STANDARD TREATMENT GUIDELINES

CRITICAL CARE MEDICINE

DEPARTMENT OF HEALTH AND FAMILY WELFARE

GOVERNMENT OF KERALA

Committee for development of Standard Treatment guidelines in Critical Care medicine

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"Driven by the inspiration drawn from Shri Rajeev Sadanandan IAS, Additional Chief Secretary , Department of Health and Family Welfare, Government of Kerala, the process of preparation of Standard Treatment Guidelines (STG) was initiated by the Director of Medical Education Dr Remla Beevi. A.The process of developing and finalizing the STG's were coordinated by Dr.Sreekumari. K. Joint Director Medical education and Dr.Suma T K, Professor of Medicine and ably supported by a dedicated team of experts, including external faculty".

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Pinarayi Vijayan
Chief Minister

GOVERNMENT OF KERALA

Secretariat
Thiruvananthapuram

The Government is taking many initiatives to ensure providing quality health care to all. Out of the five missions launched by the Government, the Aardram mission is primarily focussed to improve Primary Health Care to provide standard health care facilities to people at grassroots. This initiative is complemented by strategic investment for the improvement of infrastructure in secondary and tertiary health care institutions to provide quality health care services.

I am happy to note that the Department of Health is also taking initiatives to bring standardization in treatment for various disciplines like Cardiology, Critical care, Diabetes Mellitus, Cancer Care, etc. It is a noteworthy initiative to improve the qualitative aspects of the health service delivery. I appreciate the efforts taken by the experts from Government sector and private sector from Kerala and also the subject experts from outside the state. I am hopeful that the introduction of standard guidelines for diagnosis and treatment will ensure better quality and consistency in health care.

I wish all the success to this endeavour.

Pinarayi Vijayan
Chief Minister

K. K. SHAILAJA TEACHER

MINSTER FOR HEALTH, SOCIAL JUSTICE
WOMEN & CHILD DEVELOPMENT
Government of Kerala

Thiruvananthapuram
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Message

With health indicators comparable to the developed countries, Kerala has always been a role model for other States. Both public health infrastructure and private sector health care services have a wide network covering the entire length and breadth of the State.

But instead of sitting on its past laurels, the State is now gearing up to take public health care system to the next level under the revolutionary programme Aardram. The mission's main objective is to completely transform public health sector making it people friendly, affordable for poorest and provide substantial state of the art infrastructure facilities.

The government is committed to regulate treatment costs, provide free medicines for life style diseases. Treatment protocols will be put in place for major diseases. Universal training to doctors and support staff will keep them updated on the latest developments in their respective fields. On the whole Aardram Mission promises to bring a total revolution in health sector like never before

I am happy to inform that Government of Kerala is publishing a book on the Standard Treatment Guidelines for multiple disciplines like Cardiology, Critical Care, Diabetes, Hypertension and may other ailments. The Protocol would serve as a unified standardized protocol for initiation of treatment and follow up of cases in medical colleges and other tertiary institutions both in the private and public sector. Experts from various medical colleges, Professional Associations, Subject experts from various parts of the country had worked together for publishing the initiative. I appreciate the whole team and various stakeholders for the efforts taken and wishing everyone success in this endeavour.

K K Shailaja Teacher

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Patient care has moved away from management by an individual based on personal knowledge and skill to an evidence based, team managed operation. Decisions are reviewed more rigorously post facto and their alignment verified with standard practice. With the mode of payment for care moving from out of pocket payments to third party payers there will be a demand for rigorous documentation and evidence of having conformed to standard practice. When analysis of big data and machine learning becomes the norm it will require a standard set of procedures to act as the baseline from which to measure deviations and differences in impact.

To meet the requirement of these developments in the field of medicine, it is necessary to have explicit, objectively verifiable set of standard operating procedures. They have to be prepared based on international guidelines with the highest acceptance, but have to be modified to suit local knowledge and practice, so that there is local ownership. Government of Kerala has been trying to get the guidelines prepared for some time now. I would like to thank and congratulate Dr. Sreekumari, Joint Director of Medical Education and Dr. T.K.Suma, Professor of Medicine, T.D. Medical College, Alappuzha who took on the task of preparing standard treatment guidelines and completed it through a long, consultative process. I also thank the conveners of the different thematic groups who coordinated the work in their field as well as the innumerable number of participants, in government and private sector, who contributed their effort and knowledge to improve the guidelines. Professional associations have also contributed in their fields. Their efforts have resulted in a product they and Kerala can be proud of.

Treatment guidelines cannot be static if they are to remain relevant. They must be updated based on new knowledge and the

experience of treatment based on these guidelines. To do this the group which prepared the guidelines has to remain active and have a system for collecting data on the results of practice based on these guidelines. I hope such an activity is institutionalised and periodic revisions of the guidelines are prepared and published.

I wish that these guidelines contribute to raising the quality of patient care in Kerala.

Rajeev Sadanandan IAS

Addl Chief Secretary

Health & Family Welfare Department

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 crash if not identified early and resuscitative measures started.

Not included are patients <18 years. Details of equipment's used, basic pathophysiology, details of different modes of ventilation and ventilator graphics are not included. Recommendations may change over time and will be updated accordingly.

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

Health care setting:

Secondary and tertiary health care, patient presenting in the outpatient department, emergency department or ICUs.

Outcome:

Early identification of critically ill, resuscitation in the proper manner and hence reduction in mortality

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 Hemoglobin in arterial blood
SAT:	Spontaneous awakening trial
SBT:	Spontaneous breathing trial
SAD:	Supraglottic air way disease

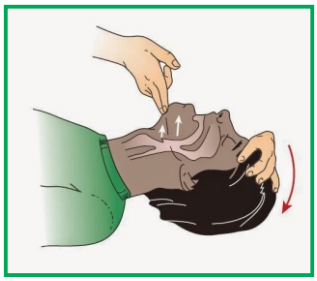
3. GENERAL ASSESSMENT & RESUSCITATION OF A CRITICALLY ILL PATIENT:

3.1. GENERAL GUIDELINES

1. All critically ill patients should be monitored adequately and steps initiated to prevent further deterioration.
2. All resuscitation is team work 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)

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.



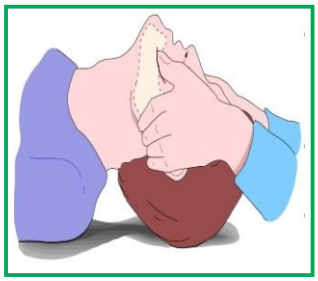
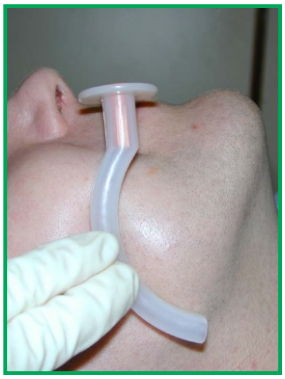


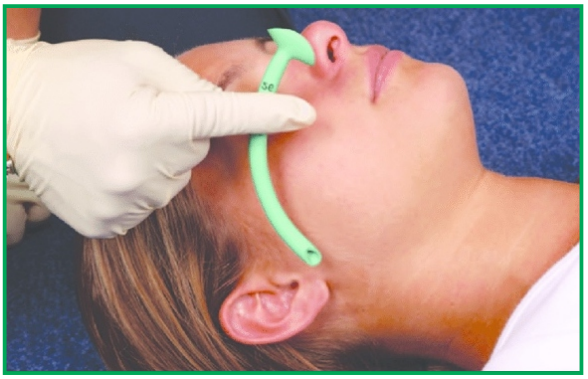
Figure1: a. Chin lift and head tilt b. jaw thrust

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/Tachypnea/ 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 hemothorax and drain immediately.
- Any evidence of massive lung collapse with desaturation requires intubation, suctioning, and positive-pressure ventilation.
- Noninvasive ventilation can be tried in relatively stable patients if they are suffering from a condition where noninvasive 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 SpO_2<90%
Post resuscitation	lowest oxygen administration to achieve SpO2>94%
Ventilated intensive care patients	Lowest O_2 to achieve SpO2 of 90-94%/PaO_2 of 60-80mmHg

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

3.4 . ENDOTRACHEAL INTUBATION:

3.4.1 Indications:

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

Hypoxemia

a. Acute respiratory distress syndrome
b. Hypoventilation
c. Atelectasis
d. Secretions
e. Pulmonary oedema

Hypercapnia

a. Hypoventilation
b. Neuromuscular failure
c. Drug overdose

3.4.2 Contraindication:

Severe airway trauma where Cricothyroidotomy /tracheostomy is indicated.

Special considerations during tracheal intubation in the intensive care unit

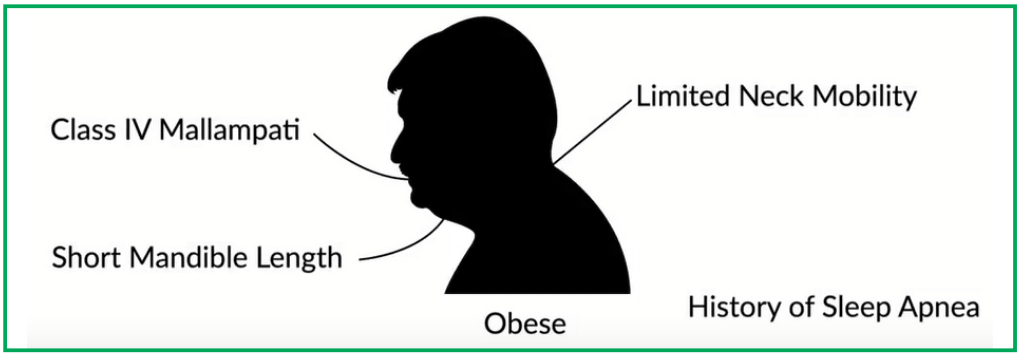
1. Presence of two operators (one experienced in airway management)
2. Call for additional help at the earliest
3. Preoxygenation: Three minutes of pre-oxygenation using non-invasive positive pressure ventilation
4. Hemodynamic instability: Drugs used for intubation like Propofol, Thiopentone, Diazepam 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.
5. Muscle relaxants: Use of neuromuscular blocking agents has been shown to improve the first-attempt intubation success.
6. Device: video laryngoscopy has been shown to increase the first-attempt success and improve glottis visualisation during intubation in the ICU.

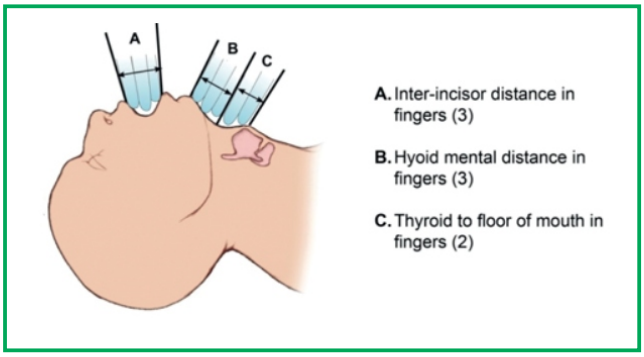
3.4.3. Difficult airway:

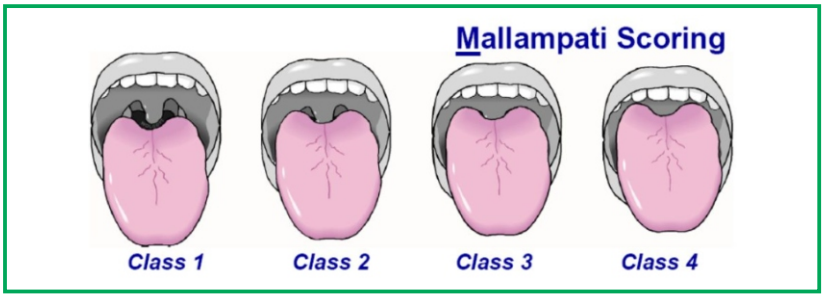
Look for signs of difficult airway:

- 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)
- Syringes for inflating the cuff
- Magill's forceps
- Stylet, Bougie, Tube fixation tapes/ties
- End tidal CO_2 monitor/disposable CO2 detector device
- Fiber-optic bronchoscope/ Video laryngoscope if available
- Drugs: Induction agents and muscle relaxants, topical anesthetics 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
Face Masks, ETT, Magill's Forceps, Stylet, Bougie	Video laryngoscope
Oropharyngeal/Nasopharyngeal airways	Flexible fiber optic bronchoscope
Manual self-inflating bag	Equipment for high flow nasal oxygenation e.g. THRIVE
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	

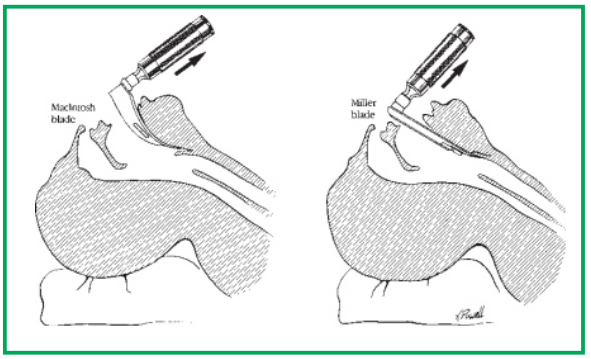
3.4.4 Drugs Used to Facilitate Intubation

(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 choline	1-2	45-60	Hyperkalemia, Increased ICP, Intragastric pressure
Rocuronium	0.6-1.0	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.

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.

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:

- – $EtCO_2$
- – 5-point auscultation
- – Direct visualization
- – CXR if clinically indicated

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

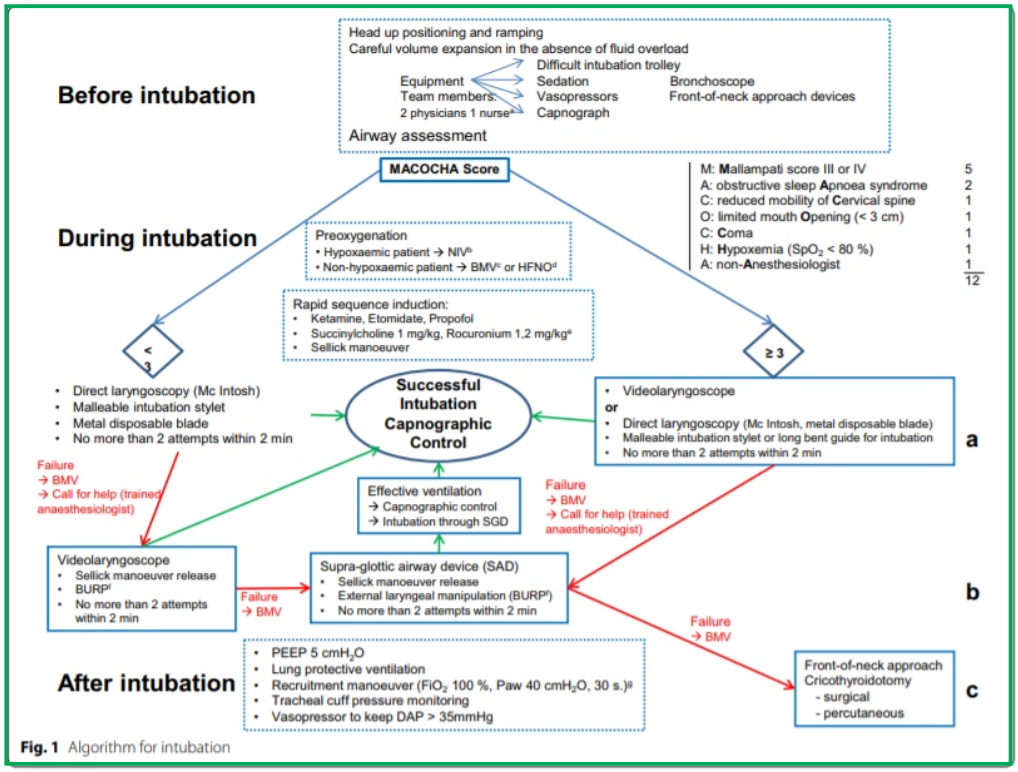


Fig. 1 Algorithm for intubation

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).

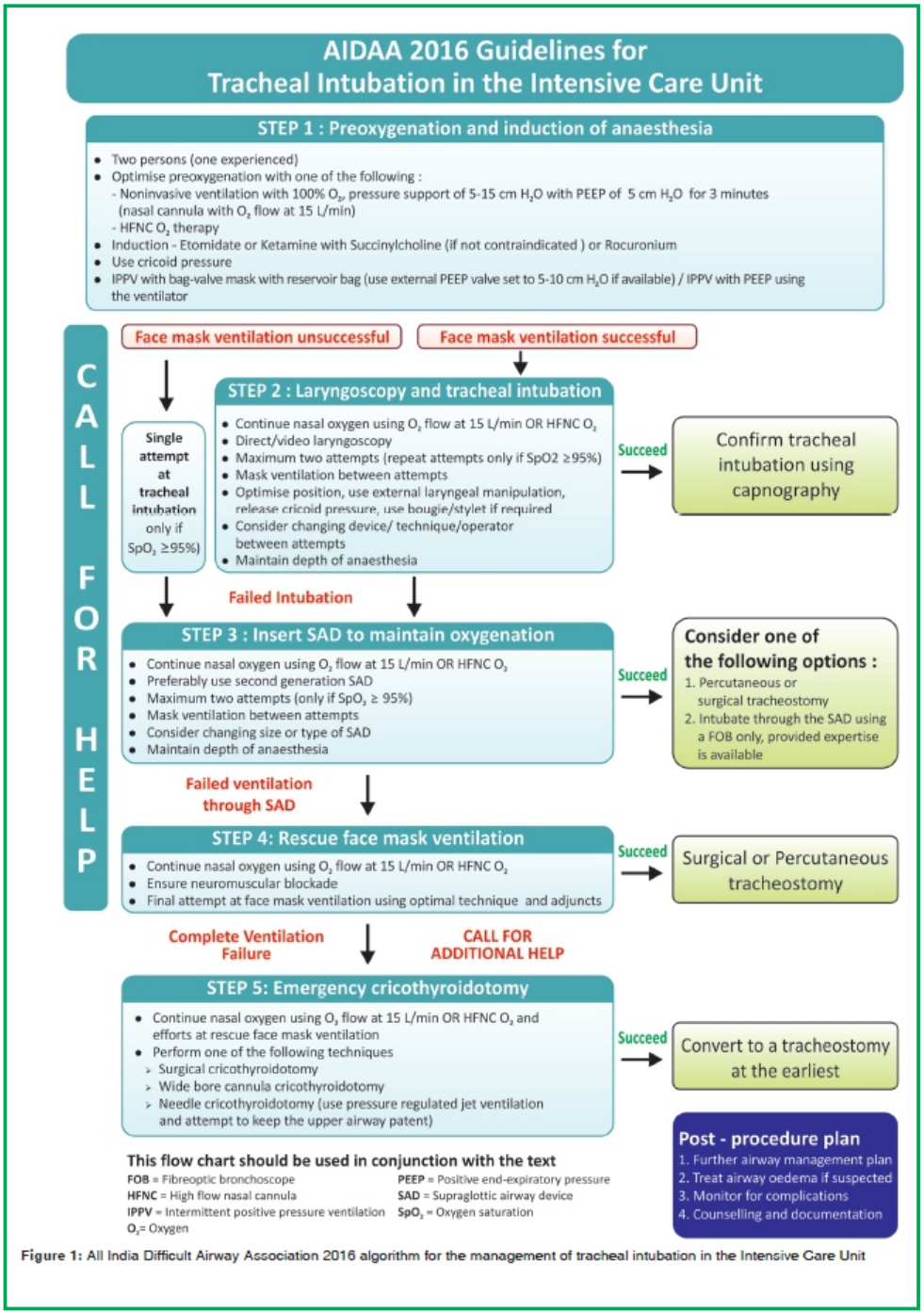
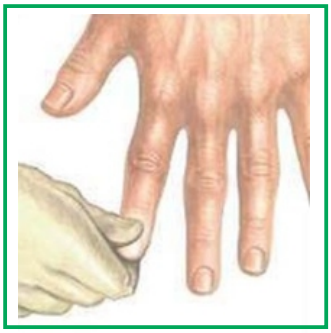


Figure 1: All India Difficult Airway Association 2016 algorithm for the management of tracheal intubation in the Intensive Care Unit

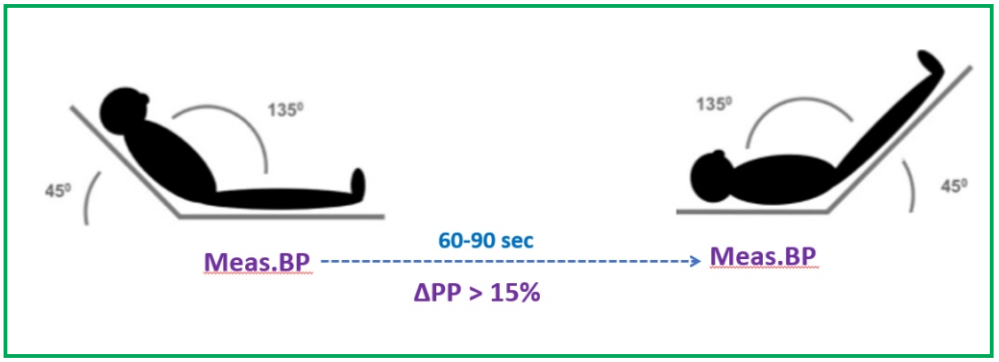
From The All India Difficult Airway Association 2016 guidelines for tracheal intubation in the Intensive care unit.

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
- 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
- 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 hypoglycemia and correct urgently.
- Control ongoing seizures with appropriate measures.
- Consider urgent antibiotics for patients with features suggestive of bacterial meningitis.

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

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°C and with an absolute humidity of 44 mg H_2O 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 intuabtion

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°C
4. FiO_2: Increase in FiO2 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, dyspnea 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 edema: 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 edema.
- 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.

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: 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 asthm a exacerbation		No recommendation
Immunocompromised	+++	Conditional recommendation for
De novo respiratory failure		No recommendation
Post operative patients	+++	Conditional recommendation for
Palliative care	+++	Conditional recommendation
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 H_2O and PEEP at 4 cm H_2O.
– 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:

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 noninvasive ventilation.
- In ventilator setting, look for air leaks, triggering and patient–ventilator

interaction.

- Monitor carefully the worsening respiratory distress, sensorium, tachypnea, 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
- In Pressure controlled ventilation set Pressures so as to meet target tidal volume (6-8ml/kg predicted body weight) and in volume-controlled ventilation measure /monitor the pressures needed to deliver the tidal volume.

6.3. CARE OF PATIENT ON VENTILATORY SUPPORT

- Analgo-sedation with Dexmeditomidine, Opioids, Benzodiazepines
- Comfortable, Co-operative and Calm
- Muscle relaxants are used sparingly (Severe ARDS, Intubation, prone ventilation etc)

- 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 $PaCO_2$.

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
- 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.

6.4 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. 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 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

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 hemoglobin 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. Wearing 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. Dyspnea 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: PaO_2/FiO_2>150-200 with FiO_2 <50% and PEEP <8mmHg, $PaCO_2$ 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	PaO_2<50-60mmHg on FiO_2 >0.5 or SaO_2<90%
	$PaCO_2$>50mmHg or an increase in$PaCO_2$>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, Dyspnea

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 noninvasive 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 apnea.

- 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.

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 edema/ 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
Hypokalemia
Hypomagnesemia
Hypocalcemia
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.
- Have good glycemic control (110–140 mg/dL).
- Correct metabolic factors (especially metabolic alkalosis).
- Maintain hemoglobin 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 etiology 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".

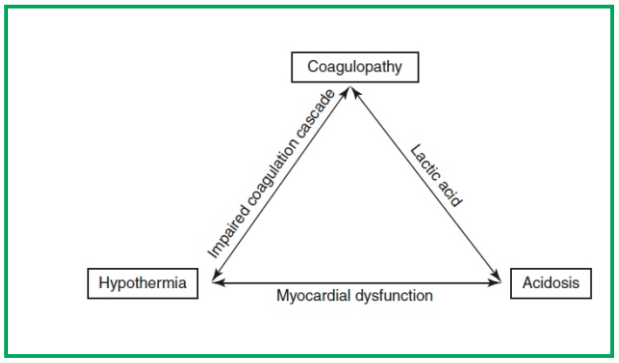
7.2. CLASSIFICATION:

	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:

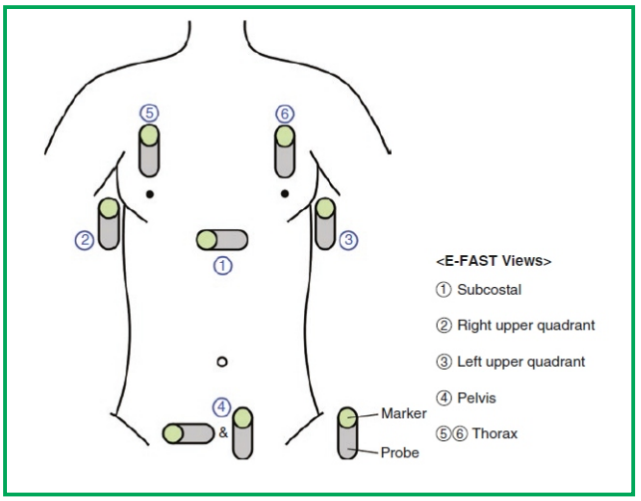
1.Hemmorhagic shock:

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



b. SOURCE: OBVIOUS

c. SOURCE: Unidentified: Imaging: USG EFAST



d. Serial measurements of hematocrit, 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 hemodilution induced by aggressive intravenous fluid
Transfusion of RBC:Palsma: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 hemorrhagic 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 Hemostasis 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 edema, an appropriate amount of fluid can be administered. If there is no improvement in perfusion with fluid challenge, or there is hypoperfusion with pulmonary edema, 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 decompressionICD

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	SpO_2>94%
Hemoptysis	1	No hemoptysis
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.

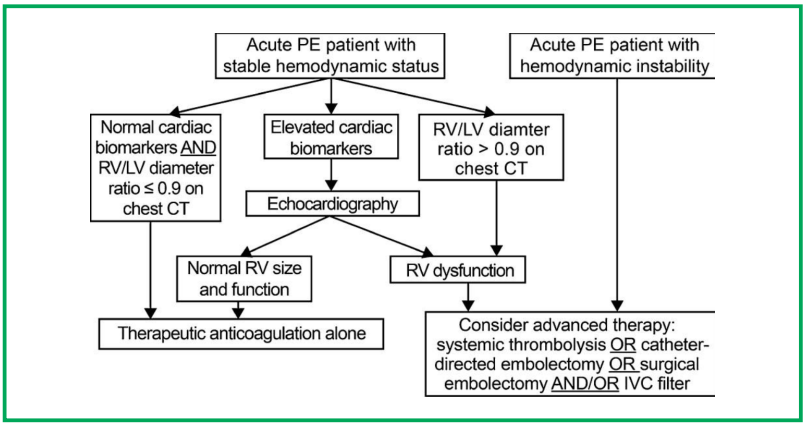
Catheter embolectomy can be considered when cardiopulmonary deterioration is evident or in sub-massive PE when patients have clinical evidence of adverse prognosis.(when cardio pulmonary deterioration is evident.)

Indications of thrombolysis:

1. Acute massive pulmonary thromboembolism (high-risk pulmonary

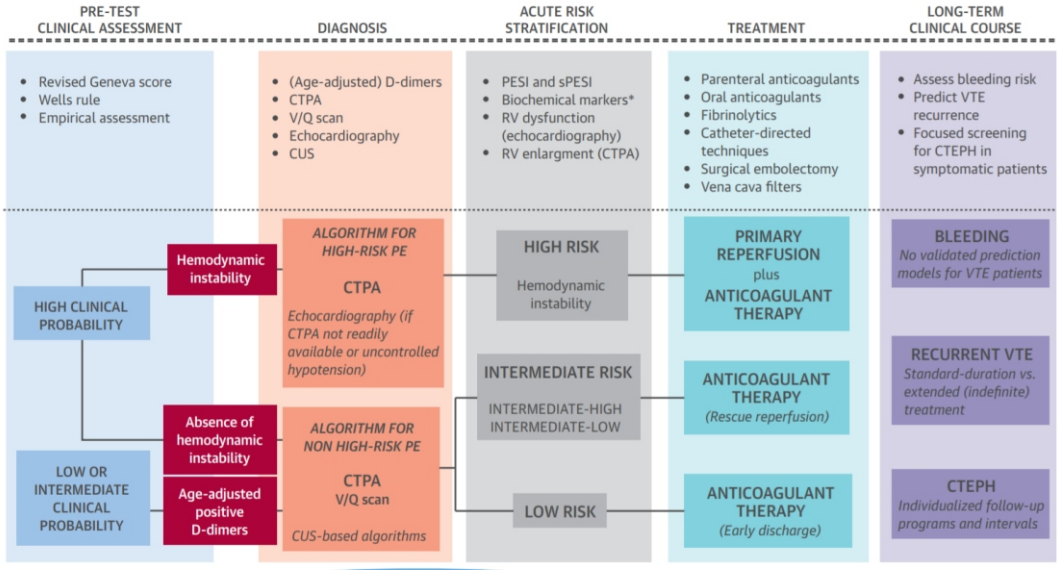
embolism, systolic blood pressure <90 mmHg, or a decrease in systolic arterial pressure of at least 40 mmHg for at least 15 min)

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

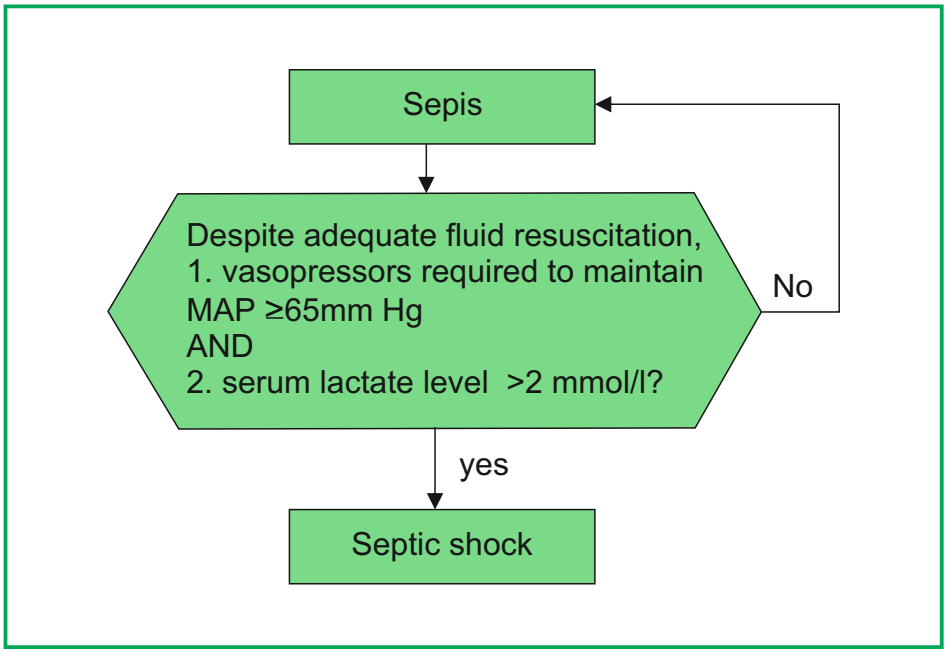
7.6. Distributive Shock:

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 5-15min
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 + H_2-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 first-line 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:

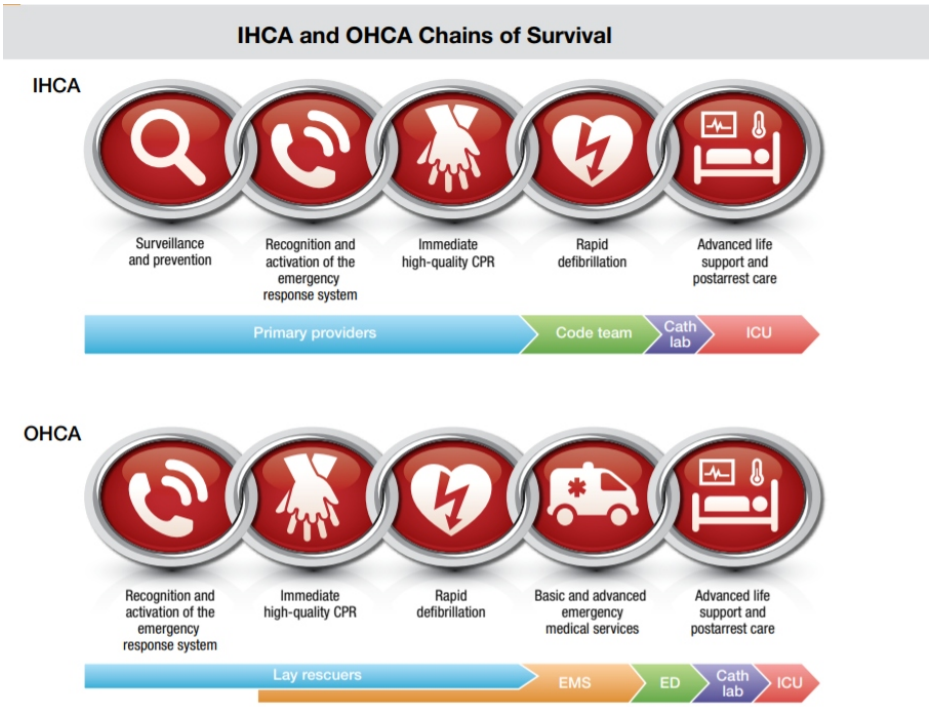
- Epinephrine auto injector.

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:

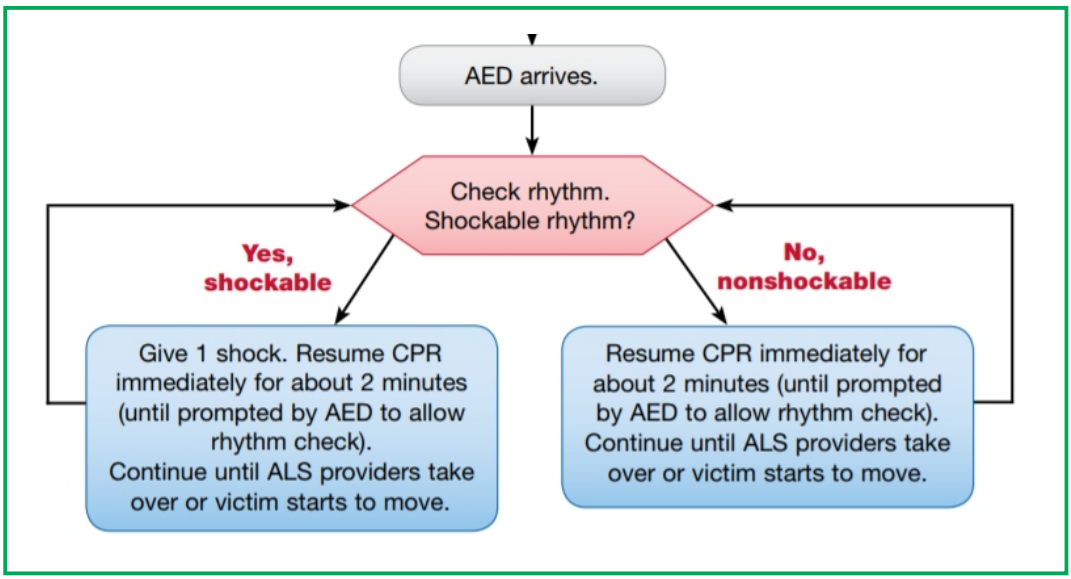
For witnessed adult cardiac arrest when an AED is immediately available, it is reasonable that the defibrillator be used as soon as possible. For adults with unmonitored cardiac arrest or for whom an AED is not immediately available, it is reasonable that CPR be initiated while the defibrillator equipment 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:

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 etiology 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^{0}C 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

CPCR IN PREGNANCY:

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 anesthetic systemic toxicity and particularly to patients who have premonitory neurotoxicity or cardiac arrest due to bupivacaine toxicity

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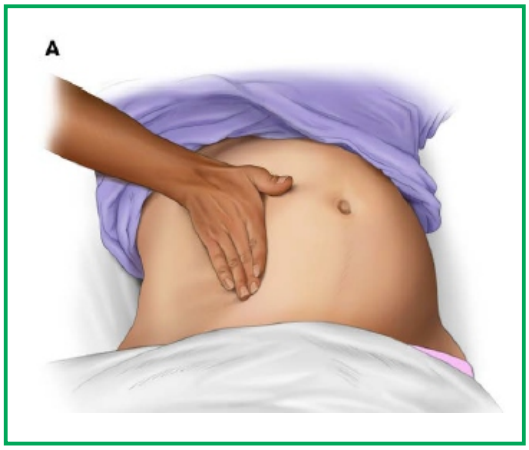

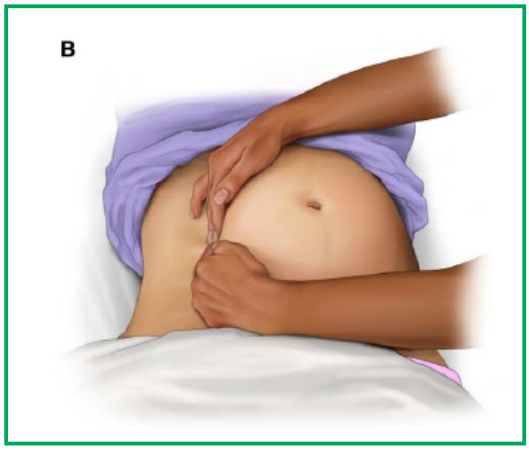

CARDIAC ARREST ASSOCITED WITH LOCAL ANESTHETIC TOXICTY:

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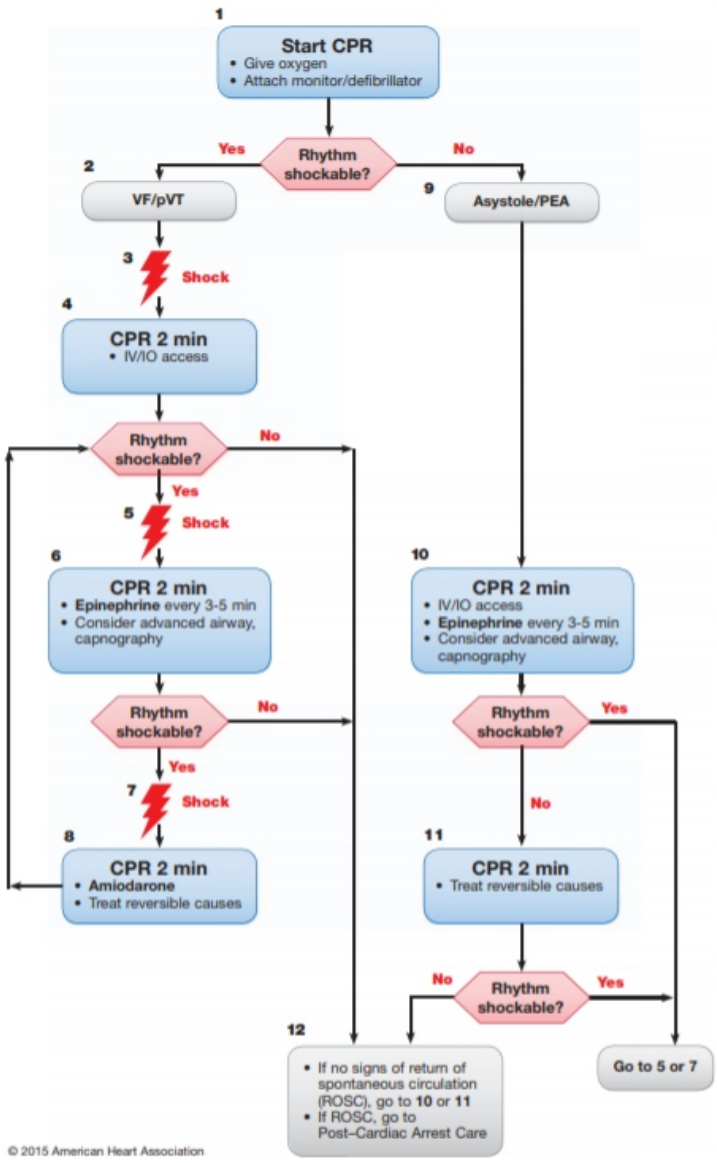
Adult Cardiac Arrest Algorithm—2015 Update



CPR Quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Rotate compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality.
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve 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.

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- 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

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

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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