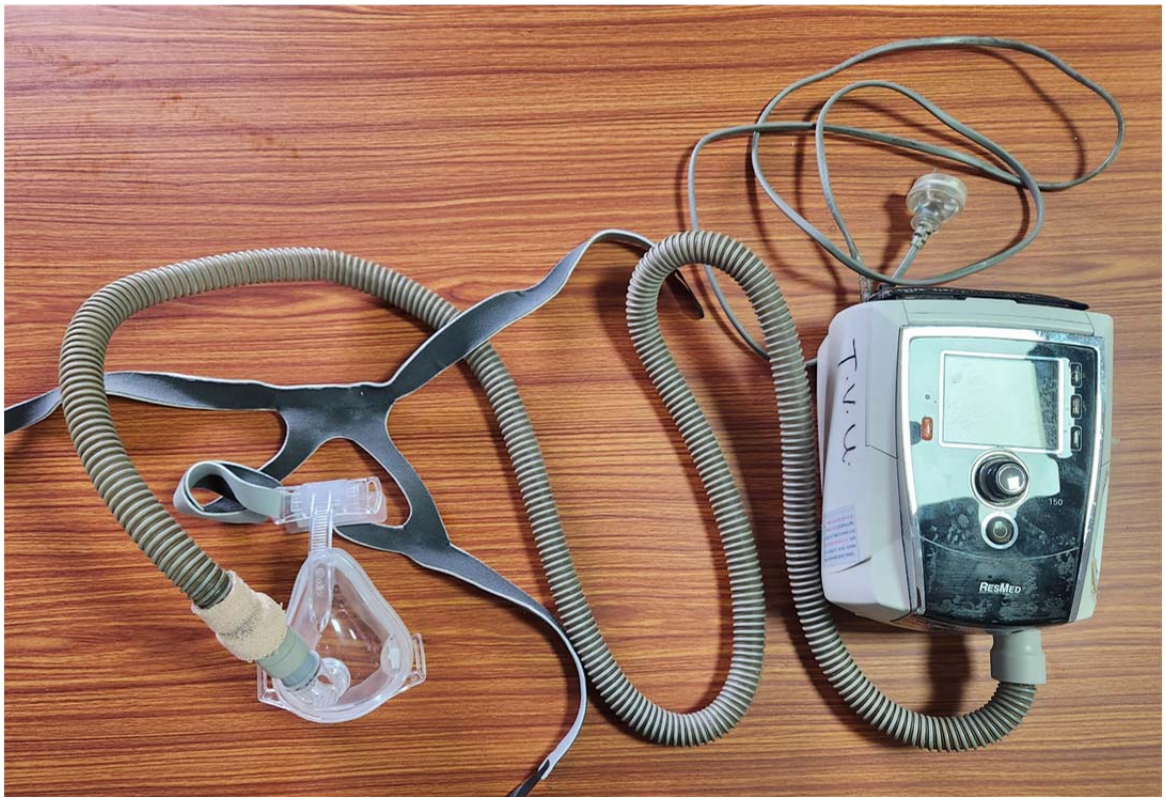


Figure No. 1: Non-invasive ventilator with oronasal mask interface.

## Monitoring patients on NIV:

1. **Bedside Observation**
   - Consciousness level
   - Comfort level
   - Chest movements
   - Accessory muscle use
   - Patient-ventilator synchrony

2. **Vital Signs**
   - Respiratory Rate
   - Exhaled tidal volume (and flow, volume, and pressure waveform for poor synchrony problems)
   - Heart rate
   - Blood Pressure
   - Continuous electrocardiography

3. **Gas Exchange**
   - Pulse oximetry
   - Arterial blood gas analysis as clinically indicated.

## Adverse effects and complications

Major adverse effects of NIV seldom occur in appropriately selected patients and are minimized when the technique is applied by experienced caregivers. The most frequent complications are related to the interface, ventilator flow or pressure, or patient-ventilator interaction.

The pressure of the mask over the bridge of the nose may induce discomfort, erythema, or ulceration. There are various remedies to ameliorate this complication such as application of a hydrocolloid sheet over the nasal bridge or switching to alternative interfaces.

**Air leakage** under the mask into the eyes may cause **conjunctival irritation** and excessive pressure may be responsible for **ear pain**. To minimize these problems, proper fitting of the mask or lowering inspiratory pressure may be useful. **Patient-ventilator asynchrony** is a common cause of NIV failure and is often related to patient agitation or the inability of the ventilator to sense the onset of patient expiration because of excessive air leaking. A judicious use of sedatives may be safe and effective in the treatment of NIV failure due to low tolerance and minimizing air leaks may improve patient- ventilator synchrony.

Presumably because of the low inflation pressure used as compared to invasive ventilation, NPPV is well tolerated hemodynamically, but it should be **avoided** in patients with an **unstable hemodynamic status, arrhythmias, or uncontrolled ischemia** until these problems are stabilized. Gastric insufflation occurs commonly, but it is usually well tolerated. **Aspiration pneumonia** has been reported in as many as 5% of patients. The risk of aspiration is minimized by excluding patients with compromised upper airway function or problems clearing secretions and positioning a nasogastric tube in those with excessive gastric destination, an ileus, or nausea or vomiting. Although **pneumothoraxes are uncommon**, inspiratory pressures should be kept to a minimum effective level in patients with bullous lung disease.

# 3. Weaning From Mechanical Ventilation

The process of withdrawing the patient from mechanical ventilation in a staged manner is termed as weaning. The goal of weaning may be achieved through good nutrition and the application of a planned and well monitored program.

Before considering a patient as a candidate for weaning, a basic level of physiological readiness must be established.

**Approach to weaning:**

- Determine readiness for weaning.
- Develop a weaning plan, including mode, duration of weaning trial, end points and period of rest.
- Prepare the patient psychologically.
- Plan to initiate weaning during daytime.
- Stop sedation, assess GCS, and hold feeding.
- Suction the airway.
- Assess baseline cardiac, pulmonary and neurological function.
- Perform ongoing assessment of patient's tolerance.
- Throughout the process, provide assurance and psychological support to the patient.

**Before starting the weaning process:**

1. Assure proper sleep and rest. Ensure adequate nutrition and an appropriate period of work and rest.

2. If the patient is not tolerating SBT, enquire about the cause of failed SBT. Causes of SBT failure should be corrected before attempting further SBT. It should be performed every 24 hours.

3. Those patients who fail SBT must be comfortably ventilated for 24 hours.

4. Protocols aimed at minimizing continuous use of intravenous sedation can reduce the duration of weaning.

Weaning strategy in the prolonged mechanically ventilated patient should be slow paced and include gradually lengthening spontaneous breathing trials.

## Answer the following questions before attempting to wean:

- Initial problems have improved or resolved, for which the patient was ventilated.
- $SpO_2 \geq 90\%$ or $PaO_2 \geq 60$ mmHg and $FiO_2 \leq 0.5$, $PaO_2/FiO_2 \geq 200$, and PEEP $\leq 8$ cm $H_20$, PS < 10,
- MV < 15 l/min, RR < 30/min
- Good ventilatory drive and patients have spontaneous breathing efforts.
- Cardiovascular stability (no active cardiac ischemia, none or low dose of vasopressors/inotropes)
- Normal electrolytes (including Mg, Phosphate)
- Normal body temperature
- Adequate nutritional status
- Absence of major organ system failure

If the answer to the above questions is yes, start weaning. Put the patient on PS/PEEP mode, stop sedation and feeding, assess the patient's GCS, look for agitation. If everything is acceptable, give a short period of SBT for 30 mins. If SBT is successful, increase the duration.

## Extubation:

**Consider Extubation**:

- Rapid shallow breathing index (f/Vt) ≤105 or
- RR < 35 per min.
- Patients able to protect airway, adequate cough present with suctioning.
- Minimal tracheal secretions.
- No upper airway obstruction.
- GCS > 12

**Criteria for failure of a SBT:**

- RR > 35/min
- SpO2 < 90%, PaO2 < 60 mmHg
- Respiratory distress (>2 of following):
- HR > 140/min or sustained 20% increase in HR
- SBP > 180 mmHg, diastolic > 90 mmHg
- Marked used of accessory muscles.
- Abdominal paradox
- Marked dyspnea.
- Agitation, anxiety, and diaphoresis
  If there is failure, restart mechanical ventilation with previous settings. Start sedation and reassess next morning.

# 4. Mechanical Ventilation: Complications and Care

## Complications of mechanical ventilation

### A. Associated with Patient's Response:

1. **Decreased Cardiac Output-** Due to increase intrathoracic pressure, venous return to Right atrium decreases by positive pressure ventilation.

*Symptoms*– Tachycardia, Hypotension, Decrease CVP, Cool Clammy Skin

*Treatment* – Increase the preload by fluid administration and decreasing the airway pressure by decreasing inspiratory flow rate and TV

2. **Barotrauma** – Caused by excessive airway pressure/ over distension of alveoli, may results into pneumothorax, pneumomediastinum, pneumoperitonium or subcutaneous emphysema.

*Treatment* – Aim is to reduce $V_T$, caution to set PEEP.

3. **Pneumonia** – aim to decrease the risk of VAP, can be reduced by hand washing

*Treatment*- Decrease the risk of aspiration (adequate cuff inflation, use of small-bore NG tube).

Suctioning when needed with sterile technique.

Ensure adequate nutrition.

### B. Associated with ventilator malfunction:

1. **Low exhaled volume**- This may be due to cuff leak, patient disconnection.
   Evaluate cuff; reflate; if rupture change ETT
   Evaluate connection; tightened and reconnect it
   Check ETT; if dislodged, change it
2. **High pressure**- this may be due to secretion in airway, conscious patient biting ET tube, tube kinking, cuff herniation, bronchospasm, endobronchial intubation, pneumothorax, pneumonia, patient. Fighting with ventilator, pain, anxiety etc.
3. **Low $O_2$ pressure** – immediately switch the patient on bain's circuit or AMBU.

### C. Other complications related to endotracheal tube

1. **Sinusitis & nasal injury**

*Prevention* – Avoid nasal intubation.

2. **Tracheoesophageal Fistula**– This is the result of Pressure necrosis of posterior Wall of trachea caused by over inflated ET tube cuff and Nasogastric tube.

*Prevention* – Avoid excessive Cuff Pressure and monitor cuff pressure regularly (8 hourly).

Position the cuff of ET tube distal to fistula; use gastrostomy tube for enteral feeding; placean esophageal tube for suction proximal to fistula.

3. **Laryngeal or Tracheal Stenosis**

*Prevention* – Avoid excessive Cuff Pressure and monitor cuff pressure regularly (8 hourly).

*Treatment* – Tracheostomy

## Care of Patients

### Chest physiotherapy:

**Aim –** to inflate collapsed alveoli, mobilize the secretions, reflate collapsed lung segments.

**Contraindications** –raised ICP and undrained pneumothorax.

**Techniques-**

1. **Hyperinflation** – Hyper inflate the lung to 50% above the TV, aiming to expand the collapsed lung. Recommended tech. is slow inflation, 1-2 sec plateau phase and then a rapid release of bag to stimulate a huff & mobilize secretion. Preoxygenation is needed. Cardiac output often falls.
2. **Percussion & vibration**- drumming & shaking action on chest wall to mobilize secretions.
3. **Postural drainage to assist drainage**– depends upon the affected lung area.
4. **Suction-** tenacious secretion can be loosened by instillation of 2-5ml normal saline.

**General care to avoid the need of physiotherapy**

- adequate humidification avoids tenacious sputum & mucous plugs.
- semi recumbent position to optimize the use of respiratory muscles.
- ensure nutrition is adequate to maintain muscle strength.
- Mobilization & deep breathing may avoid infections.

**Nutritional assessment and management:**

Dietary intake should be individualized for patients need and social belief. It should aid in patient recovery and early mobilization.

**Role of ICU nurse:**

1. Identify the indication of ventilation.
2. List the steps for intubation.
3. Determine FiO2, TV, Rate and mode of ventilator.
4. Aware about the complications of ventilator and what steps are essential for preventing these complications.
5. Keen watch on the alarm of ventilator.
6. Complete the care list of ventilator patient.
7. Complete the suction checklist.

**Preparation for intubation-**

1. Recognize the need for intubation.
2. Do not forget consent, unless it is an emergency.
3. Arrange and check all the equipment required during intubation.
   - Sterile Suction catheter (14Fr.) with pre checked suction assembly, including suction Jar, regulator and connecting tubing.
   - Sterile gloves.
   - Saline for instillation.
   - Nasogastric tube and lignocaine jelly 2%.
   - Manual resuscitation bag/ Bain's circuit.
   - Laryngoscope with blades.
   - Elastic gum bougie and stylet.
   - ETT.
   - ETT fixing tape.
   - Airways oral & nasal.
   - Face masks of different sizes.
   - O2 source.
   - Call for Chest X-ray to confirm positioning of ETT.
   - Notify and ensure family for any change in condition.

Section

3

# Fluid and Blood Products

# 1. Fluid Management in ICU: Fluid Choices

## Introduction

Fluid therapy plays a critical role in the management of patients in the Intensive care unit (ICU). It is aimed at restoring and maintaining hemodynamic stability, which is vital for the proper functioning of organs and tissues. In the ICU, patients often face complex medical conditions, trauma, or surgical procedures that can lead to various disturbances in their fluid balance. Effective fluid therapy can help address these imbalances and improve patient outcomes. Both the type and amount of intravenous fluid administered are important for adequate management of these conditions.

## Body fluid compartments

The body contains 60% water by weight. The fluid inside cells is called intracellular fluid (ICF). ICF constitutes 60% of all body water (35% of body weight). Fluid outside the cells is known as extracellular fluid (ECF). ECF consists of 40% of all body water (25% of body weight). Fluid in the blood vessels is known as intravascular fluid (plasma). Fluid outside blood vessels and the cells is called interstitial fluid.

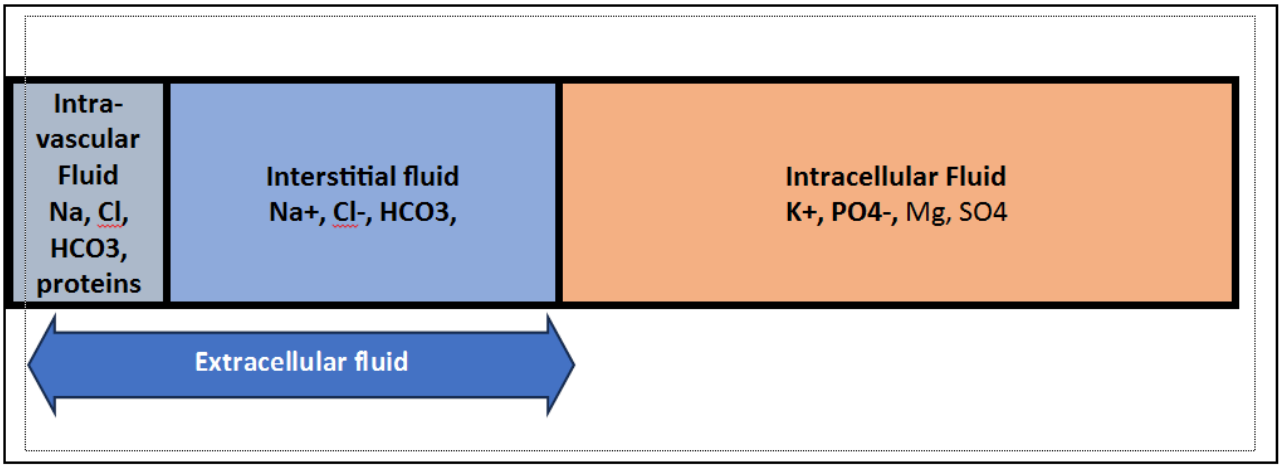


Figure No. 1: Fluid compartments of body and their composition.

## Types of fluid:

**Crystalloid solutions:**

Crystalloid solutions easily cross semi-permeable membrane. Crystalloid solutions contain inorganic ions such as sodium, chloride, potassium, magnesium, and calcium. These may also contain organic substances such as glucose, acetate, or lactate. Commonly used crystalloids are compared in Table No.1.

**Normal Saline**: Normal saline has osmolarity similar to plasma. However, the electrolyte composition differs from the plasma. High amount of chloride result in hyperchloremic metabolic acidosis when normal saline is infused in large quantities.

**Balanced salt solutions** aim to mimic the electrolyte composition of extracellular fluid more closely than normal saline. Commonly used balanced salt solutions include:

- **Lactated Ringer's Solution**: Lactated Ringer's solution contains sodium, potassium, calcium, and lactate (a buffer) in addition to chloride. It is similar in composition to plasma and is preferred for fluid resuscitation in patients with trauma, burns, or surgical interventions.
- **Plasma-Lyte**: Plasma-Lyte has acetate or gluconate as buffers. It is used to replace fluids lost through surgical procedures, electrolyte imbalances, and metabolic acidosis.

- **Hartmann's Solution**: Hartmann's solution, is similar to lactated Ringer's, and is used for resuscitation and as a maintenance fluid.

**Dextrose containing fluids** are not used for resuscitation as they distribute rapidly move out of the intravascular compartment. They also cause undesirable hyperglycaemia. Various commonly used crystalloids are shown in Figure No. 2.

Table No.1 Comparison of commonly used crystalloid solutions.

| Crystalloid solution | Components (mEq in 1,000 ml) | pH | Osmolarity (mOsmol/l) |
|---|---|---|---|
| **Lactated Ringer's / Hartmann's solution** | $Na^+$ 130, $Cl^-$ 109, $K^+$ 4, $Ca^{2+}$ 3, Lactate 28 | 6 to 7.5 | 273 |
| **Ringer's acetate** | $Na^+$ 130, $Cl^-$ 112, $K^+$ 5.4, $Ca^{2+}$ 0.9, $Mg^{2+}$ 1, Acetate 27 | 5.1 to 5.9 | 276 |
| **Normal saline** | $Na^+$ 154, $Cl^-$ 154 | 4.5 to 7 | 308 |
| **Normosol-R, Plasmalyte A** | $Na^+$ 140, $Cl^-$ 98, $K^+$ 5, $Mg^{2+}$ 3, Acetate 27, gluconate 23 | 7.4 | 295 |
| **Dextrose** | $H_2O$, Dextrose | 3.2 to 6.5 | 252 |


Figure No. 2: Commonly used crystalloid fluids.

**Colloid Solutions:**

Colloids contain larger molecules that remain within the intravascular compartment for a more extended period. They also increase the plasma osmotic pressure. Theoretically, this makes colloids a useful choice for fluid resuscitation. But immediate volume expansion is same as crystalloid. In addition, colloids have other disadvantages like increased cost, risk of allergic reactions, renal dysfunction, and coagulation abnormalities. Commonly used colloids are compared in Table No.2. Clinical trails have not shown any benefit of colloids over crystalloids for volume resuscitation. They may be used if large amount of crystalloids are required for volume resuscitation. Commercially available colloid fluids are compared in Table No. 2.

Table No. 2: Comparison of commonly used colloid solutions.

| Colloid solution | Components (mEq in 1,000 ml) | pH | Osmolarity (mOsmol/l) | Remarks |
|---|---|---|---|---|
| **Albumin 5%** | $Na^+$ 130-160, $Cl^-$ 130, $K^+$ 2, | 6.4 to 7.4 | 309 | Good fluid for resuscitation. High rate of allergic reactions. Costly. Human source. |
| **Albumin 20%** | $Na^+$ 160, $Cl^-$ 112, $K^+$ <2 | 6.7-7.3 | 130 | |
| **6% HES in NS** | $Na^+$ 154, $Cl^-$ 154 | 4-5.5 | 308 | High rate of impaired platelet function & coagulation, renal injury. Allergic reaction. |
| **6% HES in buffered solution** | $Na^+$ 137, $Cl^-$ 110, $K^+$ 4, $Mg^{2+}$ 1.5, Acetate 34, | 5.7-5.5 | 286 | |
| **Succinylated Gelatin 4%** | $Na^+$ 154, $Cl^-$ 120 | 7.4 | 274 | Adverse effects are similar to HES. |
| **Polygeline 3.5%** | $Na^+$ 145, $Cl^-$ 145, $K^+$ 5.1, $Ca^{2+}$ 6.25 | 7.3 | 301 | |
| **Dextran 6%** | $Na^+$ 143, $Cl^-$ 124, $K^+$ 3, $Ca^{2+}$ 5, Lactate 28 | 5.9 | 307 | Renal injury. Allergic reaction. |

# 2. Fluid Management: How Much Fluid to Give?

Hemodynamic instability is frequently encountered in critically ill patients. Intravenous fluid bolus is the most appropriate therapy for all patients with hypovolemic (bleeding and dehydration) and distributive (septic and anaphylactic) shock. Fluid bolus in these patients increases the cardiac preload, improving the cardiac output and tissue perfusion. Small amounts of cautiously administered fluid bolus may also be beneficial for a few patients of obstructive shock. Conversely, fluid bolus will harm patients with cardiogenic shock with fluid overload. Excess resuscitation with large positive fluid balance increases the risk of pulmonary edema, impairs oxygenation, and prolongs hospital stay. This chapter focuses on the correct amount of fluid to be administered hemodynamically unstable patients.

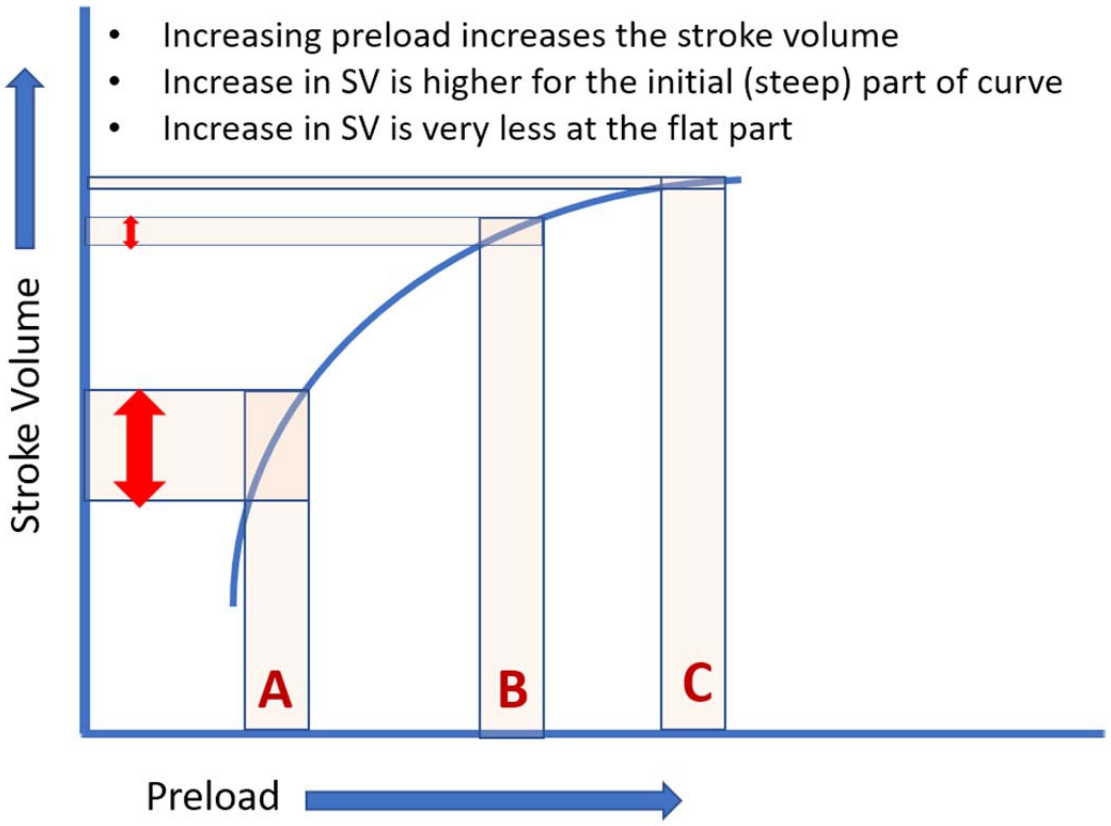


Figure No. 1 Frank-Starling relationship between preload and stroke volume

## Goals of fluid resuscitation:

Fluid resuscitation is aimed at providing adequate preload to the heart, which increases the stroke volume and cardiac output. This is explained by the Frank-Starling relationship of the heart as shown in Figure No.1. For the same heart, when the preload is low (situation A), the increase in preload improves the stroke volume significantly. However, further increases in preload increase the stroke volume to a lesser extent. On the flat part (situation C) of Frank-Starling curve, the increase in stroke volume is negligible and may cause harm. Our aim is to identify patients at the steep portion (situation A). These patients will potentially benefit from fluid bolus. It is essential to know the fluid status of the patient before administering fluid. Giving fluid to patients with inadequate preload (situation A) leads to improvement in cardiac output. Giving fluid to patients with adequate preload (situation C) does not increase the cardiac output and may cause pulmonary edema. These patients require inotropes. Measures of preload are classified as static indices and dynamic indices.

# Estimating fluid status

## Traditional/static measures of preload/fluid status

Traditional measures of intravascular fluid status are not reliable indicators cardiac preload. **Central venous pressure** is raised in patients with right ventricle failure and tricuspid regurgitation. CVP can also be semi-quantitatively estimated with the help of IVC diameter collapse/distension with respiration (**Table No. 1**), but also suffers from the same unreliability. In patients breathing spontaneously, there is a decrease in IVC diameter on inspiration. In mechanically ventilated patients, expiration causes a decrease in IVC diameter.

**Pulmonary artery capillary wedge pressure** is a better measure of left ventricle pressure (preload of LV) but is falsely elevated in mitral valve disease.

## Dynamic measures of Preload/fluid status

Dynamic measures are useful indicators of intravascular fluid status in patients who are on mechanical ventilation. During inspiratory phase, the right ventricle preload decreases (due to increased intrathoracic pressure impeding venous inflow of SVC & IVC). At the same time the positive pressure squeezes the pulmonary veins and increases the preload to the left ventricle. Thus, the left ventricle output increases immediately after inspiration and right ventricle output decreases during inspiration. The reverse occurs during expiration: LV output decreases and RV output increases. This phasic respiratory variation is exaggerated in patients with low intravascular volume and preload. LV stroke volume and output can be measured, and the respiratory variation estimated using following methods: **Stroke volume variation (SVV)** can be measured using various monitors.

$$SVV = \frac{(SVmaximum - SVminimum)}{Mean\ SV}\ X\ 100$$

Patients with SVV >12% are fluid considered responsive and fluid bolus administration is expected to increase their cardiac output. It should be noted that many patients without hemodynamic instability/shock have increased SVV and other dynamic indices of fluid responsiveness. Fluid bolus is not recommended in these patients.

Similarly, **pulse pressure variation (PPV)** can be used to predict fluid responsiveness. PPV has the advantage of being less invasive- requiring only an arterial cannula. A PPV > 12% is considered positive.

$$PPV = \frac{(PPmaximum - PPminimum)}{Mean\ PP}\ X\ 100$$

Table No. 1: Semi-quantitative estimation of CVP from IVC diameter variation.

| IVC diameter variation with respiration | IVC diameter | Decrease in IVC diameter on respiration | CVP mm Hg |
|---|---|---|---|
|  | <2.1 cm | <50% | 3 |
|  | <2.1 cm | <50% | 8 |

| | | | |
|---|---|---|---|
|  | >2.1 cm | >50% | 8 |
|  | >2.1 cm | <50% | 15 |

Respiratory variation in IVC diameter: Respiratory variation in the diameter of IVC of greater than 12% is also considered an indicator of fluid responsiveness.

Disadvantages of PPV, SVV, & IVC diameter variation is that the patients should be on positive pressure ventilation with a tidal volume ≥8 ml/kg and in sinus rhythm. Increased intra-abdominal pressure or open chest also make these indices unreliable.

**Passive leg raising** is a useful test for assessing fluid responsiveness in non-intubated patients. Figure No. 2 shows the positions for the bed for passive leg raising. This infuses the central circulation with 300-500 ml bolus, raising the cardiac output by >10% in fluid responsive patients. This technique requires continuous cardiac output monitoring.

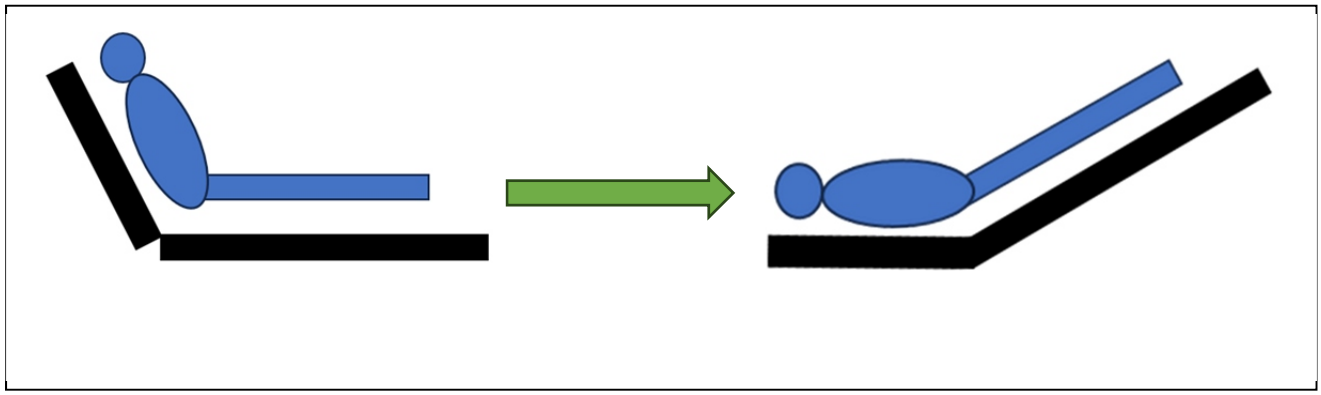

Figure No. 2: Bed positions for passive leg raising test. Note that the whole bed has to be tilted.

A **traditional fluid challenge** (300-500 ml) with an increase of cardiac output by 15% also predicts fluid responsiveness. A **mini-fluid challenge** (100 ml) with an increase of cardiac output also identified fluid responsive patients. Both these methods require measurement of cardiac output and have a risk of fluid overload to non-responsive patients. The dynamic indices of preload are tabulated in Table No.2.

Table No. 2: Dynamic indices of preload estimation and predicting fluid responsiveness.

| Method | Threshold | Remarks |
|---|---|---|
| SVV | 12% | Require ventilation at tidal volume >8 ml/kg |
| PPV | 12% | |

| | | |
|---|---|---|
| **IVC variation** | 12% | |
| **Passive leg raising** | 10% | Require continuous cardiac output monitoring |
| **Fluid challenge (300-500 ml)** | 15% | Risk of fluid overload if non-responsive |
| **Mini-fluid challenge (100 ml)** | 6% | |

**Monitoring fluid resuscitation:** Other than the dynamic parameters, cardiac output, blood pressure and heart rate, other variables are also monitored. Blood lactate begins to progressively decrease and urine output, $SvO_2$, skin perfusion (capillary refill time, temperature, mottling), and mental status improves.

## Phases of fluid therapy

Fluid resuscitation using fluid boluses is the '**rescue'** phase of fluid therapy in patients with hemodynamic instability. It is one of the four phases (Figure No. 3) of fluid therapy. Dynamic indices can be used to guide fluid therapy. Fluid therapy should be decreased when dynamic indices indicate adequate preload (SVV or PPV <12%), acceptable hemodynamic goals (e.g., MAP > 90 mm Hg, HR< 100 per minute) are attained or markers of tissue perfusion (capillary refill time, urine output, blood lactate) are achieved. Next phase is '**optimization**', a phase in which tissue perfusion is optimized using conservative use of fluid challenges. The third phase is '**stabilization'**, which aims for zero-fluid balance. In this phase, only maintenance fluid is given to the stabilized patients. '**De-escalation'** is the last phase in which the removal of the fluid accumulated in the initial phases of fluid therapy is done. This is important as fluid overload is associated with lung injury, delayed recovery, increased cost, impaired wound healing, and increased length of hospital stay. The total body fluid status is shown in Figure No. 4.

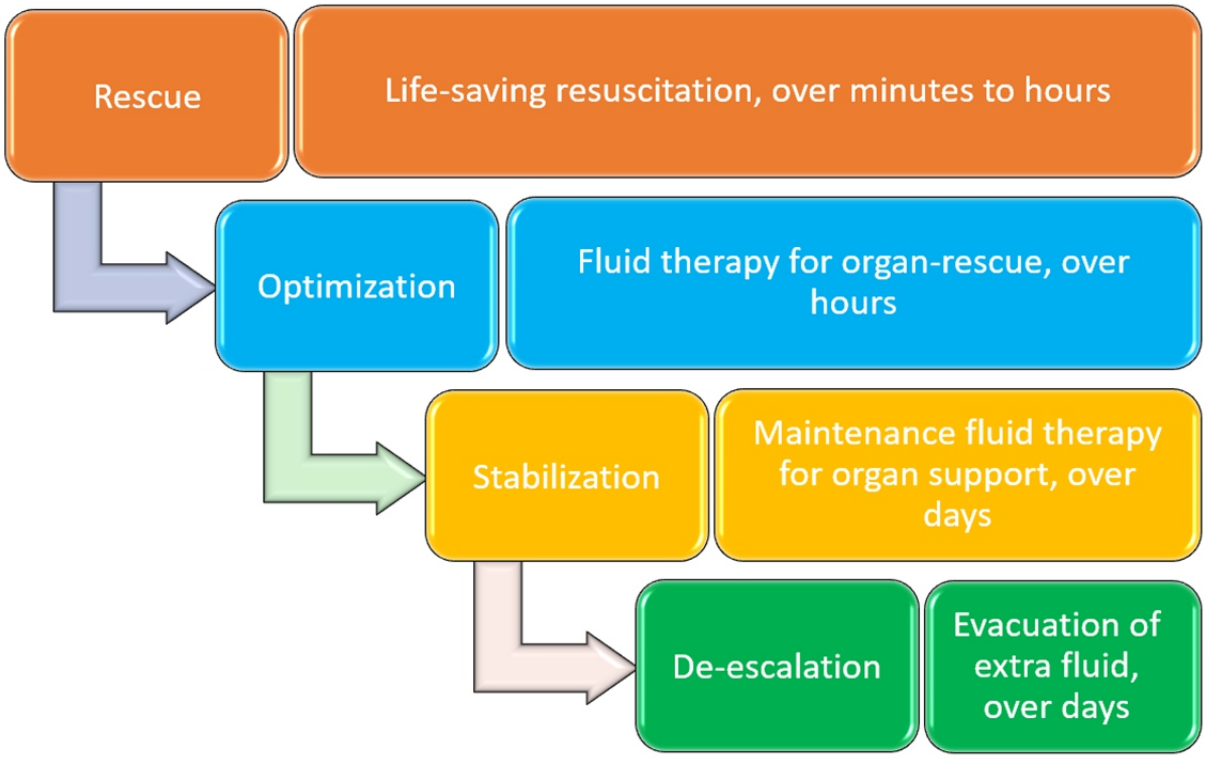


Figure No. 3: Phases of fluid therapy in ICU patients

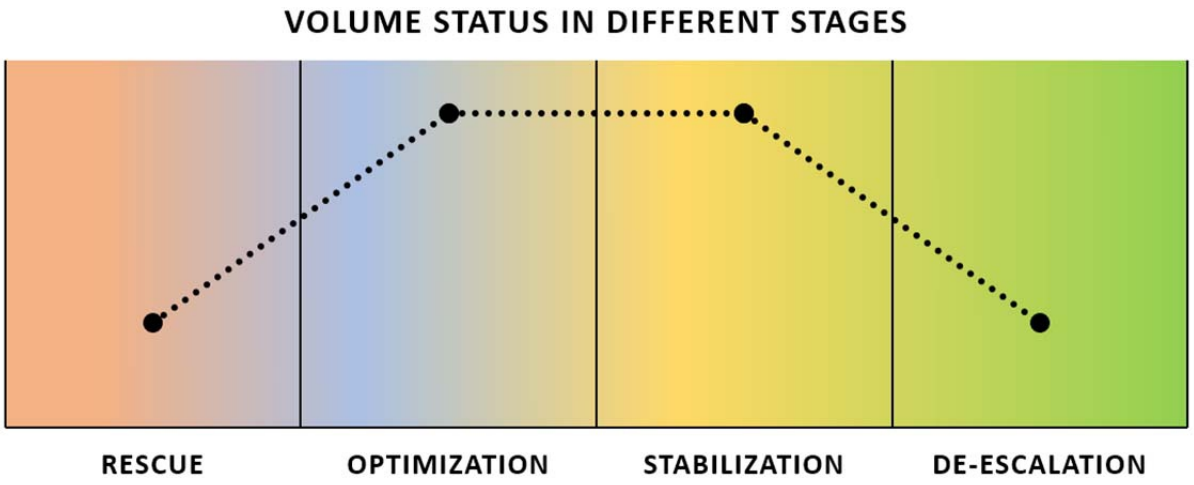


Figure No. 4: Total body fluid status is in fluid therapy stages.

# 3. Blood Component Therapy: Blood Products

Blood component therapy is used to optimize blood supply management. The donated whole blood is divided into specific components, used for targeting specific needs of the patients. Hence, the blood donated by one donor can be used to treat several patients. Also, fluid overload to patients is avoided as volume of packed red blood cells (PRBC) is 250-350 as compared to the 500-550 ml in whole blood. Blood component therapy also optimizes the use of coagulation factors as their levels rapidly decline after collection.

**PRBC:**

PRBC (Figure No. 1) contains the RBC at a hematocrit of 60-80%, WBCs and few platelets. It is stored at 2 to 6o Celsius for 5-6 weeks. PRBC is indicated for patients with anemia and hemorrhage. Transfusion of PRBC is indicated for patients with hemoglobin ≤7 g/dL. However, patients with cardiac disorder may require blood transfusion at Hb ≤8gm/dl. For correction of anemia and hemorrhage, PRBC is better than whole blood is it provides same amount of hematocrit in lesser volume.


Figure No. 1: PRBC bag.

**Fresh frozen plasma:**

Fresh frozen plasma (FFP, Figure No. 2) is derived by centrifugation of whole blood and is frozen. In frozen state, it has a shelf life of 1 year but once thawed, it must be used within 24 hours (at 1-6$^{o}$ C). It contains all coagulation factors. It has a volume of 200-250 ml. FFP is used for:

1. Replacing clotting factors. Generally, 15-20 ml/kg is used. If the patient is deficient in a specific clotting factor, it is better to use specific factor concentrates (PCC or recombinant factor VII) as they can be given rapidly without risk of fluid overload.
2. Massive transfusion: PRBC, FFP, and platelets units are given in a ratio of 1:1:1.

FFP should not be used for volume expansion and for improving albumin levels.


Figure No. 2: Fresh frozen plasma.

**Platelets:**

Platelets are derived from whole blood (random donor platelets, RDP) or by apheresis technique (single-donor platelets, SDP). Unlike other blood components, platelets are stores at 22°C under constant agitation. This restricts their storage duration to 5 days. Various thresholds for platelet transfusion are:

1. 50,000/$mm^3$: for invasive procedures.
2. 100,000/$mm^3$: for invasive procedures in closed space e.g., brain and eyes or if there is massive bleeding.
3. 30000/$mm^3$: for bleeding patients.
4. 10,000/$mm^3$: for all patients. There is a high risk for spontaneous bleeding in below 5000/$mm^3$.

Prophylactic transfusions are not useful chronic bone marrow failure, immune thrombocytopenia, heparin-induced thrombocytopenia, and thrombotic thrombocytopenic purpura.

**Cryoprecipitate**:

It is derived from FFP by controlled thawing at +4°C and re-suspending in 10-20 mL plasma. It is stored at -25°C or colder for up to 1 year after the date of phlebotomy. It contains fibrinogen, factor VIII and XII. It is indicated for bleeding patients having fibrinogen levels < 150 mg/dl. Prophylactic cryoprecipitate transfusion maybe considered for patients having fibrinogen levels < 100 mg/dl planned for invasive procedure. Each unit of cryoprecipitate raises the fibrinogen level by 7-10 mg/dl.

Table No. 1: Components of commonly used blood products

| Component | Content | Volume | Shelf-life |
|---|---|---|---|
| **PRBC** | RBCs (hematocrit 60-80%), WBC, platelets, plasma | 250-350 | 21-42 days |

| | | | |
|---|---|---|---|
| **Platelets: SDP** | Platelets, Few WBC & RBC | 200-350 ml | 5 days |
| **Platelets: RDP** | | 50 ml | |
| **Fresh frozen plasma** | Plasma with coagulation factors, albumin, globulin | 200-250 ml | 1 year at -18°C, 24 hours after thawing at 1-6°C |
| **Cryoprecipitate** | F VIII, fibrinogen, vWF, F XII | 20 ml | 1 year at -18°C, 4-6 hours after thawing at 1-6°C |

## Special processing

**Leukoreduction**: Leukoreduction eliminates 99.9-99.99% of the leucocytes using a leuko-reduction filter. Leukocytes are implicated in febrile nonhemolytic transfusion reactions, CMV transmission, and immunologically mediated reactions. Use of leukoreduced blood products significantly reduces or eliminates these reactions. Leukoreduction does not prevent graft-vs-host disease (GVHD).

**Irradiation**: Irradiated with gamma radiation inactivates all the leucocytes in the blood component and prevents GVHD. It is used for immunocompromised patients such as organ transplant recipients, stem cell transplant, Hodgkin's lymphoma etc. Irradiation reduces the shelf-life to 28 days.

**Washed RBCs**: Washing of the RBCs with normal saline prevents the complications associated with plasma components. Also, it does not increase potassium levels.

### Practical Administration of Blood Components:

1. Indications: Transfusion of blood components has potentially life-threatening complications. They should be transfused only in presence of specific indication.
2. Informed consent: It is compulsory for all non-emergency transfusions.
3. Patient identification: It is critical to ensure that the blood component is transfused to the intended recipient.
4. Inspection of blood component: The blood component should be inspected before transfusion. Abnormal units should be returned back to the blood bank.
5. Blood filters and infusion set: All blood components should be transfused using a blood transfusion set with 170-260 micrometer filter. Concomitant administration of non-compatible intravenous fluids should not be done through same venous access. Normal saline is compatible with all blood products. Calcium containing intravenous fluids (example ringer's lactate) are not compatible with blood components as they can cause clotting of blood components by binding citrate anticoagulant. Change infusion set every 12 hours.
6. Blood warmers: Transfusion of cold blood component, especially in large volumes, can cause significant hypothermia. An in-line blood warmer like Level 1 or Fluid-O can be used to warm the blood. Temperatures above 41°C should be avoided as it can cause hemolysis.
7. Rate: The rate at which blood is administered is given in Table No….. Blood components should be started slowly to ensure early detection of complications with infusion of even small amount of blood. For example, acute hemolytic transfusion reaction occurs with 10-15 ml of blood and the mortality rate is directly related to the amount of non-compatible blood transfused. High transfusion rate can also cause circulatory overload.
8. Monitoring: The patients should be monitored for complications of blood transfusion. Tachycardia, hypotension, hypertension, fever, chills, rash, urticaria, dyspnea, restlessness, chest pain and hematuria are some signs of blood reaction. Presence of any side-effect warrants stopping the

infusion, investigating the cause and institution of appropriate therapy. Therapy is supportive in most of the cases.

9. Post transfusion:
    a. Documentation: Pre and post transfusion vitals, specific components and number with blood bag number, blood reaction etc. are documented.
    b. Blood test: If needed, blood samples can be taken 15 minutes-1 hours after transfusion to measure improvement due to transfusion.

Table No.2: Maximum rates of blood transfusion

| Adult | Rate |
|---|---|
| **Whole blood** | 150-200 mL/hour |
| **PRBC** | 100-150 mL/hour |
| **Platelet /Plasma** | 150-300 mL/hour |

# 4. Blood Component Therapy: Complications

Complications due to blood component transfusion can vary from mild like rash to severe life-threatening anaphylaxis. Based on severity, the complications are classified as category 1 to category 3. The treatment of the side effects is based on the category of blood reaction.

## Guidelines for recognition and management of acute transfusion reactions

### Category 1: Mild reactions:

| Signs | Symptoms | Possible cause |
|---|---|---|
| Localized cutaneous:<br>Urticaria or Rash | Pruritus | Hypersensitivity<br>(mild) |

**Immediate Management of Category 1: Mild reactions**

- Slow the transfusion.
- Administer **antihistaminic** drugs.
- If there is no clinical improvement within 30 minutes, or if signs and symptoms worsen, this is re-classified as Category 2.

If clinical features improve, restart transfusion slowly.

### Category 2: Moderately severe reactions

| Signs | Symptoms | Possible cause |
|---|---|---|
| Flushing<br>Urticaria<br>Rigors<br>Fever<br>Restlessness<br>Tachycardia | Anxiety<br>Pruritus<br>Palpitations<br>Mild dyspnea<br>Headache | Hypersensitivity |

**Immediate Management of Category 2: Moderately severe reactions**

- Stop transfusion.
- Return the blood unit with transfusion administration set, freshly collected urine and paired blood samples (1 clotted and 1 anticoagulated), drawn from a vein opposite site to the transfusion site, to the blood transfusion center for laboratory investigations.
- Give **antihistaminic, antipyretic and corticosteroids**. Avoid aspirin.

- Symptomatic management.
- If condition improves, restart transfusion slowly with new blood unit and observe carefully.
- If the condition worsens or not improved within 15 minutes, treat as category 3.
- Send 24 hour urine sample for examination of hemolysis.
- If available, a leucocyte reduction filter (WBC filter) should be used in repeated transfusion.

**Category 3: Life-threatening reactions**

| **Signs** | **Symptoms** | **Possible cause** |
|---|---|---|
| Rigors<br>Fever<br>Restlessness<br>Hypotension (fall of 20% in systolic BP)<br>Tachycardia (rise of 20% in heart rate)<br>Hemoglobinuria (Hb in urine)<br>Unexplained bleeding (DIC) | Anxiety<br>Chest pain<br>Pain along the transfusion<br>Respiratory distress/shortness of breath<br>Loin/back pain<br>Headache<br>Dyspnea | Acute intravascular haemolysis (mismatched blood transfusion).<br>Bacterial contamination and septic shock.<br>Fluid overload.<br>Anaphylaxis.<br>Transfusion Related Acute Lung Injury (TRALI) |

**Immediate Management of Category 3: Life-threatening reactions**

- Stop transfusion. Flush IV line with normal saline.
- Resuscitate patients with intravenous fluid.
- Maintain airway, breathing and circulation.
- Provide oxygen supplementation.
- Give adrenaline (as 1:1000 solution) 0.02 mg/kg body weight by IM injection.
- Use antihistaminic, corticosteroids, bronchodilators, and diuretics.
- Check a fresh urine specimen visually for signs of hemoglobinuria.
- Send the blood unit with transfusion set, fresh urine sample and new blood samples (1 clotted and 1 anticoagulated), drawn from a vein opposite the infusion site, with the appropriate request from to the blood transfusion center for investigation.
- Symptomatic management.
- Send 24-hour urine sample for examination of hemolysis.
- Give antipyretics if fever is present, avoid aspirin.

## Specific complications

The following are the selected specific causes and mechanism of specific blood component- related side effects.

**Acute hemolytic transfusion reaction (AHTR):** This happens when incompatible (e.g.: ABO or RH) blood is transfused, and the recipient's plasma antibodies attack the donor's RBCs. There is hemolysis and a severe inflammatory cascade is activated leading to hemolysis, causing hypotension, vascular collapse, and DIC. Free hemoglobin from the lysed RBCs and immune complex reactions cause nephrotoxicity. It commonly occurs within minutes of transfusion but can take up to 24 hours. Common clinical features are **fever**, **chills, back/flank pain, hypotension, hemoglobinemia** (appears as dark colored blood), hemoglobinuria (clinically like hematuria), oliguria, bleeding from puncture sites or epistaxis (suggesting DIC), nausea, dyspnea, and hemodynamic instability. Specific management of AHTR is summarized in Box No. 1.

Box No. 1: Immediate management of hemolytic blood transfusion reaction.

- Stop blood transfusion immediately.
- Maintain urine output of at least 1-1.5 ml/kg/minute by:
    - Intravenous fluid bolus.
    - Mannitol and/or furosemide.
- Alkalinization of urine by sodium bicarbonate.
- Send samples:
    - Urine and blood free hemoglobin.
    - Test for DIC: PT, PTT, fibrinogen, platelet count, thromboelastogram.
- Send blood sample and the blood bag to blood bank for repeat crossmatch**.**

**Delayed hemolytic transfusion reaction (DHTR):** Patients develop antibody against donor blood (incompatible Rh and Kidd antigens) components. These antibodies persist in the recipient's plasma in low levels. When the next transfusion is done, these antigen-containing RBC are not immediately lysed as the antibodies level is low. When the antibody levels increase to a sufficient amount (taking 2-21 days), the lysis of foreign RBCs start to occur- resulting in delayed hemolytic transfusion reaction. Clinical features are less severe than AHTR. Decreased hemoglobin, icterus, and rare kidney dysfunction is seen, but mortality is rare. Due to the time gap, the causal association can be difficult to establish. Management is supportive, but glucocorticoids may be helpful.

**Febrile non-hemolytic reactions (FNHR):** These are the most common blood reactions. Features include fever, chills, nausea, headache, myalgia, and cough. Very few patients have chest pain, dyspnea, and hypotension. It is due to the leucocyte-released pyrogenic cytokines in the stored blood. More common with PRBC and platelets. Management is supportive with paracetamol. As WBCs are central in pathogenesis of FNHR, patients with FNHR are given leuko-reduced blood products. As fever is also found in AHTR and TRALI, differentiation is necessary.

**Transfusion-associated acute blood reaction (TRALI):** TRALI is a life-threatening, acute lung injury due to sequestration of activated neutrophils in the pulmonary circulation. These activated neutrophils cause a severe inflammatory cascade and result in lung injury. Typical features include sudden-onset (within **6 hours**) **non-cardiogenic pulmonary edema, bilateral pulmonary infiltrates**, fluid in the endotracheal tube, **fever**, **hypoxemia**, hypotension, and leucopenia. This may progress to ARDS in a few cases. Management is supportive with supplemental oxygenation/ intubation and hemodynamic support. It results in resolution within 4-5 days in most patients. The role of steroids is not well established but may be useful in a few cases.

**Plasma** and PRBC of multiparous donors are most common inciting agents. Use of male predominant FFP source has reduced the occurrence of TRALI.

**Transfusion associated circulatory overload (TACO)**: It is the **volume overload** due to excess volume of blood components transfused, causing cardiogenic pulmonary edema. Patients with pre-existing cardiac or renal dysfunction receiving large volumes of PRBC or FFP are at increased risk. Like TRALI, patients with TACO have acute dyspnea during/following blood transfusion. However, **fever is absent, neck veins are distended, and CVP and PCWP (> 18 mm Hg) are elevated**. NT-Pro BNP is frequently raised, unlike TRALI. TACO can be prevented by using low volumes and slow infusion rates. Treatment includes diuretics, fluid restriction, and maintaining oxygenation using supplemental oxygen or positive pressure ventilation.

Section 4

# Nutritional Support in Critical Care

# 1. Nutritional Support: Introduction

Adequate nutrition in critically ill patients is essential as:

1. Most of the Patients in ICU are in catabolic state.
2. Proper and adequate nutrition is necessary to strengthen the immune system.
3. Adequate nutrition improves patient outcomes.

In absence of nutritional support there is rapid energy, protein, and micronutrient deficit causing decreased immune function, prolonged ventilator duration, and increased mortality. Patients with malnutrition at the time of admission are more likely to benefit from nutritional support. NUTrition Risk in the Critically ill (NUTRIC) score is an objective method of assessing the nutritional status at the time of admission.

**Indications for nutrition support:** Nutritional support should be considered for all patients staying in the ICU for >48 hours. Greater benefits are seen in the following patient groups:

1) Patients with high caloric requirements (e.g., burns, sepsis, major, surgery, or trauma).
2) Patients who sustain high protein losses (e.g., corticosteroid or tetracycline usage, nephrotic syndrome, or draining fistulas); and
3) Patients who are already malnourished at the time of ICU admission.

Once a decision has been made to provide supplemental nutrition, three basic questions need to be answered:

A. What route of feeding will be used?
B. The amount of proteins and calories to be given.

**Route of feeding:** Oral route is the preferred choice. Enteral nutrition (EN) is used if patients' oral feeding is not feasible. Parenteral nutrition (PN) is the least preferred option and is used only if above two fail/contraindicated. Starting EN early (within 48 hours) reduces the incidence of pneumonia and may also reduce mortality. Enteral nutrition (even in small amounts) prevents atrophy of gut mucosa. This prevents the transfer (translocation) of gut bacteria to systemic circulation.

**Type of feed:** Blenderized food is widely available and cheap but has variable nutritional content and there is a high contamination risk. Commercially available tube feeds are available in sterile packaging and given using closed/sterile system. These have a known nutritional content and have reduced risk for contamination. Continuous feeding is preferred to bolus feeding.

## Enteral Nutrition

**Candidates for enteral nutrition (EN):**

- Functional GIT
- Can not eat for medical reasons.
  - Trauma
  - Surgery
  - Unconscious
  - Dysphagia
  - Major full-thickness burns
- Risk for malnutrition
  - Inadequate nutritional intake for 5 days
  - Wt. loss>10% in 6 m/>5% in 1 m
  - Wt. <20% of ideal body wt.

**Advantage over parenteral nutrition (PN):**

1. Maintains gut mucosal structure.

2. Decrease bacteria and toxin translocation.
3. Does not require central venous catheter.
4. Reduced risk of sepsis and central line related complications.
5. Promotes enteric hormone secretion.
6. Buffers gastric acid.
7. Less likely to cause hyperglycemia.
8. Provides unique and complex nutrients not available by TPN.
    - Glutamine
    - Dietary fiber
    - Medium- and short-chain fatty acids
9. Dramatically reduced cost

**Contraindications of EN:**

- Intestinal obstruction
- Paralytic ileus
- Bowel perforation
- Intestinal ischemia
- Intractable vomiting
- Circulatory shock
- GI hemorrhage
- Pancreatitis
- Enterocutaneous fistula (>500ml/day)
- Severe diarrhea (>200gm/24hrs)

## Complications of EN:

- Complications due to insertion – injury to nasal passages, pharynx or esophagus, bleeding, sinusitis.
- Pulmonary aspiration - prevented by 30□ head up position and attention to NG aspirate volume.
- Alteration in drug absorption and metabolism.
- Diarrhea – minimized by early commencement of isotonic feed before mucosal atrophy develops (24-48 hours). If diarrhea occurs, Clostridium difficile should be excluded, prokinetics ceased, hypertonic feeds replaced with isotonic and unnecessary antibiotics ceased. If diarrhea persists, motility reducing agents and probiotics may be prescribed.
- Refeeding syndrome.
- Exacerbation of ileus or obstruction.
- Tube misplacement in respiratory tract – detected on chest radiograph.
- Tube blockage by feeds or medications – prevented by flushing after all medications or if feeding is stopped.

Constipation– in patients on established feeding with structurally normal bowel, who have not passed stool for 48h, colonlytely may be administered enterally at 200ml QID. If this has no effect, neostigmine may be infused intravenously at 10mg/day, until stool is passed. Bradycardia or cardiac conduction defects are contraindications to neostigmine.

**Table No.1:** Energy requirements in critically ill patients

| Daily caloric requirements | Using body weight |
|---|---|
| Sedated mechanically ventilated patients | 20-24 kacl/kg |
| Mechanically ventilated patients without sedation | 22-24 kacl/kg |
| Spontaneously breathing critically ill patients | 24-26 kacl/kg |

## Nutritional requirements

There are several methods for calculating the energy requirement in critically ill patients. Indirect colorimetry is the gold-standard method for calculating the energy expenditure of patients. But it is cumbersome and not widely available. Instead, energy requirements can be calculated as shown in Table No. 1

**1. Glucose and carbohydrates**

Approximately 70% of total calorie requirements should be provided using carbohydrates. Excess carbohydrates can cause hyperglycemia. The human body has a limit of 5 mg/kg/minute or 7.2 gm/kg/day for glucose metabolism. Rates greater than this will cause significant hyperglycemia.

**2. Lipids:**

Lipids should provide approximately 30% of energy requirements. Usual lipid doses are in the range of 0.5 to 1.5 gm/kg/day. In most patients the limits of lipid metabolism are approximately 1-1.5 gm/kg/day. Linoleic acid is the only essential fatty acid in the diet. Deficiency causes dermopathy, cardiac dysfunction, and decreased immune function. It is present in safflower oil.

Injection propofol contains intralipid and provides 1.1 Kcal/ml. It should also be added when calculating the delivered energy.

**3. Protein and amino acid:**

Critically ill patients require 1.2-2 gm/kg protein per day. If adequate calories are not provided, most of protein is diverted for energy production.

**4. Vitamins, trace elements, and other additives:**

Vitamins and trace elements serve as antioxidants and play key roles as intracellular cofactors for enzymatic and energy generating reactions. **Thiamine** (vitamin B1) is an important co-factor in glucose metabolism. Its deficiency results in Wernicke's encephalopathy, peripheral neuropathy, cardiomyopathy, and lactic acidosis.

**Vitamin E** is the most important lipid-soluble antioxidant in the body. It is present in all cell membranes and protects body from oxidative injury. **Glutamine** is a fuel for the mucosa and has a protective effect on gastric mucosal. It is more important in patients with trauma and burns. Omega 3 fatty acid (fish oil) may be useful in patients with ARDS. Carnitine transports fatty acids to mitochondria. Deficiency causes cardiomyopathy and hypoglycemia.

**Monitoring EN:**

- Weight (at least 3 times/week).
- Signs/symptoms of edema (daily).
- Signs/symptoms of dehydration (daily).
- Fluid I/O (daily).
- Adequacy of intake (at least 2x weakly).
- Serum electrolytes, BUN, creatinine (2-3 x weekly).
- Serum glucose, calcium, magnesium, phosphorus (weekly).
- Stool output and consistency (daily).

## Access for EN:

### Short-term access for EN:

- **NG (nasogastric) tube:** This is the preferred method for EN.
  - Made of soft silastic material
  - Narrow (6-8Fr) tubes are more comfortable.
  - Distance: tip of nose to ear lobe to xiphisternum (50-60 cm)
- **Duodenal**: Tip of the feeding should be advanced beyond pylorus in patients at risk for aspiration. Endoscopic or fluoroscopic guidance may be needed for successful placement.
- **Correct placement: This should be confirmed using:**
  - X-ray.
  - Aspiration of gastric content.
  - Air.
  - Litmus paper.

### Long term access:

- **Gastrostomy:**
  - Indication: Need for nasogastric tube>8 weeks.
  - Generally preferred: Less diarrhea.
  - If pulled out can be replaced.
  - Larger bore tube- less clogging.
- **Jejunostomy:**
  - Useful when there is an upper GI obstruction.
  - Small bore (12 fr tube).
  - Causes more diarrhea.

## *Practical points for EN:*

1. All patients who are on mechanical ventilation should have a nasogastric tube in situ. The position of the nasogastric tube should be confirmed by radiology or by injecting 10-20 ml of air and auscultating the epigastric area.
2. Patients with recent abdominal and bowel surgeries may require prior discussion with the surgeon and intensivist before starting enteral feeding.
3. Bowel sounds (BS): absence of bowel sound is a common finding in a ICU patient and it should not be considered as a contraindication of enteral feeding.
4. Initially in acute phase start with slow continuous feeding.

5. Emesis: Single episodes of emesis does not require holding of enteral feeding.
6. NPO periods >4 hours should be avoided.
7. Start EN within 24-48 hours of admission with low calories delivered initially. Progressively increase the dose with target of achieving 70-100% of calories over next 5-7 days.
8. Try to achieve 25 kcal/kg/day and at least 1.2-1.5 g/kg/day of protein.
9. Flush with 30 ml NS after feed/medication.
10. Each medication is given separately and the feeding tube is flushed with 5ml NS between medications.
11. Enteral feed should be stopped 30 minutes before Trendelenburg position.
    Enteral feed should be stopped 15 minutes before and after medication.

- **Continuous feeding**

  **Continuous feeding** should be started initially at the rate of 20-40ml/hour. Aspirate the feeding tube every 4 hours.

  1. If the amount aspirated is < 200ml, return all aspirates. Increase feeding flow rate by 20 ml/hour every 3 cycles till a flow rate is achieved that fulfill the caloric requirement of the patient. Once caloric target achieved, the feeds may be further diluted with water to address the fluid requirements of the patient.
  2. In case of aspirate >200ml, return all the aspirate to patient and reduce the rate of feeding to half. Rule out bowel obstruction first. If there is no clinical evidence of bowel obstruction, administer prokinetic agents like Erythromycin or metoclopramide.
  3. If amount of aspirates continue to > 200 ml after the above has been carried out, consider the use of small bowel feeding and elemental formulas.

- **Intermittent bolus feeding**

  Start intermittent bolus feeding with 50 ml at 3-hour interval. Do not forget to aspirate before every feed.

  1. If the aspirate amount is less than 200 ml, return back aspirate to patient. Increase the amount by 50 ml after every 4 feeds. Increase by 100 ml/feed every 24 hours till caloric requirements of the patient is fulfilled. Once target calories are achieved, the feeds may be diluted with water to address the fluid requirements of the patient.
  2. If aspirate is more than 200 ml, return the 200 ml aspirate to patient and reduce by 50 ml per feed. Rule out bowel obstruction first. If there is no clinical evidence of bowel obstruction, administer prokinetic agents like erythromycin, metoclopramide etc.
  3. If aspirates continue to exceed 200 ml after the above has been carried out, consider the use of continuous feeding.
  4. Use a closed system for enteral feeding wherever feasible. Closed system refers to the “prefilled enteral feeding bag”.
  5. Feeding must be given with patient in semi-recumbent position with head end elevated 30-45$^0$.
  6. Use polymeric formulas as these are nutritionally complete and made up of mostly intact nutrients for feeding.
  7. Nasojejunal/nasoduodenal i.e., small bowel feeding is used for the patients who are not tolerating enteral feeding due to high inotropic support, continuous infusion of sedatives, or paralytic agents or with high gastric residual volumes or in pancreatitis.
  8. Prokinetic agents such as IV metoclopramide 10-20mg 6-8 hourly and/or IV erythromycin 125 mg QID or 250 mg BID can be used in patients who do not tolerate the enteral feeds.

# 2. Nutritional Support: Parenteral Nutrition

It is the delivery of nutrients intravenously, e.g., via the bloodstream. These are of two types:

- Central Parenteral Nutrition: often called Total Parenteral Nutrition (TPN) delivered into a central large vein.
- Peripheral Parenteral Nutrition (PPN): delivered into a smaller or peripheral vein.

**The nutrient components are in elemental or "pre-digested" form:**

- Protein as amino acids
- Carbohydrates as dextrose
- Fat as lipid emulsion
- Electrolytes, vitamins, and minerals

**Common indications for PN:**

- Patients have failed EN with appropriate tube placement.
- Severe acute pancreatitis.
- Severe short bowel syndrome.
- Mesenteric ischemia.
- Paralytic ileus.
- Small bowel obstruction.

## Components of PN

**Macronutrients: Carbohydrate**

- Source: monohydrate dextrose
- Properties: Nitrogen sparing
- Energy source 3.4 kacl/gm
- **Recommended intake:**
  - Stable: Not >5 mg/kg/min.
  - 70% of total calories.
  - Dextrose limited to 5-10% final concentration.

**Macronutrients: Amino Acids**

- Source: crystalline amino acids—standard or specialty
- Properties: hyperosmolar
  Essential amino acids 40-50%, non-essential amino acids 50-60%
  Glutamine/Cysteine
- **Recommended intake:**
  1.2-2.0 gm/kg/day
- Amino acids 3% final concentration

**Macronutrients: Lipid**

- Source: Sunflower and/or soyabean oil. 2-5% should be linoleic acid, present in safflower oil.
- Properties: long chain triglycerides
  Isotonic or emulsion
  9 kacls/gm
- Recommend intake:
  0.5-1.5 gm/kg/day (not >2gm/kg)
  12-24 hour infusion rate

  30% of total calories

  Max. 60% of kacl or 2gm fat/kg

**Other components:**

- Fluid- 30 to 50 ml/kg (1.5 to 3 l/day)
    - Sterile water is added to PN admixture to meet fluid requirements.
- Electrolytes
    - Use acetate or chloride forms to manage metabolic acidosis or alkalosis.
- Vitamins: multivitamin formulations.
- Trace elements.


Figure No. 1: TPN bag.

Section 5

# Infection Prevention and Control

# 1. Personal Protection

## Healthcare facility recommendations for standard precautions:

1. **Hand hygiene:**
   - Hand washing (40-60 sec): With chlorhexidine containing hand wash. Wet hands and apply soap; rub all surfaces; rinse hands and dry thoroughly with a single use towel; use towel to turn off faucet.
   - Hand rubbing (20-30 sec): Use 3 ml of 0.5 % chlorhexidine and 70% isopropyl alcohol handwash (Figure No.1). Apply enough product to cover all areas of the hands; rub hands until dry.

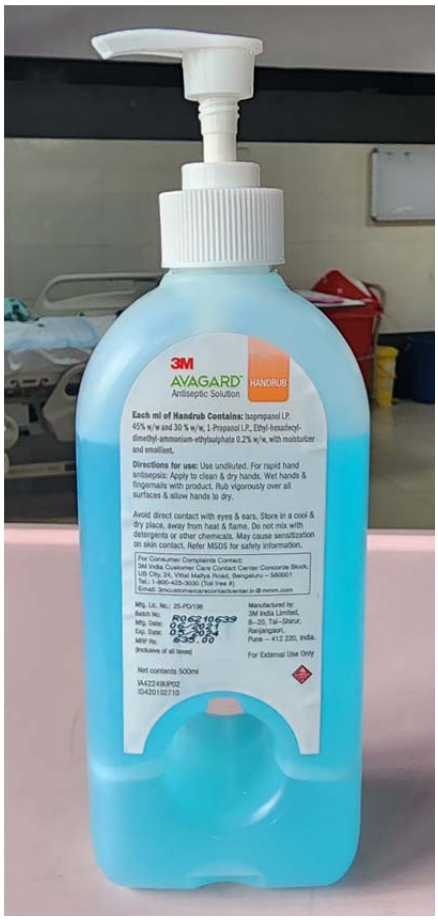


Figure No.1: Alcohol-based hand sanitizer.

2. **Gloves:**
   - Wear when touching blood, body fluids, secretions, secretions, mucous membranes, non-intact skin.
   - Change between tasks and procedures on the same patient after contact with potentially infectious material.
   - Remove after use, before touching non-contaminated items and surfaces, and before going to another patient. Perform hand hygiene immediately after removal.
3. **Facial protection (eyes, nose, and mouth):**
   - Wear (1) a surgical or procedure mask and eye protection (eye visor, goggles) or (2) a face shield to protect mucous membranes of the eyes, nose, and mouth during activities that are likely to generate splashes or sprays of blood, body fluids, secretions, and excretions.

4. **Gown:**
   - Wear to protect skin and prevent soiling of clothing during activities that are likely to generate splashes or sprays of blood, body fluids, secretions or excretions.
   - Remove soiled gown as soon as possible and perform hand hygiene.
5. **Masks:**
   - A three-ply normal mask should always be used to prevent infection spread.
   - An N-95 mask should be used while dealing with patients suspected to have infectious respiratory pathogens like tuberculosis, covid 19, swine flu, influenza, etc.

# 2. Infection Prevention

**Increased risk of infection is associated with:**

- Severity of illness.
- Multiple invasive devices and procedures.
- Contact with healthcare personnel.
- Longer ICU stays.
- Limited space.

It is common to see ICU patients with multiple invasive devices such as urinary catheter, central venous catheter, dialysis catheter, arterial line, tracheostomy etc. therefore risk of catheter related infections is high in these patients.

- Ventilator associated pneumonia is a common cause of mortality and morbidity in ICU.
- Attributable mortality ranges between 5-14% for pneumonia developing in the ICU patients.

**Cross-infection in the ICU: sources**

- Hands of staff and attendants
- Handwashing and communal towel or no handwashing
- Assisted ventilation equipment.
- Suction and drainage bottles,
- I.V. lines – central and peripheral,
- Urinary catheters,
- Wound and wound dressings
- Disinfectant containers
- Dressing trolleys

**Strategies to reduce infection risk**

**I. Identify the patients as potential source of infection.**

- Patients have Diarrhea, skin rashes.
- Known to have any communicable diseases.
- Recognized carrier of an epidemic strain of micro-organism
- Patients with suspicion of having communicable disease admitted to an isolation ward.

**II. Hand hygiene:**

- Hands of health care worker are the most common source of transmission of infection from one patient to other
- It is advised that family members of patient and nursing staff should wash their hands before touching the patients.
- hand wash with soap and water or alcohol-based hand rub should be performed:
  - Before entering the ICU.
  - Before performing any invasive procedure including peripheral cannula insertion and removal.
  - Before administration of IV drugs or fluids or using multidose vials
- Routine hand wash should be performed:
  - Before assisting or performing any invasive procedure
  - When hands are visibly soiled

**III. Procedures requiring aseptic technique:**

- Intravenous drug therapy,
- urinary catheterization
- While performing respiratory care

**A. IV care practices**

- Scrub the hub.
- Cap all ports when not in use.
- While placing a central venous catheter practice full aseptic precaution. Use cap, mask, sterile gown, sterile gloves and large sterile sheet.
- Central venous catheter and dialysis catheter on new onset of fever or if any pus discharge or sign of infection at insertion site. These lines need not to be changed on a routine basis.
- Do not remove Central Venous Catheters on the basis of fever alone. Assess clinically the removal of intravenous lines and catheters. Keep the possibility source of infection elsewhere or fever of noninfectious origin is also as a probability. Peripheral arterial lines should not be replaced routinely.

**B. Respiratory care- patient based interventions:**

- In mechanically ventilated patients' elevation of head end 30-45$^0$ is recommended to reduce the risk of aspiration if not contraindicated
- Drain the condensate collected in tubing's regularly as this increase the resistance. care must be taken not to allow drain towards patient end.
- If possible, use an endotracheal tube with subglottic suction to allow continuous suction.
- Use stress related mucosal disease prophylaxis in the form of sucralfate, H2-blockers, and/or antacids, in a patient on mechanical ventilation.
- High-risk preoperative patients who are undergoing any abdominal, thoracic, head or neck surgery or whose pulmonary functions are compromised, advise them deep breathing exercises and try to mobilize them as early as possible.
- Use only sterile fluid for nebulization and procedure should br performed aseptically.
- If multi dose vials are used with sterile techniques.

**Bundle approach to infection prevention:**

***VAP (Ventilator Associated Pneumonia) Care Bundle:***

1. Daily sedation vacation (unless contraindicated) or assessment using sedation score.
2. Daily assessment of readiness to wean.
3. Stress ulcer prophylaxis using proton pump inhibitors.
4. Nursing patients in a semi recombinant position (35-45 degree).
5. Use of subglottic suction.

***CAUTI (Catheter Associated Urinary Tract Infection) Care Bundle:***

1. Ensure proper indication for catheter insertion.
2. Proper barrier precautions while insertion.
3. Hand hygiene and standard precaution while inserting and maintenance.
4. Unobstructed urinary flow.
5. Removal as early as possible.

***CLABSI (Central Line Associated Blood Stream Infection) Care Bundle:***

1. Proper indication for insertion.
2. Choose an appropriate site to avoid infection.
3. Maximum barrier precaution and strict aseptic technique while insertion.
4. Use chlorhexidine 0.5% in alcohol for site preparation.
5. Daily assessment of the need for central catheter and prompt removal.
6. Standard aseptic technique while handling central catheters.

Section 6

# Specialty Critical Care

# 1. Cardiovascular System: Shock

Shock is the clinical condition of ***organ dysfunction*** **resulting from an** ***imbalance between cellular oxygen supply and demand.*** It results from decreased supply, increased demand, or inadequate oxygen utilization. Early recognition and management of shock are essential because advanced stages of shock are challenging to treat and have high mortality. According to pathophysiology, shock is commonly classified into the following categories:

**Hypovolemic Shock**: This happens when there is a significant **loss of intravascular volume**, leading to decreased blood volume and subsequent decreased cardiac output. Hemorrhagic shock occurs due to blood loss, e.g., polytrauma, abruptio placentae, and gastrointestinal bleeding. Non-hemorrhagic shock occurs with severe diarrhea, vomiting, polyuria, and burns.

**Distributive shock**: This occurs when there is **widespread vasodilatation** and loss of vascular tone, leading to **relative hypovolemia** despite adequate intravascular volume. Examples include septic shock, neurogenic shock, anaphylactic shock, and adrenal insufficiency.

**Cardiogenic shock**: This occurs due to the primary inability of the heart to pump adequate blood into the body organs. Examples include myocardial infarction, myocarditis, arrhythmias, and valvular heart disease.

**Obstructive shock**: In obstructive shock, the heart is unable to pump adequate blood due to **external obstruction to the blood flow**. Examples include tension pneumothorax, cardiac tamponade, restrictive pericarditis, pulmonary embolism, and aortic dissection.

Mixed shock is the condition having more than one mechanism of shock. A patient of septic shock developing cardiomyopathy has two types of shock: distributive and cardiogenic shock.

## Stages

All types of shock have three stages:

**Compensated shock (pre-shock)**: This is the initial phase of shock. In this phase, the body utilizes a variety of physiological responses to counteract the initial insult and attempts to reestablish adequate perfusion and oxygen delivery. There are no overt signs of organ dysfunction. If the shock is recognized in this phase, management is relatively easy.

**Decompensated shock**: In this phase, organ dysfunction starts, and the patient's compensatory response to the insult is overwhelmed. Organ failure starts in this phase.

**Irreversible shock**: This is the late phase of shock in which multi-organ failure occurs. Recognition of the shock in this phase is easy, but managing irreversible shock is very difficult with high mortality.

## Features of shock:

**Hypotension** is common in shock, **but it is not mandatory for the diagnosis of shock**. **Tachycardia** is more common than hypotension and reflects the body's physiological response to improve oxygen supply to tissues. Tachycardia is absent in patients with heart block and patients taking beta-blockers.

Table No. 1: Comparison of shock types

| | Preload CVP/PCWP | Afterload/ SVR | Cardiac Output/ Pulse pressure | Lactate | Urine output | SvO2 | Cold/clammy extremities |
|---|---|---|---|---|---|---|---|
| **Hypovolemic** | ↓ | ↑ | ↓ | ↑ | ↑ | ↓ | Yes |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| **Cardiogenic** | ↑ | ↑ | ↓ | ↑ | ↑ | ↓ | Yes |
| **Obstructive** | ↑/↓ | ↑ | ↓ | ↑ | ↑ | ↓ | Yes |
| **Distributive** | ↓/N | ↓↓ | ↑/N | ↑ | ↑ | ↑ | No |

**Tachypnea** is common as the body compensates for reduced oxygen delivery and metabolic acidosis. **Skin is cool, clammy, and mottled** due to vasoconstriction causing hypoperfusion of skin. **Capillary refill time** (CRT) is also raised (>3 seconds). However, the skin is warm in distributive shock because of peripheral vasodilation. CRT is also normal in distributive shock. **Oliguria** (urine output <0.5 ml/kg/hour) results from hypoperfusion of the kidneys and is an easily identifiable feature present in all types of shock. It is also useful for evaluating the effectiveness of shock treatment. In the initial shock phase, blood is diverted from other organ systems to the brain, and brain function is normal. Mental state changes are seen in advanced and decompensated shock. **Altered mental status** should not be ignored as they may represent decompensated or irreversible shock. Due to inadequate oxygen delivery in shock, anaerobic metabolism starts in the tissues. **Blood lactate levels increase** in all shock types as lactate is a byproduct of anaerobic respiration. **Mixed venous oxygen saturation (SvO2) is decreased** due to the decreased oxygen supply to the tissues. The features of different shock types are compared in Table No. 1.

## Specific types of shock

### Hypovolemic Shock

Hypovolemic shock is due to dehydration, internal or external bleeding, or gastrointestinal/urinary losses. Hemodynamically, there is reduced preload- reflected as reduced filling pressures (low pulmonary capillary wedge pressure, central venous pressure, and jugular venous pressure). Cardiac output, stroke volume, and pulse pressure are also decreased. The blood pressure is frequently (but not always) low, with rapid heart rate. *Shock index* is the ratio of heart rate and systolic blood pressure. The normal shock index is 0.5-0.7. A shock index of ≥1 suggests hypovolemic shock. $SvO_2$ decreases as the oxygen delivery to the tissues is decreased. Clinical features include hypotension, tachycardia, reduced jugular venous pressure, decreased pulse pressure, tachypnoea, oliguria, cool, clammy, mottled extremities, and altered mental status.

Replacement of intravascular fluid is the mainstay of hypovolemic shock management. The fluid of choice is balanced crystalloid solutions (e.g., ringer's lactate). Colloid solutions (albumin, gelatins, and dextrans) are used if large amounts of crystalloids are required. Dynamic indices of intravascular fluid deficit (pulse pressure variation, stroke volume variation, and passive leg raising) are preferred for diagnosing and monitoring ongoing fluid resuscitation. Packed blood cell transfusion is needed to keep hemoglobin level >7 gm/dl. If fluid resuscitation cannot maintain tissue perfusion, vasopressors (norepinephrine, vasopressin) are added, and a second etiology of shock is searched.

### Distributive Shock

Distributive shock results from widespread severe vasodilatation, leading to a condition of relative hypovolemia. Septic and anaphylactic shock are two important examples of distributive shock. The hemodynamic profile is of reduced systemic vascular resistance, increased/normal cardiac output, and decreased/normal preload. Hypotension is common but not universal. Tachypnea, tachycardia, reduced urine output, and altered mental status are present. However, the capillary refill time is normal, and the skin is warm and well-perfused.

**Septic shock**: Septic shock is **one of the most common types of shock encountered in the ICU**. It is defined as acute circulatory failure secondary to life-threatening sepsis. $SvO_2$ is paradoxically raised as oxygen utilization at the cellular level is also impaired in septic shock. Management includes intravenous fluid bolus (30 ml/kg within initial 3 hours), empiric antibiotics, and, if needed, vasopressors (norepinephrine and vasopressin).

**Anaphylactic shock**: Anaphylaxis is a severe, life-threatening allergic reaction that can occur after exposure to an allergen- e.g., antibiotics, latex, succinylcholine, and insect stings. Rash, urticaria, itching, angioedema, rhinorrhea, wheezing, cough, and stridor are also seen in addition to the usual clinical features of distributive shock. Management includes the removal of the trigger. If intravenous access is not present, **intramuscular 0.3 to 0.5 mg epinephrine** is administered every 5-10 minutes. If intravenous access is present, intravenous epinephrine boluses are given. Epinephrine infusion is needed if the shock persists after 3-5 boluses. Fluid bolus (1-2 liters) is given for relative hypovolemia. Adjunctive agents for the treatment of anaphylaxis include $H_1$ antihistaminic (e.g., diphenhydramine, cetirizine, hydroxyzine), $H_2$ antihistaminic (e.g., famotidine) drugs; both relieve itching and hives. Bronchodilators are given to relieve bronchospasm (albuterol, salbutamol). Glucocorticoids (e.g., dexamethasone, hydrocortisone, and methylprednisolone) do not relieve the initial symptoms and signs of anaphylaxis. Oxygen supplementation is given if required.

## Cardiogenic shock

Cardiogenic shock is caused due to **cardiac dysfunction** that leads to inadequate tissue perfusion. Features of reduced organ perfusion include cool, clammy, mottled extremities, weak pulse with narrow pulse pressure, reduced urine output, altered mental status, increased blood lactate, and reduced SvO2. Cardiac filling pressures (pulmonary capillary wedge pressure with left heart failure, central venous pressure for right ventricle failure) are typically elevated. Cardiac output is reduced (cardiac index <2.2 L/minute/$m^2$), and there is a compensatory increase in systemic vascular resistance.

**Treatment**: Most common cause of cardiogenic shock is acute myocardial infarction. Immediate revascularization is required in ST elevation MI. Hemodynamic support with vasopressor (e.g., noradrenaline) and inotropic agents (e.g., dopamine, dobutamine, adrenaline, milrinone, and levosimendan). If the pharmacological therapy fails, mechanical devices like intra-aortic balloon pump and veno-arterial ECMO are indicated. Ventilatory support is required in many patients.

## Obstructive shock

Obstructive shock is due to ***extracardiac*** **obstruction of blood flow,** leading to tissue hypoperfusion. Tension pneumothorax and pericardial tamponade cause decreased filling of the right ventricle. Pulmonary embolism causes impedance to right ventricle outflow and reduced left ventricle filling. Aortic dissection causes an impedance to left ventricle outflow. The clinical features vary according to specific conditions.

**Management**:

Tension pneumothorax: Decreased breath sounds with hyper resonant percussion notes, and hemodynamic instability is common. Immediate needle thoracotomy or ICD placement in $5^{th}$ or $4^{th}$ intercostal space anterior axillary line.

Pericardial tamponade: Engorged neck veins, pulsus paradoxus, and muffled heart sounds are found. Pericardiocentesis should be done.

Pulmonary embolism: Anticoagulation (e.g., unfractionated heparin, low molecular weight heparin) is the mainstay of therapy in patients with pulmonary embolism. It decreases blood's ability to clot and prevents further blood clot formation. If pulmonary embolism is life-threatening, fibrinolytic are used. Thrombolytic therapy (e.g., recombinant tissue plasminogen activator) dissolves the clot and restores pulmonary blood flow.

Aortic dissection: Immediate hemodynamic support and surgical repair or reconstruction with a synthetic graft is needed.

# 2. Cardiovascular System: Pulmonary Embolism

## Introduction:

Pulmonary embolism is defined as the obstruction of one or more branches of the pulmonary artery or its major branches by a thrombus, fat, air, tumor, or other material, originating from a distant site. The most common cause of PE is the dislodgment of thrombi from deep vein thrombosis in the lower extremities. It is one of the commonest causes of cardiovascular death. The incidence of PE is even higher in intensive care unit (ICU) as multiple risk factors are commonly present in ICU patients. The mortality due to PE is higher than myocardial infarction. Figure No.1 summarizes the overview of PE diagnosis and management. Management of PE requires an understanding of following:

1. **Risk factors** of PE: At least 80% of PE occurs in patients with risk factors.
2. Recognition of **sign and symptoms**: The sign and symptoms are commonly non-specific and can mimic other diseases.
3. Pre-test **probability of PE**: Wells score and revised Geneva score predict the probability of a patient having PE, based on presence of clinical features and predisposing factors.
4. **Diagnostic tests**: Patients having a low or intermediate pre-test probability should have PE ruled out using d-dimer. If the d-dimer is raised or a patient has a high pre-test probability, imaging tests should be undertaken.
5. Bedside **echocardiography**, evaluating mainly right ventricle (RV) function, is the test of choice in hemodynamically unstable patients. RV dysfunction or presence of presence of deep vein thrombosis in proximal veins of lower limb suggest presence of PE in hemodynamically unstable patients.
6. In hemodynamically stable patients, **CT angiography** has become the gold-standard for diagnosing PE due to high specificity, sensitivity and providing results in a short time. But it exposes the patients to ionizing radiation and potentially nephrotoxic radiocontrast agents.
7. **Massive PE (high-risk PE)** is defined as presence of shock/persistent hypotension. Patients who are hemodynamically stable but have RV dysfunction/strain/enlargement or raised ProBNP/troponins are classified as **sub-massive PE (intermediate risk PE)**. Hemodynamically stable patients without RV dysfunction are considered as **non-massive/low-risk PE**.
8. All patients **suspected of pulmonary embolism** should be treated with systemic **anticoagulation** using unfractionated heparin (UFH) or low-molecular weight heparin (LMWH) in absence of contraindications.
9. All hemodynamically unstable patients with confirmed PE (using echocardiography, lower extremity ultrasound, etc.) should be treated with **fibrinolytic therapy** in absence of contraindications.
10. Other available therapies include inferior vena cava filter placement, catheter directed thrombolysis, surgical embolectomy, and extracorporeal life support.
11. As morbidity and mortality due to PE is high, appropriate **prophylaxis** against deep vein thrombosis (DVT) is essential in the presence of risk factors.

## Risk factors for PE:

Virchow's triad for pathogenesis of venous thrombosis include blood stasis, hypercoagulability and endothelial injury. Increase in one or more factors predispose patients for DVT. Table No 1 shows the risk factors of pulmonary embolism.

Table No. 1: Risk factors of pulmonary embolism

| | |
|---|---|
| Surgery: especially lower limb, orthopedic, cancer surgery | Trauma: burns or brain, spine, pelvis, and hip trauma |
| Immobility | Obesity |

| | |
|---|---|
| Advanced age: >65 years | Malignancy especially adenocarcinoma |
| Previous thrombo-embolism | Pregnancy |
| Acute illness: sepsis, stroke, pneumonia, heart failure, heparin-induced thrombocytopenia | Chronic inflammatory states: antiphospholipid syndrome, inflammatory bowel disease |
| Factor V Leiden mutation, deficiency of protein C, S or antithrombin. | Drugs: estrogen containing OCP |

**Clinical features:**

The signs and symptoms of PE are non-specific. Dyspnea is the most common symptom in PE. Onset is rapid and develops in seconds to minutes. Cough, tachypnea, tachycardia, and rales are also found. Pleuritic chest pain and hemoptysis are seen in smaller and peripheral PE. As lower extremities are the most common source of PE, signs of lower limb thrombosis (pain, swelling) are also present.

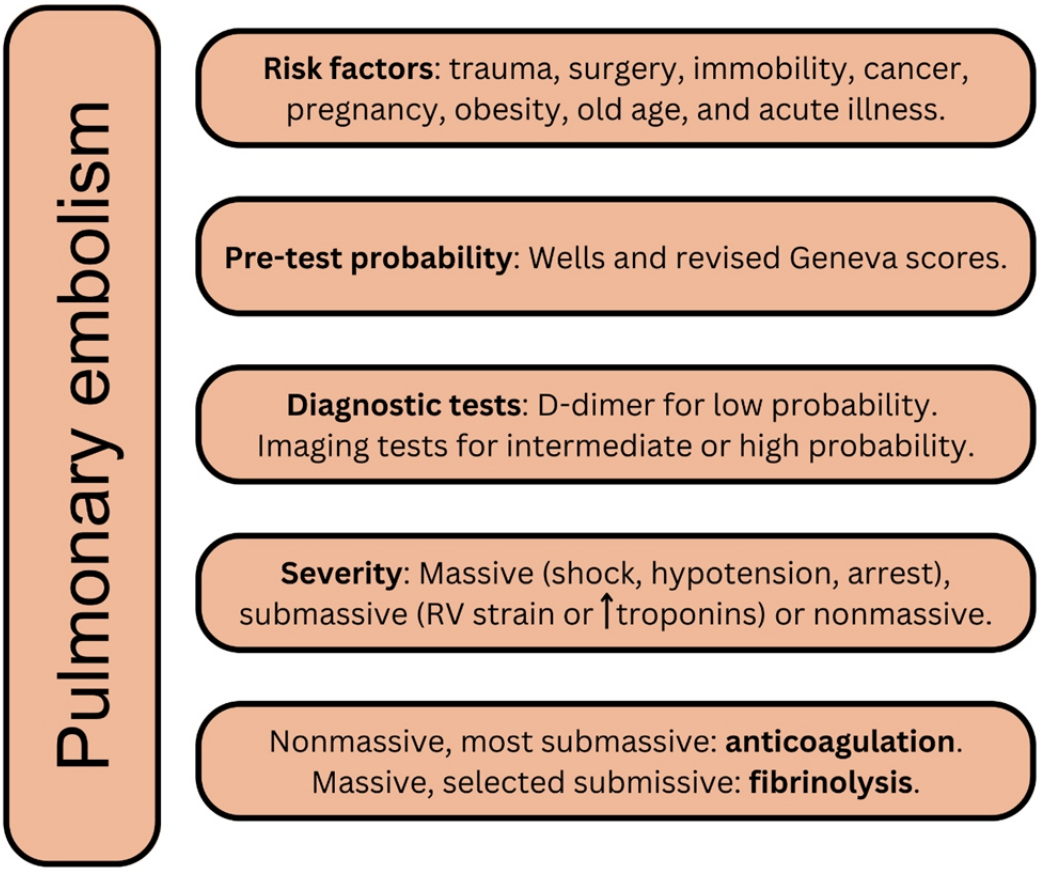


Figure No. 1: Overview of pulmonary embolism.

**Pre-test probability of PE:**

As the clinical features are non-specific, Wells score (Table No. 2) and revised Geneva score (table No.3) are used for predicting the probability of PE. Based on risk factors and clinical features of PE, the patients are classified as having low, intermediate or high risk (probability) of PE. This is important as patients having a high probability of PE are directly subjected to imaging tests of PE, while those of low or intermediate probability have their d-dimers tested first.

## Investigations:

**D-dimer**: D-dimer produced by breakdown of fibrin clot (in the pulmonary embolism). It has a high sensitivity (>90%) but a low specificity (<30%) for PE. A normal d-dimer excludes a diagnosis of PE. But increased d-dimer can be due to several causes and is very common in patients in ICU. D-dimer is **used to exclude PE in patients with low or intermediate probability of PE**. Patients with elevated d-dimer should undergo additional imaging tests to diagnose PE. D-dimer is used only in patients at a low-risk of PE.

Table No 2: Wells Score and modified Wells score:

| Criteria | Points | Interpretation |
|---|---|---|
| Clinical features of DVT (leg swelling, pain on palpation) | 3 | **Wells criteria:**<br>High probability:> 6<br>Intermediate probability: 2-6<br>Low probability: <2 |
| Other diagnosis less likely than pulmonary embolism | 3 | |
| Heart rate >100 | 1.5 | |
| Immobilization (≥3 days) or surgery in the previous four weeks | 1.5 | **Modified Wells criteria:**<br>PE likely: >4<br>PE unlikely: ≤4 |
| Pervious DVT/PE | 1.5 | |
| Malignancy | 1 | |
| Hemoptysis | 1 | |

Table No.3 Revised Geneva score

| Criteria | Points | Interpretation |
|---|---|---|
| Age >65 years | 1 | High probability: 0-3<br>Intermediate probability: 4-10<br>Low probability: >10 |
| Previous PE/DVT | 3 | |
| Surgery/fracture within 1 month | 2 | |
| Active cancer | 2 | |
| Hemoptysis | 2 | |
| Unilateral lower extremity pain | 3 | |
| Leg pain on palpation/unilateral edema | 4 | |
| Heart rate: 75-94 bpm | 3 | |
| Heart rate: >94 bpm | 5 | |

## Imaging tests:

These are used for patients with a **high probability of PE** or for patients who have low risk with elevated d-dimer. The choice of imaging tests depends on the hemodynamic stability of the patients. For unstable patients who are unstable and cannot be transported to radiology suite, echocardiography and lower-extremity ultrasound are the useful options. Hemodynamically stable patients, who can be transported to the radiology suite, have the following options:

**CT angiography (CTA):** This is now the gold-standard test because of high (>90%) sensitivity and specificity, rapid results, and low rates of inconclusive tests. This has excellent diagnostic value for thrombus in large and intermediate sized vessels. Detection of small sub-segmental thrombus is not accurate, but these are never hemodynamically significant. It can also help in diagnosing non-embolic causes of acute dyspnea (pneumothorax, aortic dissection). RV strain for classifying PE severity can also be assessed. CTA exposes patients to radiation and requires potentially nephrotoxic contrast agents.

**Radionuclide lung scan:** These are indicated in patients with renal disease or patients with radiocontrast allergy. Radio-labelled aggregated albumin is injected intravenously. These particles get trapped in the pulmonary circulation and are imaged using scintigraphy. A perfusion defect may indicate an increased/absent flow. In the second part, the patient inhales radio-labelled tracers. The presence of ventilation in the areas of perfusion defect confirms PE. Lung scan has a very high equivocal report incidence. The radiation dose is lower than the CTA.

**Pulmonary angiography:** It was the gold standard for diagnosis of PE but has been replaced by CTA. It is less accurate than CTA for detecting smaller PE and carries all the risks of invasive catheterization. The only advantage is that invasive catheter-based interventions can be done in the same setting if a PE is found.

### Hemodynamically unstable patients

As these patients cannot be transported to radiology suite, findings of either echocardiography or lower-extremity ultrasound can be used for diagnosing PE.

**Echocardiography**: Echocardiography in patients with PE shows RV dilatation and strain. Tricuspid annular plane systolic excursion (TAPSE), an indicator of the RV function is decreased. The interventricular septum becomes flattened due to raised RV volume and pressure. ‘Thrombus in transit’ in the right sided chambers may be seen; it is an indication for fibrinolysis. IVC is dilated with decreased respiratory variation. Echocardiography is frequently abnormal in patients with larger and hemodynamically significant.

**Ultrasound of the lower extremity veins**: Duplex ultrasound is a combination of color doppler and compression ultrasound with a high sensitivity and specificity for DVT detection. Thrombus in a vein result in loss of compressibility of the vein and compression ultrasound is considered a better method for detection of DVT. Since DVT of larger veins (iliac, femoral, and popliteal veins) are the principal source of PE, presence of thrombus with clinical features of PE is taken as a surrogate marker of PE.

Presence of RV dilatation or DVT of large veins of lower extremity, when combined with clinical features of PE is enough for starting the treatment of PE. Absence of abnormal echocardiography or lower extremity ultrasound does not exclude PE.

### Other ancillary tests:

**ECG**: S1Q3T3 pattern (conspicuous s wave in lead I, Q wave in lead III, and T wave in lead III) RV strain and incomplete right bundle branch block are uncommonly found. Sinus tachycardia and ST-T changes are more common.

**Chest X-ray**: Well-described findings of PE are very uncommon. Hampton hump, Westermark sign, and Palla sign are seen in PE. Non-specific findings are common. Chest X-ray is more useful for diagnosing/excluding alternate causes of dyspnea.

**BNP and cardiac troponins**: Both are raised and have a prognostic value.

**ABG**: ABG shows hypoxemia and raised alveolar-arterial gradient. Hypocapnia is common in conscious patients due to raised respiratory rate that overcompensates for the additional dead space. Mechanically ventilated patients frequently have hypercarbia. Figure No. 2 summarizes the diagnostic algorithm of PE.

## Severity of PE:

Patients of acute PE are classified into three classes:

1. Massive/high-risk PE: These are hemodynamically unstable with either of the following:
    a. Hypotension- systolic BP <90 mm Hg or a decrease of >40 mm Hg from baseline or needing inotrope support for >15 minutes,
    b. Cardiogenic shock, or
    c. Circulatory collapse or arrest.

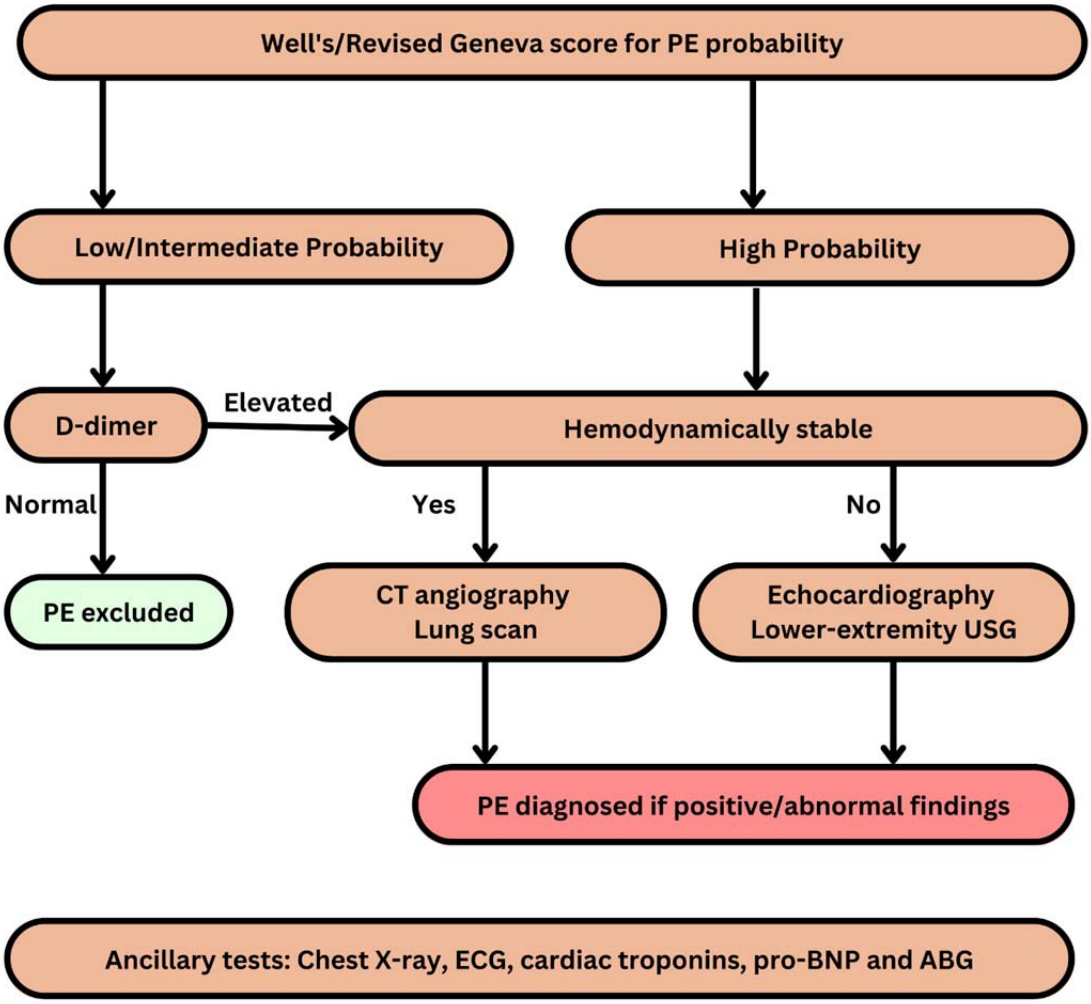


Figure No. 2: Diagnostic algorithm of pulmonary embolism.

2. Submassive/intermediate risk PE: RV dysfunction, pulmonary hypertension, or elevated cardiac troponins.
3. Nonmassive/low-risk PE: Systolic BP>90 mm Hg and no evidence of RV dysfunction, pulmonary hypertension or myocardial marker elevation.

## Treatment:

Treatment of PE is shown in Figure No.3. Treatment is divided into supportive, definitive, and advance therapies.

**Supportive management:**

Airway, breathing and circulation are supported.

**Oxygenation**: Patients are frequently hypoxemic. Supplemental oxygen may help keep peripheral oxygen saturation >90%.

**Intubation and mechanical ventilation**: If needed, endotracheal intubation should bed done carefully as these patients are at risk of cardiovascular collapse and rapid desaturation during intubation. Mechanical ventilation increases the pulmonary vascular resistance and increase RV afterload. Normocarbia, 100% oxygen, low PEEP, low inspiratory pressures, and avoidance of acidosis reduce the pulmonary vascular resistance.

**Fluid management**: Small boluses of fluid (250-500 ml) may increase the cardiac output in hypovolemic patients. Large amounts of intravenous fluids may cause RV distension and flattening of the interventricular septum.

**Vasoactive drugs**: Vasoactive drugs may help maintain blood pressure. A combination of inotropes (nor-epinephrine, dobutamine) and vasopressor (vasopressin) may be needed in some cases. Pulmonary vasodilators (milrinone and inhaled nitric oxide) reduce pulmonary vascular resistance and improve RV function.

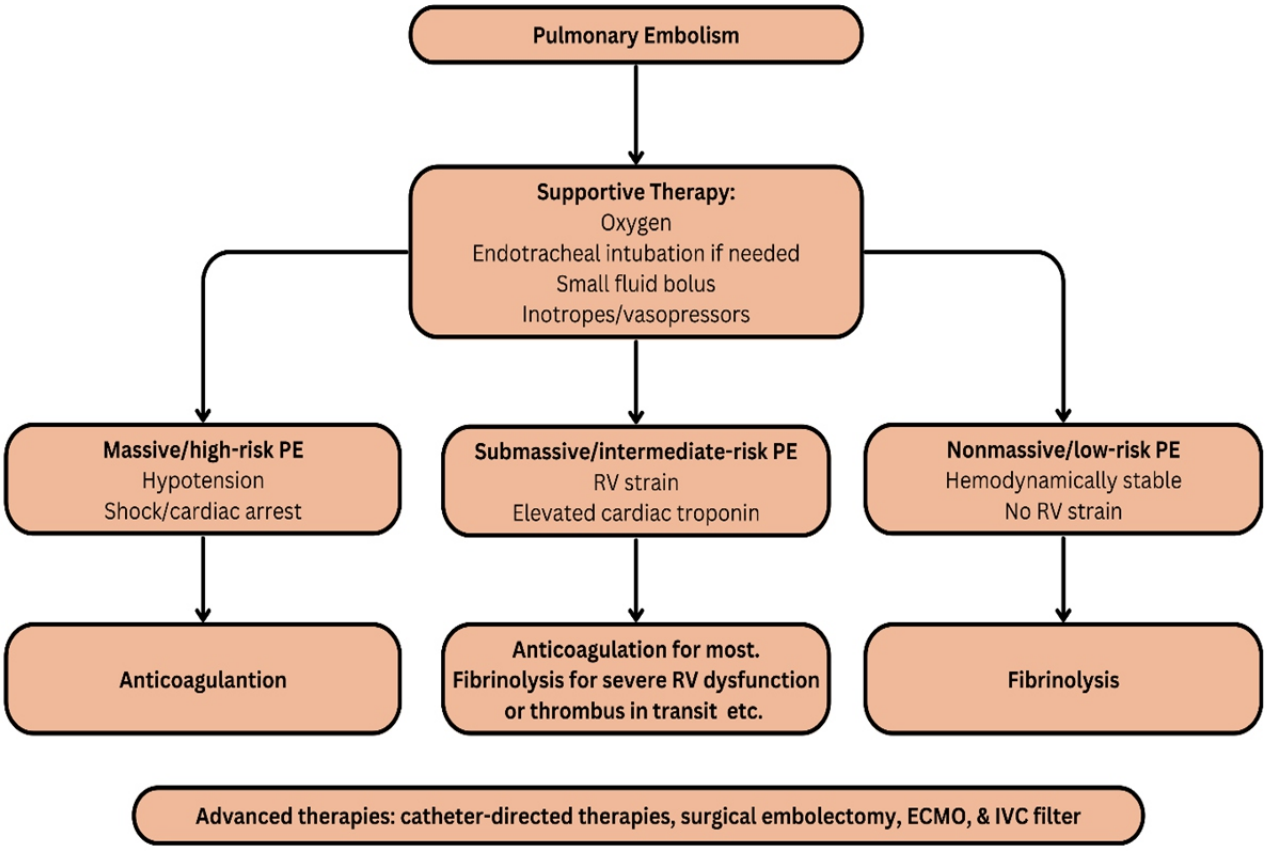


Figure No. 3: Management of pulmonary embolism.

**Definitive therapy:**

**Anticoagulation**: All hemodynamically stable (submassive/nonmassive PE) patients should receive immediate anticoagulation, in absence of contraindications. Anticoagulation can be started even before confirmation of PE using imaging (CT angiography, lung scan, and pulmonary angiography) tests. If PE is excluded on imaging tests, anticoagulation is stopped. Anticoagulants prevent further clot formation and propagation but have a minimal effect on already formed thrombus.

Choice of anticoagulation: Subcutaneous low-molecular weight heparin (LMWH), fondaparinux or intravenous unfractionated heparin (UFH) ae common choices of anticoagulation in these patients. LMWH and fondaparinux are preferred in hemodynamically stable patients. (UFH is better for patients with renal failure, planned for thrombolysis and hemodynamic instability).

**Fibrinolysis**: In absence of contraindications, all hemodynamically unstable patients are treated with fibrinolytics. Fibrinolytics (reteplase, alteplase, streptokinase) cause breakdown of the thrombus in pulmonary vessels and resolution of PE with a high success rate. It is associated with risk of bleeding. Greatest benefit of thrombolysis is seen if thrombolysis is done within 48 hours of symptom onset, but it can be efficacious up to 2 weeks.

Patients with submassive/intermediate risk PE can be treated with fibrinolytics if there is severe respiratory failure, new hemodynamic instability, thrombus in transit, or severe RV dysfunction.

**Advanced therapies:**

**Surgical embolectomy**: For patients of acute massive PE, it is indicated for patients for patients with contraindications to or failed thrombolysis. It requires cardiopulmonary bypass and has a high mortality rate.

**Catheter directed thrombolysis**: These include inserting a catheter in the pulmonary artery branches and lysing the thrombus using targeted fibrinolytic, ultrasound-assisted thrombolysis, and suction embolectomy.

**Inferior vena cava filter**: These are indicated for patients with recurrent PE and contraindication to anticoagulation.

# 3. Acute Kidney Injury

Acute Kidney Injury (AKI) is characterized by a sudden (over hours to days) decline in kidney function. This results in an inadequate excretion of urea and nitrogenous waste products with water and electrolyte imbalance. It is defined as an increase of serum creatinine by ≥0.3 mg/dl in 48 hours, or >1.5-times the baseline. A urine output <0.5 ml/kg/hour for 6 hours also defines acute kidney injury and can be used for **early** identification of AKI. A urometer (Figure No.1) helps to accurately measure hourly urine output. AKI can be due to several etiologies and its management depends on the mechanism of ALI. AKI can be broadly divided into three categories:

**Prerenal AKI** is due to inadequate perfusion of kidney due to hypovolemia or inadequate cardiac output. This is seen in hemorrhagic shock, severe dehydration, and cardiogenic shock.

**Renal/intrarenal AKI** is caused by intrinsic kidney disease. Common examples include acute tubular necrosis (ATN) and acute interstitial nephritis. ATN is caused by ischemic or toxic injury to the renal tubules. Severe sepsis, radiocontrast agents, and aminoglycosides are common causes of ANT in ICU.

**Postrenal/obstructive AKI** is caused by obstruction of urine outflow after the renal collecting ducts. Obstruction can be caused by nephrolithiasis, extraluminal masses, or prostatic hyperplasia.


Figure No. 1. Urometer

## Differentiating the types of AKI:

Bedside ultrasound is usually the first investigation for this purpose. Postrenal AKI is easily identified by marked dilatation of collecting system and hydroureteronephrosis. If an obstructive reason for AKI is found, it should be promptly treated. The rest of the investigations focus on differentiating prerenal and renal AKI. In prerenal AKI, the kidneys preserve sodium and urinary sodium on spot urine examination is low (<20 mEq/L). In renal cause, the urinary concentrating ability is lost, and spot urine examination is >40 mEq/L. Similarly, the urine is concentrated in prerenal cause. Fractional excretion of sodium and urea is low while urine osmolality is higher in prerenal AKI.

## Clinical Features:

Oliguria/anuria, and hypertension/hypotension are commonly seen in patients with AKI but are not universal. Uremia is due to accumulation of nitrogenous waste products (generally at BUN> 100mg/dl) causing systemic symptoms. Uremia causes anorexia, nausea, vomiting, cognitive impairment, pruritis, hypertension, pericarditis, arrhythmias, pleural effusion, anemia, thrombocytopenia, and metabolic acidosis.

## Management:

**Prerenal AKI**: Management is directed towards improving the renal perfusion. Hypovolemic patients require fluid bolus and if required vasopressors. Colloidal starch solutions should be avoided as they aggravate AKI. Improving the cardiac output patients with inotropes and mechanical devices is indicated in patients with cardiac failure.

**Renal AKI**: There is no specific treatment. Stopping/removing the offending drug/toxin is necessary. N-acetyl cystine is protective against contrast-induced injuries.

**Postrenal AKI**: removal of obstruction is necessary and frequently requires surgical/percutaneous placement of a drain.

### General Management and treatment of complications:

**Fluid overload**: As the patients' ability to excrete fluids is impaired, they may have fluid overload, edema, pulmonary congestion, and hypertension. Fluid intake should be restricted, and fluid intake and output are closely monitored. Fluid overload causing severe complication or very low urine output may require dialysis for excess fluid removal. Furosemide can also help excess fluid removal but only in recovery phase of AKI. Use of dopamine is not useful and may cause harm.

Patients with hypovolemia require fluid boluses. Administration of diuretics may aggravate hypovolemia and worsen AKI.

**Electrolyte management**: Hyperkalemia is common and is prevented by avoiding potassium containing fluids. Management includes calcium gluconate, insulin + glucose, albuterol, potassium binders and hemodialysis. Other derangements commonly seen are hypocalcemia, hypermagnesemia, acidosis, and hyperphosphatemia.

**Metabolic acidosis**: It is commonly seen in patients with AKI. The goal is to keep pH> 7.2 and bicarbonate level > 22 mEq/L. Sodium bicarbonate can be given to patients who are not in volume overload and have acceptable urine output. Patients in volume overload and oliguria may need dialysis to correct acidosis and hypervolemia.

**Uremia**: The development of uremic signs and symptoms (vomiting, cognitive impairment, pruritis, pericardial frictional rub, and arrhythmia) in AKI is an indication for dialysis.

### AKI bundle:

These are the group of interventions aimed at preventing or treating AKI. This includes:

1. Hemodynamic optimization: Maintain a mean blood pressure >70-75 mm Hg and diastolic blood pressure > 55 mm Hg. A mean blood pressure >80-85 mm Hg is recommended in patients with chronic hypertension.
2. Volume optimization: Correction of intravascular is required for prevention of further kidney injury in prerenal AKI. Volume resuscitation should be done using a balanced salt solution (ringer's lactate). Large amounts of normal saline use can cause hyperchloremic metabolic acidosis. Colloids containing starch, gelatins, or dextran are not recommended as they can cause renal injury and coagulation defects.
3. If a vasopressor is needed, norepinephrine is the first choice. Dopamine is not useful. In hepatorenal syndrome, vasopressin and terlipressin are useful.
4. Glycemic control: Maintaining blood glucose <180 mg/dl is beneficial for patients at risk for AKI.
5. Avoiding nephrotoxic drugs: Replacement of nephrotoxic drugs with non-nephrotoxic drugs is required. Dose of drugs affected by renal failure and renal replacement therapy need to be reduced.
6. Perioperative AKI prevention: Use of statins in major vascular surgery and optimization of hemodynamic status reduces risk of AKI.

## Renal Replacement Therapy

**Indications for renal replacement therapy (RRT):** Patients with any of the following may require RRT:

1. Severe metabolic acidosis (pH <7.1).
2. Life threatening electrolyte disorders e.g., refractory hyperkalemia ($K^+$ >6.5 mEq/L).
3. Uremia (altered mental status, pericarditis, neuropathy).
4. Severe hyponatremia ($Na^+$ > 160 mEq/L) or hypernatremia ($Na^+$<115 mEq/L).
5. Significant fluid overload, not responding to diuretics.
6. Oliguria or anuria.
7. Dialyzable drug overload/toxin.

**Modalities for RRT**: Out of the several available modalities conventional intermittent hemodialysis, continuous renal replacement therapy and sustained low-efficiency dialysis are commonly used. These modalities differ in their mechanism, efficiency, tolerability, and speed.

**Intermittent hemodialysis (IHD):** IHD is preferred option for hemodynamically stable AKI patients. Even in hemodynamically stable patients, the incidence of hypotension is 10-40% due to intercompartmental fluid shifts or excessive fluid removal. The rate of dialysis is higher and IHD typically lasts for 4 hours and is conducted every alternate day. It is recommended for patients with hyperkalemia. Rapid fluid shifts can cause increased intracranial tension. It is also widely available and cheap. IHD is feasible without anticoagulation. Figure No. 2 shows a hemodialysis machine.

**Continuous renal replacement therapy (CRRT)**: CRRT allows for slow removal of fluid and solutes. It is a less efficient method and not considered optimal when urgent removal of a solute (severe hyperkalemia, toxins) is required. Slow removal of fluids makes it suitable for hemodynamically unstable patients. Slow but continuous removal of drugs and toxins allows for better equilibration in body fluid compartments, reducing the risk of rebound. As the rate of correction is slow, fine adjustments during the CRRT are possible. The resulting disadvantage is the requirement of continuous use over 24 hours a day. The equipment for CRRT is relatively simple and can be easily performed by ICU staff at bedside. CRRT is costly and limits mobility of the patients. Figure No. 3 shows a CRRT machine.

**Sustained low-efficiency dialysis (SLED)**: SLED has features of IHD and CRRT. SLED can be performed by standard intermittent hemodialysis machines, using a slow dialysate flow for prolonged periods (8-12 hours). This allows for excellent solute removal with hemodynamic stability. Its properties are hybrid between IHD and CRRT. It is possible to use SLED without anticoagulation.

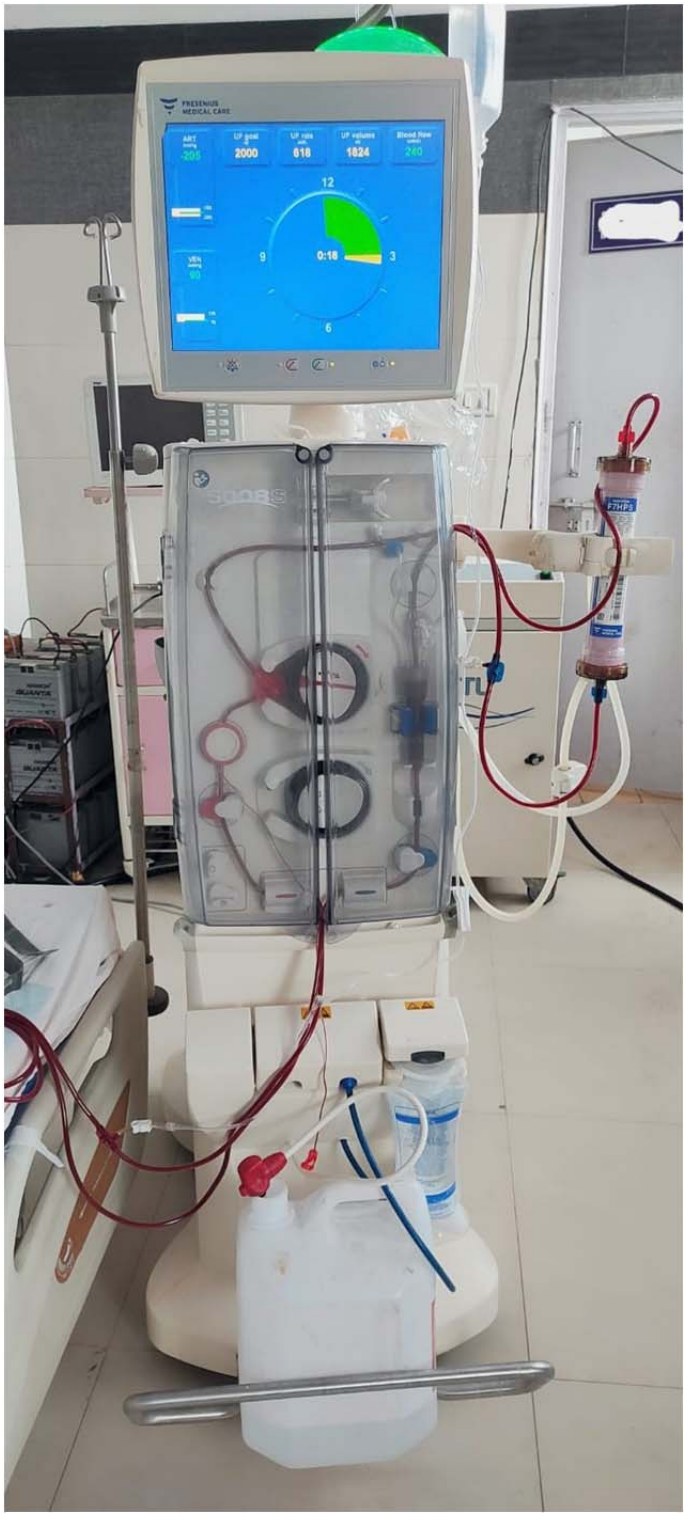


Figure No. 2: A hemodialysis machine.


Figure No. 3: A CRRT machine

# 4. Acute Liver Failure

Liver is the largest abdominal organ and is vital for carbohydrate, lipid and protein metabolism, detoxification, synthesis of coagulation factors (all except III, IV, and VIII), heme and bilirubin metabolism, hormone synthesis and deactivation, and immune regulation.

## Liver function tests

**Indices of Hepatocellular injury**: These include alanine aminotransferase (ALT), aspartate aminotransferase (AST). ALT is more specific for liver injury, and AST/ALT <1 is found in viral hepatitis. AST/ALT> is commonly found in alcoholic liver disease (which affects other organs also). The amount of rise does not correlate with the severity of the disease. An end-stage 'burnt-out' cirrhotic liver may have only moderately raised aminotransferase levels due to few viable hepatocytes. Lactate dehydrogenase is a non-specific marker of liver injury. Glutathione-s-transferase (GST) is a sensitive and early marker of liver injury.

**Markers of Cholestasis (obstruction of bile flow):** Common indicators of cholestasis are conjugated bilirubin, alkaline phosphatase (ALP), 5′-nucleotidase (5-NT), gamma-glutamyl transferase (GGT). ALP is non-specific as it is also present in bone, placenta, and intestine. GGT and 5-NT are very specific for liver disease.

**Markers of synthetic function**: Serum prothrombin and albumin are commonly used as indicators of hepatic-synthetic function. Factor VII has a rapid turnover due to a half-life of 6 hours, leading to an early rise in INR with acutely decreased hepatic synthetic function.

Other tests: These include measurements of liver blood flow (indocyanine green clearance), radiological methods (ultrasound, CT, MRI), and liver biopsy.

***Patterns of liver injury:***

**Hemolysis/prehepatic**: Predominant unconjugated hyperbilirubinemia is seen.

**Hepatocellular injury/parenchymal dysfunction**: ALT and AST are typically raised but can be only moderately elevated or even normal in end-stage liver disease. Conjugated bilirubin is raised. PT and albumin are deranged.

**Cholestatic disorders**: Conjugated bilirubin, ALP, 5-NT, and GGT are typically raised. PT is also raised but returns to normal on vitamin K administration.

## Acute liver failure (ALF):

Acute liver failure is a rapid (<26 weeks or <6 months) deterioration of liver function **(defined as INR>1.5 and hepatic encephalopathy)**, in patients without known liver disease. Acute-on-chronic liver failure is much more common and occurs in patients with chronic liver disease due to acute precipitating factors like infection, hepatotoxins or upper gastrointestinal bleeding.

Acute liver failure is classified into hyperacute (<7 days), acute (7 to 21 days), or subacute (>21 days and <26 weeks). In hyperacute and acute ALF cerebral edema is present. Cerebral edema is uncommon in subacute ALF. Common causes of ALF are viral hepatitis, paracetamol poisoning, HELLP syndrome, hypoperfusion/ischemia, and autoimmune hepatitis. Initial clinical features are non-specific and consist of nausea, vomiting, fatigue, anorexia, pruritis, and right upper quadrant pain. Later, jaundice develops with hepatic encephalopathy bleeding and ascites. Typically, large elevations of AST and ALT are seen due to hepatic necrosis. Conjugated bilirubin is elevated.

## General Management of ALF:

**Medications to avoid**:

All drugs (paracetamol, aspirin, isoniazid) causing hepatic injury should be stopped. Sedatives should be avoided as patients of ALF already have encephalopathy. If needed, propofol is preferred over benzodiazepines. As renal failure is also common in patients with ALF, nephrotoxic agents should be avoided.

**Hemodynamic management**:

Patients with ALF have low systemic vascular resistance, leading to hypotension. They are also fluid deficient due to decreased intake and third-space fluid loss. The pattern of shock closely resembles septic shock. Hypotensive patients with ALF should be given fluid bolus. If it does not improve the hemodynamics, norepinephrine and vasopressin are the vasopressor of choice. Adrenal insufficiency can also be present, and steroids may also be needed in refractory shock.

**Deranged hemostasis**:

As most of the pro- and anticoagulants are synthesized in the liver, acute liver failure reduces their levels. Factor VII has smallest half-life and prothrombin time is raised. Prothrombin time or INR correlates with the severity of ALF and has a prognostic significance. But it does not directly correlate with risk of bleeding as other anti- and procoagulant factors are also decreased. Thromboelastography is a better detects the which factor is deficient and aids in selecting the specific blood product needed to stop bleeding. Patients with ALF should be given stress ulcer prophylaxis (e.g., ranitidine/pantoprazole) to reduce risk of gastric mucosa erosion and gastrointestinal bleed. Initiating early enteral nutrition also reduces the risk of gastric mucosal atrophy and erosion. It also reduces the risk of bacterial translocation, which can lead to sepsis and pneumonia.

**Metabolic derangements**:

Hyperlactatemia develops due to increased lactate production (inadequate perfusion) and decreased hepatic clearance of lactate.

Hypoglycemia occurs in severe ALF due to depletion of glycogen stores and failure of hepatic gluconeogenesis. Hypoglycemia can be refractory, requiring continuous dextrose infusion.

Hyponatremia is less common than chronic liver failure but relates with poor prognosis.

**Infection Prevention:**

Patients with ALF are at risk of infection and sepsis. Infection prevention measures must be followed strictly. Typical signs of sepsis may also be masked in ALF. There should be low threshold for starting antibacterials and antifungals for these patients.

**Hepatic encephalopathy, cerebral edema and altered mental status:**

Hepatic encephalopathy is seen in both ALF and decompensated chronic liver failure. It is due to accumulation of metabolic toxins, false neurotransmitters, benzodiazepine line molecules, and inflammatory mediators. Cerebral edema is also seen in ALF due to accumulation of ammonia. Ammonia crosses the blood-brain barrier and causes cytotoxic cerebral edema, contributing to hepatic encephalopathy. Hepatic encephalopathy is graded from I to IV (Table No. 1). Hepatic encephalopathy is an indicator of prognosis.

**Table No. 1. Grading hepatic encephalopathy**

| Grade | Mental status |
|---|---|
| I | Euphoria/depression/anxiety, mild confusion, shortened attention span, disordered sleep |
| II | Lethargy/apathy, personality change, moderate confusion |
| III | Marked confusion and disorientation, incoherent, sleeping but arousable |
| IV | Coma |

Management of hepatic encephalopathy:

1. Correction of metabolic abnormalities.
2. **Lactulose**: It is fermented to fatty acids, reducing the bowel pH. This kills ammonia producing gut bacteria and reduces ammonia absorption. Dose 30-45 ml two-three times a day.
3. Non-absorbable antibiotics: **Neomycin or rifamaxin** can be used to kill the ammonia producing gram-negative bacteria.
4. Avoiding fluid overload.
5. Protein restriction is not indicated.
6. Early dialysis in renal failure. Reduces ammonia levels and corrects electrolytes.
7. Airway protection and mechanical ventilation. Aim for mild respiratory alkalosis.
8. Elevate the head of bed by 15-30 degrees.
9. Intracranial pressure monitoring for grade III and IV hepatic encephalopathy.
10. Liver transplant reverses hepatic encephalopathy.

# 5. Care of Patients with Neurological Disorders

The unconsciousness is the state of unawareness to surrounding and decrease response to sensory stimulus. The common causes of unconsciousness are traumatic brain injury, stroke, tumor and drug overdose or poisoning.

### Airway and Breathing:

**a.** Maintain a patient airway. Position the patient laterally with the chin extended. Tongue fall is the commonest cause of airway obstruction. This lateral recumbent position is the safest position for unconscious patient also referred as "coma position".
**b.** Suction the mouth, pharynx, and trachea as often as necessary to prevent aspiration of secretions.
**c.** Lateral position prevents the pooling of mucous and secretions in the lungs.
**d.** Oxygen supplementation as per patient's requirement.
**e.** Suction of blood, secretion, or vomitus if present.

### Nutritional Needs:

**a.** Start enteral feeding to unconscious patient through naso-gastric tube (NG tube).
  **1)** **Always** observe the patient carefully when administering anything by NG tube.
  **2)** Do not leave the patient unattended while feeding.
  **3)** Keep accurate record (feeding, formula, water, liquid medications)
  **4)** **When** RT feeding an unconscious patient, it is best to place the patient in a sitting position (Flower's or semi flowers) and support with pillows.
**b.** Fluids are given by intravenous access.
  1) Keep accurate records of IV intake and urine output.
  2) Observe the patient for signs of dehydration or fluid overload.

### Skin Care:

**a.** The unconscious patient should be thoroughly cleaned with disinfection wipes. Moisturizing agents should be applied to prevent skin drying. Nails and hair should be trimmed as required.
**b.** Do complete oral hygiene at least twice per shift. Include the tongue, all tooth surfaces, and all soft tissue areas. The unconscious patient is often a mouth breather. This causes saliva to dry and adhere to the mouth and tooth surfaces.
**c.** Keep the nostrils clean and moist.
**d.** Normal blink reflexes and tear-washing mechanism may be absent in comatose patient. Lubrication with eye drops/ ointment should be maintained.
**e.** The perineal area must be washed and dried thoroughly after each defecation.
  1) Change the bed linen if damp or soiled.
  2) Observe the skin for evidence of skin breakdown.
**f.** Skin care should be provided each time the patient is turned.
  1) Examine the skin for an area of irritation or breakdown.
  2) Apply lotion.
  3) Gently massage the skin to stimulate circulation.

### Elimination – urine:

The bladder should be regularly emptied to prevent infection or stone formation.

1) Adequate fluids should be given to prevent dehydration.
2) Keep accurate intake and output records.
3) Monitor hourly urine output and report low urine output.
4) Provide catheter care at least once per shift to prevent infection in catheterized patients.

**Positioning:**

**a.** When positioning the unconscious patient, pay particular attention to maintaining proper body alignment. The unconscious patient cannot tell you that he is uncomfortable or is experiencing pressure on a body part.

1) Limbs must be supported in a position of function. Do not allow flaccid limbs to rest unsupported.
2) When turning the patient, maintain alignment and do not allow the arms to be caught under the torso.
3) Utilize a foot board at the end of the bed to decrease the possibility of foot drop
    - When joints are not exercised in their full range of motion each day, the muscles will gradually shrink, forming what is known as a contracture. Passive exercises must be provided for the unconscious patient to prevent contractures.
    - Exercises with a range of motion (ROM) are performed under the direction of the physical therapist.
    - Precaution must be taken to prevent the development of pressure scores.
    - Use a protective mattress such as a flotation mattress, alternating pressure mattress, or egg crate mattress.
    - Change the patient's position at least every two hours.
    - Unless contraindicated, get the patient out of bed and into a cushioned, supportive chair.

**Post-Operative Care consideration:**

**1)** Accurately monitor and record all vital signs and neurological signs.
   **a)** Post-operative cerebral edema peaks between 48- and 60-hours following surgery.
   **b)** Patient may be lucid during the first 24 hours, then experience a decrease in level of consciousness during this time.
**2)** Bone flaps may not have been replaced over surgical site; turning patient to the affected side, if the flap has been removed, can cause irreversible damage in the first 72 hours.
**3)** Maintain head of bed 30° elevation.
**4)** Perform a passive range of motion exercises to all extremities every 2-4 hours.
**5)** Institute seizure precautions at patient's bedside. (tongue blade, airway)
**6)** Monitor intake and output accurately.
**7)** Prevent pulmonary complications associated with bed rest.
   **a)** Cough and deep breath every 2 hours.
   **b)** Perform gentle chest percussion, with the patient in the lateral decubitus position, if tolerated.
**8)** Consciously talk to the patient while providing care, reorienting him to person, place and time.

**Management in Traumatic Brain Injury:**

The goal of primary TBI management is to prevent the Secondary brain injury.

**1)** Assessment of consciousness level is very crucial if the patient deteriorates rapidly.
**2)** Secure the airway:
   **a)** Endotracheal Intubation and hyperventilation may be helpful in early hours in reducing carbon dioxide induced vasoconstriction and intra cranial pressure.
   **b)** Stimulation of coughing when suctioning increases intracranial pressure and may precipitate seizure activity.
**3)** ICP management:
   - Elevation of head end of bed 30°-45$^0$ promotes the return of venous blood and decreases intracranial pressure.
   - Hypotonic fluids like Dextrose cross the blood-brain barrier and worsen cerebral edema and intracranial pressure.
   - Protect patients from injury if seizures occur.
   - Avoid hypotension, Hyperthermia, hypoxia, hyper/hypocarbia, hyper/hypoglycemia.
   - Correct electrolyte imbalance

# 6. Obstetric Critical Care

Maternal mortality is a great concern to any society and India is constantly working to improve maternal mortality rate (MMR). The cause of maternal mortality includes hemorrhage, pre-eclampsia, eclampsia, infections, heart diseases and pre-existing comorbid diseases.

National Guidelines on Obstetric ICUs/HDUs (2016) provide recommendations for establishing Obstetric ICUs/HDUs. These units are considered to have a very high impact on maternal mortality. The following are the recommendations from guidelines:

1. All medical colleges should have an Obstetric ICU & Obstetric HDUs
2. All district hospitals should have and Obstetric HDUs
3. Later, states can plan for Obstetric HDUs in High delivery load CHCs and Block PHCs
4. 4 bedded HDU should be established at facilities with monthly deliveries up to 250, 8 bedded units for facilities with monthly deliveries of 251-500 and proportionate increase in beds as per monthly deliveries.

These guidelines provide a detailed framework of resources required to operationalize these units and a guideline for their functioning.

## Management of various life-threatening obstetric conditions

**A. Hemorrhage**: Bleeding per vagina in a pregnant female always requires attention. It can be due to following causes-

1. Trauma
2. Ante partum hemorrhage: Placenta previa or placental abruption
3. Post partum hemorrhage: Placenta accreta, Atonic or traumatic
4. Hemorrhage associated with coagulation disorders or thrombocytopenia.

**Management**:

1. Fluid resuscitation- Normal saline or Ringer lactate cane be used.
2. Blood and blood products: Packed RBC and other blood products should be transfused as per requirement.
3. Continuous monitoring of mother and fetus.
4. Termination of pregnancy may be required in some conditions.
5. Oxytocin, methyl ergometrine or misoprostol can be used to contract uterus in atonic PPH. Removal of retained placenta, uterine tamponade, ligation of uterine vessels or sometime hysterectomy may also be required.

**B. Pre-eclampsia/Eclampsia**: It is characterized by hypertension with proteinuria after 20 weeks of gestation in previously normotensive and non proteinuric pregnant female. It may occur early in H. mole or multiple fetus. Preeclampsia is defined as the presence of (1) a systolic blood pressure (SBP) greater than or equal to 140 mm Hg or a diastolic blood pressure (DBP) greater than or equal to 90 mm Hg or higher, on two occasions at least 4 hours apart in a previously normotensive patient, OR (2) an SBP greater than or equal to 160 mm Hg or a DBP greater than or equal to 110 mm Hg or higher. (In this case, hypertension can be confirmed within minutes to facilitate timely antihypertensive therapy.). In addition to the blood pressure criteria, proteinuria of greater than or equal to 0.3 grams in a 24-hour urine specimen, a protein (mg/dL)/creatinine (mg/dL) ratio of 0.3 or higher, or a urine dipstick protein of 1+ (if a quantitative measurement is unavailable) is required to diagnose preeclampsia. Preeclampsia with severe features is defined as the presence of one of the following symptoms or signs in the presence of preeclampsia:

- SBP of 160 mm Hg or higher or DBP of 110 mm Hg or higher, on two occasions at least 4 hours apart while the patient is on bed rest (unless antihypertensive therapy has previously been initiated).

- Impaired hepatic function as indicated by abnormally elevated blood concentrations of liver enzymes (to double the normal concentration), severe persistent upper quadrant or epigastric pain that does not respond to pharmacotherapy and is not accounted for by alternative diagnoses, or both.
- Progressive renal insufficiency (serum creatinine concentration >1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal diseases).
- New-onset cerebral or visual disturbances.
- Pulmonary edema.
- Thrombocytopenia (platelet count < 100,000/μL)

In a patient with new-onset hypertension without proteinuria, the new onset of any of the following is diagnostic of preeclampsia:

- Platelet count below 100,000/μL.
- Serum creatinine level above 1.1 mg/dL or doubling of serum creatinine in the absence of other renal disease.
- Liver transaminase levels at least twice the normal concentrations.
- Pulmonary edema.
- Cerebral or visual symptoms.

**Eclampsia** is defined as seizures that cannot be attributable to other causes in a woman with preeclampsia. HELLP syndrome (hemolysis, elevated liver enzymes, low platelet count) may complicate severe preeclampsia. Complications of preeclampsia include seizures, intracranial bleeding, liver dysfunction, renal failure, cardiac failure, fetal compromise.

Investigations:

1. Complete hemogram
2. LFT
3. KFT
4. Coagulation profile
5. Urine analysis
6. USG Abdomen
7. Fetal monitoring
8. CT head: if post ictal disorientation is prolonged, focal neurological deficit, severe headache or atypical presentation.
9. 2D echo may be required to assess pulmonary oedema,

**Management:**

1. The only cure of preeclampsia is termination of pregnancy. Fetal maturity and maternal compromise should be weighed to reach a decision.
2. Seizure prophylaxis and treatment: Airway, breathing and circulation should be maintained. Magnesium sulfate is given 4 gm IV loading over 10-15 minutes followed by 1gm/hr infusion. IM regime is also followed by some centers. Signs of magnesium toxicity should be constantly monitored. Therapy should be continued for 24 hours after the last seizure. Lorazepam, Phenytoin or levetiracetam can be added as second line drug. Magnesium sulfate prophylaxis should be given to all patients with severe pre-eclampsia. Refractory seizures should be managed by an expert intensivist in ICU.

3. Hypertension: The goal is to maintain BP around 140/90 mmHg. First line antihypertensive is Labetalol or Hydralazine. Nifedipine or Sodium Nitroprusside ca be added as second line drug.
4. Maintaining euvolemia and avoiding aggressive fluid resuscitation can prevent pulmonary oedema and AKI.
5. Diuretics can be used in pulmonary oedema and volume overload. But it should be better avoided.
6. Fetal monitoring should be done.
7. Patients should be monitored in postnatal period for at least 24 hours after stabilization of blood pressure and control of seizures.

**C. Sepsis**: Sepsis is a dysregulated inflammatory response to a confirmed or suspected infection. Sepsis is clinically diagnosed using qSOFA score. The presence of at least two of these three clinical variables may be prone to the poor outcome typical of sepsis: (1) low blood pressure (SBP ≤ 100 mmHg), (2) high respiratory rate (≥ 22 breaths per min), or (3) altered mentation (Glasgow coma scale < 15) (quick SOFA). Confirmation can be done by positive cultures and raised serum procalcitonin levels. Multi organ dysfunction can occur in sepsis and patients may land up in septic shock. Septic shock is defined as hypotension in setting of sepsis not corrected by fluid resuscitation and requiring vasopressor support. It is life threatening condition with high mortality rates.

Common sources of sepsis in pregnancy are pneumonia, urinary tract infection, puerperal sepsis, surgical site infection and blood stream infection.

**Management**: Sepsis and septic shock is managed as per survival sepsis guidelines 2021 by Society of Critical Care Medicine (SCCM)

1. Take out all appropriate cultures.
2. Send serum lactate.
3. Antibiotics: Broad spectrum antibiotics should be administered within 1 hour if sepsis is confirmed or sepsis is suspected, and patient is in shock. In patients without shock with suspected sepsis, antibiotics should be administered within 3 hours.
4. Fluids: Balanced salt solution, Ringer lactate or normal saline should be administered up to 30ml/kg to maintain MAP more than 65 mmHg.
5. Vasopressors: Noradrenaline is a drug of choice and should be administered if MAP remains below 65 mmHg or serum lactate >2 mmol/l. Dopamine can be added if bradycardia or signs of cardiac dysfunction is present.
6. CVP and hematocrit should be maintained within normal range.
7. Patients should be admitted to ICU within 6 hours of suspicion of sepsis.
8. Blood sugar should be controlled, and fetal monitoring should be started.
9. Patients may require mechanical ventilation.

**D. Pregnancy with heart diseases**: It is not uncommon in India for a primigravida to be diagnosed as heart disease after mid pregnancy. These patients usually present with worsening dyspnea, pedal oedema, cough with pink frothing, fetal growth retardation and overt heart failure. Most common cause is rheumatic heart disease especially mitral stenosis.

WHO has classified heart diseases as per their mortality risk into five groups, as shown in Figure No.1.

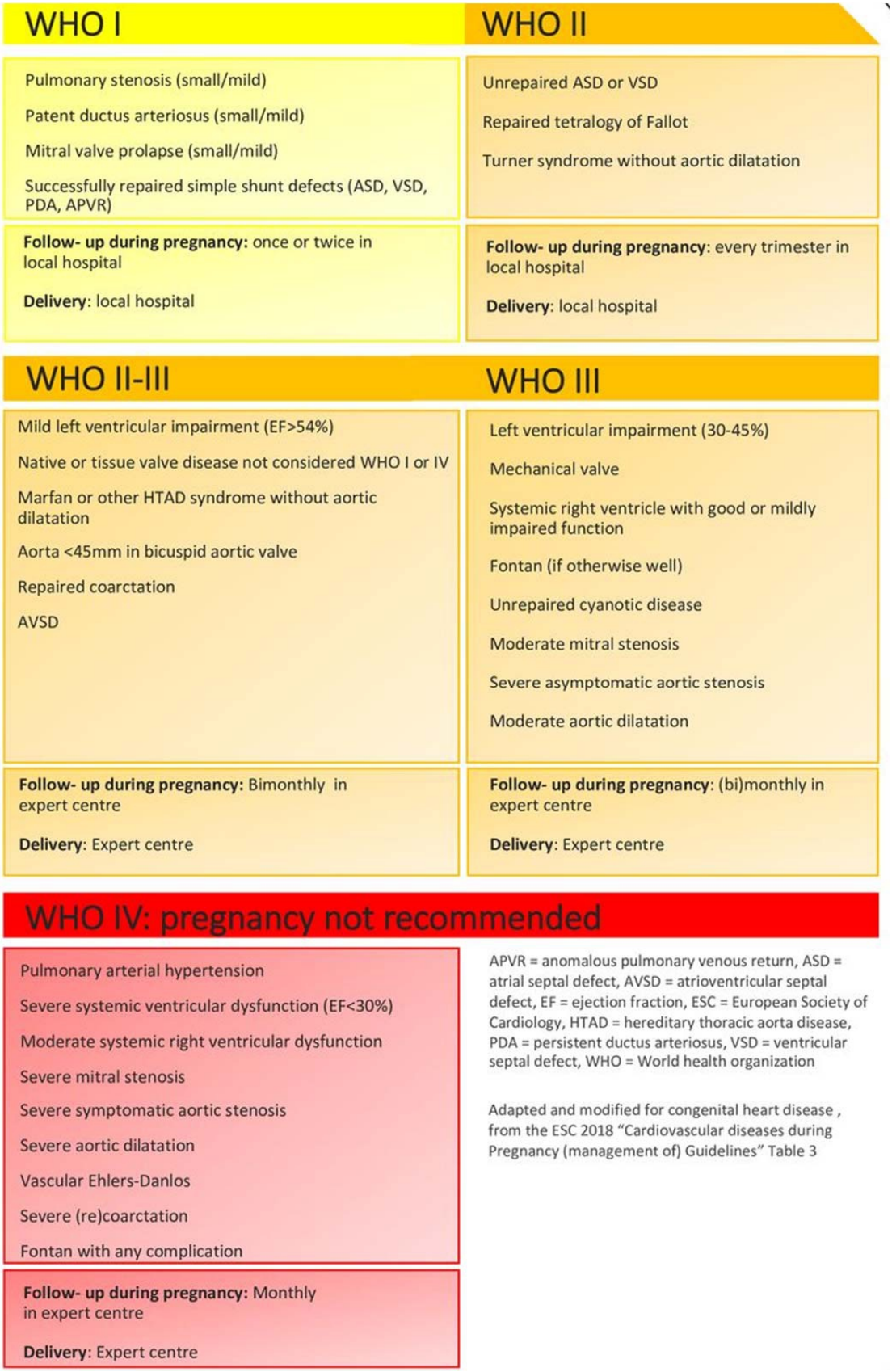


Figure No.1. Risk categories with pregnancy with heart diseases.

Section 7

# Miscellaneous Topics

# 1. Microbiological Sampling

- Microbiological samples are sent before commencement of antibiotics.
- Identify the possible source of infection. If it is not possible to identify the specific site of infection, blood urine and sputum/ETA samples are sent.
- After taking samples start broad spectrum antibiotics.

## Blood culture

- Samples are withdrawn under full aseptic precaution in blood culture vial (Figure No. 1).
- 10-20 ml sample is required.
- Samples are taken from two different sites.
- Samples are labeled carefully.
- Any Gram –ve or Staph. aureus growth is significant.


Figure No. 1: Blood culture vial

## Urine culture

- Samples are withdrawn using aseptic precaution in a sterile container (Figure No. 2).
- The sample should be sent immediately to the laboratory.
- All catheter specimens show growth if catheters are placed for more than 2 days.
- The isolation of the same organism from blood is significant.

Figure No. 2: Sterile container for urine culture.

## Sputum

- Samples easily get contaminated during sampling.
- Samples from intubated patients can be taken by using sterile suction catheters (Figure No. 3).
- Blood culture should be sent along with sputum/ETA to confirm the diagnosis of pneumonia.
- Protected catheter brush to be used for BAL.
- The sample should be sent immediately to the laboratory.

Figure No. 3: Mucus extractor used for extracting sputum sample from intubated patients.

### Wound swabs and pus

- Pus is preferable for bacterial isolation.

# 2. Needlestick Injury

**Epidemiology**

- Average risk for HIV: 3 per 1000 injuries.
- HBV and HCV infection is more likely than HIV in health care providers.
- Risk of transmission of infection following occupational exposure:
    - HIV 0.3%.
    - Hepatitis B 18-30%.
    - Hepatitis C 1.8%.
- High risk:
    - Deep injury
    - Terminal HIV related ill patient.
    - Visible blood on the device which caused injury.

**Management:**

- Needlestick injury - Wash with soap and running water.
- Blood or Body fluid in eyes or mouth - irrigate with cold water.
- Report incidence and discuss with local health consultant as soon as possible.
- Post exposure prophylaxis (PEP) needs to be started as soon as possible.
- PEP consists of 28 days course of triple combination of ART, has significant side effects, and needs careful follow up.
- Investigations of blood examination for virology (HIV, HBV, HCV) of injured health care provider.

**Prevention:**

- Vaccination to all health care provider should be encouraged.
- Hand wash before and after contacting each patient.
- Use separate gloves for each patient.
- Cover all wounds/skin lesions with waterproof dressings.
- Sharps should be used with particular care.
- Wear close footwears in situations where blood/discharge may be split.
- Clear up spillage of blood/discharge and disinfect surface.
- Follow universal precautions.
- Always use new single disposable syringes and needles.
- Discard contaminated sharps immediately without recapping into puncture proof container.
- Document quality of sterilization for all instruments used for any procedure.

# 3. Sterilization and Waste Management

- Hospital waste should be properly managed to maintain hygiene.
- Follow the institutional waste management guidelines or WHO guidelines properly.

## Categories of healthcare waste

- Sharps waste- Used or unused sharps (e.g., hypodermic, intravenous or other needles; auto-disable syringes; syringes with attached needles, infusion sets; scalpels; pipettes; knives; blades; broken glass)
- Infectious waste - Waste suspected to contain pathogens and that poses a risk of disease transmission (e.g., waste contaminated with blood and other body fluids; laboratory cultures and microbiological stocks; waste including excreta and other materials that have been in contact with patients infected with highly infectious diseases in isolation wards)
- Pathological waste - Human tissues, organs, or fluids; body parts; fetuses; unused blood products
- Pharmaceutical waste, cytotoxic waste - Pharmaceuticals that are expired or no longer needed; items contaminated by or containing pharmaceuticals. Cytotoxic waste containing substances with genotoxic properties (e.g., waste containing cytostatic drugs often used in cancer therapy, genotoxic chemicals)
- Chemical waste- Waste containing chemical substances (e.g., laboratory reagents; film developer, disinfectants that are expired or no longer needed; solvents; waste with high content of heavy metals, e.g., batteries; broken thermometers and blood-pressure gauges)
- Radioactive waste- Waste containing radioactive substances (e.g., unused liquids from radiotherapy or laboratory research; contaminated glassware, packages or absorbent paper, urine and excreta from patients treated or tested with unsealed radio nuclides; sealed sources)
- Non-hazardous or general health-care waste- Waste that does not pose any particular biological, chemical, radioactive or physical hazard.

## WHO recommended segregation scheme:

- Highly infectious waste -yellow container, marked "HIGHLY INFECTIOUS", with biohazard symbol. Strong, leak-proof plastic bag, or container capable of being autoclaved.
- Other infectious waste, pathological and anatomical waste -yellow container with biohazard symbol. Leak-proof plastic bag or container.
- Sharps- Yellow container, marked SHARPS", with biohazard symbol, Puncture proof container.
- Chemical and pharmaceutical waste - Brown container, labelled with appropriate hazard symbol. Plastic bag or rigid container.
- Radioactive waste- Labelled with radiation symbol. Lead box.
- General health-care waste - Black Plastic bag.

**Minimum Approach to Segregation, Storage and Transport** – The minimum standard to segregating health-care wastes is the "three-bin system" (Figure No. 1), where separate containers are provided for infectious waste, used sharps and general waste. The basic features of a minimal level of waste segregation and storage are as follows:

- Wastes are segregated at their place of production to reduce the health risk from the smaller potentially infectious factions (typically waste items contaminated with body fluids and used sharps).
- Infectious waste, general waste and used sharps waste are stored in separate colour-coded containers and locations within medical areas, and subsequently at a central storage site at a health-care facility.
- Central storage area(s) are fenced, lockable and isolated from patients and the public.
- Maximum storage times before treatment or disposal of infectious waste are not longer than temperate climate: 72 hours in winter and 48 hours in summer warm climate: 48 hours during the cooler season and 24 hours during the hot season.

- Staff receive instruction on three-bin waste segregation and safe handling and storage of health-care wastes.
- Staff are aware of how to protect themselves from injuries and infection from waste.
- Waste containers and storage areas are cleaned regularly.
- Many health-care waste-treatment systems are commercially available today.
- The choice of technology depends on the characteristics of the waste of the health-care facility, the capabilities and requirements of the technology, environment and safety factors, and costs.
- Treatment technologies employ thermal, chemical, irradiative, biological or mechanical processes.


Figure No 1: Segregation of health-care wastes in "three-bin system".

**The common types of treatment technologies are:**

- Autoclaves (Figure No. 2).
- Integrated or hybrid steam-based treatment systems.
- Microwave treatment technologies.
- Dry-heat treatment technologies.
- Chemical treatment technologies.
- Incinerators.

Figure No. 2: Autoclave.

# 4. Arterial Blood Gas Analysis

Analyzing arteria blood through autoanalyzer helps in determining acid-base status of the body. It also helps in ascertaining oxygenation and $CO_2$ removal in the blood. Arterial blood gas (ABG) analysis also gives information about serum lactate, electrolytes, and glucose levels. Systematic analysis of ABG helps us in determining the cause of acid-base disorder.

An arterial blood sample is used for analysis, and it should be quickly analyzed as with passage of time the $PCO_2$ and $PO_2$ level changes. The table below shows the values for interpretation of ABG.

| Arterial Blood Gas Analysis | | | | | | |
|---|---|---|---|---|---|---|
| **ABG Parameter** | | | **ABG Result** | **Calculation and interpretation** | | |
| **pH** | >7.45 | Alkalemia | | **pH** | **$pCO_2$** | **Interpretation** |
| | 7.36-44 | Normal | | | | |
| | <7.35 | Acidemia | | ↓ | ↓ | Metabolic acidosis |
| **$pCO_2$** | >45 | High | | ↑ | ↑ | Metabolic alkalosis |
| | 35-45 | Normal | | ↑ | ↓ | Reparatory alkalosis |
| | <35 | Low | | ↓ | ↑ | Reparatory acidosis |
| **$HCO_3$** | >26 | High | | **Corrected standard AG for Albumin** | | |
| | 24±2 | Normal | | $\frac{Albumine}{4} + 1.5$ X phosphate | | |
| | <22 | Low | | | | |
| **AG** | >16 | High | | **Anion Gap Calculation** | | |
| | 12±4 | Normal | | $\{[Na+] - Cl- + HCO_3]\} = 12 \pm 4$ | | |
| | <8 | Low | | **Corrected Na+ for AG in hyperglycemia** | | |
| **Glucose** | >10 | High | | $\text{Corrected } Na^+ = Na + \frac{\text{Glucose} - 5}{3}$ | | |
| | <2 | Low | | | | |
| **Gap: Gap** | $\frac{\Delta AG}{\Delta HCO^3} = \frac{AG - 12}{24 - HCO^3}$ | | | **Gap: Gap Calculation for metabolic acidosis** | | |
| | | | | **<0.4** | Low or Normal AG metabolic acidosis | |
| | | | | **0.4-0.8** | Normal + high AG metabolic acidosis | |
| **Lactate** | <1.9 | Normal | | 08-2.0 | Pure high metabolic acidosis | |
| | >2.0 | High | | >2.0 | Metabolic acidosis with metabolic alkalosis/respiratory acidosis | |
| **$pCO_2$** | 80-100 | Normal | | **$PAO_2 = [713 \times FiO_2] - [pCO_2 \times 1.25]$** | | |

| | | | | |
|---|---|---|---|---|
| | <80 | Hypoxia | | **A-a gradient = $PAO_2 - PaO_2 = \frac{Age}{4} + 4$** |

| **Compensation rules for** | | | | |
|---|---|---|---|---|
| **Expected $pCO_2$** | **Metabolic acidosis** | | **Metabolic alkalosis** | |
| | 1.5 X [$HCO_3$] + 8 (+/-2) | | 0.7 X [$HCO_3$] + 20 (+/-5) | |
| **Expected $HCO_3$** | **Respiratory acidosis** | | **Respiratory alkalosis** | |
| | **Acute** | **Chronic** | **Acute** | **chronic** |
| | $24 + \frac{pCO_2 - 40}{10} X_1$ | $24 + \frac{pCO_2 - 40}{10} x4$ | $24 - \frac{pCO_2 - 40}{10} x\ 2$ | $24 + \frac{pCO_2 - 40}{10} X_5$ |

Section

8

# Anesthesiology

# 1. Preoperative Assessment and Optimization

Anesthesia plays a pivotal role in ensuring patient safety and comfort during surgical procedures. Anesthetic management is broadly divided into preoperative assessment and optimization (PAC), premedication, induction, monitoring and maintenance, extubation, and postoperative management. This chapter explains the preoperative part of anesthesia.

## Preoperative assessment and optimization

After a surgery is planned, the patients should be evaluated by anesthesiologist, ideally in an outpatient PAC clinic. PAC has the following advantages:

1. Reduced rates of cancellations on the day of surgery.
2. Selective ordering of the lab tests, resulting in fewer investigations.
3. Shorter hospital stays and lower costs.
4. Reduced anxiety.
5. Better counselling for regional anesthesia and other procedures.

PAC has several purposes. It gives the anesthesiologist to **assess the surgical condition and co-morbid conditions**. The severity and optimization of co-morbidities should be assessed. If the co-morbid condition is not fully optimized, its management need to be optimized to achieve an acceptable risk during the perioperative period. For example, a patient with heart failure without optimization may need additional diuretics, beta-blockers, and/or inotropes.

Frequently, patients are diagnosed with diseases in PAC that have not been detected before. Common examples include hypertension, diabetes mellitus, thyroid disorders, valvular heart disease, coronary artery disease, and obstructive airway diseases. PAC gives the opportunity to diagnose and optimize these conditions before surgery. Patients may need referral to appropriate specialties for preoperative optimization.

**Estimation of the perioperative risk** is also an important component of the PAC. Surgery can itself be classified into **low risk/minor** (cataract surgery, hemorrhoidectomy, superficial debridement, cystoscopy, ureteroscopy, dilatation and curettage), **intermediate risk** (intraperitoneal, intrathoracic, orthopedic, prostate surgery, head and neck surgery) or **major/high risk** (major vascular surgery, joint replacement, spine surgery, modified radical mastectomy, flap reconstruction surgery, liver transplant, and Wertheim's hysterectomy) having a risk of **<1, 1-5% and >5% risk of major complications and death** respectively. Perioperative risk can also be estimated using:

1. **American Society of Anesthesiologists (ASA) physical status** classification,
2. **Revised cardiac risk index** (RCRI), and Myocardial infarction and cardiac risk (MICA) score for cardiovascular risk,
3. **Child-Turcotte-Pugh-classification** and **Model for end-stage liver disease** for patients with liver disease
4. The **National Surgical Quality Improvement Program** provides an estimate of 30-day morbidity and mortality for a large variety of surgeries.

PAC also gives an opportunity to plan the perioperative management, patient counselling, and obtain consent.

**Metabolic equivalent of tasks:**

Dyspnea, chest pain, cough, palpitation, hemoptysis, edema, and syncope are features that suggest cardiovascular disease. For most cardiac diseases, functional capacity of a patient gives a good estimate of the cardiovascular system and perioperative cardiovascular risk. Metabolic equivalent of tasks (MET) is an objective measure of physical status. One MET is the amount of oxygen consumed by body at rest. ***Patients who have four or more METs have lower risk of cardiovascular complication than those who don't***.

Activities requiring four or more are climbing one flight of stairs, gardening, cycling, jogging, and playing golf or tennis.

**Routine preoperative investigation:**

Indian Society of Anesthesiologists 2022 guidelines suggest routine investigations for ASA I and II patients are advised according to surgical risk.

Minor surgery: complete blood count (CBC), ECG if age ≥45 years.

Intermediate surgery: CBC, serum creatinine, ECG if age ≥45 years, chest X-ray (CXR) if age ≥ 50 years.

Major surgery: CBC, serum creatinine, liver function test, ECG for all patients, chest X-ray (CXR) if age ≥ 50 years.

Without any change in clinical status blood investigations are valid for two months while CXR and ECG are valid for one year. For patients belonging to ASA III-V, additional investigations should be advised based on the surgical and co-morbid conditions.

## Pre-operative medication management

Cardiovascular drugs: Generally, all cardiovascular drugs should be continued till the morning of the surgery. Exceptions include **ACE inhibitors and angiotensin-receptor blockers**. They may have to be **stopped before surgeries having major fluid shifts and hypotension**. Beta blockers should not be stopped before surgery. Beta blockers should not be started within 24 hours before surgery, as it increases the risk of cardiovascular complications.

Similarly, all respiratory drugs, and anti-seizure medication should be continued.

Oral anti-diabetics can be safely continued up to night before surgery, but morning dose of surgery needs to be stopped. Metformin should be stopped one day before in patients with renal dysfunction or major surgery. In patients planned for minor surgery metformin can be safely continued. If a patient is on insulin, give only 2/3 of night dose of NPH, regular, Glargine, Lispro and Aspart insulin. Omit morning dose of all insulins.

Patients who are taking more than 10 mg/day of prednisolone or an equivalent dose of other steroids within three months of surgery are require perioperative steroid supplementation (hydrocortisone 50-100 mg) to maintain adequate blood cortisol levels.

Aspirin should be continued perioperatively. Surgery in a closed space (intracranial, retinal) requires cessation of aspirin 5-7 days before surgery. For patients with stents, dual antiplatelet drugs (aspirin + clopidogrel, or aspirin + ticagrelor) need to be continued for a certain mandatory duration. This duration is 30 days for bare-metal stents and 1 year for drug eluting stents. After this, clopidogrel/ticagrelor are stopped (5-7 days before surgery) while aspirin is continued as described above. **Under no circumstances, clopidogrel/ticagrelor should be stopped before 30 days of bare-metal stent and 180 days for drug-eluting stent.**

**Warfarin** should be stopped **5 days** before surgery and INR should be ≤1.4 before surgery. Anticoagulation is bridged with low-molecular weight heparin (LMWH). LMWH is stopped 12-24 hours before surgery.

### Premedication:

The purpose of premedication is to reduce the level of anxiety, stress response, nausea, pain, and risk of aspiration.

**Anxiolysis and sedation**: Midazolam, given one hour before surgery, is a common premedication drug used for this purpose. Other drugs are given in table No. 1.

**Table No.1: Dose and route of common sedative/anxiolytic premedication drugs**

| Drug | Route | Dose |
|---|---|---|
| **Midazolam** | Oral | 0.3-0.7 mg/kg |
| | Nasal | 0.1-0.2 mg/kg |
| | Intravenous | 0.05-0.15 mg/kg |
| **Ketamine** | Oral | 5-6 mg/kg |
| | Nasal | 2-4 mg/kg |
| | Intravenous | 1-2 mg/kg |
| | Intramuscular | 3-7 mg/kg |
| **Clonidine** | Oral | 4 mcg/kg |
| | Nasal | 4 mcg/kg |
| **Dexmedetomidine** | Oral | 2.5 mcg/kg |
| | Nasal | 1 mcg/kg |

**Aspiration prevention:** Risk reduction of pulmonary aspiration requires **low gastric content volume (<25 ml)** and **lower acidity (higher pH, >2.5) of its contents**. Pharmacologic prophylaxis against aspiration is needed only for patients at increased risk for aspiration (Table No. 2). The drugs used for aspiration prophylaxis are given in Table No. 3.

**Table 2: Risk factors for pulmonary aspiration**

| | |
|---|---|
| **Emergency surgery** | Gastro-esophageal reflux disease |
| **Inadequate fasting duration** | Morbid obesity |
| **Diabetes mellitus** | Ascites |
| **Pregnancy** | Raised intracranial pressure |
| **Gastrointestinal obstruction** | Hiatus hernia |

**Table No. 2: Drugs used for aspiration prophylaxis.**

| Drug | Route | Dose | Timing |
|---|---|---|---|
| **Ranitidine** | Oral | 150-300 mg | Night before surgery and 2 hours preoperatively |
| | IV | 50 mg | Night before surgery and 2 hours preoperatively |
| **Pantoprazole** | Oral | 40 mg | Night before surgery and 2 hours preoperatively |
| | IV | 40 mg | Night before surgery and 2 hours preoperatively |
| **Metoclopramide** | IV | 10 mg | At least 1 hour preoperatively |
| **Sodium Citrate (0.3 M)** | Oral | 30 ml | 30 minutes preoperatively |

**Anti-sialagogues**: These are not routinely used due to tachycardia, dry mouth, decreased mucus clearance, and disorientation. Glycopyrrolate is used in dose of 200 mg IM or IV before fiberoptic intubation.

**Pre-emptive analgesia**: Analgesia before the painful surgical stimulus can potentially provide better postoperative analgesia. Commonly used drugs are paracetamol and NSAIDs.

# 2. Anesthesia: Intraoperative Management

General anesthesia, neuraxial anesthesia, and peripheral nerve blocks are commonly used methods used for anesthesia.

## General anesthesia

General anesthesia is divided into three phases: induction, maintenance, and reversal.

### Induction:

Before induction, standard anesthesia monitors are applied to the patients. All patients are monitored using ECG, non-invasive blood pressure (measured at least every 5 minutes), pulse oximeter (pulse rate and arterial blood saturation). Confirmation of endotracheal tube placement and continuous ventilation are done using waveform capnography. Temperature monitoring is used when changes in body temperature are anticipated. Optional monitoring includes arterial blood pressure, central venous pressure, neuromuscular monitoring, and depth of anesthesia monitoring. Contemporary anesthesia workstations (Figure No. 1) have integrated monitors and ventilators.

Induction of anesthesia requires careful titration of anesthetic agents to achieve unconsciousness and amnesia while maintaining physiological stability. Induction using intravenous drugs (propofol, thiopentone, or etomidate) is the most common induction choice as it is rapid and smooth. Before induction, opioids are administered to blunt the stress response of intubation and to provide analgesia. Muscle relaxants (vecuronium, rocuronium, atracurium, or cis-atracurium) are used for muscle relaxation to aid laryngoscopy and endotracheal intubation.

After achieving adequate depth of anesthesia and muscle relaxation, direct laryngoscopy is undertaken, and endotracheal intubation is done. This should be done carefully and additional equipment for airway management should be present (Figure No. 2). The correct tube placement is confirmed using bilateral chest auscultation and waveform capnography.

### Maintenance of anesthesia:

This includes unconsciousness, amnesia, muscle relaxation, analgesia, and suppression of autonomic reflexes. Unconsciousness and amnesia are primarily maintained using inhalational anesthetic agents (sevoflurane, isoflurane, or desflurane). A continuous infusion of propofol or dexmedetomidine can also be used for the above purposes. Dexmedetomidine infusion also provides analgesia. Muscle relaxation is provided using intermittent boluses of muscle relaxation. Maintaining adequate depth of anesthesia using inhalational agents and opioids suppress the autonomic reflexes.


Figure No. 1: Contemporary anesthesia workstation with integrated ventilator and monitors.

## Emergence and reversal:

After the end of surgery, the anesthetic agents are discontinued. The effect of sedative anesthetic agents is allowed to wane spontaneously with time. Muscle relaxants are reversed with anticholinesterases (neostigmine, combined with glycopyrrolate). This antagonizes the effects of muscle relaxants within 5-10 minutes. After regaining adequate muscle power, the endotracheal tube is ready to be removed. The oral cavity is suctioned and with a suction catheter and the endotracheal tube is removed after deflating the tube cuff.

After extubation, the patients are transferred to a post-anesthesia care unit for further monitoring as they recover from anesthesia.

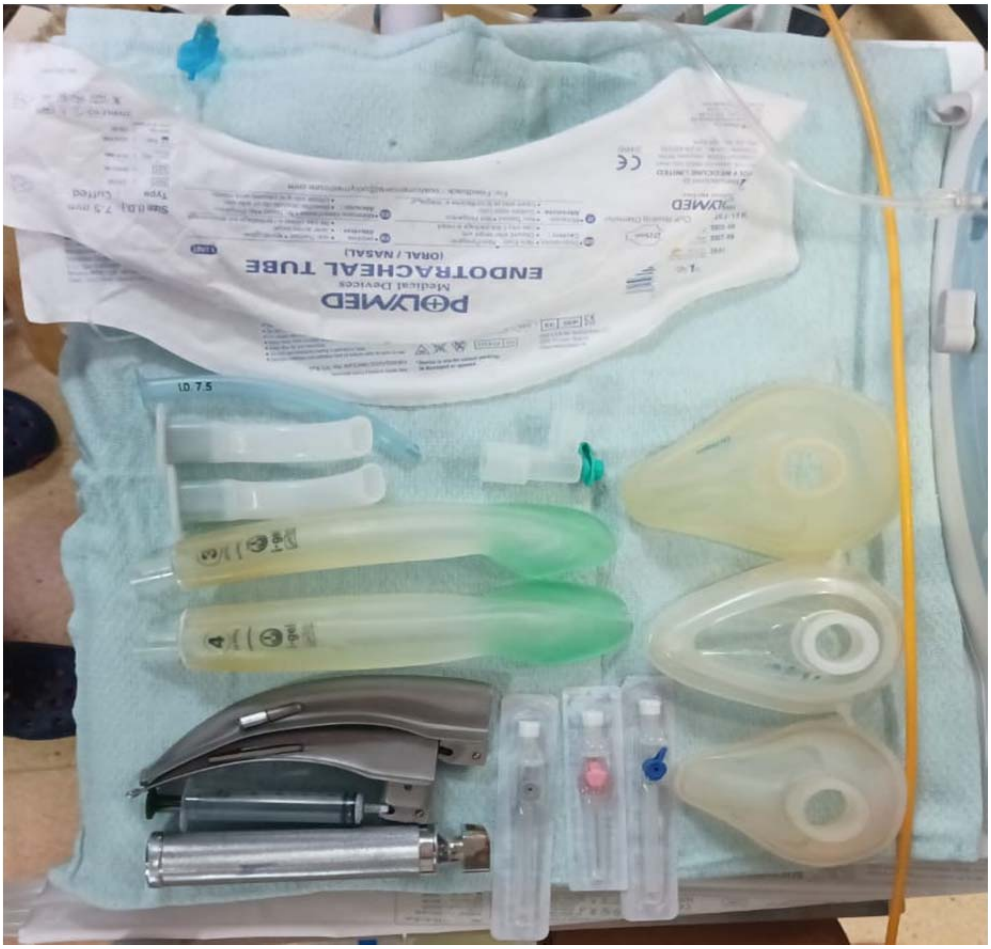


Figure No. 2: Equipment for airway management.

## Neuraxial anesthesia

Neuraxial anesthesia involves the administration of local anesthetic agents into the epidural or subarachnoid space, near the spinal cord and nerve roots. This blocks the sensory and motor function in lower body for surgery. Spinal and epidural anesthesia are commonly used types of neuraxial anesthesia.

**Spinal anesthesia**: In this, a fine needle is inserted through skin, connective tissue, and dura mater and local anesthetic is injected into the cerebrospinal fluid at the lumbar level. This results in a rapid and profound loss of sensation and motor function in the lower part of the body. Its advantages are rapid onset, profound anesthesia, lower dose of local anesthetic use, and avoidance of airway complications. Disadvantages include hypotension, limited duration of anesthesia, unintended high block and post-dura puncture headache. It is frequently used for surgeries of lower half of the body e.g., cesarean section, lower limb surgery, gynecological surgeries, urological surgeries and inguinal hernia repair.

**Epidural anesthesia**: Epidural space is a potential space immediately outside the dura mater. In epidural anesthesia, a blunt-tip epidural needle is inserted till its tip is in the epidural space. Confirmation of this is done using loss-of resistance technique. A thin catheter is inserted through the epidural needle. The tip of the catheter of the needle is located in the epidural space. The local anesthetic is injected through the catheter into the epidural space. Epidural anesthesia provides anesthesia similar to spinal anesthesia with some differences. The onset of epidural anesthesia is slower. As there is a catheter placed in this technique, additional doses can be given prolong the duration of anesthesia. Additional doses can be given to increase the level of anesthesia. Epidurals can also be used to provide postoperative analgesia. For this, a lower concentration of local

anesthetics is used. This blocks only the sensory and pain transmitting nerve fibers. Epidural anesthesia used higher dose than spinal anesthesia, hence the chances of local anesthetic toxicity are much higher. Indications for epidural anesthesia are the same as spinal anesthesia, but it can be used for surgeries of longer duration. Few surgeries needing rapid anesthesia onset (emergency cesarean section) may not be suitable for epidural anesthesia.

A third technique, **combined spinal epidural anesthesia**, has advantages of rapid onset with advantage of prolonging the duration and height of neuraxial anesthesia. In this technique, the epidural needle is placed in the epidural space. After this the spinal needle is placed through the epidural needle and local anesthetic for the spinal anesthesia is injected. After removal of the spinal needle, the epidural catheter is inserted into the epidural space.

## Peripheral nerve block

Peripheral nerve blocks are regional anesthesia techniques that involve the administration of local anesthetics near specific nerves. This results in temporary loss of sensation and motor function in a targeted area of the body. This provides excellent anesthesia and analgesia for a particular part of the body. Brachial plexus block is the most used block for upper limb surgery. Peripheral nerve blocks can also be used to provide postoperative analgesia to the patients. Examples include transversus abdominis plane block and erector spinae plane block.

Advantages of peripheral nerve block include early recovery, less exposure to multiple anesthetic agents, lesser hemodynamic and respiratory derangements, and excellent analgesia. Disadvantages include trauma to nearby anatomical structures, local anesthetic systemic toxicity, nerve injury, and bleeding. To improve the success rate and minimize complications, ultrasound-guided (Figure No. 2) or nerve-stimulator guided blocks are used.

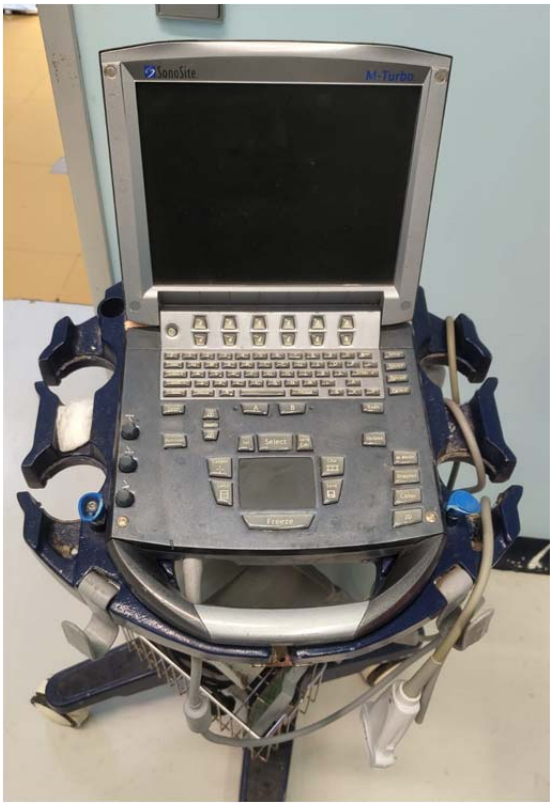


Figure No. 2: A portable ultrasound machine perioperative use. It has numerous uses like peripheral nerve blocks, vascular access, and intravascular fluid assessment.

# 3. Anesthesia: Postoperative Care

This chapter focuses on the postoperative care of surgical patients. Patients recovering from anesthesia are monitored in postoperative care unit (PACU), before transferring to surgical ward or discharge.

**PACU admission:**

A detailed handoff is important to transfer vital preoperative and intraoperative details. Common information includes the surgical condition and procedure, comorbid conditions and their management, anesthesia technique used, drugs given, fluid intake and output, intraoperative complications, and postoperative instructions.

**Initial assessment and care**: Airway (patency), breathing (respiratory rate and saturation), circulation (heart rate, blood pressure, ECG), mental status, temperature, nausea, and pain are immediately assessed. After this, these parameters are assessed at least every 15 minutes. Urine output, bleeding and fluid intake help in estimating the hydration status of the patient. Common problems in PACU should be immediately identified.

A. **Hypoxia**: Hypoxia should be rapidly detected and treated. Common causes of hypoxia in PACU are:
   1. **Upper airway obstruction**: This results from residual effects of sedatives, opioids, and neuromuscular blocking agents. Residual effects of anesthetic agents result in loss of pharyngeal tone causing collapse of airway on inspiration. This results in paradoxical breathing. In paradoxical breathing, there is retraction and collapse of chest wall and excessive abdominal wall movement on inspiration. Upper airway obstruction is more common in patients with obesity and obstructive sleep apnea. Management includes jaw thrust, neck extension and supplemental oxygen. If this doesn't improve ventilation, insertion of oro/nasopharyngeal airway, CPAP, and endotracheal intubation may be required.
   2. **Residual neuromuscular block**: Inadequate reversal is relatively common. Contributing factors include hypothermia, electrolyte disorders, hepatic or renal disorders, and drugs like amikacin, lignocaine, calcium channel blockers, and steroids. Residual neuromuscular block is best diagnosed using neuromuscular monitoring. Additional small doses of anticholinesterases may reverse the residual neuromuscular block. The rest of the management is similar to the other causes of upper airway obstruction.
   3. **Atelectasis**: It is collapse of the alveoli due to compression, absorption of oxygen, or due to obstruction (mucus, endobronchial intubation). Atelectasis is common in the postoperative period and is one of the leading causes of hypoxemia, particularly in abdominal and chest surgery under general anesthesia. It has no specific clinical features, but some patients may have dyspnea. Management includes the use of CPAP and chest physiotherapy. Intraoperative use of lower oxygen concentrations and recruitment maneuver decreases the risk of atelectasis.
   4. **Bronchospasm**: It is a reflex constriction of the bronchial smooth muscles. It is more common in patients with obstructive airway diseases like COPD and asthma. Clinical manifestations include dyspnea, wheezing, tachypnea, small tidal volumes, and hypercapnia. Management includes bronchodilators (beta-2 agonist and anticholinergics) and steroids.

**B. Cardiovascular instability**
   1. **Myocardial infarction**: Postoperative myocardial ischemia is non-ST segment elevation type. It is commonly followed by a period of tachycardia causing imbalance of oxygen supply and demand. Postoperative MI is commonly asymptomatic because of analgesics and sedatives during this period. Patients have hemodynamic instability and ECG changes. ECG changes include ST-T changes, left bundle branch block, ectopic beats, and other arrhythmias. Left ventricle failure may cause presence of S3, crepitation, wheezing and systolic murmur of mitral regurgitation. 12-lead ECG and cardiac troponins are used for diagnosis. Management includes supplemental oxygen (if hypoxemic), non-enteric coated aspirin (165-325 mg), nitroglycerin and morphine. For ST-elevation MI, fibrinolysis risks bleeding from the surgical site. Percutaneous coronary intervention is safer in patients with

STEMI and hemodynamically unstable NSTEMI patients, but risk of bleeding is still present.

2. **Hypotension**: Hypovolemia, cardiac dysfunction, vasodilatation, pneumothorax, and pleural effusion are causes of hypotension in the postoperative period. Hypovolemia is the commonest cause of hypotension in PACU. Intraoperative fluid deficits and diuretic use may result in hypovolemia in PACU. Blood loss in the surgical drains and other sites should be monitored and excess losses should be promptly corrected. Use of neuraxial techniques may also cause hypotension due to vasodilation. Management depends on the etiology of hypotension. Hypovolemia requires fluid/blood transfusion; vasodilation requires fluid and vasopressor infusion; cardiac dysfunction requires inotropes. Management of primary cause should also be done.

C. **Postoperative nausea and vomiting (PONV):** PONV is one of the commonest complications in the PACU. Risk factors for PONV include female gender, age<50 years, non-smoker, opioid use, strabismus surgery, and history of PONV. All patients with risk factors for PONV should be given prophylactic drugs for PONV. Options for prophylaxis and treatment of PONV include serotonin (5-HT3) receptor antagonists (ondansetron 4mg, granisetron 0.35-3 mg), glucocorticoids (dexamethasone 4-8 mg), antidopaminergics (droperidol 0.625-1.25 mg), and antihistamines (dimenhydrinate 1mg/kg, diphenhydramine 1mg/kg). use of multiple drugs with different mechanisms of action are more useful.

**Table No. 1: Modified Aldrete score**

| Variable Evaluated | Score |
|---|---|
| ***Activity*** | |
| Able to move four extremities on command | 2 |
| Able to move two extremities on command | 1 |
| Able to move no extremities on command | 0 |
| ***Breathing*** | |
| Able to breathe deeply and cough freely | 2 |
| Dyspnea | 1 |
| Apnea | 0 |
| ***Circulation*** | |
| Systemic blood pressure ≤20% of the preanesthetic level | 2 |
| Systemic blood pressure is 20% to 50% of the preanesthetic level | 1 |
| Systemic blood pressure ≥50% of the preanesthetic level | 0 |
| ***Consciousness*** | |
| Fully awake | 2 |
| Arousable | 1 |
| Not responding | 0 |
| ***Oxygen Saturation (Pulse Oximetry)*** | |
| Greater than 92% while breathing room air | 2 |
| Needs supplemental oxygen to maintain saturation >90% | 1 |
| Less than 90% with supplemental oxygen | 0 |

D. **Acute kidney injury (AKI):** It is defined as an increase of serum creatinine ≥0.3 md/dl in 48 hours, or >1.5 time baseline. A urine output <0.5 ml/kg/hour also defines acute kidney injury. Prerenal cause (inadequate perfusion of kidney due to hypovolemia or inadequate cardiac output) is the most common cause of AKI in PACU. Other causes are obstructive and intrinsic renal failure. As most common cause of AKI in PACU is hypovolemia, it is reasonable to administer fluid boluses for hypovolemic patients with AKI. An ultrasound KUB helps in detecting obstructive and intrinsic renal etiologies of AKI.

E. **Delayed emergence**: Failure to regain consciousness in a timely fashion after discontinuation of anesthetic drugs is known as delayed emergence. PACU receives patients with delayed emergence. The probable etiology and their management have to be done. Most cases are caused by residual effects of the anesthetics, either due to reduced elimination (hepatic/renal dysfunction, hypothermia) or excess doses. Other causes are electrolyte imbalance, hypoglycemia, hypothermia, hypo/hyperthyroidism, hypercapnia, raised intracranial pressure, stroke, intraoperative seizures, and hypoxic brain injury.

## Discharge from PACU:

Adequate recovery is required before transfer or discharge from PACU. Modified Aldrete score (Table No.1) and modified post-anesthesia discharge scoring system (PADSS, Table No.2) are used to determine readiness for transfer from PACU home. Both the scoring systems require a score of ≥9 for discharging patients from PACU.

**Table No. 2: Modified Post-Anesthesia Discharge Scoring System**

| Variable Evaluated | Score |
|---|---|
| **Vital Signs (Stable and Consistent with Age and Preanesthetic Baseline)** | |
| Systemic blood pressure and heart rate within 20% of the preanesthetic level | 2 |
| Systemic blood pressure and heart rate 20% to 40% of the preanesthetic level | 1 |
| Systemic blood pressure and heart rate >40% of the preanesthetic level | 0 |
| **Activity Level (Able to Ambulate at Preoperative Level)** | |
| Steady gait without dizziness or meets the preanesthetic level | 2 |
| Requires assistance | 1 |
| Unable to ambulate | 0 |
| **Nausea and Vomiting** | |
| None to minimal | 2 |
| Moderate | 1 |
| Severe (continues after repeated treatment) | 0 |
| **Pain (Minimal to No Pain, Controllable with Oral Analgesics; Location, Type, and Intensity Consistent with Anticipated Postoperative Discomfort)** | |
| Acceptability: | |
| Yes | 2 |
| No | 1 |
| **Surgical Bleeding (Consistent with That Expected for the Surgical Procedure)** | |
| Minimal (does not require dressing change) | 2 |
| Moderate (up to two dressing changes required) | 1 |
| Severe (more than three dressing changes required) | 0 |


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