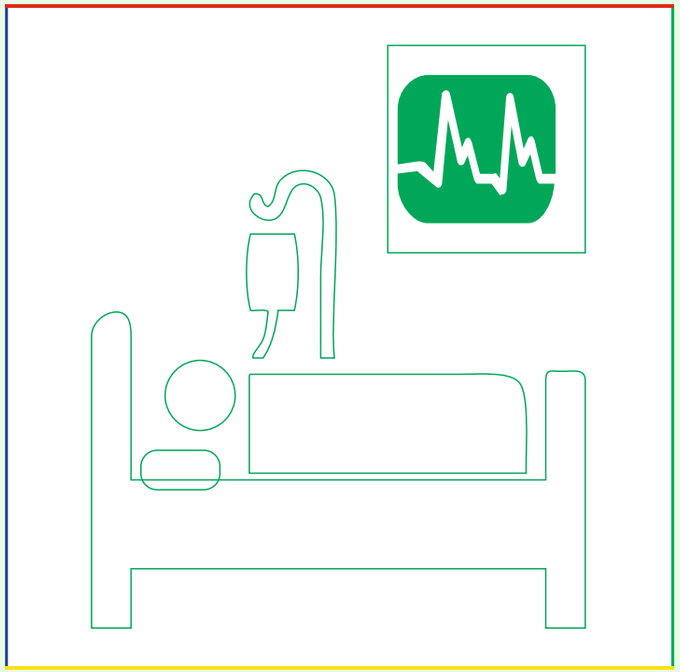
STANDARD TREATMENT GUIDELINES

CRITICAL CARE MEDICINE



DEPARTMENT OF HEALTH AND FAMILY WELFARE

GOVERNMENT OF KERALA

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May 2026

STANDARD TREATMENT GUIDELINES
for
CRITICAL CARE MEDICINE

Foreword

At the outset, I appreciate the work done by the respective thematic teams and coordination done by the DME. The Standard Treatment Guidelines (STG) were prepared and published in 2021 in the thick of the Covid pandemic. On the last page of these volumes the road map was mentioned. The few points are mentioned here for the recall.

"The Department of Health has been taking a systematic approach of creating and enabling multiple initiatives with a focus on prevention along with improving health care services. Health care service delivery is one of the most important services and is always seen as a barometer to assess the Governance. While it is important to develop infrastructure, an essential prerequisite is to develop systems and processes to bring in standardization in management of patient care. ……The foundation is laid and we take up the challenge to work on the unfinished agenda."

It was mentioned in the road map to have institutional mechanism to ensure updation of Standard Treatment Guidelines. The next step that was suggested was to do analysis of Karunya Arogya Suraksha Padhati (KASP) and standard treatment guidelines to work on developing a Balance Score Card to give information regarding compliance from the Hospitals and to build a "feedback loop" to improve. These initiatives remained at concept level on the last page! But following detailed discussions with Dr Vishwanathan, Director Medical Education, some of the foundational things were prioritized and given an impetus to take it to finality. In this journey, many committed doctors from various Medical Colleges of respective specialties participated. The previous coordination team members and experts were also consulted and they also participated in discussions and these Standard Treatment Guidelines are prepared.

The standard treatment guidelines will be made available in the Kerala Health portal (health.kerala.gov.in). This will enable the resource book availability not only to people within the state but to all in the country and outside our borders as well. I am confident that it will be used by students and practicing doctors. We request inputs based on the research from the Specialists and Experts. The teams shall continue to update and make any required changes in the STG by doing periodic updates.

The most important thing we all need to internalize is to have a shared vision and

work as a team to reach to a state of 'excellence'. If we take a look at the preparation of the Directorate Medical Education Management Information System, documents of each Medical Colleges, it provides information regarding 'what we are, what we do and what we aspire to do', pandemic preparedness, AMR accreditation and many more such initiatives taken on scale, which are all outcomes of collective TEAM work. This has laid a foundation for involving all the stakeholders including undergraduate and postgraduate students. This should encourage the teams in Medical Colleges to believe in themselves and build future initiatives on such a sound platform.

I express my sincere thanks to Dr Vishwanathan for his patience and bearing with relentless follow ups! I also take this opportunity to thank each and every team and their members and everyone from Directorate Medical Education and Medical Colleges who supported these initiatives.

I would like to express my sincere gratitude to all those who have contributed to publish these Standard Treatment Guidelines.

I wish all the success to DME team to make Kerala MCH as a premier knowledge hub in Medical Science.

Dr Rajan Khobragade IAS

Additional Chief Secretary
Health & Family Welfare and
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Govt of Kerala.

Message

Patient care today demands evidence-based, standardized, and contextually relevant clinical practice. In this regard, the publication of the **Second Edition of the Standard Treatment Guidelines** marks an important step forward in strengthening the quality, consistency, and accountability of healthcare delivery in Kerala.

The first edition laid a strong foundation for uniform clinical practice across specialties and super specialties. Since then, advances in medical knowledge, evolving treatment modalities, and the growing need for periodic updating have made it essential to revisit and refine these guidelines. The present edition reflects this commitment to continuous improvement and clinical excellence.

I am pleased to note that subject experts from various disciplines of Government Medical Colleges, private institutions and professional bodies have contributed as resource persons in the preparation of these guidelines. Their academic expertise, practical insight, and dedicated involvement have greatly enriched this edition. I deeply appreciate the sincere efforts of all the conveners, contributors, and coordinators whose collective commitment and teamwork made this publication possible.

These guidelines will serve as a valuable reference for clinicians, teachers, trainees, and healthcare institutions, helping to promote evidence-based decision-making and improve patient outcomes. I am confident that this edition will further support standardization of care and contribute to the advancement of medical education and clinical practice in the State.

I congratulate everyone involved in this commendable effort and commend this publication to all healthcare professionals.

Dr. K. V. Viswanathan
Director of Medical Education
Government of Kerala

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Contents

1. Scope

The guidelines have incorporated the basic resuscitation of a Critically ill adult patient in clinical scenario and starts with initial assessment and resuscitation. These aim to improve the standard of care while handling this group of patients who are likely to deteriorate if not identified early and resuscitative measures started. The second part deals with separate topics like Fluid management in ICU, Hemodynamic monitoring, sedation in critically ill, Traumatic brain injury-critical care management.

Population

Adults more than 18 years of age; not applicable to paediatric population

Key clinical issues covered:

General assessment & resuscitation Oxygen therapy
Ventilatory support Liberation from ventilator Shock
Cardiac arrest

Clinical issues not covered:

Detailed description of drugs and interventions; Ventilator graphics and machine support and maintenance
Applicable to all healthcare facilities catering to critically ill adult patients including Emergency department and ICU.

Areas like ACS, Toxicology, Pancreatitis is not covered in this version. An update is considered in 2026.

Outcome:

Improving patient outcomes through evidence-based practices

1. Early detection
2. Risk stratification
3. Appropriate treatment
4. Monitoring and supportive care

2. Abbreviations

ARF:	Acute respiratory failure
ARDS:	Acute respiratory distress syndrome
BP:	Blood Pressure
BPM:	Beats per minute
FiO2:	Fraction of Oxygen in inspired gas
HFNO:	High flow nasal Oxygen
HFNC:	High flow nasal cannula
HR:	Heart rate
LPM:	Litre per minute
NE:	Nor epinephrine
NIV:	Non-invasive ventilation
RR:	Respiratory rate
SaO2:	Saturation of Haemoglobin in arterial blood
SAT:	Spontaneous awakening trial
SBT:	Spontaneous breathing trial
SAD:	Supraglottic air way disease

3. General assessment & resuscitation of a critically ill patient:

3.1 General guidelines

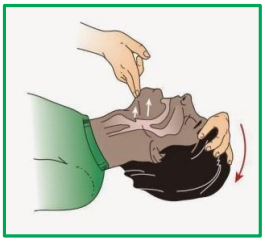
1. All critically ill patients should be monitored adequately and steps initiated to prevent further deterioration.
2. All resuscitation is teamwork and job responsibilities of each member should be clear and appropriate.
3. Team to be adequately staffed
4. Take early assistance whenever needed from other members of the team.
5. Initial aim is to determine immediate life-threatening problems.
6. Correcting physiological abnormalities should take precedence over arriving at an accurate diagnosis.
7. Working diagnosis is essential for deciding treatment options once physiological stability is achieved.
8. For hemodynamically unstable patients, resuscitation should be systematic and aimed toward assessment and management of A (airway), B (breathing), and C (circulation).
9. All three components can be managed simultaneously; sequential approach is not necessary. (If adequate number of trained personnel available)

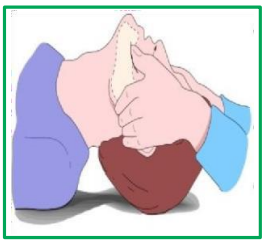
All the above domains in general guidelines should be included in a sop formed by each organization

3.2 Airway:

Obstruction may be partial or complete. The latter is characterized by total lack of air exchange. The former is recognized by inspiratory stridor and retraction of neck and intercostal muscles. If respiration is inadequate, the head-tilt–chin-lift or jaw-thrust manoeuvre should be performed. In patients with suspected cervical spine injuries, the jaw-thrust manoeuvre (without the head tilt) may result in the least movement of the cervical spine.

Figure1: a. Chin lift and head tilt b. jaw thrust





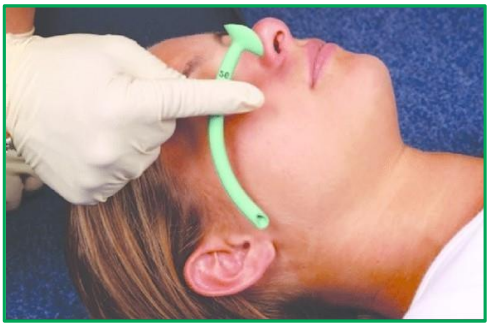
Clear upper airway obstruction if present:

- Snoring, gurgling sound, paradoxical movement of the chest wall (inward movement during inspiration) and abdomen and inadequate/absent chest rise during ventilation may suggest upper airway obstruction.
- Perform an oral or nasal (with soft malleable catheter) suctioning for no more than 10 s at a time and resume oxygenation soon after.
- Use an oropharyngeal or nasopharyngeal airway if obstruction is not cleared by suctioning. The airway should have a length equivalent to distance from the tip of the nose/angle of the mouth to the tragus.
- Nasopharyngeal airway diameter should be less than the patient's nostril.
- Complete airway obstruction is silent—intubate.
- Assess the need for oxygen and ventilation. SpO_2, ABG.
- Signs of distress: Breathlessness/Tachypnoea/ Inability to talk/ Open-mouth breathing/ Ala nasi flaring/accessory muscle use/Paradoxical breathing/Restlessness/ Delirium/ Drowsiness/Cool extremities/Cyanosis/ Tachycardia/ Arrhythmia/ Hypotension/ Flapping tremor
- Look for features of tension pneumothorax and evidence of massive pleural effusion or haemothorax and drain immediately. NEEDLE THORACOTOMY for Tension Pneumothorax.
- Any evidence of massive lung collapse with desaturation requires intubation, suctioning, and positive-pressure ventilation.

- Non-invasive ventilation can be tried in relatively stable patients if they are suffering from a condition where non-invasive ventilation has been shown to be effective

NP airway, selecting correct size.

Sizing of nasopharyngeal airway



Oropharyngeal airway

Nasopharyngeal Airway

3.3 Oxygen therapy

Oxygen treatment is often life-saving, but multiple studies in recent years have yielded evidence that the indiscriminate administration of oxygen to patients in the intensive care unit and emergency room can cause hyperoxia and thereby elevate mortality. Hypoxemia should certainly be avoided.

Oxygenation goals*	
Acute exacerbation of COPD	Target: 88-92%
Myocardial Infarction	Oxygen administration if SpO2<90%
Post resuscitation	lowest oxygen administration to achieve SpO2>94%
Ventilated intensive care patients	Lowest O2 to achieve SpO2 of 90-94%/PaO2 of 60-80mmHg
ARDS (moderate to severe)	PaO2: 55-80 mmHg

*Oxygen Treatment in Intensive Care and Emergency Medicine Jorn Grensemamm et al

3.4 Endotracheal intubation:

3.4.1 Indications:

- Inability to maintain airway patency. Trauma/Foreign bodies/ Infection/ Hematoma/ Tumour/ Congenital anomalies/ Laryngeal oedema/ Laryngeal spasm
- Inability to protect the airway against aspiration: Head injury/Drug overdose/Cerebrovascular accident, GCS<8
- Anticipation of a deteriorating course that will eventually lead to respiratory failure. E.g. airway burns
- Respiratory Failure:

Hypoxemia

a. Acute respiratory distress syndrome
b. Hypoventilation
c. Atelectasis
d. Secretions, Copious; > 4suctions/hr
e. Pulmonary oedema

Hypercapnia

a. Hypoventilation leading to acidosis

b. Neuromuscular failure

c. Drug overdose

3.4.2 Contraindication:

Severe airway trauma where Cricothyroidotomy /tracheostomy is indicated.

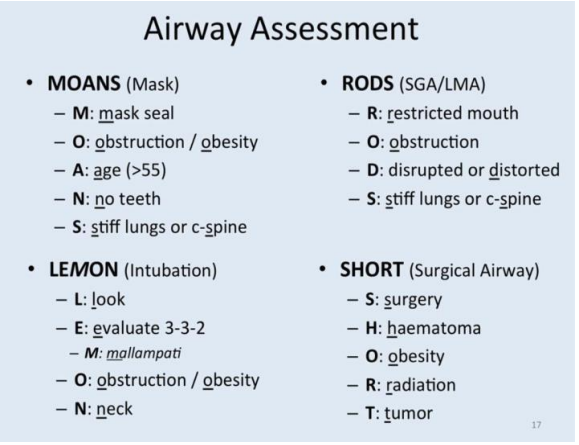


Table showing Airway assessment acronyms.

Time	Step
Zero minus 10 min	Preparation
Zero minus 5 min	Preoxygenation—100% oxygen for 3 min or 8 vital capacity breaths
Zero minus 3 min	Preintubation optimization—as indicated
Zero	Paralysis with induction • Etomidate, 0.3 mg/kg • Succinylcholine, 1.5 mg/kg
Zero plus 30 s	Positioning—Sellick maneuver optional
Zero plus 45 s	Placement • Laryngoscopy and intubation • End-tidal carbon dioxide confirmation
Zero plus 2 min	Postintubation management • Sedation and analgesia as indicated • Initiate mechanical ventilation • NMBA only if needed after adequate sedation, analgesia

Table showing time line for Rapid Sequence Intubation

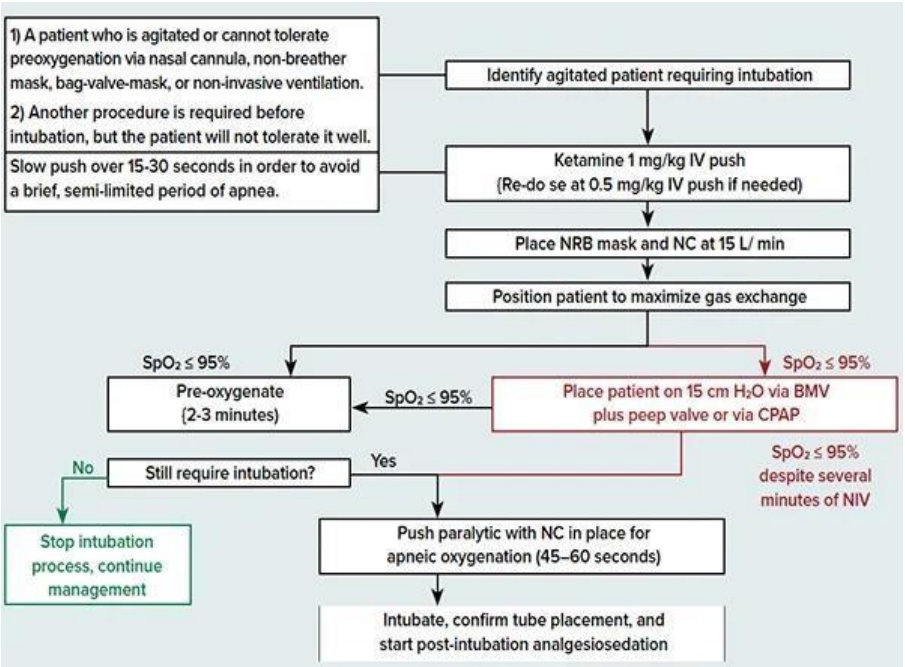


FIGURE 1. Delayed Sequence Intubation (DSI) Algorithm.
Adapted from Nickson, C. (2016). Delayed sequence intubation. LITFL. Life in the Fast Lane Medical Blog.

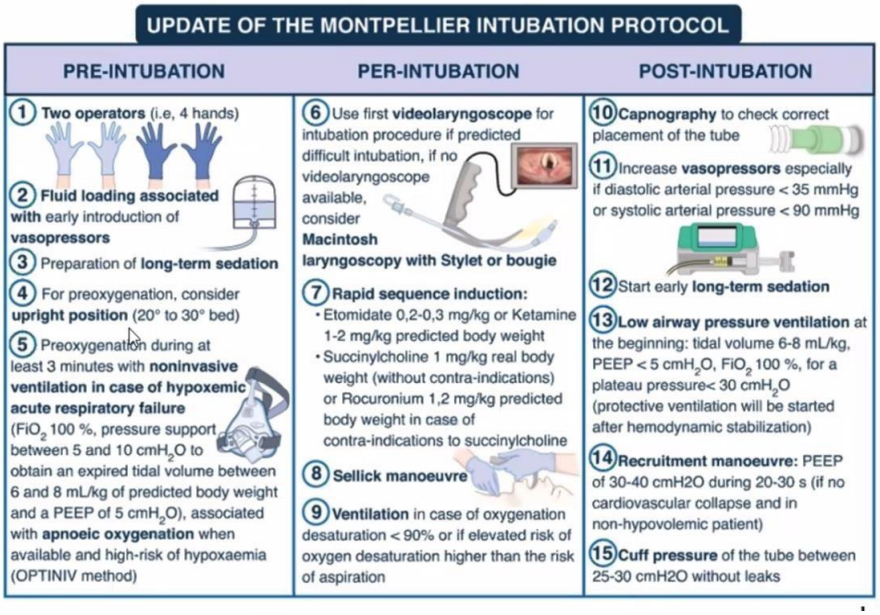
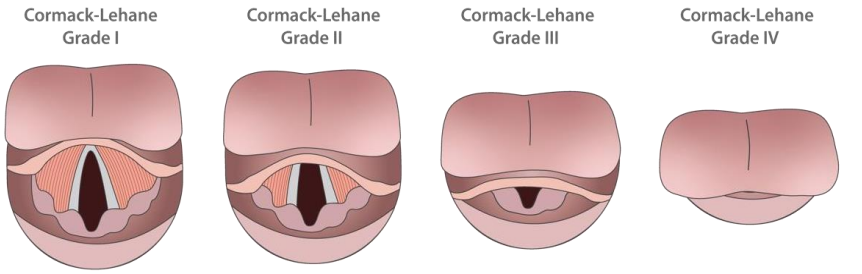


Table 2. MACOCHA Scoring

Factor	Points Awarded
Patient-related factors	
Mallampati score of III or IV	5
Obstructive sleep apnea	2
Reduced cervical spine mobility	1
Limited mouth opening <3 cm	1
Disease-related factors	
Coma (Glasgow score <8)	1
Severe hypoxemia	1
Operator-related factor	
Non-anesthesiologist	1

MACOCHA: **M**allampati score of III or IV, sleep **A**pnea syndrome, decreased **C**ervical mobility, mouth **O**pening <3 cm, **C**oma defined by a Glasgow score <8, severe **H**ypoxemia, and if the practitioner is not an **A**nesthetist.



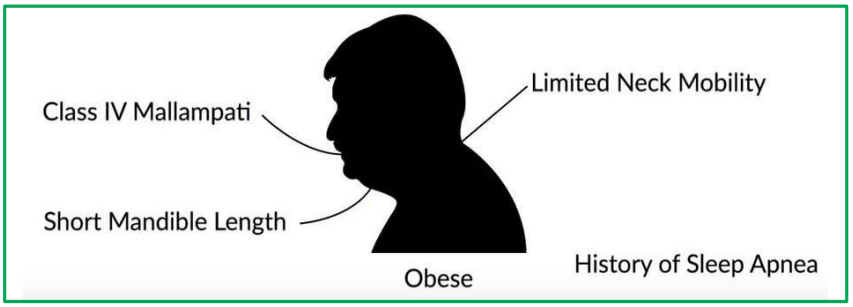
Special considerations during tracheal intubation in the intensive care unit

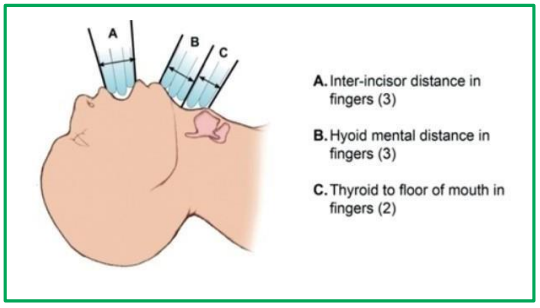
- Presence of two operators (one experienced in airway management)
- Call for additional help at the earliest
- Preoxygenation: Three minutes of pre-oxygenation using non-invasive positive pressure ventilation
- Hemodynamic instability: Drugs used for intubation like Propofol, Thiopentone, Diazepam can worsen hypotension. Hence hemodynamic resuscitation may be initiated it time permits prior to administration of these agents for intubation and a lower dose may be used. Ketamine, due to its sympathomimetic effects, helps maintain blood pressure during intubation and is preferred over other agents in unstable patients.
- Muscle relaxants: Use of neuromuscular blocking agents has been shown to improve the first-attempt intubation success.
- Device: video laryngoscopy has been shown to increase the first-attempt success and improve glottis visualisation during intubation in the ICU.

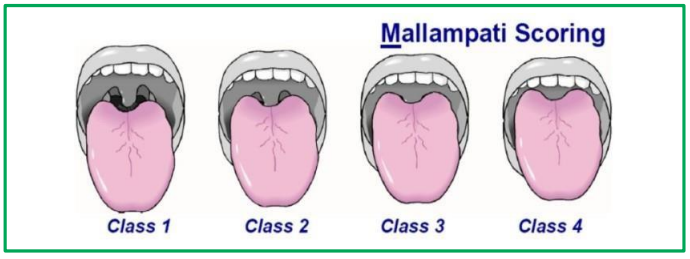
3.4.3. Difficult airway:

Look for signs of difficult airway:

- Length of upper incisor—relatively long • Inter incisor distance—less than two fingers (3 cm) • Overbite—maxillary incisors override mandibular incisors
- Temporomandibular joint translation—cannot place mandibular incisors anterior to maxillary incisors
- Mandibular space compliance—small, stiff, indurated, or occupied by mass
- Thyromental distance—less than three fingers (6 cm) Mallampati class—III and IV
- Neck—short, thick • Limited neck mobility—cannot touch chin to chest or cannot extend neck







Intubation cart:

- Self-
- inflating bag with reservoir bag
- Face mask of different sizes
- Oropharyngeal/ Nasopharyngeal airway
- Endotracheal tubes-Appropriately sized.7.5 to 8.5 for males, 6.5 to 7.5 for females
- Endotracheal

- tubes with sub glottic suction preferably used in all patients in whom prolonged intubation is anticipated • Lubricating jelly
- Working Laryngoscope: At least 2 blades (assortment of Miller and Macintosh Blades) Video Laryngoscopy to be available as part of Intubation cart.
- Syringes for inflating the cuff
- Magill's forceps
- Stylet, Bougie, Tube fixation tapes/ties

- End tidal CO_2 monitor/disposable CO_2 detector device
- Fiber-optic bronchoscope/ Video laryngoscope if available
- Drugs: Induction agents and muscle relaxants, topical anaesthetics and vasoconstrictor
- Rescue devices: LMA/Intubating LMA and Cricothyroidotomy set

List of mandatory and desirable equipment for difficult airway cart	
Mandatory	**Desirable**
Working Laryngoscope blades with Mcintosh blades	McCoy Blade
Video laryngoscope	Flexible fibre optic bronchoscope
Face Masks, ETT, Magill's Forceps, Stylet, Bougie	Equipment for high flow nasal oxygenation
Oropharyngeal/Nasopharyngeal	

airways	
Manual self-inflating bag	
Cannula/catheter	
Supraglottic airway devices (preferably intubating SAD)	
Nasogastric tube	
Airway exchange catheter	
Cricothyrotomy device-wide bore cannula, scalpel, bougie, size6mmID ETT or any commercially available cricothyroidotomy kit	

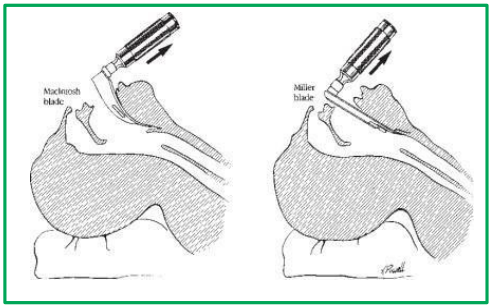
3.4.4 Drugs Used to Facilitate Intubation

(modified from Irwin & Rippe 7th ed)

Drug	Iv dose mg/Kg	Onset(sec)	Remarks
Thiopental	2.5-4.5	20-50	Hypotension
Propofol	1-2.5	<60	Hypotension, Pain
Midazolam	0.2-0.02	30-60	Hypotension
Ketamine	0.5-2	30-60	Increase ICP, Secretion Emergence reactions
Etomidate	0.2-0.3	20-50	Adrenal insufficiency, Pain on injection
Fentanyl	0.001-0.005	60-90	Cardio stable, high doses cause rigidity
Succinyl	1-2	45-60	Hyperkalaemia, Increased ICP, Intragastric pressure

choline			
Rocuronium	0.8-1.2	60-90	Long acting

- After giving adequate preoxygenation and proper position, cricoid pressure may be given just before the beginning of induction. As soon as the patient is asleep, increase the pressure.
- Use only rapidly acting muscle relaxants (suxamethonium or rocuronium) while maintaining cricoid pressure. The use of cricoid pressure is optional, applied selectively, as it may make laryngoscopy and intubation difficult if incorrectly applied and aspiration often occurs despite its use.
- Only if saturation is not maintained, gentle positive pressure ventilation (modified RSI).
- Perform laryngoscopy and intubation. Hold the laryngoscope handle in the left hand. Open the mouth of the patient with the thumb and the index finger of the right hand. Insert the laryngoscope blade gently into the mouth from the right-side angle of the mouth and move it to the left side taking the tongue along with the blade as it is inserted further inside the mouth. When the epiglottis is visualized, insert the curved blade into the vallecula and pull the laryngoscope forward and upward to expose the glottis.
- Use of IV lignocaine or low dose opioid prior to intubation to reduce stress response.
- Insert the ETT using the right hand between the vocal cords under direct vision. Use of stylet in ETT, bougie (a thin long plastic/rubber cylinder with a bent tip that is passed through the partially visible glottic opening and then the ETT is guided over it), or other airway adjuncts can aid oral intubation.



After intubation, inflate the ETT cuff just enough to avoid pharyngeal leak during ventilation. Cuff pressure monitors to be used for correct inflation pressures. Maintenance of intracuff pressures between 17- and 23-mm Hg should allow an adequate seal to permit mechanical ventilation under most circumstances while not compromising blood flow to the tracheal mucosa. The intracuff pressure should be checked periodically. (Every 8 hourly or during each shift.)

Release cricoid pressure only after intubation, cuff inflation, and confirmation of tube placement or if it makes laryngoscopy or intubation difficult

3.4.5. Confirm Tube placement:

- $EtCO_2$: At least 6 continuous, characteristic alveolar waveforms to be present
- 5-point auscultation – Direct visualization – CXR if clinically indicated – ET tip position to be kept 2 to 3cm above carina

3.4.6 Maintenance:

Proper maintenance of the airway will reduce the incidence of accidental extubation, disconnections, tube blockage, and nosocomial pneumonia.

Keep the head of bed elevated at 30–45°.

All ETT and tracheostomy tubes (TT) should be checked for position at incisor teeth/alae nasi, adequate fixation, patency, tracheal cuff pressure (<25 mmHg), and pharyngeal leak during each shift and should be documented.

3.5. Difficult airway Algorithm

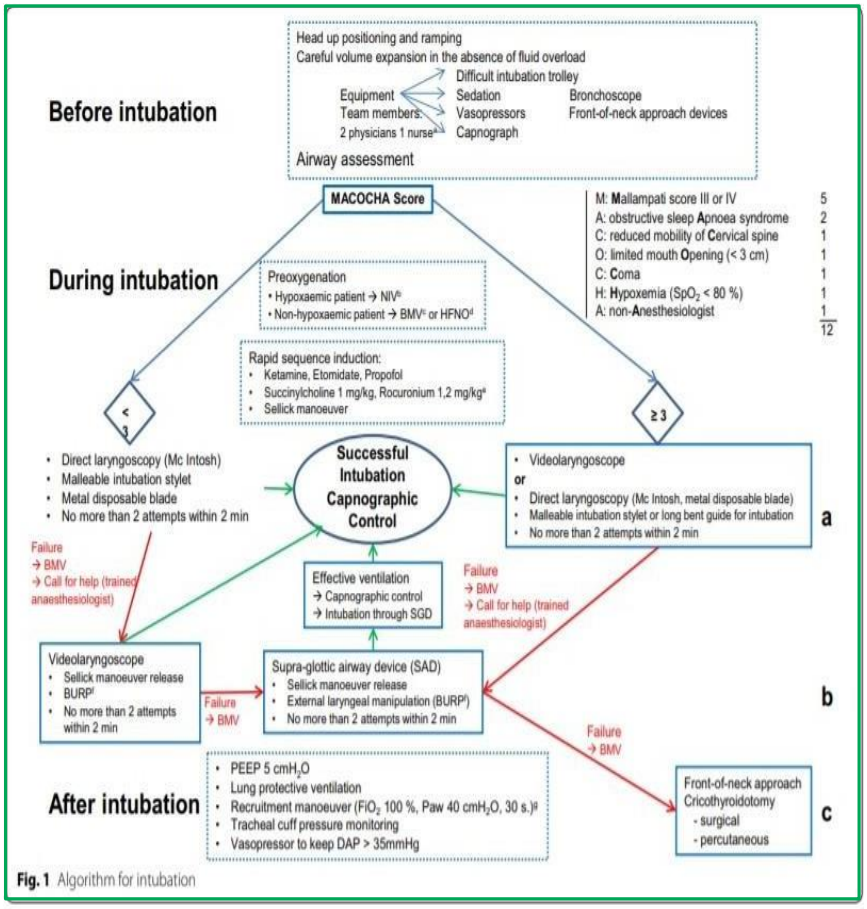


Fig. 1 Algorithm for intubation

Adapted from: Experts' guidelines of intubation and extubation of the ICU patient of French Society of Anaesthesia and Intensive Care Medicine (SFAR) and French-speaking Intensive Care Society (SRLF).

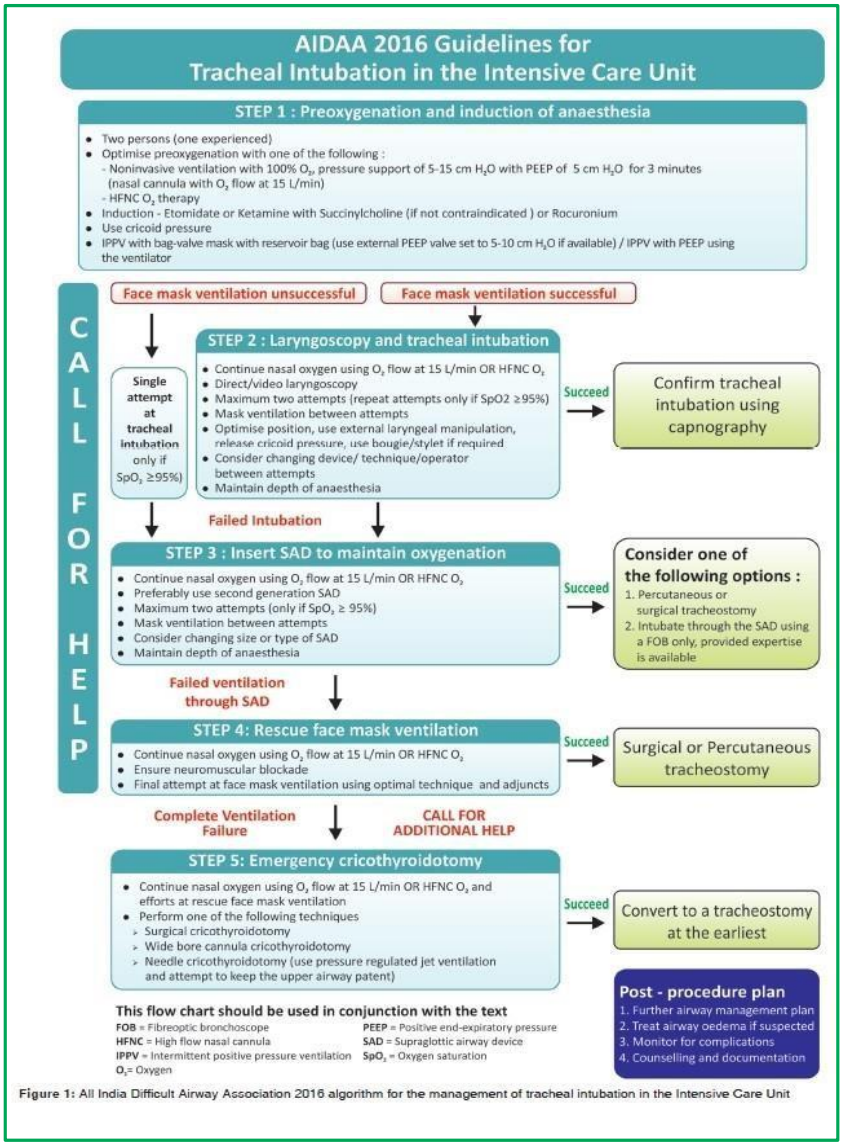


Figure 1: All India Difficult Airway Association 2016 algorithm for the management of tracheal intubation in the Intensive Care Unit

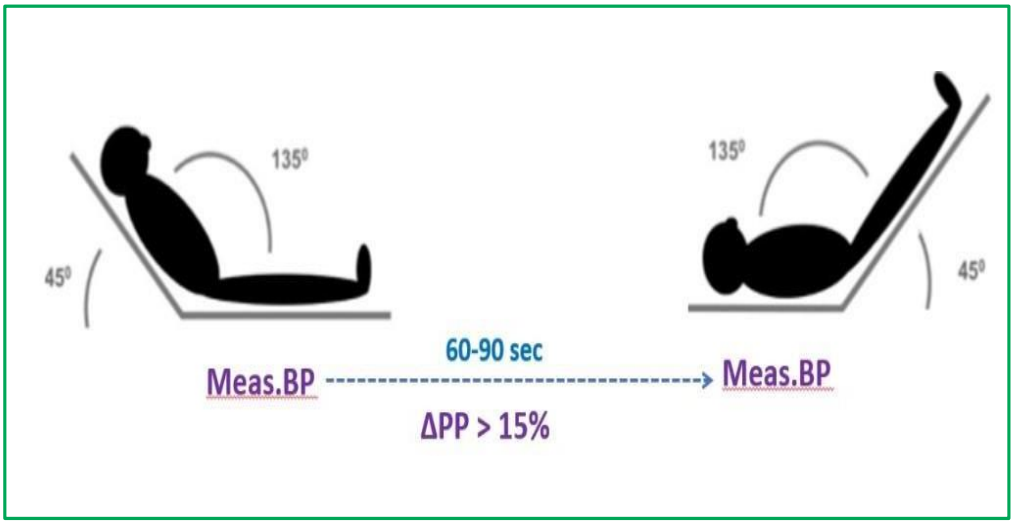
From The All India Difficult Airway Association 2016 guidelines for tracheal intubation in the Intensive care unit.

3.6 Circulation (C)

- Assess adequacy of circulation. Assessment and management should go side by side
- Peripheral and central pulse for rate, regularity, volume, and symmetry.

- Skin temperature
- Heart rate and rhythm • Blood pressure (supine and sitting for orthostatic hypotension)
    - Capillary refill • Mottling score • Jugular venous pressure
        - Urine output
    - Ultrasonography- Point of care.
- Echocardiography-Point of care • Consider invasive monitoring
- Central venous catheter insertion • Arterial catheter insertion
- Advanced hemodynamic monitoring • Passive leg raising to determine fluid responsiveness — not to be used in TBI, IAH and preferably done in a specialized bed.
- Judiciously use volume, inotropes, and vasopressor support.
- Look for pericardial tamponade causing hemodynamic instability requiring immediate pericardiocentesis.
- In patients with features of severe sepsis and septic shock, early broad-spectrum antibiotics AFTER Appropriate cultures and guided fluid resuscitation

Capillary refill:<2 sec Normal, >4 sec Abnormal, > 5sec: worsening organ function



Passive leg raising (Cherpanath et al Crit.Care Med 2016;45(5) 981-91

3.7 Disability (Neurological status)

- Frequent neurological examination needs to be performed in drowsy patients. Neuromonitoring hourly
- Lateralizing features like hemiplegia are usually a feature of neurological disease.
- A depressed conscious level in the absence of a primary neurological cause is indicative of severe systemic disease. Check for hypoglycaemia and correct urgently. - < 40mg/dl and prior administration of thiamine in case of suspected thiamine insufficiency Control ongoing seizures with appropriate measures.
- Consider urgent antibiotics for patients with features suggestive of bacterial meningitis.

Causes of Encephalopathy

Mnemonic: "VITAMINS"

Category	Causes
V: Vascular	Stroke (ischemic/hemorrhagic), hypertensive encephalopathy, cerebral venous thrombosis.
I: Infectious	Meningitis, encephalitis (e.g., HSV, CMV), sepsis-associated encephalopathy.
T: Toxic/Metabolic	Uremic encephalopathy, hepatic encephalopathy, electrolyte disturbances (Na^+, Ca^{2+}, Mg^{2+}), hypercapnia, hypoglycemia.
A: Autoimmune	Autoimmune encephalitis (e.g., anti-NMDA receptor encephalitis), paraneoplastic syndromes.
M: Medications/Drugs	Sedatives, opioids, anticholinergics, alcohol intoxication or withdrawal, illicit drugs.
I: Inflammatory	CNS vasculitis, demyelinating disorders (e.g., multiple sclerosis).
N: Neoplastic	Primary brain tumors, metastatic lesions, paraneoplastic encephalopathy.
S: Structural	Traumatic brain injury, subdural hematoma, cerebral edema, hydrocephalus.

Assessment of patients with altered mental status	
A	ALCOHOL
E	EPILEPSY
I	INSULIN

O	OVERDOSE
U	UNDERDOSE
T	TRAUMA
I	INFECTION
P	PSYCHOSIS
S	STROKE

National Early Warning Score (NEWS)*

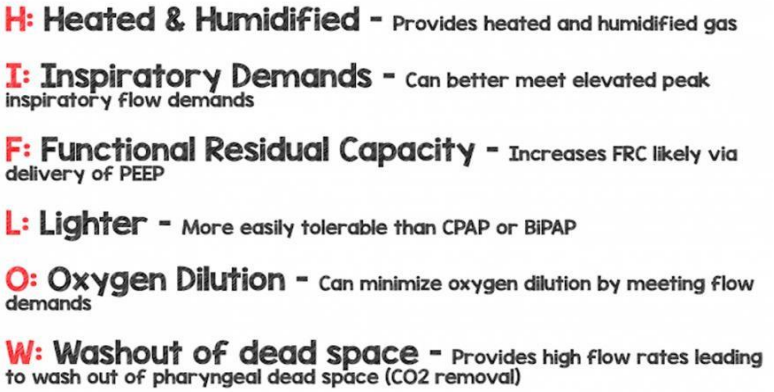
PHYSIOLOGICAL PARAMETERS	3	2	1	0	1	2	3
Respiration Rate	≤8		9 - 11	12 - 20		21 - 24	≥25
Oxygen Saturations	≤91	92 - 93	94 - 95	≥96			
Any Supplemental Oxygen		Yes		No			
Temperature	≤35.0		35.1 - 36.0	36.1 - 38.0	38.1 - 39.0	≥39.1	
Systolic BP	≤90	91 - 100	101 - 110	111 - 219			≥220
Heart Rate	≤40		41 - 50	51 - 90	91 - 110	111 - 130	≥131
Level of Consciousness				A			V, P, or U

3.9. Warning signs of severe illness:

- BP systolic <90 or mean arterial pressure <60 mmHg
- Glasgow coma score <12
- Pulse rate >150 or <50 beats/min Respiratory rate >30 or <8/min
- Urine output <0.5 mL/Kg/h Send relevant investigations
- Assess responsiveness to initial treatment
- Construct a working diagnosis and plan further management
    - Send relevant consults
- Brief relatives.

4. High flow nasal oxygen therapy/high flow nasal catheter

HFNO allows for delivering up to 60 liters min-1 of gas at 37^{O}C and with an absolute humidity of 44 mg H2O litres-1. In contrast with all the other systems for oxygen therapy, HFNO enables the administering of FIO2 up to 100%.



Strengths	Drawbacks
Easy to implement & manage	Nasal mucosal irritation
Minimal risk of skin breakdown	Discomfort
Lower nurse workload in comparison with NIV	Runny nose
Stability of nasal cannula in comparison with conventional high flow nasal mask	Pneumothorax in new born (air leak syndrome)
No claustrophobia	Alteration of smell
Eating drinking communicating permitted	Risk of delayed intubation

Setting of HFNC

1. Prongs not to totally occlude nostrils
2. Flow rate: start with 30-40 Lpm and increase to meet patients demand
3. Temperature: Set at 37^0C
4. FiO2: Increase in FiO2 by adjusting Oxygen flow in flow meter assembly until satisfactory SaO2 is achieved
5. Flow: increase till a reduction in respiratory rate and stable Sa02 is achieved
6. Water reservoir: place as high as possible above the humidifier
7. Monitoring: Continuous-HR, RR, SaO2
8. Positive response: Gas flow and FiO2 adjusted to the clinical response, reduce FiO2 by 5- 10% and reassess after 1-2hr. Consider weaning from HFNO with flow rates <25lpm and FiO2<0.4
9. Ineffective response: If there is no improvement after 60-120 min, treatment escalation must be considered

Specific scenario

- Hypoxemic (de novo) acute respiratory failure: HFNO is superior to conventional forms of oxygen administration in improving arterial oxygenation and patient comfort, while reducing respiratory rate, dyspnoea and clinical signs of respiratory distress. The patients most likely to benefit from HFNO are those with mild-to-moderate forms of hypoxemic ARF. Reserve HFNO for patients in whom standard oxygen fails and escalating to NIV prior to invasive mechanical ventilation if HFNO also fails
- Post-extubation respiratory failure Immediate post-extubation is a crucial moment in the transition from mechanical ventilation to spontaneous breathing. By guaranteeing adequate oxygenation, facilitating expectoration and reducing the breathing effort, HFNO has the potential to prevent post-extubation respiratory failure and thereby avoid re-intubation
- Acute cardiogenic pulmonary oedema: By improving oxygenation while reducing

cardiac afterload through the generation of a low intrathoracic positive pressure, HFNO might also be beneficial in acute cardiogenic pulmonary oedema, though NIV is recommended first choice

- Do not intubate and palliative care: HFNO could be an additional means for the management of these patients. In fact, HFNO can be delivered continuously for protracted periods with few side-effects, which might allow more effective symptom palliation.
- Apnoeic oxygenation using OPTINIV method
- Immunocompromised
- Procedural sedation for bronchoscopy and endoscopy

5. Non-invasive ventilation

5.1 Criteria for NIV

1. In COPD exacerbation:
   a. Recommend bilevel NIV for patients with ARF leading to acute or acute on-chronic respiratory acidosis (pH ≤7.35) due to COPD exacerbation.
   (Strong recommendation, high certainty of evidence.)
   b. Recommend a trial of bilevel NIV in patients considered to require endotracheal intubation and mechanical ventilation, unless the patient is immediately deteriorating. (Strong recommendation, moderate certainty of evidence.)
   c. NIV not be used in patients with hypercapnia who are not acidotic in the setting of a COPD exacerbation.
2. In ARF due to Cardiogenic pulmonary oedema
   a. Recommend either bilevel NIV or CPAP for patients with ARF due to cardiogenic pulmonary oedema. (Strong recommendation, moderate certainty of evidence.)
3. In acute Asthma: No recommendation
4. De novo respiratory failure: Respiratory failure occurring without prior chronic respiratory disease. Group includes Significant hypoxaemia (PaO_2/FiO_2 ≤200),

tachypnoea (>35) and a non- COPD diagnosis (e.g. pneumonia and/or acute respiratory distress syndrome (ARDS)). Not included: Cardiogenic pulmonary oedema, post-operative respiratory distress.: The ability of NIV to achieve optimal pressures to reduce the work of breathing reliably in acute hypoxemic respiratory failure is challenging because the high pressures often required increase air leaks, gastric insufflation and patient intolerance. Thus, the ability to use lung protective ventilator strategies (such as maintaining a low tidal volume of 6 mL·kg−1 of predicted body weight) may be more difficult with NIV than with invasive ventilation. Some evidence even supports the idea that spontaneous ventilation can induce harm similar to ventilator-induced lung injury in situations of severe lung injury, which raises a note of caution when using NIV that combines spontaneous effort with ventilator support.

No recommendation on the use of NIV for de novo ARF.

5. Post-operative respiratory failure: NIV for patients with post-operative ARF recommended. (Conditional recommendation, moderate certainty of evidence.)
6. Palliation: Offering NIV to dyspnoeic patients for palliation in the setting of terminal cancer or other terminal conditions. (Conditional recommendation, moderate certainty of evidence.)
7. NIV for chest trauma patients with ARF: Recommended
8. Pandemic viral illness: No recommendation
9. ARF following extubation from invasive mechanical ventilation:
    - NIV be used to prevent post-extubation respiratory failure in high-risk patients post- extubation. (Conditional recommendation, low certainty of evidence.)
    - Suggest that NIV should not be used to prevent post-extubation respiratory failure in non- high-risk patients. (Conditional recommendation, very low certainty of evidence.)
    - Suggest that NIV should not be used in the treatment of patients with established post- extubation respiratory failure. (Conditional

recommendation, low certainty of evidence.)

10. NIV to facilitate weaning from mechanical ventilation: (weaning in progress) NIV be used to facilitate weaning from mechanical ventilation in patients with hypercapnic respiratory failure. (Conditional recommendation, moderate certainty of evidence.) NO recommendation for hypoxemic patients.

Clinical Indication	Certainty of evidence	Recommendation
Prevention of hypercapnia in COPD exacerbation	++	Conditional recommendation against
Hypercapnia with COPD exacerbation	++++	Strong recommendation for
Cardiogenic pulmonary oedema	+++	Strong recommendation for
Acute asthma exacerbation		No recommendation
Immunocompromised	+++	Conditional recommendation for
De novo respiratory failure		No recommendation
Post operative patients	+++	Conditional recommendation for
Palliative care	+++	Conditional recommendation for
Post extubation in high-risk patient	++	Conditional recommendation
Post extubation respiratory failure	++	Conditional recommendation against
Weaning in hypercapnic patients	+++	Conditional recommendation for

Adapted from Official ERS/ATS clinical practice guidelines: non invasive ventilation for acute respiratory failure Bram Rochwerg

5.2. Contra indication

- Inability to protect the airways—comatose patients, patients with CVA or bulbar involvement, confused and agitated patients, upper airway obstruction
- Hemodynamic instability—uncontrolled arrhythmia, patients on very high doses of inotropes, recent myocardial infarction
- Inability to fix the interface—facial abnormalities, facial burns, facial trauma, facial anomaly
- Severe gastrointestinal symptoms—vomiting, obstructed bowel; recent gastrointestinal surgery, upper gastrointestinal bleeding
- Life-threatening hypoxemia Copious secretions
- Non-availability of trained medical personnel

CHOOSE NIV option on ventilator–as leak compensation is better Use Non vented connector/mask for dual limb circuit

Use Vented connector/mask for single limb circuits

5.3. Mode

- NIV-PS-PEEP (in ICU ventilator) In BiPAP machine: IPAP-EPAP (Difference is the driving pressure)
- NIV-PCV (In BiPAP machine: PAC mode) BIPAP machine: Spontaneous, Spontaneous/Triggered, PAC, iVAPS are available

BIPAP MACHINE:

  - Using spontaneous/S/T MODE
  - Start with low settings such as inspiratory pressure support at 5–6 cm H2O and PEEP at 4 cm H2O.
  - Initiate NIPPV while holding the mask in place and confirm optimum fit. If it is big or small or loose, change it.
  - Hold the mask. Do not fix the headgear.

- Increase PEEP until inspiratory efforts are able to trigger the ventilator.
- If the patient is making inspiratory effort and the ventilator does not respond, it indicates that the patient has not generated enough respiratory effort to counter auto-PEEP and trigger the ventilator (in COPD patients).
- Increase PEEP further until this happens.
- Once the patient's inspiratory efforts trigger the ventilator, start increasing IPAP further, keeping the patient's comfort in mind. (Reduced respiratory rate, reduced use of respiratory accessory muscle, etc.)
- Difference BETWEEN IPAP AND EPAP is the driving pressure and a gap of minimum 4-5 to be maintained.
- Ensure that there are no major leaks.
- Secure interface with the headgear. It should be tight, but not overtight. Small leaks are acceptable.
- A peak inspiratory pressure of more than 25 cm is rarely required in COPD, but higher pressures can be used when using NIPPV for other indications. PEEP is usually titrated between 5 and 10 cm H_2O to improve triggering and oxygenation.

5.4 Monitoring during niv: hacor score

Variable	Value	Score
HR	≤ 120	0
	≥ 121	1
pH	≥ 7.35	0
	7.30-7.34	2
	7.25-7.29	3
	< 7.25	4
Glasgow	15	0
	13-14	2
	11-12	5
	≤ 10	10
PaO_2/FiO_2	>201	0
	176-200	2
	151-175	3
	126-150	4
	101-125	5
	≤ 100	6
RR	≤ 30	0
	31-35	1
	36-40	2
	41-45	3
	≥ 46	4

1. Mask comfort
2. Tolerance of ventilator settings
3. Respiratory distress
4. Respiratory rate
5. Sensorium
6. Accessory muscle use
7. Abdominal paradox
8. Ventilator parameters
9. Air leak
10. Adequacy of pressure support
11. Adequacy of PEEP
12. Tidal volume (5–7 mL/kg)
13. Patient–ventilator synchrony
14. Continuous oximetry (until stable)
15. ABG, baseline and 1–2 h, then as indicated

- The patient will show improvement in parameters if NIPPV is effective.
- ABG sample should be sent after 30 min to 1 h after the application of non-invasive ventilation.
- In ventilator setting, look for air leaks, triggering and patient–ventilator interaction.
- Monitor carefully the worsening respiratory distress, sensorium, tachypnoea, and deteriorating blood gases, and intervene early because delay in intubation is a very common major complication of NIPPV.
- Most complications are minor that can be managed very easily, and so every attempt should be made to continue NIPPV.

- Distension of the stomach due to aerophagia and aspiration can occur secondary to vomiting. A nasogastric tube can be used to relieve the distension while still allowing the mask to seal.
- Adverse hemodynamic effects from NIPPV are unusual, although preload reduction and hypotension may occur. Give intravenous fluids if tolerated.
- It is very important to know when to discontinue NIPPV and intubate and ventilate the patient.
- NIPPV failure-Consider intubation and ventilation Worsening mental status
    - Deterioration of pH and $PaCO_2$ after 1–3 h of therapy
    - Refractory hypoxemia—when even a brief discontinuation of NIPPV leads to significant fall in oxygen saturation
    - Intolerance to NIPPV
    - Hemodynamic instability.
    - Inability to clear secretions.

5.5 Weaning from NIV:

Initially, give NIPPV continuously as long as possible.

Once the patient is tolerating periods off NIPPV, start discontinuing during daytime and give during night time. In 2–3 days, the patient can be weaned off the NIPPV.

6. Invasive ventilation

6.1 Indications:

1. Apnoea/Respiratory arrest
2. Failed NIV/NIV contraindicated
3. Hypoxemic respiratory failure: moderate to severe ARDS
4. GCS 8 or less
5. Hemodynamic instability

- For full ventilatory support start with A/CMV: Volume Controlled ventilation or Pressure Controlled Ventilation.- **Physician Preference**

6.2. Care of patient on ventilatory support

- Analgo-sedation with Dexmedetomidine, Opioids, Benzodiazepines
- Sedation strategy: light sedation, nurse-driven, RASS targeted, polypharmacy, avoid benzodiazepines
- Comfortable, Co-operative and Calm
- Muscle relaxants are used sparingly (Severe ARDS, Intubation, prone ventilation etc) . Cis- atracurium is preferred, maximum for 48 hours as an infusion.
  - Once patient is stabilized and spontaneous efforts present can change to PSV mode
  - Monitoring and adjustments during mechanical ventilation
    - Patients should be closely monitored. Plateau pressure should be measured at least every 4 h and after any changes in tidal volume and PEEP.
    - Institute inspiratory hold in Volume controlled ventilation to measure Plateau pressure (gives idea regarding alveolar pressure).
    - In Pressure controlled ventilation Peak pressure is taken as alveolar pressure.
  - The ventilatory setting should be adjusted as per guidelines for mechanical ventilation in ARDS.net trials.
  - In ARDS: Lung protective ventilation with low tidal volume (Plateau pressure <30cmH_2O, Transpulmonary pressure <30 cm H_2O, driving pressure <14cm H_2O), Respiratory rate to a maximum of 35, Set according to pH and $PaCO_2$. Mechanical power <17J/min

Lower PEEP/higher FiO2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12

FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

- In Obstructive airway diseases like COPD: prolonged expiratory phase with increased expiratory time, decrease respiratory rate.
- Watch for auto PEEP and set external PEEP to match at least 80% of Auto PEEP while the patient is triggering the ventilator.
- If there is frequent high-pressure alarm, then look for bronchospasm, pneumothorax, atelectasis, blockade of endotracheal tube with secretions, right main bronchus intubation, leaks.
- Once the patient improves and the respiratory muscles are adequately rested, the patient should assume some of the work of breathing and be evaluated for weaning from the mechanical ventilation. The patients fulfilling the weaning criteria are extubated.

6.3 Liberation from mechanical ventilation

1. Recommend protocolized rehabilitation directed toward early mobilization for acutely
2. hospitalized adults who have been mechanically ventilated for more than 24 hours, (conditional recommendation, low certainty in the evidence). There is insufficient evidence to recommend any rehabilitation protocol over another.
3. Recommend ventilator liberation protocol for adults who have been mechanically ventilated for more than 24 hours (conditional recommendation, low certainty in the evidence). The ventilator liberation protocol may be either personnel driven or computer driven. Closed loop ventilation aids in early weaning. There is insufficient evidence to recommend any ventilator liberation protocol over another.
4. Cuff leak test and steroids before extubation:

- Recommend performing a cuff leak test in mechanically ventilated adults who meet extubation criteria (>110ml) and are deemed high risk for post extubation stridor (traumatic intubation, intubation more than 6 days, large endotracheal tube, female sex, and reintubation after unplanned extubation. (conditional recommendation, very low certainty in the evidence).
- For adults who have failed a cuff leak test but are otherwise ready for extubation, we suggest administering systemic steroids at least 4 hours before extubation, (conditional recommendation, moderate certainty in the evidence).
- A repeat cuff leak test is not required after the administration of systemic steroids
- Cuff leak test not well validated.

6.4.1. Pre-requisites

Step 1: Identify readiness for weaning

Any patient on MV should be considered for weaning if he/she fulfils the readiness criteria as mentioned below. Prerequisites "readiness criteria"

- The underlying reason for MV has been stabilized and the patient is improving.
- The patient is hemodynamically stable on minimal-to-no pressors.
- Oxygenation is adequate (e.g., $PaO_2/FiO_2 > 200$, PEEP < 5–8 cm H_2O, $FiO_2 < 0.5$).
- The patient is able to initiate spontaneous inspiratory efforts.
- Besides these criteria, the patient should be afebrile (temperature <38°C), have stable metabolic status (pH>7.25), adequate haemoglobin and adequate mentation (e.g., arousable, Glasgow coma scale >13).

6.4.2. Predictors of successful weaning

The predictors of successful weaning have been designed from physiologic parameters to help the decision-making process. Clinical judgment is of paramount importance.

- Rapid shallow breathing index (RSBI) is assessed by putting patient on PS PEEP
  - RSBI=RR/TV in litre
  - Less than 100 is predictor of successful weaning.
  - The threshold of 100 is not binding and can be relaxed by 10–20 in patients with endotracheal tube size less than 7 and in women.
- Minute ventilation less than 10 L/min.
- Respiratory rate (RR) less than 35 breaths/min.
- Maximum inspiratory pressure more negative than −30 cm H_2O.

6.4.3. Weaning Process

Step 1. SAT:

- Stop continuous infusion of sedation daily to awaken the patient to do spontaneous awakening trial
- Communicate with patient, explain the procedure, and calm them.
- Record baseline parameters and keep flow sheet at the patient's bedside.
- Keep a calm peaceful environment and have the nurse or physician remain at the bedside to offer encouragement and support.
  - If patient fails SAT, restart sedation on half of the previous dose.
  - If patient passes the SAT after stopping sedation, assess the patient for spontaneous breathing trial (SBT) based on the prerequisites criteria mentioned.

Step 2. Identification of failed SAT:

1. Anxiety, agitation, or pain
2. Respiratory rate >35/min
3. SpO_2 <88%
4. Respiratory distress

5. Acute cardiac arrhythmia

Step 3: Do spontaneous breathing trial (SBT)

- Whenever possible, position the patient upright in bed.
- Thoroughly suction the endotracheal tube and ensure patency.
- Any of the following modes can be chosen for SBT: A. T-piece:
  - Patients are disconnected from the ventilator and made to breathe humidified oxygen— air mixture through a T-piece connected to the endotracheal/tracheostomy tube for 30– 120 min.
  - Increased respiratory load is offered by the endotracheal tube. Dyspnoea and fatigue should be carefully avoided.

B. Pressure support ventilation • The pressure support level is to be gradually reduced, titrated to RR and patient comfort.

- A level of 6–8 cm H_2O pressure support is considered to overcome the tube resistance.
- Put the patient on PS of 6–8 cm H_2O and PEEP of 4 cm H_2O.

Duration:

The duration should be 30–120 min—shorter time for the patients on the ventilator for less than 1 week and longer for the patients on prolonged MV.

Step 4: Monitor closely

- Patient comfort, dyspnea, and all vital and respiratory parameters should be closely monitored. SBT should be terminated if it fails.
- SBT should be tried at least once in 24 h. More frequent SBTs do not help.
- At the end of the trial, if it succeeds, the patient is considered for extubation.

Step 5: Extubate the patient

After undergoing a successful SBT, a few more criteria should be fulfilled before deciding about extubation:

- Adequate cough reflex—spontaneously or while suctioning.
- Patient should be able to protect airways, and they should follow simple commands.
- Secretions should not be copious.
- A cuff leak of less than 110 mL measured during assist-control ventilation helps to identify patients who are at high risk of developing post extubation stridor/ obstruction of airway.
- No radiological or surgical procedure is being planned in the near future.

Checklist for Identifying candidates for a trial of spontaneous breathing	
1. Respiratory: PaO2/FiO2>150-200 with FiO2 <50% and PEEP <8mmHg, PaCO2 Normal or at baseline levels, Patient is able to initiate an inspiratory effort	
2. Cardiovascular: No evidence of myocardial ischemia. Heart rate <140 bpm, Bp adequate with minimal or No vasopressors	
3. Appropriate Mental status: Patient is arousable or GCS>13	
4. Absence of Correctable Comorbid conditions: No fever, No significant electrolyte abnormalities	
SBT Failure	
Objective measurement	PaO2<50-60mmHg on FiO2 >0.5 or SaO2<90%
	PaCO2>50mmHg or an increase inPaCO2>8mmHg
	pH<7.32 or a decrease in pH>0.07
	Rapid Shallow breathing index >105RR>35 or an increase of >50%

	RR>35 or an increase of >50%
	Heart rate >140 or increase of >20%
	Systolic blood pressure>180 or an increase of > 20%
	Systolic pressure<90
	Cardiac arrhythmias
Subjective clinical assessment	Agitation and anxiety
	Depressed mental status
	Diaphoresis
	Cyanosis
	Evidence of increasing effort: Increasing accessory muscle use, facial signs of distress, Dyspnoea

Step 6. After extubation, the patient should be observed closely for signs of extubation failure as:

- RR more than 25/min for 2 h • Heart rate more than 140 beats/min or sustained increase or decrease of more than 20%
- Clinical signs of respiratory muscle fatigue or increased work of breathing
- SaO_2 less than 90%; PaO_2 less than 80 mmHg, on FiO_2 more than
- 0.50 • Hypercapnia ($PaCO_2$>45 mmHg or >20% from pre extubation), pH < 7.33
- Step 7: Try non-invasive ventilation (NIV)
- If the signs of extubation failure are present, the physician should try
- NIV particularly in conditions where its role is proved; for example, in

COPD, postoperative failure after lung resection surgery, or decompensated obstructive sleep apnoea.

- NIV has the advantage of reduced complications and better patient interactions. However, it is important to keep in mind that it should not delay reintubation (if required), and every hour that a patient spends on NIV when intubation is clearly required increases mortality and delays recovery. Step 8: Identify difficult weaning
- Weaning success is defined as extubation and the absence of ventilator support 48 hours following extubation.
- Simple weaning: extubation success within 1st SBT attempt

Weaning failure is defined as one of the following:

- Failed SBT
- –Reintubation and/or resumption of ventilator support following successful extubation
- Death within 48 h following extubation

W*eaning in progress* is used for the patients who are extubated, but remain supported by NIV. *Difficult weaning* ––Patients who fail initial weaning and require up to three SBT or as long as 7 days from the first SBT to achieve successful weaning. *Prolonged weaning* ––Patients who fail
at least three weaning attempts or require more than 7 days of weaning after the first SBT.

Step 9: Causes of weaning difficulty

- Carry out a detailed examination of the patient, and look for the cause of difficult weaning.
  - Make a checklist based on pathophysiologic mechanisms:
  - Nutritional deficiencies ● Excess of sedatives ● Central nervous system abnormality ● Sleep deprivation ● Unresolving pneumonia ● Unresolved pulmonary oedema/ fluid overload
  - Undiagnosed pulmonary embolism

- The splinting effect of obesity, abdominal distension, or Ascites
- Respiratory muscle fatigue/weakness Nutritional and metabolic deficiencies Critical illness polyneuropathy/myopathy Hypokalaemia
- Hypomagnesemia Hypocalcaemia Hypophosphatemia Hypoadrenalism Hypothyroidism
- Corticosteroids: myopathy, hyperglycemia Chronic renal failure
- Systemic disease sepsis: impaired diaphragmatic force generation
- Refractory hypoxemia and hypercapnia ● Persistently increased work of breathing
- Ineffective triggering, auto-PEEP ● Increased resistance due to ventilator tubing or humidification devices
- Poor cardiac performance ● Neuromuscular dysfunction/disease
- Drugs ● Anxiety
  - It is difficult to distinguish anxiety from ventilatory failure. If in doubt, always presume it to be ventilatory failure. ● ·Psychological dependency in difficult weaning

Step 10: Treat all the reversible cause identified

- Provide good nutrition, but avoid overfeeding. – 25 to 30 kcal/kg/day IBW
- Feeding goals should be achieved over a period of one week. Practice PEP-UP protocol
- Watch for refeeding syndrome in malnourished patients.
- Have good glycaemic control (140 – 180mg/dl).
  - Correct metabolic factors (especially metabolic alkalosis).
  - Maintain haemoglobin above 7–8 g/dL. ● Maintain adequate cardiac output and tissue perfusion.
  - Treat arrhythmia. ● Treat hypothyroidism and steroid deficiency or excess.
  - Control the patient's underlying illness.
  - Abolish ventilator dyssynchrony with appropriate inspiratory flow and

trigger settings.

- Change of the mode of ventilation may help improve patient–ventilator interactions.
- Reverse bronchospasm as much as possible and reduce dynamic hyperinflation.
- Drain out significant pleural effusions and ascites. ● Treat intraabdominal hypertension. ● Treat pulmonary edema aggressively.
- Discontinue the use of steroids, aminoglycosides, colistin, and statins, if possible.
- Avoid fluid overload in renal failure and cardiac failure—do dialysis if indicated.
- Aggressive physiotherapy and mobilization. ● Reverse over sedation.
- Treat anxiety: Improve patient communication, use relaxation techniques, and give low-dose benzodiazepines.
- Diagnose and treat narcotic/benzodiazepine withdrawal. ● Treat delirium and depression.
- Ensuring night time sleep may be helpful.

Step 11: Weaning process in difficult weaning

1. Select the mode of ventilation

- The mode of ventilation used should provide adequate respiratory support and prevent diaphragmatic atrophy.
- Pressure support ventilation.
- Continuous positive airway pressure (CPAP): Besides the usual benefits of improved oxygenation and improved left ventricular function, it has beneficial role in selected patients with hypoxemic respiratory failure.
- Automatic tube compensation: It may be helpful in narrow endotracheal tubes to overcome tube resistance.

2. Plan tracheostomy.
    - Percutaneous tracheostomy has been shown to have fewer complications than surgical tracheostomy and to be more cost effective.
    - Potential benefits of using tracheostomy in difficult-to-wean patients are as follows:
        - Decreased work of breathing
        - Reduced requirement of sedation and improved patient comfort and cooperation
        - Earlier reinstitution of oral feeding
        - Less chances of accidental extubation
3. Do aggressive physiotherapy and mobilization Physiotherapy and mobilization are prerequisites for successful weaning. Early institution of physiotherapy in a protocol- driven approach and daily assessment to achieve maximum mobility is now an integral part of ICU management.
    - Select proper place for weaning-Cost-effective care has been shown to be provided in respiratory intermediate care units and specialized regional weaning centers.

Step 12: Decide about home ventilation Indications:

- An inability to be completely weaned from ventilatory support including NIV
- A progression of disease aetiology that requires increasing ventilatory support
- Patients should have stable physiology and proper resources, personnel, and motivation

7 Shock

7.1 Definition:

“a life-threatening, generalized form of acute circulatory failure associated with Inadequate oxygen utilization by the cells”.

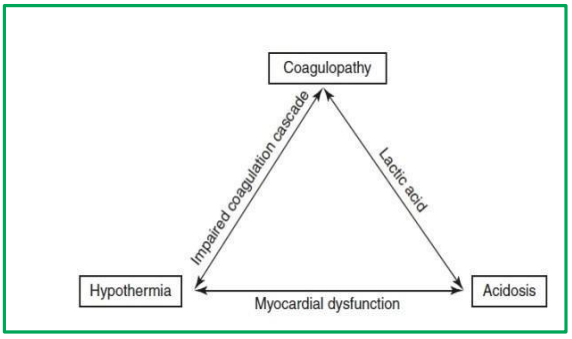
7.2 Classification:

	Hemodynamic changes	Etiologies
Hypovolemic	Decreased preload, CO, Increased SVR	Hemorrhage, capillary leak, GI losses, burns
Cardiogenic	Increased Preload, after load, SVR, Decreased CO	MI, dysrhythmias, heart failure, valvular disease
Obstructive	Decreased preload, Increased SVR, decreased CO	PE, Pericardial tamponade, pneumothorax, LV outlet obstruction
Distributive	Decreased preload, Increased SVR, Mixed CO	Septic shock, anaphylactic shock, neurogenic shock

7.3 Hypovoluemic shock:

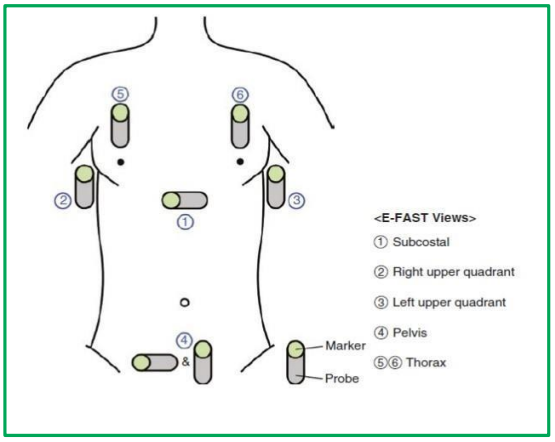
7.3.1 Hemmorhagic shock:

a. Initial assessment of the severity of the patient and identification of the source of bleeding are crucial for the patient with haemorrhagic shock.



b. Source: Obvious

c. Source: Unidentified: Imaging: USG EFAST



d. Serial measurements of haematocrit, lactate, base deficit, and monitoring of coagulation with conventional tests and viscoelastic methods are essential for diagnosis and guiding treatment

e. Initial Management: arrest ongoing bleeding, to restore the effective circulating blood volume, and to restore tissue perfusion.

f. Damage control resuscitation (DCR)

g. Target Blood pressure: systolic blood pressure of 80–90 mmHg is recommended currently in the initial resuscitation phase (Exception-TBI, Caution: chronic hypertension, elderly)

Rapid recognition of coagulopathy and shock
Permissive Hypotension
Rapid Surgical control of bleed
Hypothermia Prevention /treatment of hypothermia, acidosis and hypocalcemia
Avoidance of haemodilution induced by aggressive intravenous fluid
Transfusion of RBC: Plasma: Platelets in a high unit ratio or reconstituted whole blood in a 1:1:1 unit ratio
Early and appropriate use of coagulation factors
The use of fresh RBCs and whole blood when available

Damage Control Resuscitation

h. Type of fluid:

- Blood
- Synthetic Colloids to be avoided.
- Crystalloids:
- 0/9% Saline: Can use in TBI. Complication: Normal Anion gap metabolic acidosis and AKI.
- Avoid hypotonic crystalloids in TBI
- Balanced salt solution: can be used

I. Vasopressors: vasopressors may be required in the life-threatening hypotension despite fluid resuscitation.

j. Temperature control: Hypothermia in haemorrhagic shock should be prevented and warm the patients with hypothermia using measures such as removing wet clothing, covering the patient, infusion of warm fluid, forced warm air, and rewarming devices

k. Tranexamic Acid: tranexamic acid reduced organ failure and mortality in traumatic shock patients. Dose: loading dose of 1 g over 10 min, followed by infusion of 1 g over 8 h within 3 hrs

l. Control of bleeding:

- Tourniquets and Pelvic binders
- Angiographic embolization
- Endoscopic Haemostasis and Interventional Approach
- Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) (Weak evidence)

m. Ca++ correction

n. Correction of pH, Fibrinogen

7.4 Cardiogenic shock:

Cardiogenic shock: Systolic BP <90 mmHg despite adequate filling status with signs of hypoperfusion.

Due to primary failure of the ventricles of the heart to function effectively.

Causes:

Acute Myocardial infarction

Pump failure

Mechanical complications

RV infarction

Other conditions

End stage cardiomyopathy

Myocarditis

LV outlet obstruction

Acute MR

Cardinal manifestations include

- Oliguria and worsening renal function.
- Metabolic acidosis due to increased production and decreased clearance of lactate.
- Mental status changes ranging from confusion to coma.
- Cool, clammy skin due to intense vasoconstriction. In patients with distributive shock and low systemic vascular resistance (SVR), extremities may be warm and flushed.

Investigations: Chest X-ray, ECG, and echocardiograph, Troponins, NT Pro BNP. Hemodynamic monitoring should complement (and not replace) other markers of end-organ perfusion in CS. The optimal MAP likely differs from patient to patient, and the risks of hypoperfusion with lower MAPs must be balanced (and individualized) with the potentially deleterious impact of vasoactive agents on myocardial oxygen demand, ischemia, and arrhythmia associated with higher MAP targets.

Assess the adequacy of end-organ and tissue perfusion in response to individualized targets by integrating serial markers of systemic perfusion, including (but not limited to) arterial lactate, mixed or central venous oxygen saturations, urine output, creatinine, liver function tests, mental status, temperature, and other invasive hemodynamic variables.

Recommend an early invasive strategy with appropriate revascularization for all suitable patients with suspected ACS-associated CS, including patients with uncertain neurological status or those who have received prior fibrinolysis, regardless of the time delay from MI onset.

Prepare for revascularization in the cardiac catheterization laboratory or surgical intervention for mechanical failure

		Volume Status	
		Wet	Dry
Peripheral Circulation	Cold	Classic Cardiogenic Shock (↓CI; ↑SVRI; ↑PCWP)	Euvolemic Cardiogenic Shock (↓CI; ↑SVRI; ↔PCWP)
	Warm	Vasodilatory Cardiogenic Shock or Mixed Shock (↓CI; ↓/↔SVRI; ↑PCWP)	Vasodilatory Shock (Not Cardiogenic Shock) (↑CI; ↓SVRI; ↓PCWP)

Hemodynamic support: Fluid is given in RV infarct with hypotension. Because some patients with cardiogenic shock develop hypotension without pulmonary oedema, an appropriate amount of fluid can be administered. If there is no improvement in perfusion with fluid challenge, or there is hypoperfusion with pulmonary oedema, vasopressors or inotropes are considered. IABP, LV Assist device, extra corporeal life support can be tried

MI-associated CS who have multivessel or left main disease, PCI or CABG revascularization decisions should be made collaboratively between cardiologists and surgeons by incorporation of the patient's medical information, coronary anatomy, procedural risks, potential treatment-related delays, and expressed preferences.

Cause or presentation of CS	Medication	Hemodynamic response
Classic or wet	NE/Dopamine Inotrope	NE-preferred in tachycardia Dopamine-in bradycardia (high incidence of arrhythmias) Consider addition of inotropic agent when stabilized and after revascularization (MI only)
Euvolemic cold and dry	NE/Dopamine Inotropic agent Small fluid boluses	NE-preferred in tachycardia Dopamine-in bradycardia (high incidence of arrhythmias) Consider addition of inotropic agent when stabilized and after revascularization (MI only)
Vasodilatory warm & wet or mixed cardiogenic and vasodilatory	NE	Has low SVR
RV shock	Fluid boluses, NE, Dopamine or vasopressin, inotropes, inhaled pulmonary vasodilators	Maintain preload, lower RV after load, treat bradycardia Dopamine- decreased HR (but associated with arrhythmias), Vasopressin may raise SVR and have neutral effect on PVR Consider inotrope after hemodynamic stabilization and revascularization

Adapted from AHA Contemporary Management of Cardiogenic Shock 2017

7.5 Obstructive shock:

- Obstructive shock is a form of shock associated with mechanical obstruction of blood flow to the heart, specifically left ventricle.
- Tension pneumothorax, Cardiac tamponade, Pulmonary embolism ● Detailed history is sought ● Point of care ultrasonography:
- Presence, location, and volume of pneumothorax ● RV strain on echo- Pulmonary embolism ● Pericardial tamponade Management ● Airway
- Oxygen supplementation / Ventilatory support

7.5.1. Tension Pneumothorax:

- tracheal deviation toward the contralateral side of tension pneumothorax,
- hyper-resonance
- diminished lung sounds on the affected side ● subcutaneous emphysema neck vein engorgement.
- Persistent shock may result in the bradycardia and pulseless electrical activity arrest.
- USG lung, Chest X-ray, CT
- Immediate Needle decompression/ ICD

7.5.2. Pulmonary Embolism:

- In the absence of hemodynamic instability at presentation, the diagnostic work-up of a patient with suspected acute PE begins with the assessment of the clinical or pre-test probability of PE.
- Standardized prediction rules integrating baseline clinical parameters and the patient's history permit the classification of patients into distinct categories of clinical probability of the disease.

7.5.2 . a. Probability score

Wells Probability score for Pulmonary Embolism		Pulmonary embolism rule out criteria All 9 to be present to rule out
Clinical signs & symptoms of DVT	3	Clinical low probability (<15%probability of PE based on gestalt assessment)
Pulmonary embolism is most likely diagnosis	3	Age<50 yrs
Tachycardia>100 bpm	1.5	Pulse<100bpm during entire stay at ED
Immobilization or surgery in previous 4 weeks	1.5	SpO2>94%
Haemoptysis	1	No haemoptysis
Active malignancy	1	No prior VTE history

Low risk:<2, Intermediate risk2-6, high>6 Pulmonary embolism unlikely0-4, likely >4 points.	No surgery or trauma requiring Endotracheal or epidural anesthesia within the last 4 weeks

Preferred imaging method for the diagnosis of acute PE in patients with either a high clinical (pre-test) probability or low/intermediate probability and elevated D dimer levels:

CT pulmonary angiography

Criteria	Clinical probability category	Proportion of patients with confirmed PE	Plan
Wells score for PE/Revised Geneva Criteria	LOW	10%	D-dimer Age adjusted cut off if >50y <(Agex10ug/l)
	INTERMEDIATE	30%	D-dimer
	HIGH	65%	Imaging
2 tier classification	PE unlikely	12%	D-dimer
	PE likely	50%	Imaging

7.5.2.b. Adverse Prognosis in Acute Pulmonary Embolism

- ECG: Sinus tachycardia, new onset atrial arrhythmias, New RBBB,

  QR pattern in V1, S1Q3T3, T inversion In V1 through V4 Biomarker: Elevated Troponin, Elevated BNP and N terminal pro-

  BNP

- CT: RV diameter/LV diameter >0.9, Ventricular septal bowing from Right to left, presence of RV enlargement
- Echocardiography: RV dilatation and hypokinesis, RV/LV diameter>0.9, interventricular septal flattening and paradoxical leftward septal motion, Presence of TR., presence of PH (peak tricuspid jet velocity greater than 2.6m/s and loss of respiratory phasic IVC collapse, RV free wall hypokinesis with apical sparing (McConnell's sign)

  The severity of PE is stratified into massive (PE causing hemodynamic compromise), sub- massive (PE causing right ventricular dysfunction demonstrable by echocardiography, computed tomography or elevated cardiac biomarkers) and non-massive or low-risk (PE without evidence of RV dysfunction or hemodynamic compromise).

7.5.2.c. Management

A. Systemic thrombolysis

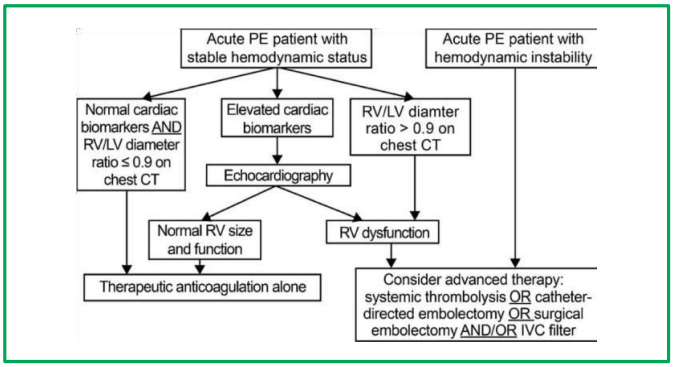
Systemic thrombolysis is associated with lower all-cause mortality in patients with massive PE and should be the treatment of choice in this subset of patients.

In sub-massive PE, use of systemic thrombolysis is associated with a mortality benefit yet significantly increases the risk of major bleeding, including intracranial hemorrhage. For this subset . of patients' guidelines currently recommend systemic thrombolytic therapy when cardiopulmonary deterioration is evident yet frank hypotension has not occurred.

Catheter embolectomy can be considered when cardiopulmonary deterioration is evident or in sub-massive PE when patients have clinical evidence of adverse prognosis.(when cardio pulmonary deterioration is evident.) Indications of thrombolysis:

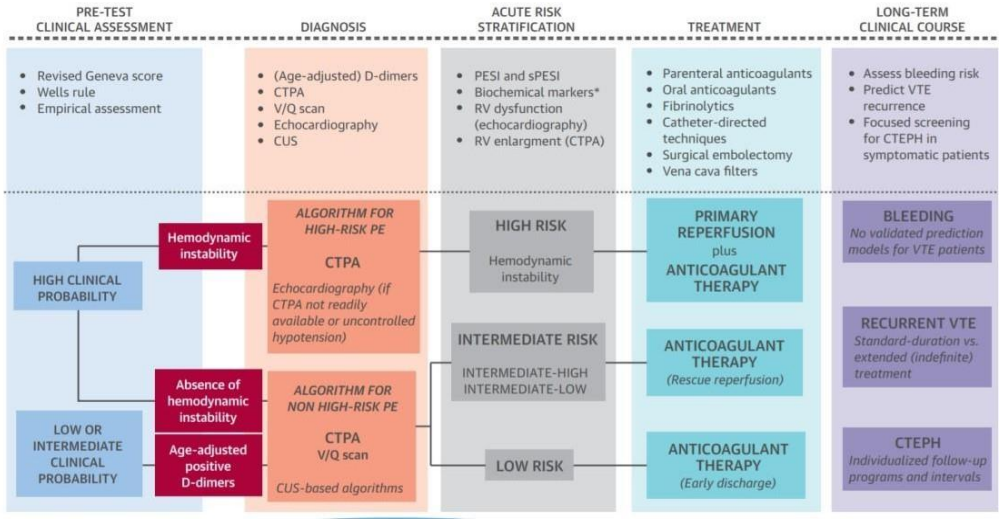
1. Acute massive pulmonary thromboembolism (high-risk pulmonary embolism, systolic blood pressure <90 mmHg, or a decrease in systolic arterial pressure of at least 40 mmHg for at least 15 min)

2. Acute sub-massive pulmonary thromboembolism (intermediate-high risk, normal blood pressure with RV dysfunction)



B. IVC filter

C. VA ECMO



Adapted from Management of Pulmonary Embolism An Update Stavros V. Konstantinides

7.5.3 Cardiac Tamponade:

Acute circulatory failure due to the compression of the cardiac chambers by the pericardial effusion.

Diagnosis:

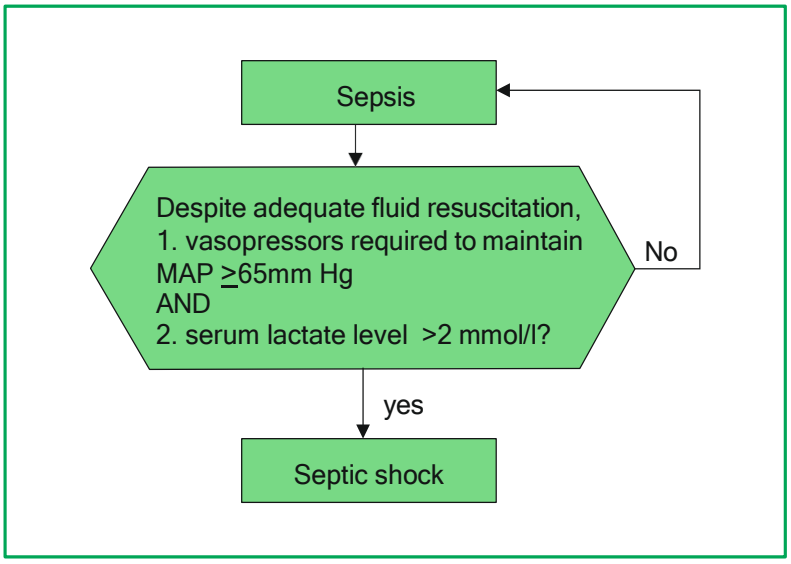
Signs & Symptoms: Dyspnoea at rest and with exertion, tachycardia, narrow pulse pressure,neck vein engorgement. Pulsus paradoxus. Chest radiography: Enlarged heart silhouette and epicardial fat-pad sign ECG: Low-voltage QRS complexes, electrical alternans. TREATMENT: Pericardiocentesis

7.6. Distributive Shock:

7.6.1. Septic shock:

Intravascular volume depletion Cardiac dysfunction

Peripheral vasodilation



Treatment

Fluid for resuscitation • Crystalloids • Synthetic colloids: Increased mortality & AKI, Do Not USE • Assess responsiveness: dynamic parameters better Antibiotics:

- Appropriate broad-spectrum antibiotics started as soon as possible (preferably after sending culture & sensitivity but don't delay antibiotics)

Vasoactive agents:

- Non responsive to Fluid resuscitation
- Noradrenaline preferred • Dopamine: ONLY if relative bradycardia present

- MAP target: 65mmHg • Add on VASOPRESSIN (1-2u/Hr) AS THE SECOND AGENT
- Steroid supplementation when on increasing dose of vasopressin. HC 200mg over 24hrs or 50mg 6 Hourly
- Stop steroids once resolution of shock

7.8 Anaphylaxis

Anaphylaxis is an acute, potentially lethal, multisystem syndrome resulting from the sudden release of mast cell, basophil, and macrophage-derived mediators into the circulation

7.8.1 Basic initial mangement

1. Remove exposure to the trigger, if possible eg: discontinue intravenous diagnostic or therapeutic agent that seems to be triggering symptoms.
2. Assess circulation airway, breathing, mental status, skin and body weight
3. Call for help
4. Inject Epinephrine IM in mid-anterolateral aspect of thigh 0.01 mg/kg of 1:1000 solution, to a maximum of 0.5 mg; repeat in 515min
5. Place patient on back, or in a position of comfort if there is respiratory distress and or vomiting, elevate the lower extremities; fatality can occur within seconds if the patient stands or sits suddenly
6. Supplementary oxygen (6-8LPM) by face mask
7. Establish intravenous access with wide bore cannula. When indicated give 1-2L of 0.9% saline rapidly
8. When indicated at any time prepare to initiate cardiopulmonary resuscitation with continuous chest compressions
9. Tryptase and compliment can help in diagnosis Airway management:
    - Rapid assessment.
    - Intubation in patients with developing airway compromise Early intubation to be considered if significant edema of tongue, uvula, or voice alteration has developed.

7.8.2. Epinephrine:

- First-line use of epinephrine is the standard of care for anaphylaxis.
- Dose: intramuscular route in the mid-anterolateral thigh as soon as anaphylaxis is diagnosed or strongly suspected, in a dose of 0.01 mg/kg of a 1:1000(1 mg/mL) solution, to a maximum of 0.5 mg in adults. Dose can be repeated every 5–15 min, as needed.
- Epinephrine can be given by slow intravenous infusion with diluted solution 1:10,000 (0.1 mg/mL), with the dose titrated according to non-invasive continuous monitoring of cardiac rate and function. (if shock is imminent or has already developed or cardiac arrest is impending, an intravenous bolus dose of epinephrine is indicated; however, in other anaphylaxis scenarios, this route of administration should be avoided)
- All patients with orthostasis, hypotension, or incomplete response to epinephrine should receive fluid resuscitation with isotonic saline.

Oxygen therapy

H1-antihistamines + H2-antihistamine GLUCOCORTICOIDS:

- Glucocorticoid is not lifesaving <u>in initial hours</u> of an anaphylactic episode. They have a delayed onset of 4 to 6 hours. Play a role in preventing rebound anaphylaxis Current systematic review failed to identify any evidence to confirm the effectiveness of glucocorticoids in the treatment of anaphylaxis, and raised concerns that they are often inappropriately used as firstline medications in place of epinephrine.

BRONCHODILATORS:

- · Should not be substituted for epinephrine because they have minimal alpha-1 adrenergic agonist vasoconstrictor effects and do not prevent or relieve laryngeal edema and upper airway obstruction, hypotension, or shock.

Medications:

First line:

- Epinephrine 1:1000 IM max 0.5mg • Fluids • Oxygen Second line:

- H1 antihistamine iv Chlorpheniramine 10 mg • Beta 2 adrenergic agonist-Salbutamol

  2.5mg/3ml via nebuliser • Glucocorticoid iv Hydrocortisone 200 mg or methyl prednisolone 50-

  100 mg • H2 antihistamine iv Ranitidine 50mg

- REFRACTORY ANAPHYLAXIS:
- Adrenaline • Norepinephrine • Vasopressin
- ECMO

Recurrent anaphylaxis:

- Epinephrine auto injector.

8 Cardiac Arrest In Adults

AHA ACC 2015 GUIDELINES • Early CPCR

- No interruptions
- C-A-B
- Early defibrillation for shockable rhythms After return of "spontaneous circulation", try to identify cause of arrest and treat Surveillance for and prevention of in hospital cardiac arrest.

Chain Of Survival

When to start CPCR • Unresponsiveness

- NOT BREATHING/GASPING
- NO DEFINITE PULSE (Pulse check not >10 seconds)

Activate emergency medical service once cardiac arrest is identified steps of CPCR:

C-A-B

C: COMPRESSIONS-Rate:100 to 120/min. Depth of at least 2 inches (5 cm) for an average adult, while avoiding excessive chest compression depths (greater than 2.4 inches [6 cm]). It is reasonable for rescuers to avoid leaning on the chest between compressions, to allow full chest wall recoil for adults in cardiac arrest.

Rescuers Should	Rescuers should not
Perform chest compressions at 100-120/min	Compress at a rate slower than 100/min or faster than 120/min
Compress to a depth of 5-6cms	Compress to a depth of less than 2 inches (5cms) or greater than 2.4 inches (6cms)
Allow full recoil after each compression	Lean on the chest between compressions
Minimize pauses in compressions	Interrupt compressions for greater than 10 seconds
Ventilate adequately (2 breaths after 30 compressions, each delivered over 1 second, each causing chest rise)	Provide excessive ventilation (i.e too many breaths or breaths with excessive force)

Note:

For witnessed adult cardiac arrest when an AED is immediately available, it is reasonable that the defibrillator be used as soon as possible. For adults with unmonitored cardiac arrest or for whom an AED is not immediately available, it is reasonable that CPR be initiated while the defibrillator equipment is being retrieved and applied and that defibrillation, if indicated, be attempted as soon as the device is ready for use.

A: AIRWAY

OPEN UP THE AIRWAY:

HEAD TILT CHIN LIFT OPEN MOUTH JAW THRUST

OROPHANRYNGEAL/NASOPHARYNGEAL AIRWAY

LMA/COPA/CRICITHYROIDOTOMY/TRACHEOSTOMY

B: BREATHING-RESCUE BREATHS

Rate: COMPRESSION RELAXATION RATIO: 30:2

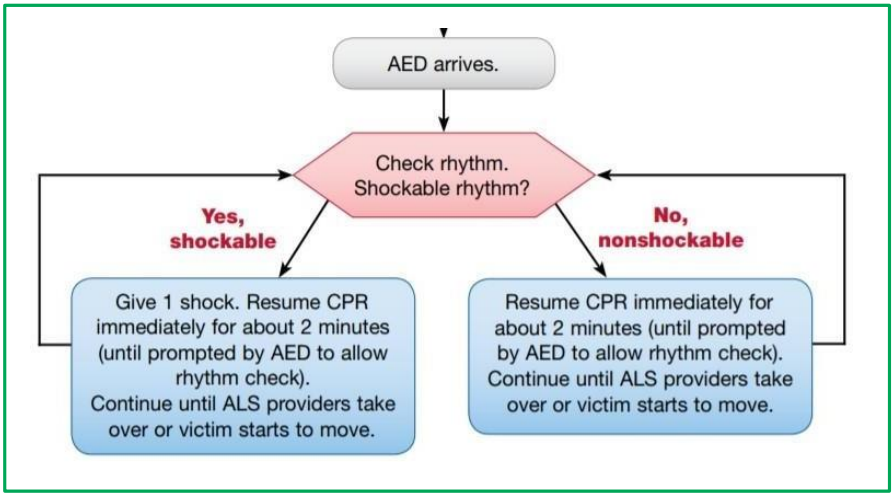
Rate: WHEN ADVANCED AIRWAY IN SITU: 10 BREATHS /MIN

TIDAL VOLUME: ENOUGH VOLUME SO AS TO GET A VISIBLE CHEST RISE ADRENALINE:

ALONG with 2nd CYCLE OR EARLIER DOSE: 1mg

EVERY 3-5minutes DEFIBRILLATION:

WHEN DEFIBRILATOR ARRIVES: Check rhythm



Shockable rhythms:

- Ventricular Fibrillation, Pulseless Ventricular Tachycardia
- ENERGY for FIRST Shock (Biphasic): DEFAULT ENERGY:120-200
- (usually marked on the dial used to select energy)
- SOON AFTER SHOCK START CPCR

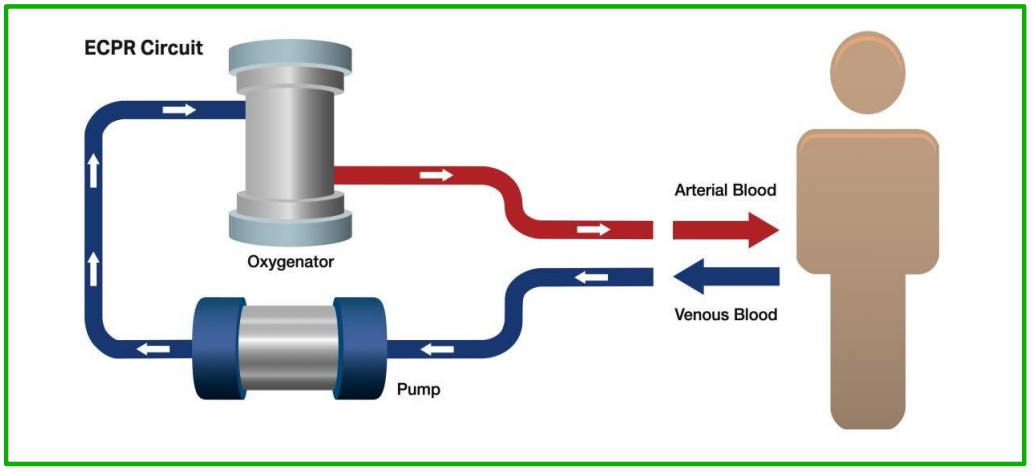
- AFTER 5 CYCLES OF 30:2 look for rhythm
- ENERGY FOR SECOND SHOCK MAY BE HIGHER/SAME AS THE FIRST
- Along with the third shock consider: Inj. Amiodarone for VF/pulseless VT NOT responding to 3 shocks

Non-Shockable rhythm: pulseless electrical activity and asystole: • Start

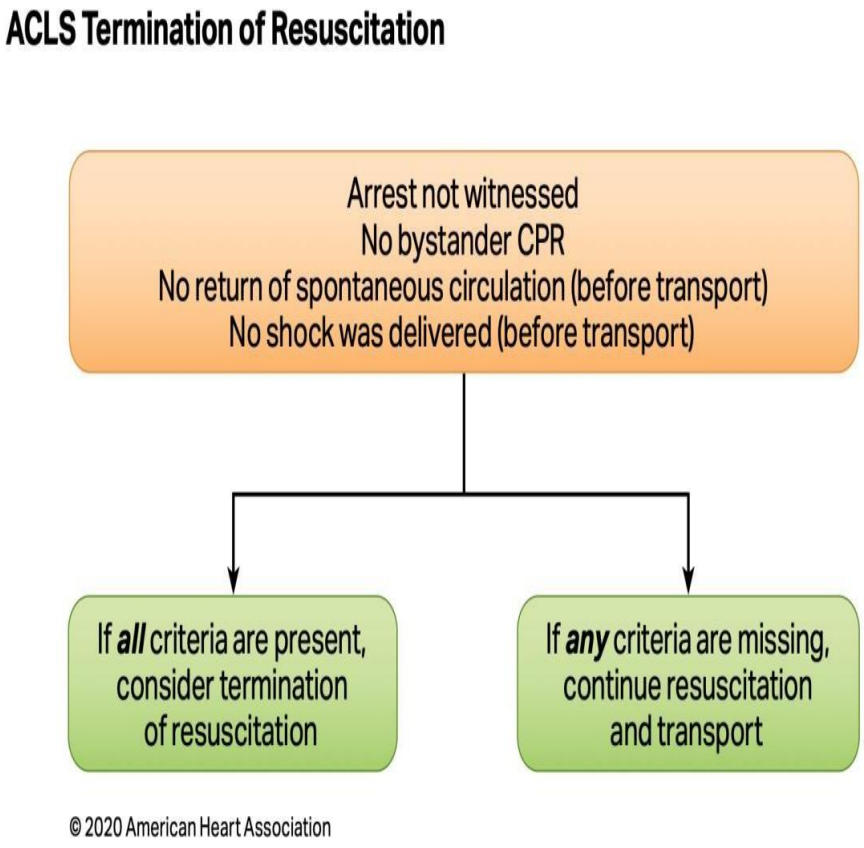
CPCR, Inj. Adrenaline.

- Check for 5Hs & 5Ts, continue CPCR
- Hs: Hypovolemia, Hypoxia, H+ ion excess, Hyper/Hypokalemia,
- Hypothermia • Ts: Tension Pneumothorax, Thrombosis-Coronary, Thrombosis-Pulmonary, Toxin, Tamponade-cardiac
- Check for return of spontaneous circulation (ROSC) during rhythm analysis Extra corporeal CPCR:

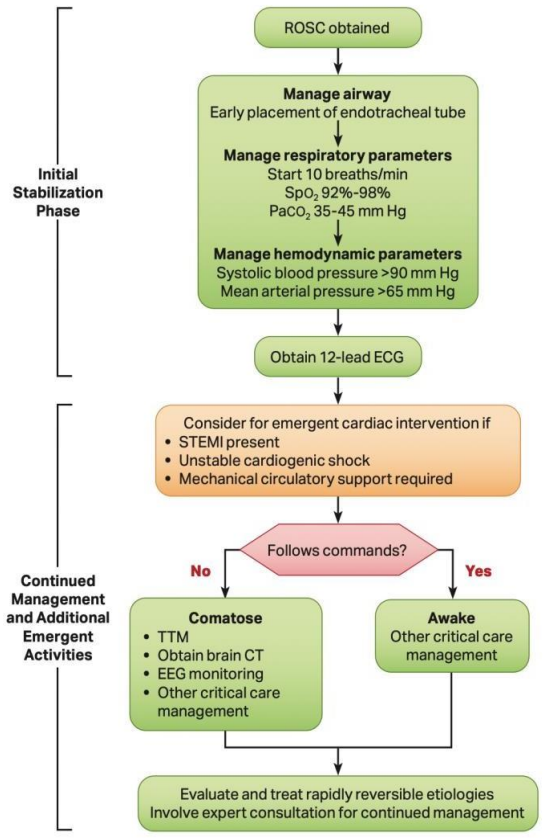
Insufficient evidence to recommend the routine use of ECPR for patients with cardiac arrest. In setting where it can be rapidly implemented, ECPR may be considered for select cardiac arrest patients for whom the suspected etiology of the cardiac arrest is potentially reversible during a limited period of mechanical cardiorespiratory support.



Stop CPCR: ROSC/Treating physician decides to stop taking into consideration many factors.



Post cardiac arrest care:



Initial Stabilization Phase

Resuscitation is ongoing during the post-ROSC phase, and many of these activities can occur concurrently. However, if prioritization is necessary, follow these steps:

- Airway management: Waveform capnography or capnometry to confirm and monitor endotracheal tube placement
- Manage respiratory parameters: Titrate FIO_2 for SpO_2 92%-98%; start at 10 breaths/min; titrate to $PaCO_2$ of 35-45 mm Hg
- Manage hemodynamic parameters: Administer crystalloid and/or vasopressor or inotrope for goal systolic blood pressure >90 mm Hg or mean arterial pressure >65 mm Hg

Continued Management and Additional Emergent Activities

These evaluations should be done concurrently so that decisions on targeted temperature management (TTM) receive high priority as cardiac interventions.

- Emergent cardiac intervention: Early evaluation of 12-lead electrocardiogram (ECG); consider hemodynamics for decision on cardiac intervention
- TTM: If patient is not following commands, start TTM as soon as possible; begin at 32-36°C for 24 hours by using a cooling device with feedback loop
- Other critical care management
  - Continuously monitor core temperature (esophageal, rectal, bladder)
  - Maintain normoxia, normocapnia, euglycemia
  - Provide continuous or intermittent electroencephalogram (EEG) monitoring
  - Provide lung-protective ventilation

H's and T's

Hypovolemia
Hypoxia
Hydrogen ion (acidosis)
Hypokalemia/**h**yperkalemia
Hypothermia
Tension pneumothorax
Tamponade, cardiac
Toxins
Thrombosis, pulmonary
Thrombosis, coronary

Components of Post–Cardiac Arrest Care	Check
Oxygenation and ventilation	
Measure oxygenation and target normoxemia 94%-99% (or child's normal/appropriate oxygen saturation).	☐
Measure and target $Paco_2$ appropriate to the patient's underlying condition and limit exposure to severe hypercapnia or hypocapnia.	☐
Hemodynamic monitoring	
Set specific hemodynamic goals during post–cardiac arrest care and review daily.	☐
Monitor with cardiac telemetry.	☐
Monitor arterial blood pressure.	☐
Monitor serum lactate, urine output, and central venous oxygen saturation to help guide therapies.	☐
Use parenteral fluid bolus with or without inotropes or vasopressors to maintain a systolic blood pressure greater than the fifth percentile for age and sex.	☐
Targeted temperature management (TTM)	
Measure and continuously monitor core temperature.	☐
Prevent and treat fever immediately after arrest and during rewarming.	☐
If patient is comatose apply TTM (32°C-34°C) followed by (36°C-37.5°C) or only TTM (36°C-37.5°C).	☐
Prevent shivering.	☐
Monitor blood pressure and treat hypotension during rewarming.	☐
Neuromonitoring	
If patient has encephalopathy and resources are available, monitor with continuous electroencephalogram.	☐
Treat seizures.	☐
Consider early brain imaging to diagnose treatable causes of cardiac arrest.	☐
Electrolytes and glucose	
Measure blood glucose and avoid hypoglycemia.	☐
Maintain electrolytes within normal ranges to avoid possible life-threatening arrhythmias.	☐
Sedation	
Treat with sedatives and anxiolytics.	☐
Prognosis	
Always consider multiple modalities (clinical and other) over any single predictive factor.	☐
Remember that assessments may be modified by TTM or induced hypothermia.	☐
Consider electroencephalogram in conjunction with other factors within the first 7 days after cardiac arrest.	☐
Consider neuroimaging such as magnetic resonance imaging during the first 7 days.	☐

12 Lead ECG angiogram:

- CAG should be performed emergently (rather than later in the hospital stay or not at all) for OHCA patients with suspected cardiac aetiology of arrest and ST elevation on ECG
- Emergency CAG is REASONABLE FOR SELECT (electrically or hemodynamically

unstable) adult patient who are comatose after OHCA of suspected cardiac origin but without ST elevation on ECG.

- CAG is reasonable in post cardiac arrest patients for whom CAG is indicated regardless of whether the patient is comatose or awake

Hemodynamic goals:

- Avoiding and immediately correcting hypotension (Systolic BP<90, MAP<65mmHg) during post resuscitation care may be reasonable.

  TARGETED TEMPERATURE MANAGEMENT: • Comatose adult patients with ROSC after cardiac arrest have TTM.

- Constant temperature between 32- 36^0C during TTM • NO routine prehospital cooling
- ACTIVELY Prevent fever in COMATOSE patients after TTM • Rewarming rate: 0.5 / hour SEIZURES:
- An EEG for the diagnosis of seizure should be promptly performed and interpreted and then should be monitored frequently or continuously in comatose patients after ROSC

Ventilation:

- Maintain $PaCO_2$ within physiological range<taking into account any temperature correction may be reasonable. OXYGENATION: SpO_2 94% OR greater <u>CPCR IN PREGNANCY:</u>

Position: Manual left uterine displacement

EVACUATION OF GRAVID UTERUS: perimortem cesarean delivery IN LATER HALF OF PREGNANCY if not achieving ROSC with usual resuscitation and LUD. Considered at 4minute of arrest CARDIAC ARREST ASSOCITED WITH LOCAL ANESTHETIC TOXICTY:

It may be reasonable to administer intra venous lipid emulsion concomitant with standard resuscitative care, to patients with local anaesthetic systemic toxicity and particularly to patients who have premonitory neurotoxicity or cardiac arrest due to bupivacaine toxicity

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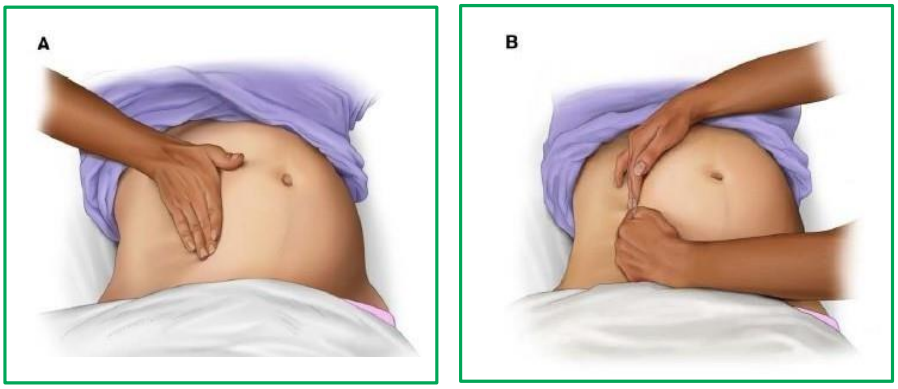
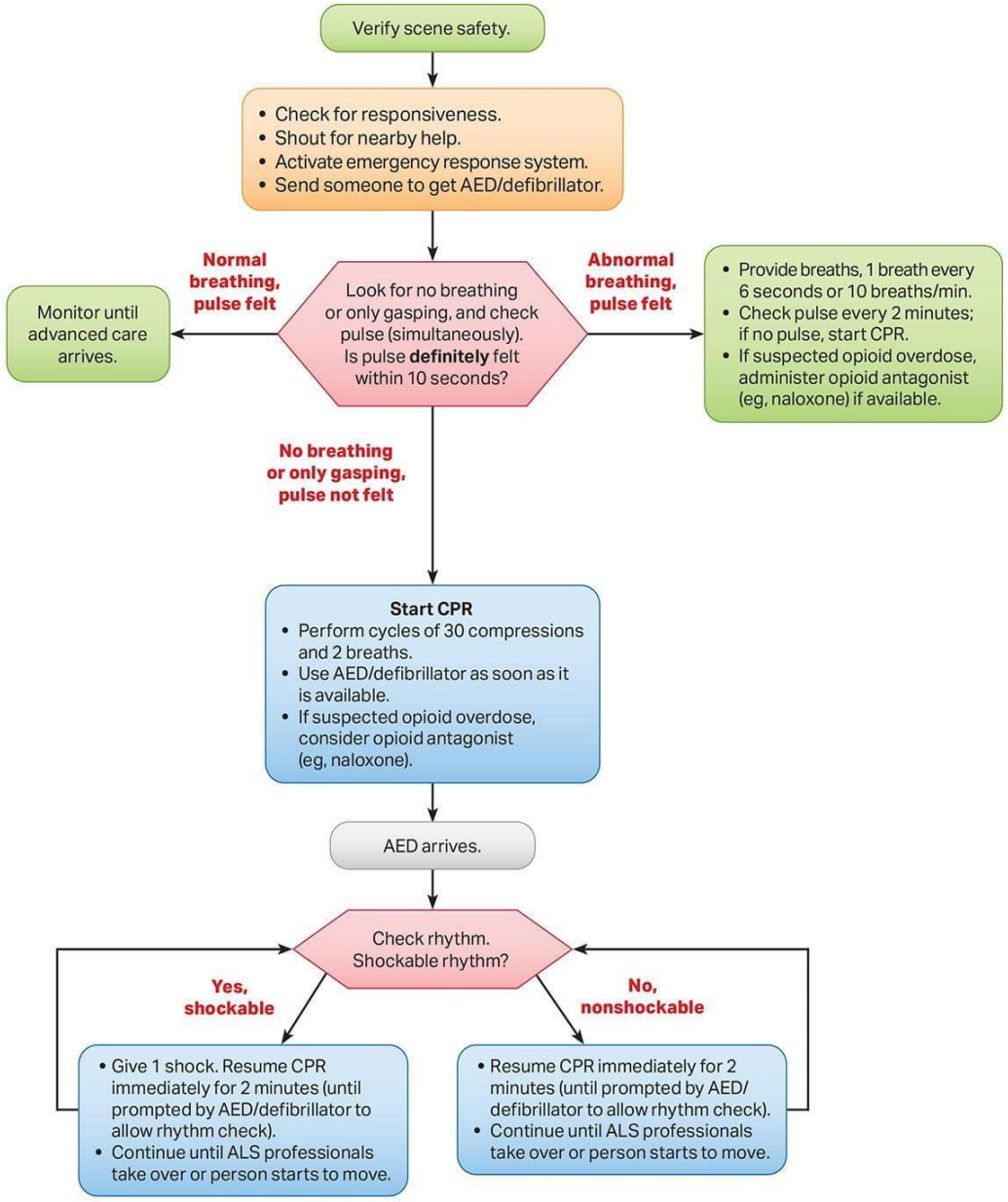


Cardiac arrest associted with local anesthetic toxicty:

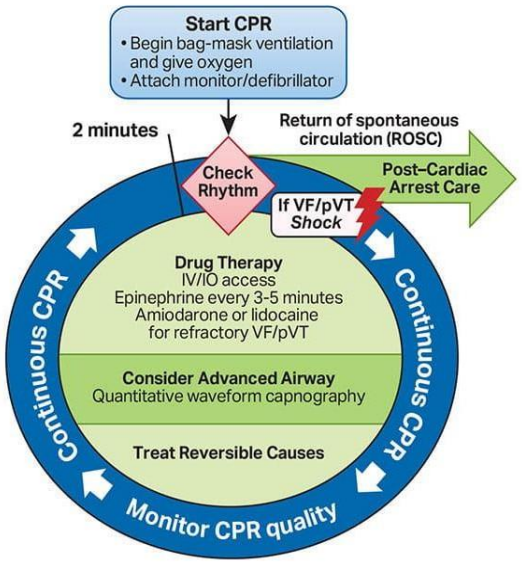
It may be reasonable to administer intra venous lipid emulsion concomitant with standard resuscitative care, to patients with local anesthetic systemic toxicity and particularly to patients who have premonitory neurotoxicity or cardiac arrest due to bupivacaine toxicity.

Adult* Basic Life Support Algorithm for Health Care Professionals



*If signs of puberty, treat as adult.

Adult Cardiac Arrest Circular Algorithm



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High-Quality CPR

- Push hard (at least 2 inches [5 cm]).
- Push fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- If advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions.
- Continuous waveform capnography
  - If $ETCO_2$ is low or decreasing, reassess CPR quality.

Shock Energy for Defibrillation

- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.
  or
- **Lidocaine IV/IO dose:** First dose: 1-1.5 mg/kg. Second dose: 0.5-0.75 mg/kg.

Advanced Airway

- ET intubation or supraglottic advanced airway
- Continuous waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

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9 Fluid management in ICU

Fluids are administered in ICU to optimize intravascular volume with an aim to improve preload and cardiac output. Fluids can be broadly divided into crystalloids & colloids. Crystalloids can be divided in isotonic solutions and balanced (buffered) solutions.

Isotonic solutions include normal saline, ringer lactate. Plasmalyte is an example of balanced salt solution. Colloids include albumin, starch, dextran and gelatin. Choice between crystalloids and colloids in intensive care has been a topic of debate for long. Studies comparing albumin and other colloids vs crystalloids yielded variable results.

The SAFE trial (Saline vs Albumin fluid evaluation), multi-centre randomized, double blind trial which enrolled 6997 patients concluded that the use of either 4 % albumin or normal saline resulted in similar outcomes at 28 days. In subgroup analysis of patients with severe sepsis there was a trend of improved survival in those treated with albumin. Similarly, the CRISTAL study (colloid Vs crystalloid in critically ill) examined outcomes for ICU patients with shock from sepsis, trauma, or hypovolemia who were randomized to treatment with colloid solution compared with crystalloids. In addition to 4% and 20% albumin, the colloid arm of CRISTAL included gelatin, dextrans, and HES. The study concluded that in ICU patients with hypovolemia, the use of colloids vs crystalloids did not result in significant difference in 28 day mortality although 90 day mortality was lower among those who received colloids.

From the available evidence it can be concluded that hydroxyethyl starch should not be used as a resuscitative or maintenance fluid in critically ill patients.

9.1 Albumin in Critically ill

Albumin infusion has been shown to prevent AKI in situations like Cirrhosis & SBP - The dose of albumin administered was 1.5 g/kg at diagnosis, with another 1 g/kg infused on day 3. Cirrhosis, Large volume paracentesis. Treatment of hepatorenal syndrome. Used in combination with noradrenaline, terlipressin or midodrine & octreotide and in ovarian hyperstimulation syndrome. Albumin administration also aids fluid removal during renal replacement therapy. Albumin can also be used in severe burns when large volumes of fluids are required. It has shown to decrease the requirement of crystalloid without any adverse effects. Albumin can be used as an adjunct to furosemide in those with hypoalbuminemia as it helps to maintain oncotic pressure thereby potentiating the effect of furosemide. Therapeutic plasma exchange — Dose to be titrated to plasma removed. There is no indication for use of albumin to correct hypoalbuminemia. In the majority of cases that require volume resuscitation in the ICU, there does not seem to be robust evidence favouring albumin or other colloids over crystalloid and crystalloids remain the first line treatment of choice in most settings

Dextrans are a mixture of glucose polymers and are approved for use in vascular surgery due to their rheologic properties especially after grafting. Dextran should not be used as a resuscitative or maintenance fluid due to concerns on coagulation, renal dysfunction and anaphylaxis.

9.2 Crystalloids

Normal saline is the most commonly used crystalloid in ICU. Infusion of large volume of normal saline can lead to hyperchloremic metabolic acidosis and renal vasoconstriction.

Among critically ill patients there is some evidence to show that use of balanced salt solution was associated with lower in hospital mortality.

The SMART trial included 15802 patients who were randomized to receive either NS versus RL or Plasma-Lyte. The cohorts were well matched for ICU admission diagnosis, vasopressor support, and baseline kidney function. There were significantly fewer adverse

kidney outcomes in the RL group compared with NS (P 5 0.04). There was also a trend toward less incidence of renal dysfunction in the RL group. In the subgroup analysis of patient with sepsis. there was a significant improvement for in-hospital 30-day mortality, when patients

received balanced fluids versus NS, and the results were also favourable in terms of 30-day mortality, adverse kidney events, and vasopressor use

The BaSICS trial (2021) in more than 11000 patients and PLUS trial (2022) with more than 5000 pts compared balanced solution vs 0.9% saline and concluded that there is no difference in 90-day mortality or adverse kidney events between the groups.

A metanalysis by Naomi et al, NEJM (2022) reviewed 13 RCTS with more than 35000 patients comparing balanced salt solution with saline in critically ill patients and concluded that using balanced solution has mortality benefit. (RR -0.96)

Patients with traumatic brain injuries has a theoretical risk of increased intracranial pressure by infusion of balanced crystalloid. Hence, it's should be avoided in TBI until further studies are conducted.

9.3 Hypotonic Solutions.

Hypotonic solutions like 5%dectrose, 0.45% NS and 5%DW and they provide free water to restore intracellular fluid deficits. Common indications for hypotonic solution are DKA, correction of hypernatremia and hyperosmolar hyperglycaemic states. Hypotonic fluids should not be used for resuscitation as they have little intravascular retention.

9.4 How much fluid?

Evidence supports that fluid resuscitation resulting in volume overload is not beneficial to critically ill patients and negatively affects clinical outcomes.

Vasopressin and Septic Shock Trial (VASST) concluded that high positive fluid balance was significantly associated with an increase in mortality.

Higher cumulative balance is also associated with ALI & ARDS. On the other side restrictive regimen with negative balance predispose to AKI. Hence titrating fluids to achieve patient specific hemodynamic goals is important.

9.5 Conclusion

The effect and adverse effect of fluid administration depends on the type and volume used. Fluid overload is associated with increased mortality. With the exception of TBI or severe hypochloraemia, ICU patients should receive crystalloids preferably balanced solution for resuscitation and subsequent fluid therapy. In TBI for volume expansion isotonic saline should be preferred over balanced solutions. In patients with cirrhosis requiring volume

expansion, albumin may have a benefit over crystalloids. Critically ill patients require individualized fluid types, volumes, infusion rates tailored to goals based on their underlying medical condition.

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10 Hemodynamic Monitoring in ICU Patients

Hemodynamic monitoring is essential in managing critically ill patients, providing real-time data on cardiovascular performance. This process involves assessing parameters like preload, afterload, cardiac output, and oxygen delivery to optimize interventions. [1]

Objectives:[2]

1. To maintain adequate tissue perfusion.
2. To guide fluid therapy and vasopressor/inotropic support.
3. To evaluate the effectiveness of therapeutic interventions.

Key Parameters

1. Preload: [3–6]

   Definition: Ventricular filling pressure at the end of diastole. Measurement:
   Central Venous Pressure (CVP). IVC diameter
   Pulmonary Artery Wedge Pressure (PAWP).

   Clinical Use: Guides fluid therapy in hypovolemia or sepsis.

2. Afterload:

   Definition: Resistance against which the heart must pump. Measurement:
   Systemic Vascular Resistance (SVR). Pulmonary Vascular Resistance (PVR).
   Clinical Use: Indicates vasoconstriction or vasodilation.

3. Cardiac Output (CO):

   Definition: Volume of blood pumped by the heart per minute. Measurement: Pulmonary Thermodilution (via Pulmonary Artery Catheter). Transpulmonary thermodilution

   Pulse Contour Analysis.

   Clinical Use: Evaluates cardiac performance in shock states.

4. Oxygen Delivery (DO_2):

   Definition: Amount of oxygen delivered to tissues.

   Measurement: Mixed Venous Oxygen Saturation (SvO_2) or Central Venous Oxygen Saturation ($ScVO_2$).

Interpretation of Data

1. Low CVP: Indicates hypovolemia; initiates fluid resuscitation.
2. High PAWP: Suggests left ventricular failure; consider diuretics or inotropes.
3. Low CO: Reflects cardiogenic shock; requires inotropic support.
4. Low SvO_2: Indicates inadequate oxygen delivery or increased consumption.

Basic parameters

Parameter	Normal Range	Purpose
Heart rate	60-100 / min	Basic cardiac function
Blood pressure	Systolic BP 90-140 mmHg	MAP > 65 mmHg ensures organ perfusion
Cardiac output	4-8 L/ min	Function of heart
Stroke volume	60-100ml/ beat	Volume of blood ejected per beat
CVP	4-12 mmHg	Approximation of right atrial pressure

Pulmonary Capillary wedge pressure	6-12 mmHg	Left ventricular preload indicator
Mixed venous Oxygen saturation SvO2	60-80 %	Balance between oxygen delivery and demand
Lactate	<2 mmol/ L	Indicator of tissue hypoperfusion

Types of Hemodynamic Monitoring Non-Invasive Monitoring [1]

1. Advantages: Lower risk of infection, Suitable for less severe conditions.
2. Limitations: Reduced accuracy in unstable patients.

Basic Non-invasive Monitoring [7]

Heart Rate (HR): Monitored via ECG.

Blood Pressure (BP): Using a cuff sphygmomanometer or Oscillo metric device.

Oxygen Saturation (SpO_2): Pulse oximetry to assess oxygenation. Respiratory Rate (RR): Monitored via visual observation or device sensors.

Advanced Non-Invasive Monitoring (8)

1. Impedance Cardiography (ICG): Measures stroke volume (SV), Cardiac output (CO), Systemic vascular resistance (SVR).
2. Doppler Ultrasound: Trans-oesophageal Doppler or transthoracic echocardiography to measure CO, SV, and fluid responsiveness.
3. Near-Infrared Spectroscopy (NIRS): Assesses regional tissue oxygenation (e.g., cerebral perfusion).
4. Volume clamp devices:
    1. Clear sight
    2. CNAP
    3. Nexfin

Advantages:

Totally non invasive

Can be used in arrhythmia

Can be used in non-ventilated patients

Disadvantages:

Inaccurate when vasopressors used (Error ~50%)

Minimally Invasive Monitoring

Utilize 4 main principles for cardiac output monitoring

1. Pulse contour analysis – non-calibrated 2.Echocardiogram and Oesophageal Doppler
3. Partial carbon dioxide rebreathing with application of Fick`s principle
4. Electrical bioimpedance/bioreactance

1. Arterial pressure monitoring

   Indications [9]

   Continuous BP measurements, beat to beat variation.

   Need for frequent ABG / blood sampling When NIBP is not reliable or possible SITE

   Most commonly radial artery

   Followed by Femoral artery, Dorsalis pedis, Brachial artery, Axillary artery

   Avoid ulnar artery due to its proximity to ulnar nerve & potential for hand ischemia

   Contraindications

   Local infections

   Lack of collateral flow - Use modified Allens test Lymphatic disruption at site

   Arterial insufficiency at site of insertion

Flash flush test / square wave test / dynamic response test is recommended to avoid over dampening and under dampening and it should be done [10]

every 8-12 hours

After opening of system

When measurement is doubtful

- Heparinized solutions for flushing have advantage on maintaining catheter patency, but higher chances for development of HIT. [11]

Transducer should be kept in phlebostatic axis.

In all patients in shock, arterial blood pressure should be monitored invasively. Target MAP of 65–70 mm Hg. [12–14]

2. Arterial pressure waveform analysis:

Devices: FloTrac, Clear Sight.

Parameters: CO, stroke volume variation (SVV), pulse pressure variation (PPV).
Indications: Fluid responsiveness, perioperative care.

FloTrac System [15]

Manufacturer: Edwards Lifesciences.

Principle: FloTrac uses arterial waveform analysis derived from an existing arterial catheter to calculate cardiac output. It relies on pulse contour analysis without requiring external calibration.

Minimal invasiveness (requires only an arterial line).

Provides dynamic parameters like stroke volume variation (SVV) and pulse pressure variation (PPV), which are useful for fluid responsiveness assessment.

Advantages

Easy setup and no need for external calibration. Continuous, real-time hemodynamic data.

Low risk of complications due to minimal invasiveness.

Limitations

Accuracy may be affected by arrhythmias, vasopressor use, or significant changes in vascular tone. Assumptions in the algorithm may lead to inaccuracies in some patient populations.

LiDCO System [16,17]

Manufacturer: LiDCO Ltd.

Principle: LiDCO combines pulse power analysis with lithium dilution calibration to monitor cardiac output. It can work with or without calibration, depending on the model.

Key Features:

Lithium dilution provides a baseline calibration of cardiac output.

Continuous real-time cardiac output monitoring through pulse contour analysis. Integration with LiDCO view software for data visualization and analysis.

Advantages

Calibration ensures higher accuracy compared to uncalibrated systems. Portable and easy to use with existing arterial catheters.

Provides dynamic parameters like stroke volume and SVV.

Limitations

Requires arterial access and, optionally, a lithium injection.

Lithium calibration contraindicated in patients on lithium therapy or with hypersensitivity.

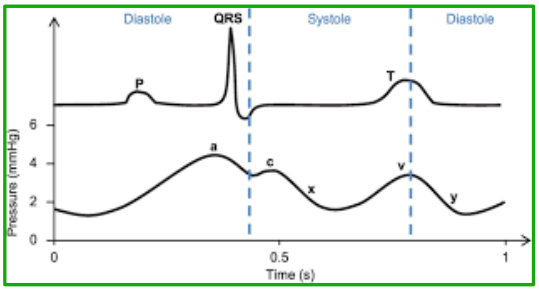
3. Central venous pressure monitoring

Indications [18]

Measurement of CVP

Surrogate marker of cardiac preload

Measurement of central mixed venous oxygenation Use for CVP wave analysis



The CVP waveform has 3 positive and 2 negative deflections. [19]

"a" wave = atrial contraction.

"c" wave = closure + bowing of tricuspid valve (TV) into atrium (ventricle contracts). "x descent" = atrial relaxation + change in ventricular geometry.

"v" wave = venous return + atrial filling; TV closed. "y descent" = opening of TV + atrial emptying.

CVP waveform anomalies [20,21] Absence of a wave- atrial fibrillation Cannon a wave - AV dissociation Tricuspid regurgitation- tall V waves Tricuspid stenosis- tall a waves Cardiac tamponade-loss of y descent

CVP does not accurately predict fluid responsiveness. [23] It has a poor correlation with blood volume. Impaired RV function, severe pulmonary diseases, valvular heart diseases affect CVP reading

4. Electrical bioimpedance in hemodynamic monitoring[25,26]

Bioimpedance technology measures cardiac output and other hemodynamic parameters by analyzing the electrical conductivity of thoracic tissues.

Principle of Bioimpedance

Bioimpedance is based on the electrical properties of tissues: The thorax conducts electrical currents differently depending on the volume of blood and fluid. Changes

in thoracic impedance during the cardiac cycle correspond to stroke volume.

How It Works

1. Electrodes Placement: Electrodes are placed on the chest to deliver and measure electrical signals.
2. Baseline Impedance: Establishes the static thoracic impedance (Z_0).
3. Dynamic Changes: Measures impedance variations during each heartbeat.
4. Cardiac Output Calculation: Derived using mathematical models incorporating stroke volume and heart rate.

Advantages

Non-invasive and easy to apply. Portable and low cost.

Continuous monitoring without arterial or central venous access.

Limitations

Accuracy reduced in conditions affecting thoracic impedance (e.g., oedema, obesity, lung disease). Motion artifacts and electrode misplacement can interfere with measurements.

5. Pulse wave transit time in hemodynamic monitoring[27]

Pulse wave transit time (PWTT) is the time interval between the onset of electrical systole (measured by the R-wave on an ECG) and the arrival of the pulse wave at a peripheral site (e.g., measured by a plethysmographic sensor).

Concept:

PWTT reflects arterial stiffness and vascular tone.

It can provide insights into cardiac output and systemic vascular resistance when correlated with other hemodynamic parameters.

A shorter PWTT often indicates higher blood pressure and increased vascular tone, while longer PWTT suggests lower blood pressure.

Applications:

Non-invasive, continuous monitoring of changes in hemodynamic status.

Limitations: Affected by factors like arterial stiffness, age, and baseline cardiovascular status.Not as accurate as invasive techniques in critically ill patients.

Invasive monitoring

Utilize the principles of pulse contour analysis and transpulmonary or pulmonary thermodilution: Calibrated system

1. Pulmonary Artery Catheter (PAC; Swan-Ganz) Measures:

   Pulmonary artery pressure (PAP).

   Pulmonary capillary wedge pressure (PCWP). Mixed venous oxygen saturation (SvO_2).

   Cardiac output (via pulmonary thermodilution).

   Indications: Severe cardiogenic shock, complex fluid management, right heart failure.

   Pulmonary Thermodilution for Assessing Cardiac Output [28]

   It involves the injection of a cold or room-temperature fluid bolus into the right atrium and the measurement of temperature changes in the pulmonary artery.

   Procedure:

   A pulmonary artery catheter (PAC) is used.

   A known volume of cold saline or dextrose is injected into the right atrium.

   The thermistor at the catheter's tip in the pulmonary artery measures the downstream temperature change.

   Cardiac output is calculated based on the thermodilution curve using the Stewart-Hamilton equation. Advantages:

   Allows simultaneous measurement of other parameters, like pulmonary

artery pressure and mixed venous oxygen saturation (SvO2).

Limitations:

Invasive, requiring expertise for catheter insertion.

Potential complications like arrhythmias, infections, or pulmonary artery rupture.

2. Transpulmonary Thermodilution (PiCCO/Volume view)

The PiCCO (Pulse Contour Cardiac Output) system is a invasive hemodynamic monitoring technique that uses transpulmonary thermodilution and pulse contour analysis to assess cardiac output (CO) and other derived hemodynamic parameters.

Advantages of PiCCO [29]

1. Less invasive compared to pulmonary artery catheterization.
2. Provides both preload (GEDV) and extravascular lung water (EVLW) measurements.
3. Continuous monitoring of CO using pulse contour analysis.
4. Measures both volumetric and pressure-derived parameters.
5. Useful for differentiating types of shock (hypovolemic, distributive, cardiogenic).

Limitations

1. Requires central venous and arterial catheters.
2. Less accurate in patients with arrhythmias or significant valvular disease.
3. Limited application in patients with poor thermodilution signals (e.g., severe pulmonary embolism) 4.Infection, Thrombosis, Vascular injury.
5. Calibration must be repeated periodically to maintain accuracy.
6. Cannot measure mixed venous oxygen saturation (SvO_2), unlike a PAC.

Parameter	Range	Unit
Cardiac index	3.0-5.0	$L/min/m^2$
Stroke volume index	40-60	ml/m^2
Systemic vascular resistance index	1200-1800	$dyn/sec/cm^{-5}/m^2$
Mean arterial pressure	70-90	mmHg
Global ejection fraction	25-35	%
Cardiac function index	4.5-6.5	L/min
Global end diastolic volume index	680-800	ml/m^2
Intrathoracic blood volume index	850-1000	ml/m^2
Stroke volume variation	<10	%
Extravascular lung water index	3.0-7.0	ml/kg
Pulmonary vascular permeability index	1-3	

3. Oesophageal Doppler Monitoring

   Measures flow velocity in the descending aorta. Parameters: CO, SV, and fluid responsiveness.

   Cardiac output determined by measuring aortic blood flow and aortic CSA by assuming a constant partition between caudal and cephalic blood supply areas.

   CSA obtained either from nomograms or by M-mode of Ultrasonography [30]

   Probe is smaller than that for TEE

   Correlate well with CO measured by thermodilution

Advantages:

Easy placement, minimal training needed. Provide continuous, real-time monitoring. Low incidence of iatrogenic complications Minimal infective risk

Disadvantages:

High cost

Poor tolerance at awake patient, so mostly patient needs to be intubated

Probe displacement can occur during prolonged monitoring and patient`s turning

High interobserver variability when measuring changes in SV in response to fluid challenges

Parameters for fluid responsiveness

Assessing fluid responsiveness involves determining whether a patient will benefit from fluid administration, resulting in an increase in stroke volume (SV) or cardiac output (CO).

Several dynamic and static parameters are used in critically ill patients, particularly in ICU settings.

A. **Dynamic Parameters**

Dynamic parameters are more reliable than static ones, as they assess changes in hemodynamic in response to fluid shifts or manoeuvres.

1. Stroke Volume Variation (SVV):(31)

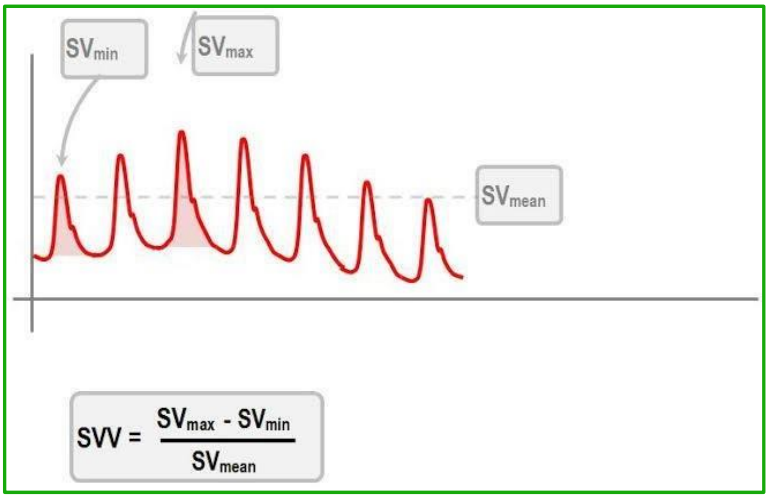
SVV is the percentage variation in stroke volume during the respiratory cycle. It is a direct indicator of preload responsiveness.

Principle

SVV relies on heart-lung interactions during mechanical ventilation.

In fluid-responsive patients, intrathoracic pressure changes cause significant stroke volume variations.

SVV is automatically calculated by advanced hemodynamic monitors (e.g.,PiCCO, LiDCO, FloTrac systems)



Threshold

SVV > 10–15% indicates fluid responsiveness. SVV < 10% suggests fluid non-responsiveness.

Advantages

Directly measures stroke volume changes.

Useful in optimizing fluid therapy in critically ill patients.

Limitations

Requires advanced hemodynamic monitors.

Valid only in:

Controlled mechanical ventilation with consistent tidal volumes. Absence of arrhythmias and spontaneous breathing.

2. Pulse Pressure Variation (PPV):[5,32]

PPV is the percentage variation in arterial pulse pressure (difference between systolic and diastolic pressure) during the respiratory cycle. It reflects the

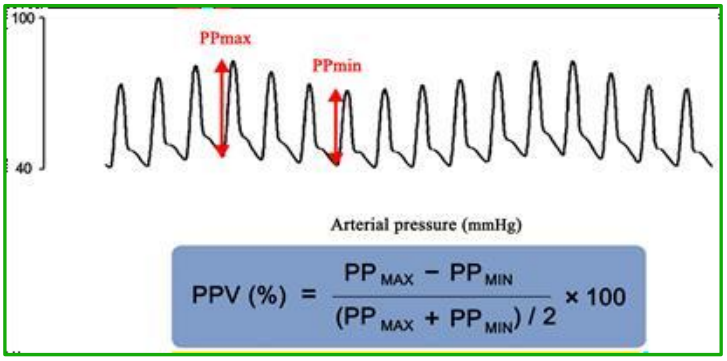
changes in preload induced by mechanical ventilation.

Principle

During inspiration, venous return to the right heart decreases due to increased intrathoracic pressure, reducing right ventricular stroke volume and subsequently left ventricular preload.

In fluid-responsive patients, these changes cause significant variation in pulse pressure.

How to Calculate



Threshold

PPV > 12–13% indicates fluid responsiveness.

PPV < 12% suggests the patient is not fluid responsive.

Advantages

Non-invasive and easily measured with an arterial catheter. Highly specific for fluid responsiveness.

Limitations

Accurate only in patients:

Under controlled mechanical ventilation with tidal volumes ≥8 mL/kg. Without significant arrhythmias or spontaneous breathing efforts.

3. Tidal Volume Challenge Test in Hemodynamic Monitoring(33)

The tidal volume (VT) challenge is a test used if the patient is getting ventilated in a lower tidal volume This method evaluates the dynamic response of cardiac output (CO) or stroke volume (SV) to an increase in tidal volume during mechanical ventilation.

Concept:

A brief increase in VT (e.g., from 6 mL/kg to 8–10 mL/kg of predicted body weight) generates larger intrathoracic pressure changes, which influence venous return and preload.

Fluid responsiveness is determined by observing whether this increase results in significant changes in stroke volume variation (△SVV >2.5%) or pulse pressure variation ((△PPV >3.5 %).

Indications: [34]

1. traditional preload responsiveness indices (e.g., passive leg raise) may not be feasible.
2. patients with low tidal volume ventilation, where dynamic indices may lose reliability.

4. Passive Leg Raising (PLR) Test:

The Passive Leg Raising (PLR) Test is a bedside, dynamic method used to assess fluid responsiveness in critically ill patients. It provides a reversible and non-invasive means of predicting whether a patient will benefit from additional fluid administration.(35)

Physiological Basis:

PLR temporarily increases venous return by shifting blood from the lower extremities to the central circulation. This mimics a fluid bolus (typically about 300 mL) without actually administering fluids.(34)

Increase in preload: Venous blood returns to the heart, increasing stroke

volume (SV) and cardiac output (CO) in fluid-responsive patients.

Non-responders: Patients who do not exhibit changes in SV or CO are unlikely to benefit from fluid resuscitation.

Steps to Perform the PLR Test

1. Patient Preparation:

Place the patient in a semi-recumbent position (head elevated at 30–45 degrees). Ensure the patient is hemodynamically stable enough to tolerate position changes.

2. Baseline Measurement:

Measure a hemodynamic variable (e.g., stroke volume, cardiac output, or blood pressure) before the PLR manoeuvre.

3. PLR Manoeuvre:

Lower the patient's upper body to a supine position and raise their legs to 45 degrees. Ensure no active muscle contractions from the patient.

4. Reassessment:

Reassess the hemodynamic variable within 30–90 seconds of the PLR manoeuvre.

5. Interpretation:

A significant increase in stroke volume (>10–15%) or cardiac output indicates fluid responsiveness. If no significant change occurs, additional fluids are unlikely to improve hemodynamic.

Indicators Monitored During PLR

Preferred indicators:

Stroke volume: Measured using echocardiography or pulse contour analysis.

Cardiac output: Measured using invasive or non-invasive methods.

Alternative indicators:

Blood pressure (less reliable but commonly used in resource-limited settings).

Advantages

1. Dynamic and reversible: No fluids are actually administered.
2. Bedside test: Requires minimal equipment and can be performed quickly.
3. Safe: Avoids risks associated with fluid overload, such as pulmonary oedema.
4. Accurate: More reliable than static indicators like central venous pressure (CVP).

Limitations

1. Equipment requirement: Accurate measurement of stroke volume or cardiac output requires advanced tools like echocardiography or a cardiac output monitor.
2. Patient factors:

Cannot be performed in patients with unstable spinal injuries.

May be less accurate in conditions like intra-abdominal hypertension or high venous resistance.

3. Technical expertise: Accurate interpretation requires trained personnel.

Resource-limited settings: Blood pressure changes during PLR can be used when advanced equipment is unavailable.

End-Expiratory Occlusion Test[34]

Temporarily halts mechanical ventilation at end-expiration to assess preload dependence. A >5% increase in stroke volume suggests fluid responsiveness.

The EEOT evaluates fluid responsiveness by assessing the hemodynamic changes during a brief pause at the end of expiration in mechanically ventilated patients.

Principle:

During the end-expiratory pause (10–15 seconds), intrathoracic pressure remains stable, increasing venous return to the right heart.

This transient increase in preload results in an increase in stroke volume in fluid-responsive patients.

How to Perform:

1. Apply an end-expiratory pause for 10–15 seconds using the ventilator.
2. Measure stroke volume or cardiac output changes during the pause.

Threshold

A ≥5% increase in stroke volume or cardiac output suggests fluid responsiveness.

Advantages:

Applicable even in low tidal volume settings (<8 mL/kg). Does not require deep sedation or paralysis.

Limitations:

Requires continuous cardiac output monitoring (e.g., echocardiography, arterial waveform analysis). Not suitable for patients with spontaneous breathing or arrhythmias.

6. Dynamic Changes in IVC or SVC Diameter:

Dynamic evaluation of the Inferior Vena Cava (IVC) and Superior Vena Cava (SVC) distensibility or collapsibility indices is a valuable bedside tool in assessing fluid responsiveness in critically ill patients, particularly in resource-limited ICU settings.

Inferior Vena Cava (IVC) or Superior Vena Cava (SVC) collapsibility during respiration measured via ultrasound.

IVC collapsibility index >40% in spontaneous breathing or >18% distensibility

during mechanical ventilation indicates fluid responsiveness.

A. Inferior Vena Cava (IVC) Indices(38) Principle

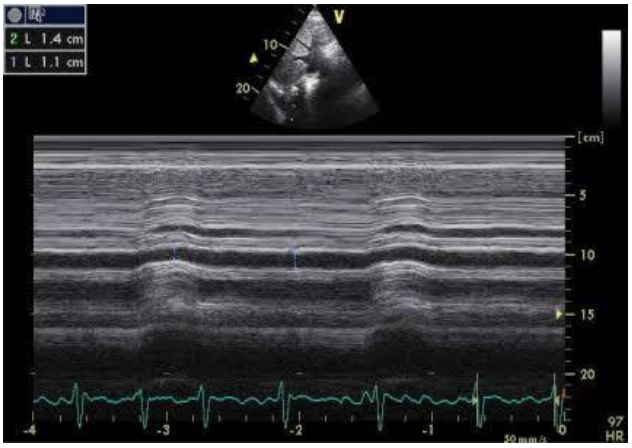
The IVC diameter changes with respiration, reflecting intravascular volume status and right atrial pressure.

Collapsibility Index (IVC-CI): Evaluates IVC diameter changes during spontaneous breathing. Distensibility Index (IVC-DI): Evaluates IVC diameter changes during mechanical ventilation.

Measurement Technique Using ultrasound:

1. Locate the IVC: Use a subxiphoid view.
2. Measure the diameter:

During inspiration (IVCmin) and expiration (IVCmax). At 1–2 cm proximal to the hepatic vein-IVC junction.



Formulas IVC max - IVC min) / IVC max

Thresholds

Spontaneous breathing: IVC-CI > 40% indicates fluid responsiveness.
Mechanical ventilation: IVC-DI > 18% suggests fluid responsiveness.

Advantages

Non-invasive, bedside, and easily performed with portable ultrasound. Useful in resource-limited settings.

Limited accuracy in:

Patients with elevated intra-abdominal pressure. Obese patients with poor ultrasound windows.

Arrhythmias or cardiac tamponade.

B. Superior Vena Cava (SVC) Distensibility Index[39] Principle

SVC diameter changes with the respiratory cycle in mechanically ventilated patients, reflecting changes in preload. It is more reliable than IVC in obese patients or those with elevated intra- abdominal pressure.

Measurement Technique

Use transesophageal echocardiography (TEE).

Measure SVC diameter during inspiration (SVCmin) and expiration (SVCmax).

Threshold

SVC-DI > 36% indicates fluid responsiveness in mechanically ventilated patients.

Advantages

Reliable in patients where IVC is difficult to assess. Less affected by external abdominal pressures.

Limitations

Requires TEE, which may not be readily available in all ICUs.

Cannot be used in non-intubated or spontaneously breathing patients.

A. Static Parameters

Static parameters assess preload but are less reliable for predicting fluid responsiveness.

1 Central Venous Pressure (CVP):

Measures right atrial pressure, indicating venous return.

CVP <8 mmHg may suggest hypovolemia but does not reliably predict fluid responsiveness.

2. Pulmonary Artery Wedge Pressure (PAWP):

Measured using a Swan-Ganz pulmonary artery catheter (PAC) which is placed in distal pulmonary artery with balloon inflated.

PAWP >18 mmHg suggests fluid overload but is not a reliable predictor of responsiveness.(23)

3. Global End-Diastolic Volume (GEDV):

GEDV represents the total volume of blood in the four heart chambers at the end of diastole. It is a preload indicator that reflects cardiac filling and is derived using transpulmonary thermodilution.(41)

Clinical Use:

A reliable preload marker that is less influenced by external factors like arrhythmias or intra- abdominal pressure.

Correlates with fluid responsiveness if combined with dynamic indices (e.g., stroke volume variation). OTHER PARAMETERS:

1. Mixed Venous Oxygen Saturation (SvO_2):

Definition: SvO_2 is the oxygen saturation of hemoglobin in the blood returning to the right atrium via the pulmonary artery, reflecting the balance between oxygen delivery and oxygen consumption.

Measurement

SvO_2 is measured from blood sampled via a pulmonary artery (PA) catheter, often using continuous monitoring devices.

Normal range: 65–75%. Clinical Significance(42)

1. High SvO_2 (>75%):

May indicate decreased oxygen extraction by tissues due to conditions like sepsis or mitochondrial dysfunction.

Can also result from high cardiac output (e.g., hyperdynamic states).

2. Low SvO_2 (<65%):

Reflects inadequate oxygen delivery or increased oxygen consumption. Common causes include hypovolemia, heart failure, hypoxia, or anemia.

Sepsis: Surviving Sepsis Campaign recommends targeting SvO_2 ≥65% during resuscitation. (13,14)

Advantages[43]

Direct measure of global oxygenation balance.

Useful in guiding fluid resuscitation, inotropic support, and oxygen therapy.

Limitations

Requires an invasive PA catheter.

May not accurately reflect regional tissue oxygenation.

2. Lactate Clearance:

Elevated lactate levels suggest tissue hypoperfusion.

A decrease after fluids may indicate improved perfusion.

Lactate clearance is the rate at which lactate levels decrease over time, reflecting improved tissue perfusion and reduced anaerobic metabolism [44]

Measurement :Serial arterial or venous lactate levels are measured over time.
Clinical Significance[42]

1. High lactate levels (>2 mmol/L):

Indicate tissue hypoxia, anaerobic metabolism, or impaired lactate clearance (e.g., liver dysfunction).

2. Lactate clearance >10% over 2–6 hours:

Associated with improved outcomes in septic shock and other critical conditions.

3. Lactate clearance vs. absolute lactate values:

Clearance provides dynamic information about resuscitation adequacy compared to static lactate levels.

Role in Hemodynamic Monitoring[13,14]

Lactate clearance is a resuscitation endpoint in the management of septic shock and hypoperfusion states

Advantages

Non-invasive and widely available.

Provides direct information about tissue hypoxia and anaerobic metabolism.

Limitations

Elevated lactate levels can result from non-hypoxic causes (e.g., beta-agonists, liver dysfunction). Clearance may lag behind clinical improvement in some cases.

3. PCO2 Gap (Arterial-to-Venous CO2 Difference)

Definition: The PCO2 gap is the difference between arterial (PaCO2) and central venous CO2 (PvCO2).

Normal Value: <6 mmHg. [45]

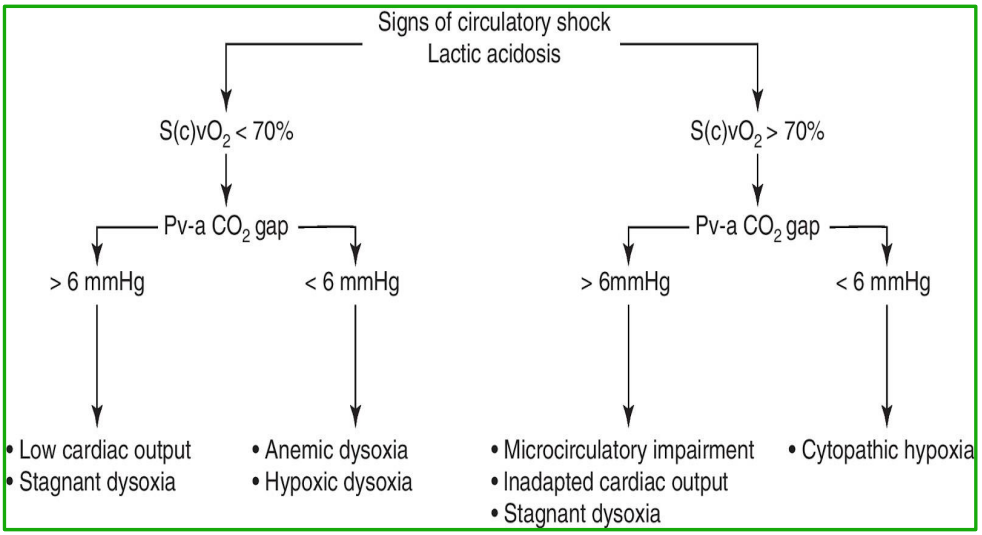
Significance in Shock:[46]

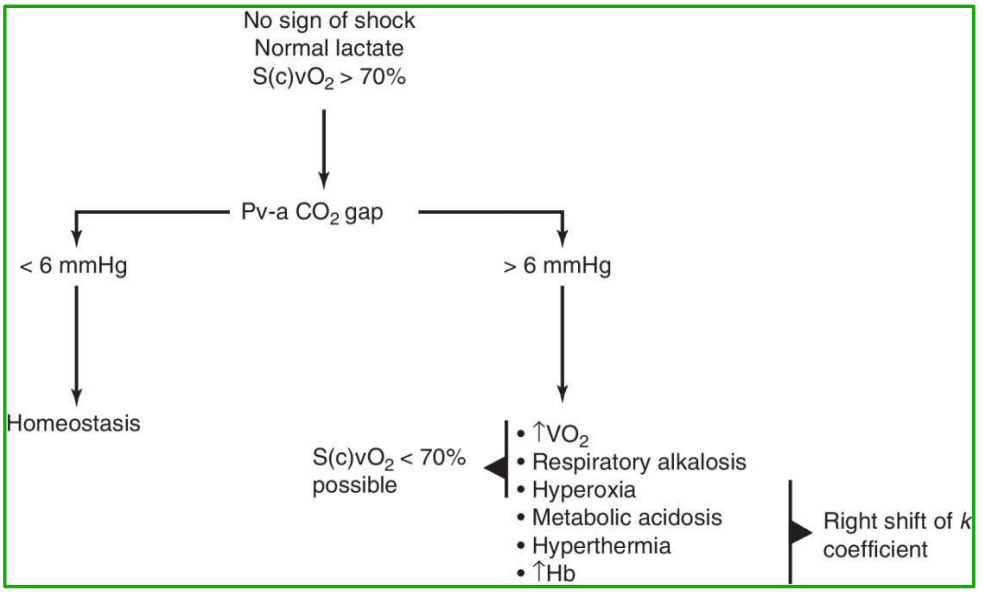
An elevated PCO2 gap (>6 mmHg) indicates inadequate cardiac output or oxygen delivery leading to impaired CO2 clearance.

A normal PCO2 gap with hyperlactatemia suggests a problem at the cellular or microcirculatory level rather than systemic hypoperfusion.

Clinical Use: PCO2 gap provides a complementary tool to lactate levels to

evaluate the adequacy of resuscitation.





B. Clinical Methods for Assessing Fluid Responsiveness

1. Fluid Challenge Test:(41) Definition

The fluid challenge test involves administering a defined volume of intravenous fluid over a short period to evaluate the patient's hemodynamic response.

Protocol

Fluid volume: Typically 250–500 mL of crystalloid solution. Duration: Administered over 5–10 minutes.

Hemodynamic parameters monitored:

Cardiac output (CO) or stroke volume (SV) via advanced monitoring devices. Surrogate parameters, such as mean arterial pressure (MAP) or pulse pressure.

A positive response is defined as a ≥10–15% increase in cardiac output or stroke volume following fluid administration.

A negative response indicates that the patient is not fluid responsive, and further fluid resuscitation may cause harm (e.g., fluid overload).

Advantages

Directly assesses fluid responsiveness.

Provides guidance on whether additional fluid therapy is needed.

Limitations

Requires invasive or semi-invasive monitoring (e.g., thermodilution, pulse contour analysis). May not be feasible in patients with cardiac or pulmonary conditions prone to fluid overload.

2. Mini Fluid Challenge Test [47]

Definition

The mini fluid challenge test is a smaller-volume variation of the traditional

fluid challenge, designed to evaluate fluid responsiveness with minimal risk of fluid overload.

Protocol

Fluid volume: 100–150 mL of crystalloid solution. Duration: Administered over 1–2 minutes.

Hemodynamic parameters monitored:

CO or SV changes using advanced monitoring (e.g., pulse contour analysis,echocardiography). A positive response is indicated by a ≥5% increase in stroke volume after the mini fluid bolus.

Suitable for patients with borderline fluid tolerance or those at high risk of fluid overload.

Advantages

Minimizes the risk of fluid overload.

Allows repeated assessments in patients with limited fluid reserve.

Can be combined with advanced techniques like echocardiographic Doppler for non-invasive monitoring.

Limitations

Requires precise hemodynamic monitoring tools.

May not provide reliable results in patients with significant arrhythmias or severe cardiac dysfunction.

3. Echocardiography:

Non-invasive assessment of cardiac function and fluid status.

Measure changes in cardiac output, stroke volume, or ventricular filling pressures.

Limitations and Considerations

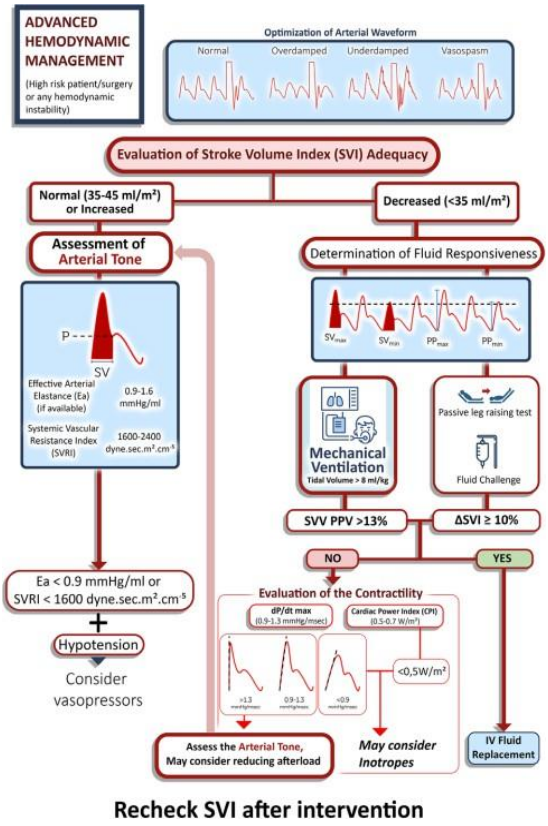
Dynamic parameters require mechanical ventilation and regular heart

rhythms.

In patients with arrhythmias or spontaneous breathing, static parameters or tests like PLR may be more appropriate.

Always consider the patient's clinical context and comorbidities to avoid unnecessary fluid administration.

By integrating these parameters, clinicians can make informed decisions to optimize fluid therapy, improve tissue perfusion, and avoid complications such as fluid overload.



Guideline for Hemodynamic Monitoring in Resource-Poor settings.

Effective hemodynamic monitoring in resource-limited ICUs can be achieved

through a combination of clinical skills, low-cost technology, and evidence-based practices. These strategies optimize outcomes and minimize the reliance on advanced tools.

Objective is to provide practical strategies for hemodynamic monitoring in resource-limited settings to optimize patient outcomes while utilizing minimal available resources.

Clinical assessment is key: [48]

Prioritize bedside evaluation over reliance on expensive equipment.

Maximize available resources: Optimize the use of accessible tools and supplies.

Train staff: Focus on skill-building to interpret clinical signs and low-tech monitoring results. Target high-risk conditions: Prioritize septic shock, cardiogenic shock, and hypovolemia.

Basic Clinical Monitoring

Vital signs:(47)

Heart rate (HR): Use pulse palpation or low-cost pulse oximeters.

Blood pressure (BP): Use manual sphygmomanometers if automated monitors are unavailable. Respiratory rate (RR): Count manually at bedside.

Temperature: Use low-cost digital thermometers.

Urine output: Monitor hourly with simple catheters. Target >0.5 mL/kg/hr.

Mental status: Use simple tools like AVPU (Alert, Verbal, Pain, Unresponsive) or GCS scoring systems

Non-Invasive Monitoring

Pulse oximetry: Monitor SpO2 levels using portable devices.

Capillary refill time (CRT): Assess perfusion, with >3 seconds suggesting hypoperfusion. Skin temperature: Cold extremities indicate poor perfusion or

low cardiac output. [49]

Laboratory and Basic Investigations

Blood gas analysis: If arterial gas is unavailable, venous gas can provide critical information (e.g., pH, lactate).

Lactate: Indicator of tissue hypoxia when available.

Haematocrit and haemoglobin: Evaluate anaemia and hypovolemia.
Electrolytes: Basic tests for sodium, potassium, and calcium.

Fluid and Volume Status Assessment

Passive leg raising (PLR) test: Predict fluid responsiveness by observing changes in BP or HR.

Bedside ultrasound: Assess IVC diameter and cardiac function, if available.[35]

Advanced Techniques in Resource-Limited Settings

Non-invasive BP monitors: Use devices that store trends for better analysis.
Handheld ultrasound: Use for cardiac and IVC assessments.

Doppler ultrasound: Assess blood flow in key vessels.

Clinical Assessment of Microcirculation

Microcirculation assessment is vital in critically ill patients to evaluate tissue perfusion and oxygenation. Dysfunctional microcirculation can lead to organ failure and poor outcomes despite normal systemic hemodynamic parameters.It is called Hemodynamic inhoherence. Below are commonly used bedside methods to assess microcirculation:

1. Central-to-Peripheral Temperature Gradient (ΔT)

This method measures the temperature difference between a central site (e.g., rectum) and a peripheral site (e.g., finger or toe).

Mechanism:

A significant gradient (>4°C) suggests vasoconstriction due to hypoperfusion.

Narrowing of the gradient (<2°C) may indicate improved perfusion but could also reflect peripheral vasodilation due to sepsis.

Measurement:

Use a thermometer or sensor to measure central (core) and peripheral (skin) temperatures. Calculate the difference:

ΔT = Central Temperature - Peripheral Temperature.

Limitations:

Influenced by external temperature and patient factors (e.g., fever or hypothermia). Not specific to microcirculatory status alone.(50)

2. Peripheral Perfusion Index (PPI)[51]

The peripheral perfusion index measures the strength of blood flow at a peripheral site using pulse oximetry.

Mechanism:

PPI is derived from the ratio of pulsatile to non-pulsatile blood flow detected by the pulse oximeter. A higher PPI (>1.4) indicates better perfusion, whereas a lower PPI (<0.5) suggests hypoperfusion

Measurement: Many modern pulse oximeters display PPI directly.

The values are affected by vasoconstriction, shock, or peripheral vascular diseases.

Advantages:

Non-invasive and continuous. Useful for trend monitoring.

3. Capillary Refill Time (CRT)(52,53)

CRT is the time taken for skin color to return to normal after applying pressure to blanch the skin. Procedure:

Apply pressure to a nail bed or skin (e.g., forehead or sternum) for 5 seconds. Measure the time for color to return after releasing pressure.

A CRT >2-3 seconds is indicative of impaired microcirculation.

Clinical Context:

CRT is prolonged in hypoperfusion and shock states.

Normal values may vary with age, skin temperature, and lighting conditions.

Limitations:

Subjective and dependent on the observer. May be unreliable in dark-skinned patients.

4. Skin Mottling Score (SMS) [54]

Skin mottling refers to patchy discoloration due to uneven blood flow in the dermis and is often assessed on the anterior knees.

Mechanism:

Mottling occurs due to peripheral vasoconstriction in shock states, indicating microcirculatory dysfunction.

A scoring system (0–5) is used to quantify the extent of mottling: 0: No mottling.

1–2: Mottling confined to the knees.

3–5: Mottling extending beyond the knees. Clinical Use:

Persistent or worsening mottling correlates with poor prognosis in septic shock.

Monitoring of Regional Circulation

1. Techniques for Monitoring Regional Circulation

A. Near-Infrared Spectroscopy (NIRS) [55]

NIRS measures tissue oxygenation in specific regions (e.g., brain or muscle) by detecting oxyhemoglobin and deoxyhemoglobin levels using near-infrared light.

Principle: Based on the absorption spectra of hemoglobin in the near-infrared range (700–900 nm). Assessing peripheral perfusion in septic shock or limb ischemia.

Advantages: Non-invasive and continuous.

Limitations: Affected by skin pigmentation, adipose tissue, and background noise.

B. Laser Doppler Flowmetry (LDF) [56]

This technique measures blood flow in microcirculation by detecting Doppler shifts caused by moving red blood cells in a laser beam.

Applications:Assessing skin or mucosal perfusion. Advantages: Real-time, non-invasive.

Limitations: Small sampling volume and sensitivity to motion artifacts.

C. Sidestream Dark Field (SDF) and Incident Dark Field (IDF) Imaging[57]

These optical imaging techniques visualize microcirculation in real-time by capturing red blood cells' flow in capillaries.

Applications:(58)

Assessing sublingual microcirculation in sepsis, trauma, and cardiac surgery. Monitoring intestinal perfusion during abdominal surgery.

Advantages: Direct visualization of capillary density and flow. Limitations: Operator-dependent and limited to superficial tissues.

D. Transcranial Doppler Ultrasound (TCD)(59,60)

TCD measures cerebral blood flow velocity in major intracranial arteries using Doppler ultrasound. Applications:

Monitoring cerebral circulation during traumatic brain injury, stroke, or vasospasm. Detecting emboli or autoregulation disturbances.

Advantages: Non-invasive and bedside capable.

Limitations: Operator-dependent and limited to large vessels.

E. Regional Oxygen Saturation (rSO_2) Monitoring(61)

This technique measures oxygen saturation in specific tissues or regions, primarily using devices like cerebral oximeters.

Applications:

Monitoring during cardiac surgery or extracorporeal membrane oxygenation (ECMO). Evaluating splanchnic perfusion in abdominal surgeries.

Advantages: Continuous monitoring and early detection of hypoxia.

Limitations: Limited resolution in differentiating arterial and venous contributions..

2. Clinical Applications of Regional Circulation Monitoring

Brain: TCD, NIRS, rSO_2 for cerebral perfusion in trauma, stroke, and surgery.

Gut/Splanchnic Circulation: NIRS, laser Doppler for ischemia monitoring in sepsis or surgery. Skin/Muscle: Laser Doppler, SDF for peripheral ischemia or shock assessment.

Kidneys: Doppler ultrasound or regional oxygenation measurements to evaluate renal perfusion. Challenges in Regional Circulation Monitoring

1. Technical Limitations: Motion artifacts, limited penetration depth.
2. Inter-patient Variability: Anatomical and physiological differences.
3. Integration with Systemic Monitoring: Requires correlation with global hemodynamic parameters for complete assessment.

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11 Guidelines for sedation/ analgesia in adult patients in the ICU

11.1 Purpose of the guideline

These guidelines are to be applied for adult patients in ICU for optimal care and to prevent untoward events and poor outcome.

11.2 Introduction

Helps alleviate anxiety, discomfort, and pain in critically ill patients. At the same time sedation can also have significant adverse effects, particularly in patients who are already hemodynamically unstable.

11.3 General Principles

- Tailor sedation to the patient: Sedation requirements vary from patient to patient and at different times in their illness.
- Use the minimum amount of sedation: Use the minimum amount of sedation to keep patients safe and comfortable.
- Monitor sedation: Use a sedation score to monitor the depth of sedation. The Richmond Agitation–Sedation Scale (RASS) ranges from −5 to +4, with more negative scores indicating deeper sedation.
- Use analgesics: Treat pain with analgesics like IV morphine or fentanyl, Paracetamol.
- Use protocolized care: Use a protocol that includes routine monitoring of sedation, pain, and delirium.

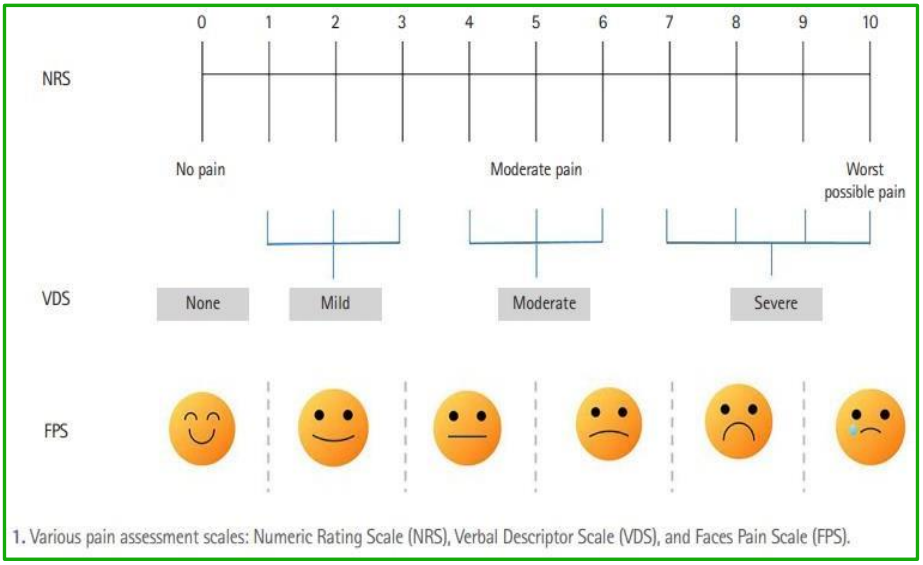
- Use spontaneous breathing: Use spontaneous breathing unless contraindicated.
- Be careful with liver and kidney failure: Use caution when sedating patients with liver or kidney failure.
- Reduce doses gradually: If a patient is on long-term sedation, reduce the dose gradually or restart a lower dose if there are withdrawal symptoms.
- Use dexmedetomidine for delirium: Dexmedetomidine is often used for patients who are delirious and require ongoing sedation.
- Use propofol: Propofol is often used because it allows for rapid and predictable de-sedation.

11.4. Importance of Sedation

1. **Anxiety and discomfort**: Critically ill patients often experience anxiety, fear, and discomfort due to their illness, pain, and the unfamiliar environment of the intensive care unit (ICU).
2. **Pain management**: Analgosedation can help manage pain, which is a common problem in critically ill patients. Pain assessment and management are crucial aspects of sedation in critically ill patients. Pain can be assessed using pain scales such as:

   Visual Analog Scale (VAS): A 10-point scale that asks patients to rate their pain from 0 (no pain) to 10 (worst possible pain).

Numeric Rating Scale (NRS): A 11-point scale that asks patients to rate their pain from 0 (no pain) to 10 (worst possible pain).



1. Various pain assessment scales: Numeric Rating Scale (NRS), Verbal Descriptor Scale (VDS), and Faces Pain Scale (FPS).

Faces Pain Scale (FPS): A scale that uses facial expressions to assess pain. Patients who cannot self-report use BPS (Behavioural pain scale)

Behavioral Pain Scale (BPS) 3-12

Item	Description	Score
Facial expression	Relaxed	1
	Partially tightened (eg, brow lowering)	2
	Fully tightened (eg, eyelid closing)	3
	Grimacing	4
Upper limbs	No movement	1
	Partially bent	2
	Fully bent with finger flexion	3
	Permanently retracted	4
Compliance with ventilation	Tolerating movement	1
	Coughing but tolerating ventilation for most of the time	2
	Fighting ventilator	3
	Unable to control ventilation	4

Critical Care Pain Observation Tool (CPOT)

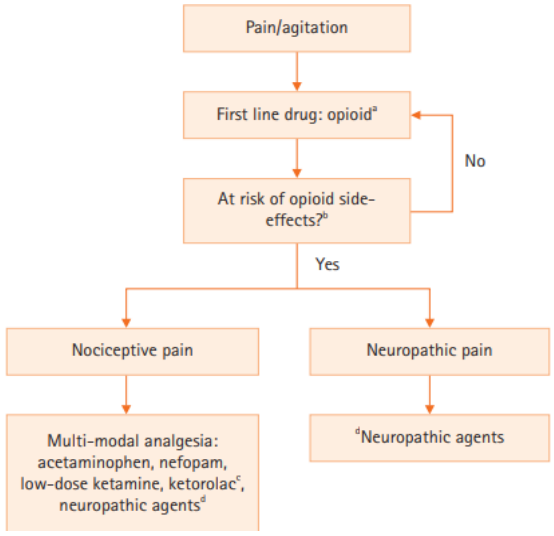
Indicator	Score		Description
Facial Expression	Relaxed, neutral	0	No muscle tension observed
	Tense	1	Presence of frowning, brow lowering, orbit tightening
	Grimacing	2	All of the above plus eyelid tightly closed or biting at endotracheal tube
Body Movements	Absence of Movements	0	Does not move at all
	Protection	1	Slow, cautious movements, touching or rubbing pain site
	Restlessness/Agitation	2	Pulling at lines, attempting to sit up, striking at staff, thrashing limbs, trying to climb out of bed
Ventilator Compliance or Vocalization	Tolerating ventilator or movement	0	Alarms not activated, easy ventilation
	Coughing but tolerating	1	Coughing, alarms activated but stop spontaneously
	Fighting ventilator	2	Asynchrony, blocking ventilation, alarms frequently activated
	Talking in normal tone or no sound	0	Talking normal tone or no sound
	Sighing, moaning	1	Sighing, moaning
	Crying, sobbing	2	Crying out, sobbing
Muscle Tension	Relaxed	0	No resistance to passive movements
	Tense, rigid	1	Resistance to passive movements
	Very tense or rigid	2	Strong resistance to passive movements

Pain management can be achieved using a variety of pharmacological and non-pharmacological interventions, including:

Opioids: Opioids, such as morphine and fentanyl, are commonly used analgesics in the ICU.

Non-opioid analgesics: non-opioid analgesics, such as Paracetamol and NSAIDs, can be used for mild to moderate pain.

Regional anaesthesia: Regional anaesthesia, such as epidural anaesthesia, can be used for patients undergoing surgical procedures.



3. **Facilitation of mechanical ventilation**: Sedation can help patients tolerate mechanical ventilation, which is often necessary in critically ill patients.
4. **Prevention of agitation**: Sedation can prevent agitation, which can lead to self-extubation, removal of lines and tubes, and other complications.

Types of Sedatives

The choice of sedative depends on the patient's underlying condition, the desired level of sedation, and the potential side effects.

Benzodiazepines: Benzodiazepines, such as midazolam and lorazepam, are commonly used sedatives in the ICU. They have anxiolytic, hypnotic, and muscle relaxant properties. They are not the first-choice sedatives as they cause confusion and delirium. When treating alcohol withdrawal syndrome, benzodiazepines are considered first line agents to reduce the severity of withdrawal, as well as the incidence of seizures.

Opioids: IV opioids are the most important treatment for non-neuropathic pain in adult ICU patients. They are also commonly used for tolerance of mechanical ventilation.

Propofol: Propofol is a short-acting sedative with anticonvulsant properties that is often used for procedural sedation. It has a rapid onset of action and can be easily titrated. Like benzodiazepines, this agent exerts its effects on the GABA receptor in the central nervous system via an alternate binding site. This is typically seen with infusion rates >65 mcg/kg/min, which is well above the normal dosing range of 0-50 mcg/kg/min, or for a prolonged duration (>48 hours) with a mortality rate reaching up to 85%

Dexmedetomidine: Dexmedetomidine is a selective α2-adrenergic agonist that has sedative, anxiolytic, and analgesic properties. It is often used for long-term sedation in the ICU. It does not appear to cause significant effects on respiratory drive. As a result, it is commonly used in non- ventilated patients and mechanically ventilated patients near extubation. It is not necessary to discontinue this agent prior to extubation. The DahLIA study found that dexmedetomidine may be beneficial in patients with delirium, though bradycardia may limit its use in some patients. During continuous infusion, dexmedetomidine can cause vasodilation resulting in hypotension. Therefore, a bolus prior to continuous infusion is not recommended.

Ketamine: Ketamine is an NMDA receptor antagonist that has sedative, analgesic, and amnestic properties. It is often used for procedural sedation and for patients who are hemodynamically unstable.

Sedative	Onset	Elimination half-life	Active metabolite	Intermittent dosing	IV infusion rate
Midazolam	2–5 min	3–11 hr	Yes (prolonged sedation, especially with renal failure)	0.01–0.05 mg/kg over several minutes	0.02–0.1 mg/kg/hr
Lorazepam	10–40 min	8–15 hr	None	0.02–0.04 mg/kg (≤2 mg)	0.02–0.06 mg/kg q 2–6 hr prn or 0.01–0.1 mg/kg/hr (≤10 mg/hr)
Diazepam	2–5 min	20–120 hr	Yes (prolonged sedation)	5–10 mg	0.03–0.1 mg/kg q0.5–6 hr prn
Propofol	1–2 min	Short-term use: 3–12 hr Long-term use: 50±18.6 hr	None	5 µg/kg/min over 5 minutes	5–50 µg/kg/min
Dexmedetomidine	5–10 min	1.8–3.1 hr	None	1 µg/kg/min over 10 minutes	0.2–0.7 µg/kg/hr

Opioids (route)	Equianalgesic dose	Onset	Elimination half-life	Intermittent dosing	IV infusion rate	Side effect and other information
Morphine (IV)	10 mg	5–10 min	3–4 hr	2–4 mg q1-2 hr	2–30 mg/hr	Accumulation in patients with liver dysfunction
Hydromorphone (IV)	1.5 mg	5–15 min	2–3 hr	0.2–0.6 mg q1-2 hr	0.5–3 mg/hr	Accumulation in patients with kidney and liver dysfunction
Fentanyl (IV)	100 µg	1–2 min	2–4 hr	0.35–0.5 µg q 0.5–1 hr	0.7–10 µg/kg/hr	Accumulation in patients with kidney and liver dysfunction, release of histamine
Remifentanil (IV)		1–3 min	3–10 min		Loading dose: 1.5 µg/kg Maintenance dose: 0.5–15 µg/kg/hr	Available regardless of liver and kidney dysfunction

Best Practices for Sedation Management

Assessment of sedation level: Regular assessment of the patient's sedation level is essential to ensure that the patient is receiving the appropriate level of sedation. A light level of sedation can be achieved and maintained using daily sedation interruption (DSI) and a nurse-protocolized targeted sedation protocol.

Use of sedation scales: Sedation scales, such as the Richmond Agitation- Sedation Scale (RASS) and the Sedation-Agitation Scale (SAS), can help clinicians assess the patient's sedation level.

Richmond Agitation-Sedation Scale (RASS)

Scale	Score	Description
Combative	4	Combative, violent towards staff
Very agitated	3	Pulls at tubes and/or catheters, aggressive towards others
Agitated	2	Frequent non-purposeful movement
Restless	1	Anxious, restless movements
Alert & calm	0	Awake and alert, calm
Drowsy	-1	Not fully alert, sustained awakening
Light sedation	-2	Awakens for < 10 seconds
Moderate sedation	-3	Movement and eye opening to voice
Deep sedation	-4	No response to voice, but opens eyes to physical stimulation
Cannot be aroused	-5	No response to verbal or noxious stimulus

Titration of sedatives: Sedatives should be titrated to achieve the desired level of sedation. This can help minimize the risk of oversedation and undersedation. Alert, calm and cooperative patient is the right level of sedation.

Regular monitoring of vital signs: Regular monitoring of vital signs, such as heart rate, blood pressure, and oxygen saturation, is essential to ensure that the patient is not experiencing any adverse effects from the sedative.

Complications of Sedation

Oversedation: Oversedation can lead to respiratory depression, hypotension, and prolonged ICU stay.

Undersedation: Undersedation can lead to agitation, anxiety, and discomfort.

Delirium: Delirium is a common complication of sedation in critically ill patients. It is characterized by acute onset of confusion, disorganized thinking, and altered level of consciousness.

Sedation-related withdrawal: Sedation-related withdrawal can occur when sedatives are stopped abruptly. It is characterized by symptoms such as anxiety, agitation, and insomnia.

Recommendation:

Level 1

- Light sedation (RASS 0 to -1) is preferred over heavy sedation unless contraindicated.
- Propofol or dexmedetomidine should be used as first line agents over benzodiazepines in critically ill, mechanically ventilated adults.

- Benzodiazepines are the treatment of choice in patients with alcohol withdrawal or seizures/status epilepticus.

Level 2

- Pain management should be guided by routine pain assessment and addressed before a sedative agent is considered.
- Continuous infusion opioids, such as fentanyl, may be used as part of an analgesia-first sedation strategy prior to adding additional sedative agents.
- Opioids may also be used as an adjunct to a first line sedative agents to achieve the desired level of analgosedation.
- Sedatives should be titrated to the appropriate level of desired sedation based on the Richmond Agitation Sedation Scale (RASS).
- Ketamine, at sedative doses (0.5-5 mg/kg/hr), may be an alternative for patients without contraindications (see below).
- Ketamine, at sub-dissociative doses (<0.5 mg/kg/hour IV), may be considered in patients with moderate to severe pain to decrease opioid requirements.
- Benzodiazepines should be used as a last line sedative agent due to the high correlation with ICU delirium.
- Dexmedetomidine should be used in patients where agitation is the main barrier to extubation.
- Daily spontaneous awakening trials should be performed in patients without contraindications.

Level 3

- Propofol is the sedative of choice when rapid neurologic assessment is needed, or intracranial hypertension is present.
- Ketamine should be avoided in patients with coronary artery disease, arrhythmias, inability to tolerate an increase in blood pressure or heart rate, severe pulmonary secretions, glaucoma, or psychiatric history.

LEVEL OF RECOMMENDATION DEFINITIONS

- **Level 1:** Supported by multiple, prospective randomized clinical trials or strong prospective, non-randomized evidence if randomized testing is inappropriate.
- **Level 2:** Supported by prospective data or a preponderance of strong retrospective evidence.
- **Level 3:** Supported by retrospective data or expert opinion.

References:

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3. Seo Y, Lee HJ, Ha EJ, Ha TS. 2021 KSCCM clinical practice guidelines for pain, agitation, delirium, immobility, and sleep disturbance in the intensive care unit. Acute Crit Care. 2022 Feb;37(1):1-25. doi: 10.4266/acc.2022.00094. Epub 2022 Feb 28. PMID: 35279975; PMCID: PMC8918705.
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12 Traumatic brain injury in adults- critical care aspects

- Leading cause of morbidity & mortality
- Mx is complex
- Severe TBI where ICP, Brain tissue Oxygenation measurement feasible
- Severe TBI where ICP, Brain tissue Oxygenation measurement NOT feasible
- Advancement in monitoring, prognostication, biomarkers

Monitoring:

- Repeated neurological assessment -hourly
- GCS: Lacks precision for prognostication except for motor sub-score
- Mild 13-15, Moderate 9-12, Severe <8
- Integrate Clinical, Biomarkers, Investigational parameters (CBI)

Qualitative pupillometry:

- more accurate
- provide early warning information before clinical deterioration,
- important especially when opioids and sedative drugs result in small pupils and hamper clinical examination

ICP monitoring

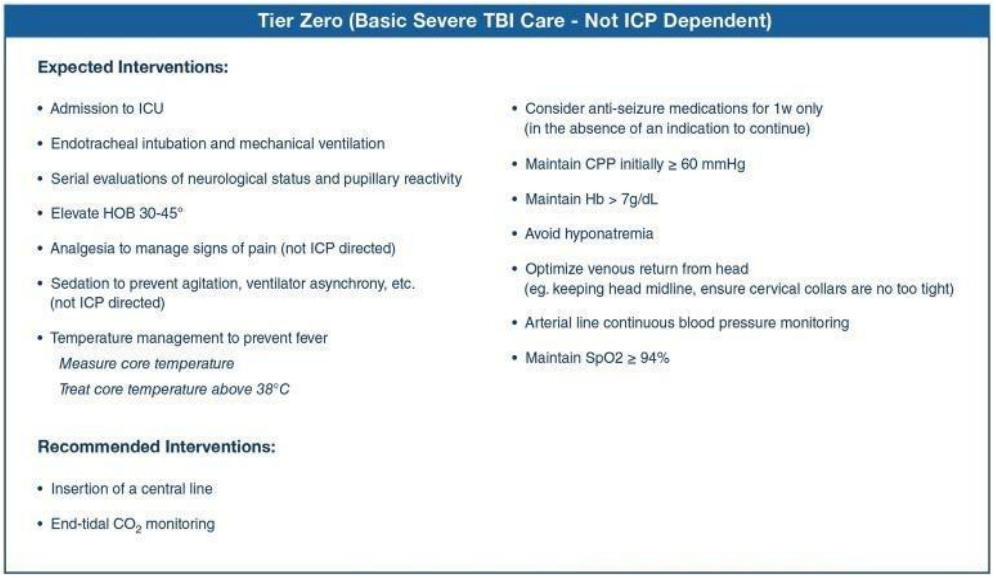
- **Invasive:**
  - GCS<8, with structural brain damage on initial CT
  - Decreased 6-month mortality
  - EVD
  - Intra parenchymal transducers
- **Non-invasive**
  - ONSD
  - TCD

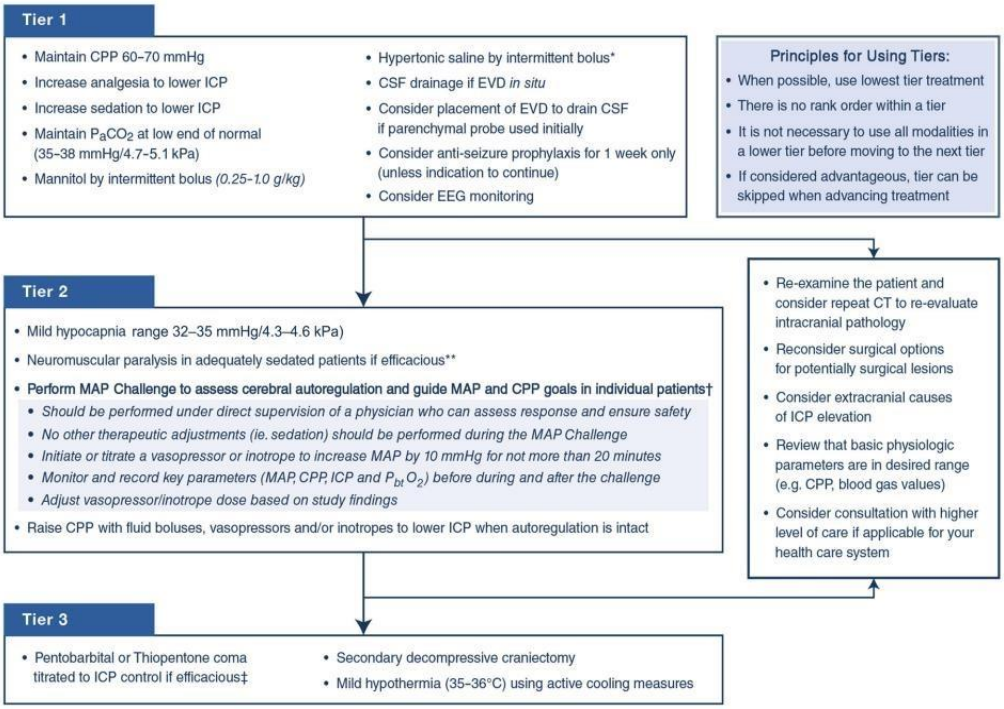
Over view of management of severe TBI:

1. Monitoring:
2. Continuous Spo2, eTco@
3. Urine out put
4. Intra arterial Blood pressure monitoring (if unit supports)
5. Central line for drugs & CVP
6. Neurological status hourly: GCS, Pupillary size & reactivity
7. CT Brain: Stat, 48Hr, 5-7 d after admission
8. Mechanical ventilation: PaO_2>60mmHg, SaO_2>90%, $PaCO_2$ 35-40mmHg
9. Sedation: Fentanyl, Propofol, Dexmeditomidine, Barbiturate: Thiopentone 1-2

mg/kg/hr infusion

10. Head of bed 30^{o}
11. Monitor temperature and treat hyperthermia>38C with cooling packs, Pharmacological
12. Early enteral nutrition
13. Seizure Prophylaxis: Inj Pheytoin
14. Stress Ulcer Prophylaxis
15. Mechanical /pharmacological DVT prophylaxis
16. Avoid Systolic BP < 90mmHg, MAP<70mmHg
17. Hyperosmolar thgerapy: Mannitol, Hypertonic Saline. Avoid Mannitol in Arterila hypotension, Hypovoluemia, Hyponatremia, Acute Kidney Injury. Avoid hyperosmolar therapy if Plasma Osmolarity > 320.





Neuro-worsening:

1. Spontaneous decrease in GCS>1 point
2. New decrease in Pupillary reactivity
3. New Pupillary asymmetry or bilateral mydriasis
4. New focal neurological deficit
5. Herniation syndrome or Cushing Triad

Response to Critical Neuro worsening:

1. Emergent evaluation to identify possible cause of neuro worsening
2. If herniation is suspected: Hyperventilation, bolus of hypertonic solution
3. Consider emergent imaging or other testing
4. Rapid escalation of treatment

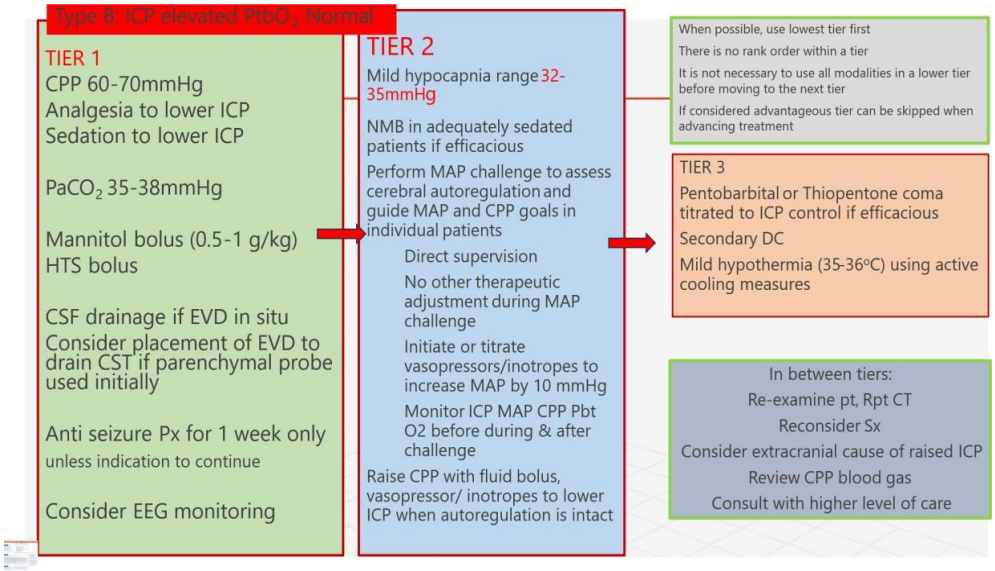
Critical Neuroworsening

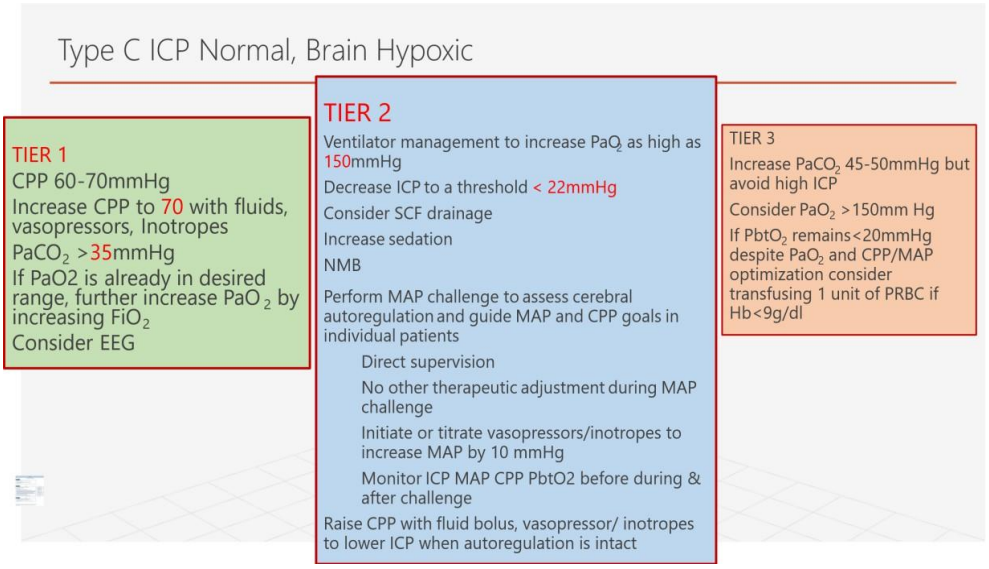
A serious deterioration in clinical neurologic status such as:

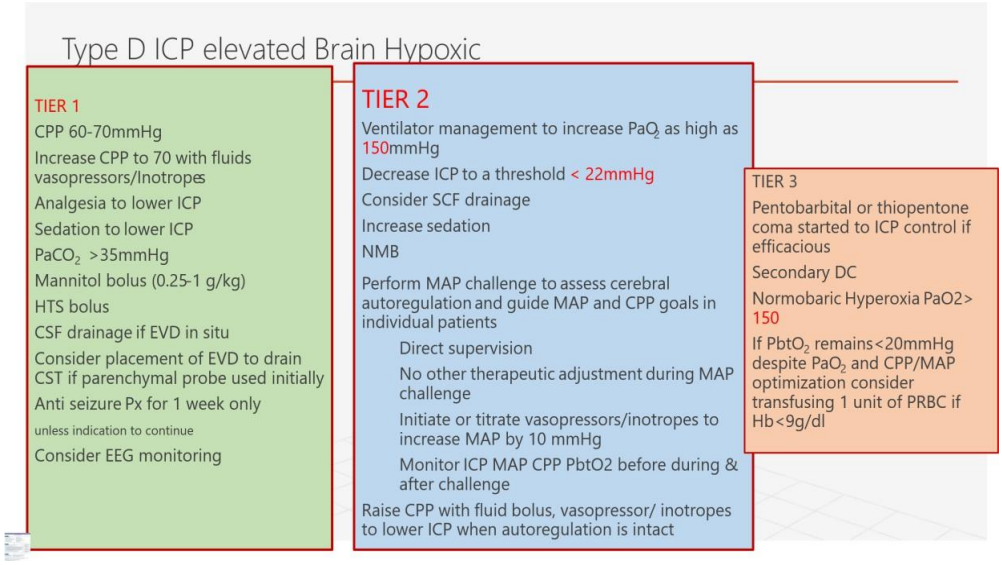
- Spontaneous decrease in the GCS motor score of ≥ 1 points (compared with the previous examination)
- New decrease in pupillary reactivity
- New pupillary asymmetry or bilateral mydriasis
- New focal motor deficit
- Herniation syndrome or Cushing's Triad which requires an immediate physician response

ICP monitoring & Brain tissue Oxygenation measurements feasible (in higher centres)

	ICP < 22 mmHg	ICP > 22 mmHg
$P_{bt}O_2$ > 20 mmHg	Type A	Type B
$P_{bt}O_2$ < 20 mmHg	Type C	Type D







Management in ICU

1. Transfusion Strategies
   a. Implement a liberal transfusion strategy, avoid Hb <9gm/dl
   b. HEMOTION trial: liberal transfusion strategy were less likely to have an unfavorable neurological outcome based on lower Glasgow Outcome Scale Extended (GOS-E)].

Hb>10 Vs <7gm/dl.

c. TRAIN study [(Hb <9 vs. <7g/dl) found an adjusted relative risk of 0.86 (95% confidence interval {CI}, 0.79–0.94); P = 0.002] for the restrictive strategy.

d. CENTER-TBI study, which indicated a significantly increased risk of unfavorable neurological outcomes and increased mortality in patient with low Hb levels (Hb ≤7.5 g/dl; OR, 3.21; 95% CI, 1.59–6.49)

2. Fluid management: Neutral fluid balance
3. Cerebral perfusion pressure:
   a. CPP = mean arterial pressure (MAP) – ICP, a determinant of cerebral perfusion and blood flow
   b. Identifying the CPP range where autoregulation is most effective, may vary among patients and change dynamically over time.
   c. Pressure reactivity index (PRx): moving correlation over time between ICP& MAP
   d. Helps in identification of personalized or population-derived optimal CPP (CPPopt)-allow individualized blood pressure targets
   e. may be higher than the conventional range in some cases, as in patients with decreased autoregulation, significant brain swelling.
   f. Without such advanced monitoring, target systolic blood pressure ≥100 mmHg and CPP between 60 and 70 mmHg
4. Hyperosmolar agents:
   a. Mannitol and HTS are both used to manage increased ICP, either alone or combined.
   b. Effect on renal function: may or may not differ: one study showed the hazard ratio for developing AKI was 2.13 for mannitol versus 1.5 for HTS.
5. Another study showed no significant difference of AKI prevalence in TBI patients receiving HTS vs no hyperosmolar therapy
6. Mechanical ventilation:
   a. Mechanical ventilation should be aimed at normal PaCO2 in TBI, since fluctuations can lead to changes in CBF, ICP.
   b. Hypocapnia causes cerebral vasoconstriction and reduced CBF
   c. Hypercapnia can increase ICP due to vasodilation and increased CBF

d. Lung protective ventilation:22% had ICP more than 22 mmHg during LPV

7. Temperature management:
   a. Fever control should be prioritized, irrespective of ICP status, especially for patients at risk of seizures or cerebral herniation.
8. Fever, whether neurogenic or infectious, is associated with unfavorable outcomes and must be promptly managed to mitigate secondary injury.
9. Temperature control (36.0–37.5 °C) is considered an essential aspect of high quality TBI care.
10. Sedation:
    a. Dexmedetomidine: selective α2-agonist that decreases sympathetic activity by reducing sympathetic outflow from the nucleus coeruleus.
    b. Pentobarbital coma seems a last resort for high ICP. Effect not well studied. Immediate ICP lowering effect with Pentobarbital-better outcome.
    c. Rescue ICP trial: in medically refractory increased ICP, DC appears superior to a strategy with barbiturates as rescue therapy.
    d. Barbiturates may still have a place in the armamentarium of ICP control but that immediate response of ICP to barbiturates is an important factor
    e. Continuous core temperature monitoring and fever management are recommended as key measures to prevent secondary brain injury
11. No role for steroids in TBI. Increased mortality.

13 Acute respiratory distress syndrome (ARDS)

Acute respiratory distress syndrome (ARDS) is the term applied to a spectrum of conditions with different aetiologies which share common clinical-pathological characteristics including:

(1) increased permeability of the alveolo-capillary membrane, resulting in inflammatory oedema

(2) increased non-aerated lung tissue resulting in higher lung elastance (lower compliance)

(3) increased venous admixture and dead space, which result in hypoxemia and

hypercapnia

Management of ARDS

The treatment of ARDS is respiratory support and identification and treatment of the predisposing cause. The supportive therapy for ARDS mainly focuses on providing adequate gas exchange with lung protective ventilation and minimizing VILI.

Lung-protective strategies are designed to reduce total stress (transpulmonary pressure) and strain (the ratio between tidal volume and functional residual capacity) on the aerated lung tissue.

Individual patient and illness characteristics should be factored into clinical decision making and implementation of these recommendations

Role of NIV/ HFNC

Mild ARDS patients are managed using non-invasive respiratory support modalities (e.g. NIV/ HFNC) which improve oxygenation and unload respiratory muscles, thereby reducing inspiratory effort and allow time for the underlying disease to be treated, thus avoiding tracheal intubation.

Concerns of HFNC/NIV

- It may cause unnecessary delay in intubation, which is associated with increased mortality.
- NIV may cause P-SILI (Patient self-inflicted lung injury) due to the high transpulmonary pressures generated either due to high level of support, strong inspiratory effort, or both.
- Patients initiated on NIV should be closely monitored for signs of respiratory distress, breathing pattern and inspiratory effort to avert P-SILI.

Contraindications to NIV/ HFNC

- Moderate to severe ARDS (P/F ratio < 150)
- Respiratory / cardiac arrest

- Hemodynamic instability
- Inability to protect airway
- Severe facial trauma / burns
- Failure / intolerance to NIV/ HFNC

When to intubate in ARDS

- Moderate to severe ARDS (P/F ratio < 150)
- Contraindications to NIV or NIV failure

The lung protective ventilation strategies to be followed include:

- Low tidal volume ventilation — tidal volume 4-8 ml /kg PBW
- To overcome hypercapnia associated with low tidal volume ventilation, increase RR upto a maximum of 35 breaths per min to target ABG pH of 7.25-7.45
- Target plateau pressure < 30 cm H2O

(P-plat is obtained on performing inspiratory hold in a sedated and paralysed patient on Volume assist controlled ventilation, provided there are no leaks in the circuit and peak pressure is less than the P limit)

- Driving pressure (P-plat – PEEP) < 15 cm H2O
- PEEP adjusted to target SPO2 88-95% (PaO2 55- 80 mmHg)
- Recruitment manoeuvres are attempted only in situations of life-threatening refractory hypoxemia

Initial ventilation settings

- Volume Assist Control mode / Pressure Assist Control Mode
- Start with FiO 2 1.0
- Tidal volume 6-8 ml /kg PBW
- Measure the P-plat every 4 hours and after each change in PEEP or VT
- If P-plat > 30 cm H2O, decrease VT to 5 ml/kg or to 4 ml/kg
- Start with PEEP 8-10 cm H2O and titrate according to oxygenation and Pplat
- RR 20-30 / min, titrate according to ETCO 2 / PaCO 2 and ABG pH, maximum RR is 35/min

- I: E ratio 1:2 (acceptable range is 1:1 to 1: 3)
- The ventilator settings are adjusted according to the lung protection strategies described above.

Peep titration

- If oxygenation goals are not met, check for recruitability of lungs. Higher PEEP is beneficial in patients with recruitable alveoli.
- If on increasing PEEP, the P-plat decreases, driving pressure decreases and Compliance increases, then patient is a PEEP responder, higher PEEP may be beneficial.
- Optimal PEEP for a patient is the one which provides best lung compliance, improves oxygenation without causing hemodynamic compromise/ barotrauma and which protects the right ventricle.
- Optimal PEEP titration strategy should be tailored to clinical expertise. The strategies may include oxygenation-based titration, titration to maximal compliance or maximum safe plateau pressure.

Recruitment manoeuvre

It is the transient, sustained increases in Transpulmonary pressure to open up collapsed alveoli. Evidence for recruitment is conflicting. Shall be done in selected cases with Intra arterial BP monitoring and RV function assessment with echocardiography in view of acute corpulmonale associated with the procedure.

- CPAP 30 -40 cmH 2 0 for 30- 40 seconds
- 3 consecutive sighs/min with a P-plat of 45
- long slow increase in inspiratory pressure up to 40 cmH 2 O (RAMP)
- Staircase Recruitment Maneuver

Staircase Recruitment Manoeuvre (SRM)

- Ventilate in Pressure controlled mode of ventilation with FiO 2 1.0
- Set Pi to 15cms H 2 O above PEEP and maintain this difference
- Increase PEEP in a stepwise manner to 20, then 30 and then 40 cm H2O with adjustments made every 2 minutes (Pi will reach 55 cm H 2 O)

- Reduce PEEP to 25, then 22.5, then 20, then 17.5 cms and so on every 3 minutes until a decrease in SaO2 of >= 1 % from maximum SaO2 is observed (point of derecruitment)
- Increase PEEP to 40 cms H 2 O again for one minute, then return to a PEEP level 2.5 cms H 2 O above the point of derecruitment (the optimal PEEP)
- Then adjust to tidal volume < 6ml/kg PBW and Pplat <= 30 cm H2O SRM should be stopped if:
- HR < 60 or > 140/min
- New dysrhythmia
- SBP< 80 mmHg
- SaO 2 < 85 %

Prolonged lung recruitment manoeuvres (PEEP >35 cm H2O for > 60 s) are not recommended in patients with moderate to severe ARDS

Use of neuromuscular blockers

NMBA are recommended in early (48 h since ARDS onset) severe ARDS (P /F ratio<= 100) who have evidence of significant ventilator dyssynchrony with associated clinical deterioration that is not mitigated by adjustments to ventilator settings or sedation. NMBA duration should be limited to a maximum of 48 hours whenever possible owing to the risk of developing ICU acquired weakness, delay in mobilisation and deep vein thrombosis.

Fluid management

Once patient is out of shock, adopt a conservative fluid management strategy. Judicious use of fluids and diuretics according to fluid responsiveness and tolerance.

Prone ventilation

- Prone ventilation is recommended in moderate to severe ARDS patients receiving invasive mechanical ventilation early after intubation (within 48 hrs) if the P/ F remains < 150 with PEEP 5 cm and inspite of lung protective ventilation strategy.
- Average duration of proning is 16- 17 hours. Multiple proning sessions may be required.
- Stop proning when P/F >= 150 with PEEP= < 10 cm and FIO2 =< 0.6 4 hrs

after making patient supine.

Contraindications to prone ventilation

- Patients with facial/neck trauma or spinal instability
- Patients with recent sternotomy / major abdominal surgery or large ventral surface burn
- Patients with raised ICP
- Patients with massive haemoptysis
- Patients at high risk of requiring CPR or defibrillation

Extracorporeal membrane oxygenation

- Selection criteria for VV-ECMO should be carefully considered and focus on maximizing access for the individuals most likely to benefit from its use, specifically those with reversible aetiologies of respiratory failure and very severe hypoxemia
  (Pa O2 /Fi O2 ratio <80 mm Hg) or hypercapnia (pH <7.25 with Pa CO2 ⩾60 mm Hg) despite optimal conventional management, who are early (<7 d) in their ARDS course, and have few risk factors for futility of treatment. For patients meeting these criteria who present to facilities without ECMO capabilities, transfer to ECMO centers should be considered when feasible.
- VV ECMO in ARDS (EOLIA inclusion criteria) P/F < 50 for > 3 hrs
- P/F < 80 for > 6 hrs
- pH < 7.25 PaCO2 >= 60 for >6 hrs
- Relative Contraindications for ECMO Irreversible etiology of respiratory failure Mechanical ventilation > 7 days Immunosuppression
- Older age
- Systemic bleeding or other contraindication to anticoagulation Chronic medical condition and life expectancy < 1 year
- CNS haemorrhage or irreversible and incapacitating CNS pathology

Weaning from mechanical ventilation

- Acute phase of primary pathology cleared

- PaO 2 /FiO 2 >200 with FiO 2 < 0.4 and PEEP<= 5, SaO 2 > 90%
- PaCO 2 < 50 or previous baseline, pH> 7.25
- Spontaneous TV >5ml/Kg, Minute ventilation >5L and < 10 L RSBI <105 (RR/TV) 130 in elderly
- Hemodynamic stability (no / low dose vasopressor) Protective airway reflexes

Steroids in ARDS

Early ARDS (within 24 hrs)

- Dexamethasone 20 mg IV daily for 5days, then 10 mg IV daily for 5 days until extubation

Early ARDS (within 72 hrs)

Methylprednisolone 1 mg/kg IV bolus, then

- Days 1-14: 1 mg/kg/d
- Days 15-21: 0.5 mg/kg/d
- Days 22-25: 0.25 mg/kg/d
- Days 26-28: 0.125 mg/kg/d
- If extubated between days 1 and 15, then advance to D15 of the regime

Unresolving ARDS (7-21 d)

Methylprednisolone 2 mg/kg IV bolus, then

- Days 1-14: 2 mg/kg/d divided Q6H
- Days 15-21: 1 mg/kg/d
- Days 22-28: 0.5 mg/kg/d
- Days 29-30: 0.25 mg/kg/d
- Days 31-32: 0.125 mg/kg/d
- If extubated before day 15, then advance to D15 of the regime

Reference

1. American Journal of Respiratory and Critical Care Medicine Volume 209 Number 1 | January 1 2024
2. An Update on Management of Adult Patients with Acute Respiratory Distress

Syndrome An Official American Thoracic Society Clinical Practice Guideline

3. 2024 Focused update: Guidelines on use of Corticosteroids in Sepsis, ARDS and Community acquired pneumonia

14 Policy for prevention of venous thromboembolism

Purpose

This policy has been developed to ensure standardization of best practices in prevention of Venous Thromboembolism in hospitalized patients

Background

VTE is one of the major cause of deaths in hospitalized patients. Without thromboprophylaxis one in seven hospitalized patients may develop a deep vein thrombosis (DVT) or pulmonary embolism (PE). Approximately 10% of hospital deaths are due to PE.

Scope

This policy applies to all hospitalized patients in all patient areas.

Prevention of VTE can be primary or secondary

Primary: The preferred method is done using drugs or mechanical methods (Intermittent Pneumatic Compression)

Drugs – Unfractionated Heparin (UFH), Low Molecular Weight Heparin (LMWH), Fondaparinux, oral factor Xa or direct thrombin inhibitors.

Secondary: Early detection and treatment of subclinical venous thrombosis by screening medical patients with objective tests that are sensitive for presence of DVT. It is not commonly used as efficacy of this tests not well established.

Reserved for patients whom primary prophylaxis is contraindicated or shown to be ineffective.

Risk assessment

The risk depends on nature of acute illness and presence of individual risk factors. A full history and examination to be obtained to assess the risk.

Risk factors commonly encountered are heart failure, acute respiratory failure, sepsis, inflammatory bowel disease, thrombophilia, prolonged immobility for ≥ 3 days, age above 60, previous VTE, elevated D- dimer.

Items	Score
Active cancer (metastases and/or chemoradiotherapy in the previous 6 months)	3
Previous VTE (with the exclusion of superficial vein thrombosis)	3
Bedrest for ≥3 days	3
Thrombophilia	3
Recent (≤1 month) trauma and/or surgery	2
Elderly age (≥70 years)	1
Heart and/or respiratory failure	1
Acute myocardial infarction or ischemic stroke	1
Acute infection and/or rheumatologic disorder	1
Obesity (BMI ≥30 kg/m^2)	1
Ongoing hormonal treatment	1

High risk of VTE: ≥4 points. VTE: Venous thromboembolism; BMI: Body mass index.

The rate of VTE was

Low risk(score < 4)- 0.3 percent

High risk(score > 4)- 2.2 (receiving adequate thromboprophylaxis) and 11 percent (not receiving adequate thromboprophylaxis)

Risk factors	Score
(a) *Major*	3 points
Cancer	
Prior VTE	
Hypercoagulability	
(b) *Intermediate*	2 points
Major surgery within previous 30 days	
(c) *Minor*	1 point
Advanced age (≥75 year)	
Obesity (>29 kg/m^2)	
Bed rest	
Hormone replacement therapy or oral contraceptives	

The risk of symptomatic VTE or VTE related death at 90 days was

Low risk (score < 3): 0.6 percent (receiving adequate thromboprophylaxis) and 0.8 percent (not receiving adequate thromboprophylaxis)

High risk (score ≥3): 3.2 percent (receiving adequate thromboprophylaxis) and 3.5 percent (not receiving adequate thromboprophylaxis)

Bleeding risk assessment

History and clinical assessment to be done to assess risk of bleeding

High risk bleeding include patients with active bleed or intracranial hemorrhage, surgery planned in immediate 6- 12 hours, moderate to severe coagulaopathy and pts in severe bleeding diathesis or thrombocytopenia (platelet count < 50000/microL or < 100,000/microL, INR >1.5 plus additional risk factor for bleeding)

Epistaxis / menstrual bleeding are not contraindication for pharmacological thromboprophylaxis.

Strongest independent risk factor for bleeding at the time admission

1. Active gastric/ duodenal ulcer
2. Bleeding within 3 months prior to admission
3. Plt < 50000

Improve bleeding risk model

Risk factors	Point
Moderate renal failure (CrCl 30 - 50 ml/min.)	1
Male Sex	1
Age 40 - 84 years	1.5
Active Cancer	2
Rheumatic disease	2
Central venous catheters	2
Admissions in Intensive Care	2.5
Severe Renal Failure (CrCl < 30 ml/min.)	2.5
Liver insufficiency (INR > 1.5)	2.5
Age ≥ 85	3.5
Thrombocytopenia (<50 × 10^9 cell/L)	4
Recent (3 months) bleeding	4
Active gastro-intestinal ulcer	
High bleeding risk when total score ≥ 7	

Method of prophylaxis

1. Low risk patients — pharmacological prophylaxis is not warranted Early ambulation with or without mechanical thromboprophylaxis
2. Moderate risk patients - at least one risk factor for VTE and no risk for bleeding, pharmacological thromboprophylaxis
3. High risk patients – low risk for bleeding, pharmacological prophylaxis

Special population

1. Heparin Induced Thrombocytopenia (HIT)- Fondaparinux may be used as an alternative to heparin
2. At risk of bleeding — Intermittent Pneumatic Compression (IPC) and

graduated compression stockings is suggested over no prophylaxis. Transition to pharmacological agent should occur as the bleeding risk becomes acceptable low

3. Other special situations
    - Patient undergoing neuraxial anesthesia
    - Stroke
    - Pregnant
    - Cancer
    - Spinal cord injury/ Traumatic brain injury
    - Orthopedic and non-orthopedic surgery patients

Duration

VTE prophylaxis should continue till the patient is fully ambulant or discharged from hospital.

Methods

Pharmacological thromboprophylaxis

1. Low Molecular weight Heparin

    ### Efficacy

    General medical population: Compared with UFH, LWMH is associated with reduced risk of bleeding High risk population:

    Stroke — LMWH is associated with reduced risk for VTE with no increase in bleeding, Critically ill — Compared to UFH, LMWH is more effective in preventive symptomatic PE, but not symptomatic or asymptomatic DVT

    Older patients- In age ≥75years, LMWH comparable with UFH in preventing DVT.

    ### Dose

    Dosage of commonly used LMWH in patients with creatinine clearance > 30 ml/minute assuming no extremes in body weight are:

    ENOXAPARIN- 40mg subcutaneously once daily DALTEPARIN- 5000 units subcutaneously once daily

Creatinine clearance (ml/min) = $\frac{Y \times (140 - age) \times Weight\ (kg)}{Serum\ creatinine\ (\mu mol/L)}$ Male Y= 1.23

Female Y= 1.04

	VTE prophylaxis*
Enoxaparin	**CrCl ≥30 mL/min:** No adjustment **CrCl <30 mL/min:** Reduce to 30 mg once daily (medical or surgical patients)
Dalteparin	**CrCl ≥30 mL/min:** No adjustment

Platelet count may be monitored frequently to detect development of Heparin Induced Thrombocytopenia.

Ideal dose of enoxaparin for extremes in body weight is is unknown.

- Use ideal body weight (IBW) in patients unless over or under weight: IBW Females = E 45.5kg + (2.3 × every inch over 5ft)] kg

  IBW Males = E 50kg + (2.3 × every inch over 5ft)] kg
- Use adjusted body weight in obese patients= IBW + 0.4× (actual body weight-IBW) kg
- Use actual body weight in underweight patients.

	VTE prophylaxis
Enoxaparin*	**BMI 30 to 39 kg/m^2:** Use standard prophylaxis dosing (ie, 30 mg every 12 hours or 40 mg once daily). **BMI ≥40 kg/m^2:** Empirically increase standard prophylaxis dose by 30% (ie, to 40 mg every 12 hours). **High VTE-risk bariatric surgery with BMI ≤50 kg/m^2:** 40 mg every 12 hours. **High VTE-risk bariatric surgery with BMI >50 kg/m^2:** 60 mg every 12 hours.
Dalteparin	**BMI 30 to 39 kg/m^2:** Use standard prophylaxis dosing (ie, 2500 or 5000 units once daily). **BMI ≥40 kg/m^2:** Empirically increase standard prophylaxis dose by 30% (ie, increase to 3250 or 6500 units once daily).

		<50kg	50-100kg	100-150kg	>150kg
LMWH	Enoxaparin	20mg daily	40mg daily	40mg BD	60mg BD

In patients with severe renal insufficiency, LMWH should be discontinued and replaced with Unfractionated heparin (UFH)

2. Low Dose Unfractionated Heparin (UFH)

EFFICACY – Effective in preventing VTE as compared with placebo or mechanical device.

DOSE- 5000 units subcutaneous twice or three times daily dose. There was no difference in between twice daily or thrice daily dosing of UFH in rates of VTE, VTE related death or major bleeding.

In obese patients UFH 5000 to 7000 subcutaneous three times daily can be used.

3. Fondaparinux

EFFICACY: As effective as LMWH

DOSE - 2.5mg subcutaneous once daily. Avoided if in creatinine clearance < 30 ml/min. A dose reduction of 1.5mg subcutaneous daily dose can be used in creatinine clearance in range of 30 to 50ml/min.

4. Aspirin

   Less effective in preventing VTE.

5. Warfarin

   Warfarin is not indicated in thromboprophylaxis.

6. Direct oral anticoagulant

   Has been studied in extended duration settings and found to be effective as LMWH, but routinely not used.

7. Mechanical device

   Mechanical devices are indicated in patients in whom pharmacological prophylaxis is contraindicated Intermittent Pneumatic Compression (IPC) is better than Graduated Compression Stockings (GCS) and venous foot pumps.

   Advise the person to wear mechanical device for as much time as possible.

8. Combined methods

   No role in combining mechanical with pharmacological thromboprophylaxis in acutely ill medical patients. However combined methods may be used in high risk patients.

Special Population

Stroke

Acute ischemic stroke

Intermittent pneumatic compression starting at admission for those who have restricted mobility.

In addition to IPC , pharmacological prophylaxis is indicated with in 48 hours of acute ischemic stroke who have restricted mobility. Exceptions include patients with transient ischemic attack (TIA), minor stroke who are treated with dual antiplatelet therapy (DAPT) and patient received full dose of heparin or anticoagulant for other indications.

1. After intravenous thrombolysis – Delay pharmacological thrombo prophylaxis for 24 hours after iv thrombolysis. Start IPC at admission itself.
2. No intravenous thrombolysis - Start IPC at admission and low dose heparin (LMWH or UFH) can be added for patients those who are not being treated with DAPT for minor stroke.
3. On dual antiplatelet therapy (DAPT) — For pts with TIA or minor stroke who are receiving short term DAPT with aspirin or clopidogrel it is reasonable to use IPC alone and avoid anticoagulation for pharmacologic VTE prophylaxis.
4. Already on anticoagulation- For patients who are receiving oral anticoagulant at the time of acute stroke, IPC is started on admission. Low dose heparin can be used for VTE prophylaxis during the interval when the full dose oral anticoagulant is stopped.

Intracerebral Bleeding

IPC should be started on admission in pts with acute intracranial hemorrhage (ICH) Once ICH has stopped, add LMW or UFH one to four days from ICH onset for patients with lack of mobility. The risk of hematoma expansion may occur in certain settings like contrast extravasation in initial CT angiography, poorly controlled hypertension, large hematoma volume in which case it is useful to avoid use of pharmacological prophylaxis.

Subarachnoid Hemorrhage

For patients with subarachnoid hemorrhage and decreased mobility, IPC is started on admission and prior to aneurysm treatment. Heparin (LMW or unfractionated) can be added once the aneurysm is secured for patients who continue to have restricted mobility.

Acute coronary syndrome

- Consider VTE prophylaxis for people who are having antiplatelets for other condition whose risk of VTE outweighs risk of bleeding.
    - If the risk of VTE outweighs risk of bleeding consider pharmacological prophylaxis.
    - If the risk of bleeding outweighs the risk of VTE consider mechanical VTE prophylaxis.
- People receiving anticoagulant drugs as part of their treatment for acute coronary syndrome do not need VTE prophylaxis.
- Consider pharmacological prophylaxis for people at risk of VTE who interrupt anticoagulant therapy.

Traumatic brain injury (TBI)

IPC is initiated at time of admission with patients with traumatic brain injury. In addition to the mechanical thromboprophylaxis pharmacologic prophylaxis may be considered if brain injury is stable and benefit outweigh the risks. There is less evidence to support recommendations regarding the preferred agent, dose or timing of pharmacologic prophylaxis for DVT. However, it is reasonable to start pharmacological prophylaxis after 48 to 72 hours after TBI if brain injury remains stable.

Acute spinal cord injury

Consider VTE prophylaxis on admission with spinal cord injury. Choose IPC initially. Reassess risk of bleeding 24 hours after admission. Consider adding pharmacological VTE prophylaxis after 24 hours of admission who are not having surgery in next 24 hours.

Continue VTE prophylaxis in people with spinal cord injury for 30 days or until the patient

is discharged or mobile, whichever is sooner.

Pregnancy

A decision regarding use of pharmacological thromboprophylaxis must be made on individual basis with careful assessment of risk of VTE and the potential harm of bleeding. Examples of women who are at moderate to high risk for VTE include admission for medical or surgical reasons, patients on prolonged bed rest (>3 days) and those with additional risk factors for VTE during pregnancy (obesity, older maternal age, malignancy, ovarian hyperstimulation, multiparity).

The risk of bleeding with pharmacological prophylaxis is increased in patients with serious or severe bleeding unrelated to pregnancy, those at risk of bleeding because of imminent vaginal or caesarean delivery or at risk of severe bleeding due to antepartum complications (abruptio placenta, placenta previa, sub chorionic hematoma).

- Consider LMWH for all patients who are admitted to hospital if they are pregnant or gave birth, had a miscarriage or had a termination of pregnancy in past 6 weeks, whose risk of VTE outweighs their risk of bleeding.
- Do not offer VTE to women who are in active labour.
- If using LMWH in women who gave birth or had a miscarriage or termination of pregnancy, start 4-8 hours after the event unless contraindicated and continue for 7 days.
- Consider combining LMWH with mechanical prophylaxis for pregnant women or women who gave birth or had a miscarriage or termination of pregnancy in the past 6 weeks who are likely to be immobilized or have significant reduced mobility, relative to their normal or anticipated mobility for 3 or more days after surgery including caesarean section.

Caesarean section-

Low risk patients- IPC may be placed on all patients not already receiving pharmacologic thromboprophylaxis before caesarean section. Encourage early ambulation after Caesarean section.

High risk patients- Both mechanical and pharmacological thromboprophylaxis in women with high risk of VTE undergoing Caesarean section. Early mobilization is recommended.

Criteria for high risk include

- Previous VTE
- Any thrombophilia
- BMI >35kg/m^2
- ≥2 less prominent risk factors for VTE (PPH or infection, pregnancy complications such as obesity, hypertension, heart disease, multiple gestation preeclampsia)

Pharmacological prophylaxis is begun 6 to 12 hours postoperatively after concerns of hemorrhage has decreased.

Cancer

VTE risk is very high in patients with malignancy admitted with other medical condition or with primary admission for malignancy.

- For hospitalized patients with cancer and reduced mobility pharmacological thromboprophylaxis is preferred over mechanical methods unless there are contraindications.
- Hospitalized cancer patients without immobility also benefit from pharmacological thromboprophylaxis.
- Consider pharmacological VTE prophylaxis for people with myeloma who are receiving chemo with thalidomide, pomalidomide or lenalidomide with steroids. Choose either
    - LMWH
    - Aspirin (75 or 150mg)
- Mechanical thromboprophylaxis can be used in patients unable to receive pharmacological thromboprophylaxis.
- Consider pharmacological VTE with LMWH for people with pancreatic cancer who receive chemotherapy.

- For patients with cancer admitted for minor procedures or short chemotherapy data is scarce regarding routine thromboprophylaxis.
- For patients with cancer who are hospitalized and require thromboprophylaxis heparin (LMWH or UFH) is preferred over direct oral anticoagulants.

Palliative care

- Consider pharmacological VTE prophylaxis for people who are having palliative care. Take in to account risk of thrombosis, bleeding risk, life expectancy and views of patient and family members. LMWH/ fondaparinux can be used.
- Do not offer VTE to people in last days of life.

Neuraxial anesthesia techniques in patient receiving VTE Prophylaxis

UFH:

- Low dose thromboprophylaxis (5000 units subcutaneously two or three times daily): Neuraxial anesthesia techniques or catheter removal to be performed at least four to six hours after last dose.
- Higher dose thromboprophylaxis (7500 to 10000 units subcutaneously two or three times daily): Neuraxial anesthesia techniques or catheter removal to be performed at least 12 hours after last dose. And confirmation of a normal aPTT.
- Low dose subcutaneous UFH may be administered 1 hour after performing neuraxial procedure or removal of a neuraxial catheter.

Low Molecular Weight Heparin (LMWH):

- For a single injection neuraxial procedure, the first postoperative dose of LMWH is administered no earlier than 12 hours of neuraxial procedure.
- Indwelling neuraxial catheters may be maintained during once daily prophylaxis. For neuraxial catheters the first postoperative dose of LMWH should be administered at least 12 hours after the neuraxial procedure and the second dose to be administered no sooner than 24 hours after the first. The catheter should be removed at least 12 hours after the last dose of LMWH, and the subsequent dose to be administered 4 hours after catheter removal.

Fondaparinux:

- Present guidelines makes no recommendation for fondaparinux.
- Neuraxial anesthesia should not be performed 36 to 42 hours after the last dose of fondaparinux.
- Should not be administered for postoperative thromboprophylaxis after difficult neuraxial procedures (more than 1 needle pass, difficulty threading catheter), and should not administered for patients with indwelling catheter. Neuraxial catheter to be removed 6 hours prior to first postoperative dose of fondaparinux.

Non orthopaedic surgery

Venous Thromboembolism is common in the post-operative setting with half of this population at moderate risk for VTE.

Asses risk for thrombosis

VTE risk depends on procedure but patient related factors also play a role:

- Procedure related- The highest risk is in those undergoing major surgery (surgery lasting more than 45minutes), abdominal and thoracic cavity surgery, prolonged surgery (≥2 hours), emergency surgeries, postoperative immobilization for ≥4 days, critically ill patients who are confined to bed (extensive burns, brain/ spine injury, multiple trauma). Risk is low for patients undergoing minor, ambulatory procedure (elective hernia repair, thyroid surgery, carotid endarterectomy, minor skin excision).
- Patient related- patient related VTE risk factors are same as those of medically ill patients.

The risk categories correspond to modified Caprini model.

Each Risk Factor Represents 1 Point

- ☐ Age 41-60 years
- ☐ Minor surgery planned
- ☐ History of prior major surgery (< 1 month)
- ☐ Varicose veins
- ☐ History of inflammatory bowel disease
- ☐ Swollen legs (current)
- ☐ Obesity (BMI > 25)
- ☐ Acute myocardial infarction
- ☐ Congestive heart failure (< 1 month)
- ☐ Sepsis (< 1 month)
- ☐ Serious lung disease incl. pneumonia (< 1 month)
- ☐ Abnormal pulmonary function (COPD)
- ☐ Medical patient currently at bed rest
- ☐ Other risk factors_____

Each Risk Factor Represents 2 Points

- ☐ Age 60-74 years
- ☐ Arthroscopic surgery
- ☐ Malignancy (present or previous)
- ☐ Major surgery (> 45 minutes)
- ☐ Laparoscopic surgery (> 45 minutes)
- ☐ Patient confined to bed (> 72 hours)
- ☐ Immobilizing plaster cast (< 1 month)
- ☐ Central venous access

Each Risk Factor Represents 5 Points

- ☐ Elective major lower extremity arthroplasty
- ☐ Hip, pelvis or leg fracture (< 1 month)
- ☐ Stroke (< 1 month)
- ☐ Multiple trauma (< 1 month)
- ☐ Acute spinal cord injury (paralysis)(< 1 month)

Each Risk Factor Represents 3 Points

- ☐ Age over 75 years
- ☐ History of DVT/PE
- ☐ **Family history of thrombosis***
- ☐ Positive Factor V Leiden
- ☐ Positive Prothrombin 20210A
- ☐ Elevated serum homocysteine
- ☐ Positive lupus anticoagulant
- ☐ Elevated anticardiolipin antibodies
- ☐ Heparin-induced thrombocytopenia (HIT)
- ☐ Other congenital or acquired thrombophilia

For Women Only (Each Represents 1 Point)

- ☐ Oral contraceptives or hormone replacement therapy
- ☐ Pregnancy or postpartum (<1 month)
- ☐ History of unexplained stillborn infant, recurrent spontaneous abortion (≥ 3), premature birth with toxemia or growth-restricted infant

Interpretation

Surgical risk category*	Score	Estimated VTE risk in the absence of pharmacologic or mechanical prophylaxis (percent)
Very low	0	<0.5
Low	1 to 2	1.5
Moderate	3 to 4	3.0
High	≥5	6.0

Assess the risk for major bleeding- Risk stratification for major bleed is estimated as:

- General/ abdominal/ pelvic surgery- 1%
- Bariatric surgery- <1%
- Plastic and reconstructive surgery- 0.5 to 1.8%
- Vascular surgery- 0.3 to 1.8%
- Cardiac surgery- 5%
- Thoracic surgery- 1%
- Neurosurgery- Craniotomy: 1 to 1.5%; Spine surgery: <0.5%
- Major trauma- 3.4 to 4.7%

Individual risk factors for bleeding- Patients with individual risk factors for bleeding include those with active bleed as an indication for surgery, patients with ICH, patients who develop moderate or severe coagulopathy, and patients with an underlying bleeding disorder or thrombocytopenia.

Methods of thromboprophylaxis:

- Very low risk thrombosis risk: The risk of VTE is considered very low when baseline risk in the absence of prophylaxis is estimated to be less than 0.5%. Examples include young healthy adult undergoing minor outpatient procedure. (eg: LASIK surgery, cataract removal, skin biopsy, benign breast biopsy,
    - diagnostic endoscopy, nasal polyp removal, dilatation and curettage, colposcopy, fluid removal from joint effusion).
    - Patients at very low risk of VTE early and frequent ambulation is recommended over mechanical or pharmacological thromboprophylaxis.
- Low VTE risk: The patients in this category include those undergoing general or abdominal/ pelvic surgery with a caprini score of 1 to 2 or patients undergoing plastic surgery with a Caprini score of 3 to 4. Examples include those undergoing minor elective abdominal- pelvic surgery (eg: appendectomy, laparoscopic cholecystectomy) or minor thoracic surgery (eg: diagnostic thoracoscopy, video assisted biopsy). Other examples include patients undergoing minor vascular

procedures (vein ablation) and elective spine surgery (spine fusion).

- Mechanical /pharmacological thromboprophylaxis is recommended over no prophylaxis.

- Moderate or high risk for VTE: Moderate risk surgical patients are defined as patient undergoing general or abdomen/ pelvic surgery with a Caprini score of 3 to 4 or patients undergoing plastic surgery with a caprine score 5 to 6. Major gynaecological and urological surgery, major cardio thoracic surgery, bariatric surgery, neurosurgical procedures, non extensive trauma come under moderate risk. High risk surgical patients are defined as patient undergoing general or abdomen/ pelvic surgery with a Caprini score of 5 or more or patients undergoing plastic surgery with a Caprini score 7 to 8. Examples distal colorectal surgery, extensive pelvic surgery, major trauma, acute spinal cord injury, or cancer surgery.

  With low bleeding risk: In non orthopedic surgical patients at moderate risk for VTE and in whom bleeding risk is low, pharmacological prophylaxis is to be initiated, while in those at high risk it is highly recommended to start pharmacological prophylaxis.

  More aggressive prophylaxis in very high risk patients in the form of increased intensity of a pharmacological agent (eg: UFH three times a day, Enoxaparin twice a day) and or the addition of mechanical device. Patients in this category include those with Caprini score >8, patients with multiple risk factors, patients undergoing craniotomy or spinal surgery for cancer, major trauma involving brain and spine.

  With high bleeding risk: For patients with contraindications to pharmacological it is recommended to start mechanical thromboprophylaxis over no thromboprophylaxis. Switching to or adding pharmacological agent is done as soon as bleeding risk becomes low.

Timing of initiation

Should be individualized as per timing of surgery, type and duration of surgery, risk of bleeding and risk of VTE.

For patients in whom thromboprophylaxis is indicated and risk of bleeding is low

mechanical methods may commence before surgery and pharmacological agents ideally should commence within 2 to 12 hours post operatively. Fondaparinux is started 6 to 8 hours after skin closure.

Duration

In most cases VTE is continued till patient is discharged from hospital. Extended pharmacological VTE prophylaxis is not routinely recommended in most non orthopaedic surgical patients except for those who undergo major abdominal or pelvic surgery for cancer. Extended pharmacological VTE with LMWH is offered to this population at very risk of VTE up to 12 weeks post discharge.

Orthopedic surgery

The risk of postoperative VTE in orthopedic patients is among highest of all surgical specialties. Assess the risk:

At baseline orthopedic surgeries are considered high risk (hip and knee arthroplasty, hip fracture surgery, pelvic and multiple fractures) and low risk (foot and ankle fractures, tibial, shoulder and elbow surgery, arthroscopy) for VTE. Caprini score may be used to stratify risk however score is validated for non-orthopedic surgery.

Patient related risk factor VTE are same as those of medically ill patients. Total Hip or Knee Arthroplasty and Hip Fracture surgery

Low bleeding risk: In patients undergoing THR, TKR and hip fracture surgery

- Pharmacological prophylaxis with or without IPC is preferred. Agents include LMWH or direct oral anticoagulants (DOAC).
- Aspirin should not be used as sole agent for VTE prophylaxis but switching to aspirin after a short course (5 days) of DOAC may be suitable for low risk patients.
- Combining pharmacological with mechanical thromboprophylaxis in high risk patients is preferred.

High bleeding risk: For patients with contraindications to pharmacological it is recommended to start mechanical thromboprophylaxis over no thromboprophylaxis. Switching to or adding pharmacological agent is done as soon as bleeding risk becomes low

Timing of initiation:

LMWH and UFH: It is preferred to administer thromboprophylaxis 12 hours or more preoperatively and 12 hours or more post operatively.

Fondaparinux: Approved to start 8 to 12 hours after skin closure.

DOAC, aspirin: It is preferred to start LMWH or UFH then change over after initial 5 days.

Duration:

In patients with THR, TKR, HFS it is recommended to administer pharmacological prophylaxis for a minimum of 10 to 14 days and continued up to 35 days after surgery. In TKR shorter course can be preferred for 10 to 14 days.

Agent selection:

LMWH is the preferred agent. After initial LMWH DOACs and strategies that switch to aspirin are being increasingly used.

Dosing:

Enoxaparin-

- THR: 30mg subcutaneous every 12 hours or 40mg subcutaneous once daily started ≥12 hours before and ≥12 hours after surgery.
- TKR: 30mg subcutaneous every 12 hours or 40mg subcutaneous once daily started ≥12 hours before and ≥12 hours after surgery.
- HFS: 30mg subcutaneous every 12 hours or 40mg subcutaneous once daily started ≥12 hours before and ≥12 hours after surgery.

Dalteparin-

- THR/ TKR: 5000 units subcutaneous once daily started either ≥12 hours before and

≥12 hours after surgery.

LMWH needs dose adjustment in renal insufficiency/ obesity.

UFH: 5000u subcutaneous twice daily can be used. In obese patients 7500 units twice daily can be used.

Fondaparinux: 2.5 mg subcutaneous once daily. Contraindicated in weight <50kg and

avoided in renal failure.

Direct oral anticoagulants:

- Rivaroxaban- 10mg once daily ≥10 hours after surgery
- Dabigatran- 110mg 4 hours after surgery and then 220 mg once daily.
- Apixaban- 2.5mg twice daily starting ≥12 hours after surgery.

Other orthopedic surgeries:

a) Foot and ankle surgery:

Consider pharmacological VTE prophylaxis

- That requires immobilization
- Total anaesthesia time is more than 90 minutes
- The patients risk of VTE outweighs risk of bleeding

b) Upper limb surgery:

VTE prophylaxis not needed if giving local or regional anesthesia for upper limb surgery. Consider VTE prophylaxis undergoing upper limb surgery if the person's total time under general anaesthesia is over 90 minutes or where their operation is likely to make them difficult to mobilise.

c) Non arthroplasty knee surgery:

VTE prophylaxis is not needed for people undergoing arthroscopic procedure of knee where:

- Total anaesthesia time less than 90 minutes
- Person is at low risk for VTE.

Consider prophylaxis after surgery for 14 days for people undergoing arthroscopic knee surgery if:

- Total anaestesia time more than 90 minutes
- Risk of VTE outweighs bleeding risk.

Consider VTE prophylaxis for people undergoing other knee surgery (osteotomy/ fracture surgery) whose risk of VTE outweighs their risk of bleeding.

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