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MINISTRY OF HEALTH - ETHIOPIA

CRITICAL CARE POCKET GUIDE

2022

FOREWORD

Ethiopia, being one of the developing countries trying to address the diverse health needs of its people. The increasing demand of intensive care units bed and equipment is one of it. The government is currently showing high commitment with proper critical care unit in place, along with the appropriate management of patients; our country can excel in providing high quality care to patients at all levels, and reduce ICU related mortalities.

This first edition of critical care pocket guide is aimed to help guide physicians and nurses to manage critically ill patients in Ethiopia. It is organized as simple as possible and context appropriate to be used in all level of health care professionals working in intensive care units of Ethiopia. The user should consider the ever changing medical practice especially in emergency and critically ill patients and recommendations may change with availability of medications and equipment.

HOW TO USE THE POCKET GUIDE

Each chapters in this pocket guide are organized in 4 sections. These sections include introduction, suggested management, management of common complications and monitoring. The suggested managements are written considering the availability and international guidelines. However, each patient's presentation as a critical illness is unique hence this guideline may not cover every patient presentation. The complete management of critically ill patients in the ICU depends solely on treating team. In addition, there are suggested documentation tools, admission, discharge guideline, and ethical guides, which can all be adopted to individual hospital.

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TABLE OF CONTENTS

LIST OF TABLES

SECTION I: GENERAL MANAGEMENT OF CRITICALLY ILL PATIENT

SECTION II: MANAGEMENT OF COMMON ILLNESS IN ICU

LIST OF FIGURES

SECTION I: GENERAL CONSIDERATIONS IN ICU

CHAPTER 1: ADMISSION AND DISCHARGE OF A CRITICALLY ILL PATIENT

1.1 ICU Admission

Patients who **requires more support than can be delivered on the general wards** and is able **to benefit** should be admitted. This will include, for example:

- All patients requiring **artificial ventilation for respiratory failure** and
- Monitoring for septic and cardiogenic shock but will exclude
    - Patients in whom terminal care is planned

Customize admission protocols for specific diseases as necessary. Whenever possible, measure of severity of illness objectively for priority setting.

- **Priority 1**: Critically ill, ***unstable patients*** in need of ***urgent intensive treatments*** which *cannot delivered elsewhere* such as ventilator support, inotropes etc.
- **Priority 2**: Critically ill patients requiring ***advanced monitoring*** and at risk for the need of immediate ICU treatment.
- **Priority 3**: Critically ill, unstable patients with underlying disease or acute illness, ***with less likelihood of recovery*** and /or benefit from ICU treatment.
- **Priority 4** : Little or no anticipated benefit (***too well to benefit***) from critical care or patients with terminal and irreversible illness (***too sick to benefit)***

Admission Procedure

- All admission should be communicated to ICU team
- **Closed or semi closed** ICUs are recommended for better patient outcome
- Emergency unit admissions for patients who need surgical intervention, the surgical team of the day should be involved in the admission of the patient

- Transfer from another hospital to ICU will be organized by Emergency Medicine with ICU team
- Patient in Operation Theater and obstetric patients get admission priority for functionality of the units

Once the decision to admit a patient has been made:

- Inform the ICU In-Charge Nurse and relatives.
- ICU team should prepare the bed and necessary materials to facilitate uninterrupted care
- Get consent for admission and ICU procedures should be signed
- ABC stabilization is a priority before proceeding to history and physical examination.
- Cardiac monitor should be attached and all vital signs are taken and recorded.
- The ICU Team, as well as the referring team should plan the patient management.
- Constant explanation and reassurance to the patient and family is mandatory to reduce anxiety

1.2 Discharge

Patients are discharged from ICU when the ***need for treatment is no longer needed*** or ***treatment has failed and/or consent for ICU treatment is witheld***.

The decision to discharge/transfer a patient finally rests with the ICU consultant or his / her representative.

Once a decision to discharge a patient has been made the ICU physician must:

- Inform and discuss with the referring Team, patient and relatives
- Inform the ICU nursing staff
- Ensure a ward bed is available and notified
- Complete a short discharge summary, medical and nursing transfer notes, investigations, x-rays and ECG strips are put together

CHAPTER 2: COUNSELING

2.1 Communication

- Patients and their families should receive timely information with or without request and when useful to make decisions
- Daily update of patient status to family members in a quit room is recommended.
- Information provided should be sensitive to the needs and preferences of patients and their families. Those who do not wish to have the information should be respected.
- Interactions with patient and family should involve nursing staff and social workers, and adequate documentation of events should reflect the spirit of discussions and not merely the decisions taken.
- Consider capacity of decision making; communicate with compassion

2.2 Information offered

- The stage of the disease
- Goals of care and outcomes
- Treatment and care options available
- Medications and their possible effects
- Availability and scope of services in the institution
- Any other help with documentation, insurance, healthcare benefits etc.

2.3 Basic ethical consideration

Patient rights: It is the patient's right to receive enough information regarding all treatments and procedures

Confidentiality: All patient related information is confidential.

Refusal of treatment: ICU staff will respect the patient or surrogates wishes to refuse specific treatments. This will be documented appropriately.

Privacy, safety and dignity: Verbal consent will be obtained as much as possible even for minor procedures (eg: nasogastric feeding, back care...)

Consent: Consent will be taken and documented for special procedures

Right to information: Patient and legally empowered next of kin have full access to all patient related records. Copies of these will be made available to them on request.

Social and Cultural needs: Every attempt will be made to recognize and fulfill cultural needs of the patients.

2.4 Care of the dying patient

The quality of ICU care is judged as much by the care of dying patients.

The following practice points are indicative of the best practices:

- If family wishes they can be at the bedside during the final moments, rrestrictions can be eased whenever possible if it does not impact on care.
- Reduce number of intrusive visits and tests.
- Ensure that patient is pain free and comfortable.
- Culturally appropriate support including sound religious leaders support can be provided as needed
- Family can bring “Holy water” or “Zam-Zam water” ….. as needed
- Discuss arrangements with family and inform them about hospital facilities for storage, embalming and paperwork and documentation.

CHAPTER 3: MONITORING IN ICU

3.1 Principle of care

- Monitoring ensures rapid detection of changes in the clinical status
- Allows for accurate assessment of progress and response to therapy
- When clinical signs and monitored parameters disagree, assume that clinical assessment is correct
- Trends are more important than a single reading and make assessment based on trends
- Use non-invasive techniques when possible
- Alarms are crucial for patient safety and should be checked frequently
- Results should be observed and interpreted in the context of the patient and treatment

3.2 Minimal noninvasive Parameters to monitor in ICU

- Respiratory rate (RR), SpO_2, Temperature (T), Heart rate (HR), Blood pressure (BP, MAP), Level of consciousness (AVPU), Urine output

Frequency of Monitoring

- During the early resuscitation phase, monitoring multiple parameters (not just one) is necessary to titrate interventions and guide actions.

Table 3-1: Parameters to be monitored continuously

Parameter	Frequency of monitoring	Limitations
RR, HR and SpO_2	Measure continuously using a non-invasive monitor Normal value is 98–100% (at sea level) goal in adults is more than 90%.	Requires a pulsatile signal – challenging with motion or poor perfusion, Does not measure ventilation (pCO_2), False readings can be seen with abnormal Hb or CO poisoning, Remember to remove nail polish if present!
BP (SBP, DBP, MAP)	Measure every 5–15 minutes during initial resuscitation of patients with shock. Once stabilized, can reduce to every 30–60 minutes. Consider invasive continuous monitor if refractory shock to fluids or need frequent arterial sample	Technically difficult to obtain in shock states Use appropriate cuff size Invasive blood pressure monitoring ,Benefits Directly measures arterial pressure, More accurate ,More reproducible, Continuous
Mental status	Monitor hourly	
(GCS or four scale)	If patient is receiving sedation, analgesia or mechanical ventilation, monitor sedation and pain with standardized scales every hour (RASS, etc.)	
Temperature	Measure at least every 3 hours	
Urine output	Measure hourly	
Physical examination	Focused examination of cardiovascular and respiratory system should be assessed every 30–60 minutes during the resuscitation phases of shock. Once patient is stabilized, can reduce to every 2–4 hours.	
Laboratory tests	As often as needed when managing shock and metabolic abnormalities (i.e. CBC, creatinine, electrolytes, glucose, and lactate). Avoid routine laboratory testing.	

Arterial blood gas analysis	Measure on arrival in patient with Severe hypoxaemia Risk of hypercapnea (e.g. COPD, depressed mental status) Risk of metabolic abnormalities (e.g. acidosis) Poor perfusion states making SpO_2 unreliable (e.g. shock) Deteriorating conditions Respiratory failure on invasive mechanical ventilation.	Invasive arterial puncture Heparinized syringe Can consider use of end-tidal CO_2 in conjunction with SpO_2 and RR to make assessment, understanding the limitations
Ventilator parameters (if patient on mechanical ventilation)	Every 2-4 hours. This includes: mode, expiratory tidal volume, respiratory rate (patient and machine), PEEP, FiO_2, I: E ratio, flow rate, compliance, plateau airway pressure, peak pressure, set inspiratory pressure (if using pressure control mode), or pressure support (if using spontaneous mode).	
Ultrasound	If possible on daily bases and as indicated in ventilated patients.	
Capnography in normal lungs, $P_{ET}CO_2$ is about 3–5 mmHg less than $PaCO_2$	During intubation and as needed	Limits: inaccurate if there is no discernable plateau: e.g. airflow obstruction. Underestimates $PaCO_2$ when there is decreased lung perfusion: Pulmonary emboli Hypotension, High PEEP, Severe ARDS, Emphysema

CHAPTER 4: NUTRITION THERAPY

4.1 Principle of care

- Critically ill patients are in catabolic stress state
- Early nutrition favorably modulate immune response. ***Feeding should be started as early as possible or within 48 hours***
- If hemodynamic instability, start after shock resuscitation
- Nutritional assessment should be done with indirect calorimetry
- Monitoring of nutrition must be done on daily bases and nutritional plan modified accordingly
- Plan for <u>parenteral nutrition</u> *if enteral nutrition cannot be achieved in 7 days.*

4.2 Feeding procedure

a. Feeding amount

- Use actual body weight unless the patient is obese
- Calories should contain ***70% carbohydrate, 30% fat and proteins***
- Start with 20kcal/kg, increase to 25 to 30kcal/kg within 5 days
- Protein requirement: 1.2-2.0g/kg actual body weight/day and 2g/kg for sever burn.
- <u>In acute kidney injury</u>*:* give 1.0-1.5 g/kg/day if not in dialysis, 1.5-2.0 g/kg/day if on hemodialysis (HD) and 2.0-2.5 g/kg/day if patient on CRRT.
- <u>In trauma patients:</u> give 1.5-2.0 g/kg/day and <u>traumatic brain injury:</u> 1.5-2.5 g/kg/day.

b. How to start enteral feeding

- It can be administered by continuous, intermittent or bolus methods.
- The head of the bed has to be elevated at 30-45 degree to prevent aspiration
- Absence of bowel sounds is not necessarily contraindication for enteral feeding.
- Look for abdominal distention, bloating, pain, increased residuals, diarrhea and dilated loops of bowel on films.

- If the patient cannot take orally 50% of required amount within 72 hr, or 100 % within 7 days start tube feeding
    - *Naso duodenal and naso jejunal tubes (transpyloric tubes):Is used when nasogastric causes frequent aspiration or if gastric emptying time is prolonged*
    - *Percutaneous gastrostomy, duodenostomy or jejunostomy tubes: these routes may be indicated in patients with esophageal stricture or following esophageal reconstruction*

Contraindication to enteral feedings

Resuscitated shocks, bowel obstruction, severe and protracted illeus, major UGIB, intractable vomiting or diarrhea, gastrointestinal ischemia

Locally Available Formula foods for enteral use in ICU

Formula diet is recommended whenever possible

- Mumbay formula is formulated with the following recipe:
    - 3 boiled eggs, 3 bananas, 3 tablespoons=50g sugar, 9 tablespoons=1.5dl full fat powder milk or 1.50dl full fat milk. Add filtered water to make totally 1 liter. Mix with blender and can be refrigerated up to 24 hours.

  The energy content of Mumbay formula per 1000ml is 1000 kcal.

  Has caloric density of approximately 1kcal/ml, protein content of about 40g/1000ml and have essential vitamins, minerals and micronutrients.
- **Plumpy'Nut:** a ready to use therapeutic food with packaging of sachet =92 g.
    - Energy/nutrient/100g is 500 kcal; 12.5g protein and 32.9g fat.

c. Parenteral Nutrition

- GI tract is not functional /cannot be accessed /Inadequate GI feeding:
- Consider TPN on day 3-7, if enteral nutrition is not possible or adequate
- Made up aseptically
- Start low and build up
- Usually given with central line in ICU – keep a clean port if PN may be needed.
    - Short term PN – can have PIC (need a different formula) or PICC

- Long-term TPN – tunneled subclavian catheter (Hickman) or subcutaneous port is usually inserted – OBSERVE STRICT ASEPSIS if handling these lines.

4.3 Manage complications of feeding

a. High gastric residual diet (residual volume is more tan 500ml or 50% of the feed)

- Decreasing the feed temporarily
- Making it continuous rather than bolus
- Addition of metoclopramide 10 mg IV or erythromycin and correct electrolyte
- Consider trans pyloric feeds

b. Diarrhea during enteral feeding

Cause could be: hyper osmolar formula, lactose intolerance, malabsorption, infectious causes or drug induced. **Management** of diarrhea during enteral feeding:
Electrolyte and rehydration therapy

- Avoid cessation of feeding
- Consider reducing rate and strength of feed
- Consider antibiotic associated colitis
- If intractable stop feeding till diarrhea stops

c. Dumping syndrome

- Inpatients with concentrated feedings
- Nausea, shaking, diaphoresis ad diarrhea.

d. Aspiration

- Prevention, motility agents, PEG, motility agents

4.4 Monitoring of nutrition

- Four to six hours gastric residual volume monitoring
- Look for Abdominal distention

Maintenance fluid

Table 4-1: Fluid requirement calculation

Maintenance (sensible &insensible losses, fever	4:2:1principle. Eg for a 50kg patient the 1st 10kgx4=40ml/hr; 2nd 10kg x2=20ml/hr; 3rd and above 30x1=30ml/hr. total 90ml/hr x24hrs= 2160ml/24hrs plus insensible loss (300-500ml/24hrs)=2460-2660ml/24hrs plus For each degree of fever above 37 , 2-2.5 ml/kg/day	0-10kg= 100ml/kg/24hrs 11-20kg =1000ml+50ml/kg for every kg above 10kg >20kg- 1500ml + 20ml/kg for every kg above 20kg/24hrs
Fluid deficit	Maintenance/hr x NPO time	
Ongoing loss	For 1ml blood loss 3ml crystalloid, for other losses 1:1	
3rd space loss	Depends on the size of the wound or surgical site and ranges from 4-8ml/kg/24hrs	

- **Maintenance volume:** consider both sensible and insensible losses, Fluid deficit – shock, NPO time, Ongoing loss – bleeding, drainages, any GI loss, 3rd space loss- fluid extravasation on the wound side

CHAPTER 5: GLUCOSE CONTROL IN ICU

5.1 Principle of care

- All oral anti hyperglycemic agents and subcutaneous insulin should be stopped. Insulin in critically ill patients should be given intravenously.
- The half-life of intravenous short acting insulin is 5 to 9 minutes, which allows for rapid reversal of hypoglycemia when it occurs.
- Reduce factors contributing to hyperglycemia including glucocorticoid therapy, dextrose-containing intravenous fluids, and dense caloric nutrition.
- Recommended **target** glucose level for ICU patients is **140mg/dl to 180 mg/dl.**

5.2 Suggested management

- Standard insulin infusion 100 U *regular human insulin* in 100 mL 0.9% normal saline.
- Divide initial BG by 70 and round to nearest 0.5 U (e.g., BG 250:250/70 = 3.57, rounded to 4, so IV bolus 4 U.)
- Give initial bolus if BG > *180* mg/dL
- After bolus, start infusion at same hourly rate as bolus (4 U/hr IV in above example).
- If BG less than 180 mg/dL, divide by 70 for initial hourly rate with NO bolus

5.3 BG Monitoring

- Check BG q1h until stable (three consecutive values in target range).
  - Once stable can change BG monitoring to q2h.
  - If stable q2h for 12–24 hours can change to q3–4h
- If no significant change in nutrition or clinical status, resume q1h BG monitoring for BG >70 md/dL with any of the following:
  - Change in insulin-infusion rate.

- Initiation or cessation of corticosteroid or vasopressor therapy.
- Significant change in clinical status.
- Change in nutritional support (initiation, cessation, or rate change).
- Initiation or cessation of hemodialysis or CVVHD.

Hypoglycemia (BG ≤70 mg/dL)

If *BG <50 mg/dL,* stop infusion and give dextrose 50% (1 amp D50) IV or 2 amp40%

- Recheck BG q10–15 min.
- When BG >90 mg/dL, recheck in 1 hr. If still >90 mg/dL after 1 hr, resume Insulin infusion at 50% most recent rate.

If *BG 50–69 mg/dL,* stop infusion.

- If symptomatic or unable to assess, give dextrose 50% or 40%.
  - Recheck BG q15 min.
- If asymptomatic, consider 12.5 g dextrose 50% (1/2 amp D50) or 8 oz. fruit juice PO.
  - Recheck q15–30 min. When, BG >90 mg/dL, recheck in 1 hr. If still >90 mg/dL after 1 hr, resume. Insulin infusion at 75% most recent rate.

Table 5-1: Current blood glucose (BG) level and rate of change

BG 70-89 mg/dl	BG 90-119 mg/dl	BG 120-179 mg/dl	BG > 180 mg/dl	Instructions (see Δ on table 5-2)
		BG ↑ by > 40 mg/dl/hr	BG ↑	INCREASE INFUSION by 2 Δ
	BG↑ by > 20 mg/dl/hr	BG↑ by 1-40 mg/dl/hr	BG UNCHANGED OR	INCREASE INFUSION by Δ
		OR BG UNCHANGED	BG ↓ by 1-40 mg/dl/hr	
BG ↑	BG ↑ by 1-20 mg/dl/hr,	BG ↓ by 1-40 mg/dl/hr	BG ↓ by 41-80 mg/dl/hr	NO INFUSION CHANGE

	BG UNCHANGED, OR			
	BG ↓ by 1-20 mg/dl/hr			
BG UNCHANGED OR	BG ↓ by 21-40 mg/dl/hr	BG ↓ by 41-80 mg/dl/hr	BG ↓ by 81-120 mg/dl/hr	DECREASE INFUSION by Δ
BG ↓ by 1-20 mg/dl/hr				
BG ↓ by > 20 mg/dl/hr	BG ↓ by > 40 mg/dl/hr	BG ↓ by > 80 mg/dl/hr	BG ↓ by > 120 mg/dl/hr	HOLD INFUSION x 30min then DECREASE by 2 Δ

Table 5-2: Change in Insulin-infusion Rate (Δ) in U/hr

Current-infusion rate (U/hr)	**Δ = Rate change (U/hr)**	**2 Δ = 2 x rate change (U/hr)**
< 3	**0.5**	**1**
3-6	**1**	**2**
6.5-9.5	**1.5**	**3**
10-14.5	**2**	**4**
15-19.5	**3**[a]	**6**[a]
20-24.5[a]	**4**[a]	**8**[a]
>25a	**5**[a]	**10**[a]

CHAPTER 6: SEDATION, PARALYSIS AND PAIN MANAGEMENT IN ICU

6.1 Pain management

I. Principle of care

- Patient COMFORT should be the goal, and includes adequate pain control, anxiolytics and prevention and treatment of delirium.
- Light/no sedation is the standard of care for most patients
- Deep sedation may cause respiratory, CVS, neurological, psychological and immunological complications and contribute to risk of death.

Table 6-1: Behavioral Pain Scale (BPS)

Item	Description	Score
Facial expression	Relaxed	1
	Partially tightened (eg. Brow lowering)	2
	Fully tightened (eg. Eyelid closing)	3
	Grimacing	4
Upper limb movement	No movement	1
	Partially bent	2
	Fully bent with finger flexion	3
	Permanently retracted	4
Compliance with MV	Tolerating movement	1
	Coughing but tolerating	2
	Most of the time fighting ventilator	3
	Unable to control ventilation	4

- BPS Score ranges from 3 no pain to 12 maximum pains
- Patient's **self-report of pain** using a10-point pain scale is most reliable (**gold standard)**
- For non-communicative or sedated, use a behavioral pain scale which is score based on facial expression, limb movement, muscle tension and ventilator compliance other method includes.

II. Suggested management of pain

- Give pre-emptive analgesia to alleviate pain prior to invasive or potentially painful procedures.
- Preferably opioid drug: *Fentanyl (0.35-0.5mic/kg, q half to one hr*, and the infusion rate is 0.7-10mic/kg/hr), and *morphine, (IV 2 – 4mg/q4 – 6 hrs or 10 – 20micg/kg* bolus according the response of the patient and the infusion rate is 5 – 40micg/kg/hr).
- *PO administered gabapentine, carbamazepine, or amitriptyline,* in addition to IV opioids, can be considered for treatment of neuropathic pain.
- *Pethidine* has neurotoxic effect especially when renal impairment is identified or expected. When there is no other drug of choice the dose is 0.5 – 1mg/kg/6 hrs.
- *Ketamine* can be used as analgesic, sedative and bronchodilator in asthmatic patients. Loading dose is 0.5mg /kg bolus, followed by 0.1 – 0.5mg/kg/hr infusion.
- *Acetaminophen 325-1000mg/4-6hr*s and the daily maximum dose is less than 4gram.
- *Regional analgesics* include epidural analgesia, peripheral nerve blocks, plane blocks (transverse abdominis muscle plane block, serratus anterior block).

6.2 Assessment and management of Delirium

Delirium is fluctuation in consciousness associated with inattention and disorganized thinking or perceptual disturbance that develops over short period of time.
Is an independent predictor of death.

Have three types: *hypoactive, hyperactive and mixed.* Hyperactive is least common but; easiest to diagnose.

It may be due to secondary conditions i.e., pain, primary intracranial process, hypoxemia, shock, infection, electrolyte abnormalities, metabolic disturbances, medications. Benzodiazepines are a common culprit or drug withdrawal.

Recognize delirium with Confusion Assessment Method (CAM-ICU) score.

Management of delirium

- Treat the underlying medical conditions
- Stop delirium producing/exacerbating drugs (i.e. benzodiazepines).
- Use non-pharmacologic interventions
  - Sleep hygiene: Protect patient sleep cycles by controlling light, reducing noise and stimuli at night, eye shades, ear plugs, cluster patient activities.
  - Orientation: re-orient patient to surroundings, provide reassurance and encourage family visits, have familiar objects in room, Provide visual aids, hearing aids, TV during the daytime, music
  - Early mobilization and exercise
  - Remove tubes and restrains as soon as possible

6.3 Assessment and management of agitation

- Patients may feel an exaggerated sense of fear, nervousness or apprehension.
- Patient may also manifest with hypo-activity and be withdrawn, distrustful or have blunted affect or can present with agitation or increased motor activity. Due to the primary illness (i.e. sepsis) or from the care itself (i.e. medication related).

Recognizing Anxiety: In adults and children, the Richmond Agitation-Sedation Scale (RASS) is easy to use. In children, the Comfort-B scale is commonly used (Table 6-2: Comfort-B scale)

Table 6-2: Comfort-B scale

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

Management of agitation

The pharmacologic options include:

Propofol (5 – 80 micg/kg/min, on average 50 – 100mg/hr in an adult),

Dexmedetomidine (0.2 – 0.7micg/kg/hr), ketamine (0.5 – 1mg/kg/hr),

Fentanyl (0.5 – 1micg/kg/hr or on average 20 – 50micg/hr), and

Midazolam (1 – 3mg/hr)

Sedation Vacations: daily sedation interruption or a light target level of sedation to minimize sedation drugs side effect, to facilitate patient assessment, to start weaning and to facilitate removal from the mechanical ventilator.

CHAPTER 7: TRANSPORT OF THE CRITICALLY ILL PATIENTS

- A person with intubation skills, and BLS/ACLS trained physician and nurses should be available during transpiration
- The safety profile of a transport of a critically ill patient should be as similar as the condition in an ICU itself.
- Before moving the patient communication with the receiving department or hospital should be maintained and confirmed
- Referral notes should completed clearly and purpose of transferring is included
- High speed journeys must be avoided except when clinically necessary,
- Light and sirens may be used to aid passage through traffic to deliver smooth journey
- To improve ease of transport of critically ill patients the following guidelines should be followed
- Fully sedate and paralyze all ventilated patients before transport. Drugs for paralyses and sedation in addition to emergency drugs should be available as syringes for bolus administration.
- All infusions, should be closed as much as possible
- Syringe drivers or volumetric pumps only allowed for essential drugs, e.g. inotropes.
- Monitoring equipment for pulse rate, SaO_2, ECG +Portable monitor (MMS)
- Check that request forms & essential x-rays are available.
- Transports will preferably not occur during rounds.
- An ICU nurse should accompany all patients.
- An ICU doctor should accompany all unstable and critical patients and should have air ways kills(preferably SR)
- Oxylog + two O_2-tank used for ventilation of transported patients
- AMBU bag for manual ventilation and essential drugs/equipment.
- Ensure appropriate emergency treatments have been given and patient is stable and ready for transport.
- Ensure appropriate documentation and handover of care to next responsible clinicians.

CHAPTER 8: PREVENTION OF ICU COMPLICATIONS

Table 8-1: Common complications in ICU to prevent includes

1. Ventilator associated pneumonia

- Oral intubation preferable to nasal
- Use a new ventilator circuit for each patient
- Keep patient in semi-recumbent position: head of bed 30°to 45°
- Perform regular antiseptic oral care:
  - Chlorhexidine mouthwash or gel preferred
- Once patient is ventilated, change circuit if it is soiled or damaged but not routinely
- Periodically drain and discard condensate in tubing.
- Use in-line closed suction system
- In adults, change heat and moisture exchanger when malfunctions, soiled, wet or every 5–7 days
- Consider specialized endotracheal with subglottic suctioning devices:
  - Limit aspiration of oropharyngeal secretions
- Preform daily, coordinated SBT

2. Gastric ulcer bleeding

Critically ill patients are at increased risk for gastric mucosal injury:

Risk factors:

- Impaired blood flow to the mucosa
- Accumulation of gastric acid
- IMV for more than 48 hours
- Presence of coagulopathy or thrombocytopenia

Prevention

- Maintain hemodynamics (e.g. early resuscitation)

- Liberate from IMV as soon as possible (e.g. SBT)
- Start early enteral nutrition for mucosal protection.
- Pharmacologic reduction of acid production:
- Histamine-2 receptor blockers (H2R)
- Proton pump inhibitor (PPI): Omeprazole 40 mg once or twise daily

3. Catheter associated blood stream infection

- Wash hand wash, use hair cap, face shield
- Wear sterile gown and sterile gloves
- Cover entire patient with full sterile sheet
- Use chlorhexidine to clean skin
- Sterile technique while using it.
- Daily reminder to remove if no longer needed
- Sterile technique when every you use the line
- Cover the tips with sterile cover
- Select subclavian site whenever possible

4. ICU acquired weakness

- ICU-acquired weakness is characterized by neuromuscular weakness and physical limitation
- Weakness due to:
    - Direct damage to nerves or muscles
    - Inflammatory states
    - Drugs (e.g. NMB or steroids)
    - Metabolic (e.g. hyperglycemia, malnutrition)
    - Immobility and atrophy.

Early mobility and exercise protocol

- Step 1: Recognize readiness to exercise
- Step 2: Conduct appropriate level of activity based on RASS score
- Step 3: Evaluate performance

- Step 4: Rest for next day
- It is safe and feasible to do in critically ill patients on mechanical ventilation.
- Improves patient outcomes:
- increases muscle strength, functional mobility and independence
- Reduces delirium
- Reduces days of IMV
- Reduces ICU length of stay

SECTION II: MANAGEMENT OF COMMON ILLNESS IN ICU

CHAPTER I: RESPIRATORY CRITICAL CARE

1.1 AIRWAY MANAGEMENT

I. Introduction and Principle of Management

Clinical management of airway includes airway maintenance and ventilation, protecting air way up to intubation and extubation and difficult airway management. Patients who cannot protect their airway or those who need invasive ventilation are the commonly admitted to ICU. The initial approach for such patient will be opening the airway and keep the patency.

II. Suggested management

How to open the airway?

a. Non equipment

Head tilt / chin lift /: cautiously in trauma patient for whom c-spine fracture is not ruled out

Jaw thrust: cautiously in facial trauma or TMJ unstable patients

b. With equipment

- Oral/nasopharyngeal airway,
- Endotracheal intubation,
- Laryngeal mask airway (LMA),
- Cricothyrotomy,
- Tracheostomy

Here are the steps of airway management:

Preparation: making ready patient, airway equipment and medications needed for the procedure

Positioning: sniffing position is ideal

***Pre-oxygenation**:* holding 100% oxygen for spontaneously breathing patient

***Paralysis**:* giving anesthetic medications for sedation, analgesia and relaxation

Positive pressure ventilation: giving 4-6 breaths with bag mask to build a reserve-contraindicated in COVID19 patients

Placement of the tube: putting endotracheal tube into the trachea and also check for proper placement

***Post tube placement care**:* securing the tube with tapes to the mouth of the patient

III. Management of complications

Complications can be:

a. During laryngoscopy and intubation

- Malpositions (esophageal or endobronchial): check tube placement immediately

Techniques checking proper tube placement in ICU:

- Look for bilateral chest movement
- Look for foggy appearance in the ETT
- Auscultate for bilateral air entry
- Direct laryngoscopic view
- Capnography(gold standard)
- Chest x ray

Trauma: intubate gently after patient is paralyzed

Aspirations: shorten time of intubation to less than 10s, GI suctioning before induction if possible

Physiologic reflexes (hypertension, tachycardia): give adequate induction medications, give IV lidocaine 2% as 1-1.5mg/kg to blunt airway reflex or use opioids

Tube malfunction (cuff perforation): check the cuff before the procedure don't push hard against the teeth

b. While tube in place

- **Mal-positioning (unintentional extubation, endobronchial)**: proper pain management, sedation and paralysis if necessary, restrain the upper limbs
- **Airway trauma:** avoid patient fight with adequate sedation-analgesia
- **Tube malfunction(kinking):** check patient position frequently especially while in prone position
- **Aspiration:** at least, check twice/day the cuff pressure with gauge if available or manually

c. Following extubation

- **Airway trauma(edema,stenosi):** nebulisation and dexamethasone 4mg iv TID for 24-48hours
- **Physiologic reflexes:** IV lidocaine, dexmedetomidine infusion for the immediate post extubation period
- Laryngospasm- encourage to cough out secretions, suctioning gently, IV lidocaine, Positive pressure ventilation, Paralysis and reintubation if not responding to the therapy
- **Aspiration:** extubate to semi sitting position, chest physiotherapy

IV. Monitoring and problem solving

- Follow BP, PR, RR and PSO_2
- Have CXR after airway management

1.2 OXYGEN THERAPY

I. Introduction and principle of care

The oxygen therapy is a universal treatment in the hospital setting, especially in the critical care units. The purpose of this therapy is to avoid hypoxemia and to ensure an adequate supply of oxygen to the tissues. The major indications of oxygen therapy are hypoxemia (PaO_2<60mmHg), disability to deliver oxygen to tissues and hypercarbia ($PaCO_2$>45mmHg), failure in the elimination of CO_2.

Table 1-1: Causes of hypoxemia

Causes of hypoxia	PaO_2	$PaCO_2$	P (A-a) O_2 gradient	Alveolar ventilation	Response to FiO_2 of 1
Reduction of FiO_2	↓	↓	=	↑	Yes
Hypoventilation	↓	↑	=	↓	Yes
V/Q mismatch	↓	↑↓	↑	= ↑	Yes
Shunting	↓	↓	↑	↑	No
Diffusion alteration	↓	↓	↑	↑	Yes

Table 1-2: Main ranges of arterial oxygen saturation ($SatO_2$) in different situation

Indication	Example	Initial therapy	SaO_2target
Critical patients	Polytrauma Shock	Mark with reservoir bag at 15 lmp	94% -98%
ARF type I	Asthma Pulmonary embolism Pulmonary edema Pneumonia	Nasal cannula at 2-4 lmp Or Facial mask at 5-10 lmp	94% -98%

ARF type II	COPD Bronchiectasis Obesity Sleep apnea Neuromuscular diseases	Nasal cannula at 2-4 lmp Or Facial mask at 5-10 lmp	88% -92%
Special situations	Carbon monoxide poisoning Pneumothorax	Mask with reservoir bag at 15 lmp	94% -98%

II. Suggested management

a. Nasal cannula: the tip of the catheter will be put nasopharynx through one nostril

- Oxygen flow up to 5L/min
- Commonly used in pediatrics

Contraindication: epistaxis, nasal obstruction

b. Nasal prong: gives comfort for the patient

- Oxygen flow: up to 5L/min

Contraindication: epistaxis, nasal obstruction

c. Simple facemask: for patient requiring higher oxygen

- Oxygen flow: 5-10L/min

Contraindications: upper airway obstruction

d. Mask with reservoir: for patient requiring 10-15L/min

- With non-rebreathing facemask, possible to give up to 20L/min

Contraindications: upper airway obstruction

e. High flow nasal cannula/HFNC: for patient requiring >20L/min

- Maximum oxygen flow is 60L/min
- Washes the dead space in the oropharynx and improves oxygenation.
- Creates continuous pressure in the airway with normal PEEP (up to 5cmH_2O)

f. Non-invasive ventilation: With CPAP or BiPAP

- Indicated for patients who are not responding for low/high flow oxygen or severe hypoxia with $PaO_2/FiO_2<150$

g. Invasive ventilation: is the last resource for patients who responded earlier for NIV

- Is indicated for severe hypoxia with $PaO_2/FiO_2<150$ not responding to NIV

III. Management of complications

Delayed intubation: follow the clinical condition while on oxygen therapy and intubate earlier if indicated

Tissue hyperoxia: decrease oxygen flow once SaO_2 is in the target.

IV. Monitoring and problem solving

- Monitor vital signs
- Follow the respiratory pattern, mental status
- ABG if available.

1.3 ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

I. Introduction and principle of care

Introduction: It is an end result of aggressive inflammatory process in the lungs. Acute hypoxia with bilateral lung infiltration which is non cardiac origen.

Causes could be:

1. **Local:** Pneumonia, aspiration, inhalation, drowning etc…
2. **Systemic:** Sepsis, trauma, pancreatitis, and transfusion related etc…

Clinical features: Increased work of breathing, tachypnea and progressive hypoxemia resistant to supplemental oxygen, signs and symptoms of respiratory distress

Classification:

1. **Mild:** PaO2/ Fio2 200- 300 on 5cm of CPAP,
2. **Moderate:** PaO2/ Fio2 100- 200 on 5cm of CPA,
3. **Severe:** PaO2/ Fio2 < 100 on 5cm of CPA,

Timing	**Within 7 days of known clinical insult**
Origin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload
Chest imaging	Chest imaging findings of new infiltrate(s) consistent with acute pulmonary parenchymal disease
Oxygenation	PaO2:FiO2 ≤ 300 on 5 cm of CPAP

Diagnosis: Using the Berlin criteria - standard

Diagnosis using **Kigali's modification for low resource settings:** Acute onset, SpO2/FIO2 ≤315 without CPAP, bilateral opacities on chest ultrasound/CXR)

Imaging: CXR /Chest CT/: Usually have extensive bilateral infiltrates

: Chest ultrasounds: Bilateral B lines ± consolidations

Principles of care

- Treat the underlying cause
- Lung protective ventilation and ventilatory management
- Fluid restriction
- Supportive care: Sedation, Nutrition, GI prophylaxis, glucose control(140-180mg/dl)

II. Suggested management

- **NIPPV/ High flow nasal oxygen:** Consider for mild cases
- **Invasive management:** AC/VC mode is preferred (Start at FiO2 100,)

  Low tidal volume 4-6ml/Kg (use ideal body weight)

  Higher PEEP (8-10 initially and increase subsequently)

  Plateau pressure (≤30cmH_2O), pH = 7.30 - 7.45

  Prone positioning: (PaO2:FiO2 ≤150) , within 48 hours

  Neuromuscular blockers for severe cases (PaO2:FiO2 ≤150) within 48 hours

  Oxygenation goal: PaO_2 = 55–80 mmHg or SpO_2 = 88–95%

Recommended steps when necessary

- If plateau pressure is > 30 cm H2O, decrease TV in 1 mL/kg until it drops below 30 cm H2O or Tidal volume down to 4 mL/kg.
- If pH 7.15 - 7.30, increase respiratory rate (RR) until pH > 7.30 or RR = 35 bpm.
- If pH <7.15, increase RR to 35 bpm. If pH still, <7.15, increase TV at 1 mL/kg increments until pH >7.15.

Special considerations

Lung recruitment maneuvers: Increase PEEP to 40 cmH20 for 40 seconds, then slightly increase the previous baseline PEEP, monitor vital sign for hypotension pneumothorax

ECMO: Extra corporal membrane oxygenation can be considered in best setups for those with no response and hypoxia

III. Management of Complications

- **Ventilatory:** ***Ventilator induced lung injury*** (Volutrauma (excessive volume), Barotrauma(from excessive pressure), Bio trauma with multi organ failure(from cytokines released during lung injury))
- **Pneumothorax**
- **Auto PEEP** (*Management*: Sedation, decrease respiratory rate, increase PEEP, increase E time in I:E ratio),
- **Hypotension,**
- **Others** - DVT/PE, Sepsis and super infection,
- **Long term complications**- lung fibrosis, oxygen dependence, pulmonary hypertension and Cor-pulmonale.

IV. Monitoring and problem solving

- Continuous monitoring of vital signs and ventilatory parameters (plateau pressure, respiratory rate <35, PH >7.3) and ABG if available
- Daily sedation interruption, early weaning and tracheostomy, early mobilization

1.4 PNEUMONIA

I. Introduction and principle of care

Community-acquired pneumonia (CAP) is a new radiographic change with evidence of lung infection in a patient, not hospitalized or residing in a long-term care facility for 14 or more days before presentation

Hospital-acquired pneumonia (HAP) is a new radiographic change with evidence of infection occurring ≥ 48 hours after hospital admission

Ventilator Associated Pneumonia (VAP) is a new radiographic change with evidence of infection occurring after ≥ 48 hours of intubation/mechanical ventilation.

Diagnosis: Patient should have clinical, radiologic and microbiologic evidences

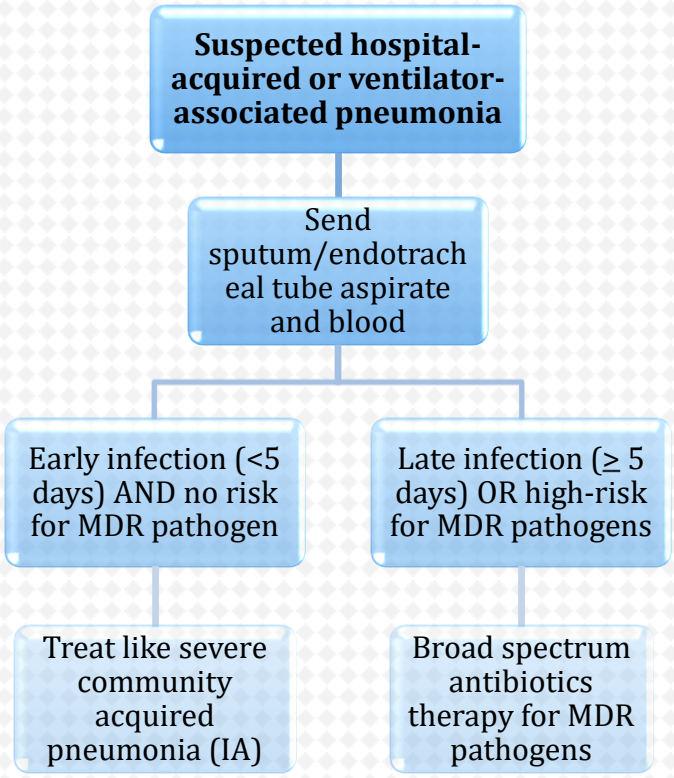
Clinical: Respiratory decline, Fever and or Productive cough

Radiologic: New / worsening lung infiltrates on chest imaging

Microbiologic: Blood culture, sputum culture, semi-quantitative sputum samples (for intubated), and invasive sampling only for immunosuppressed and continued clinical deterioration despite treatment.

Principle of care

- **Preventive measures:** VAP bundle, hand hygiene and infection prevention
- **Supportive therapy:** early enteral nutrition; **chest physiotherapy**, ventilatory management as indicated
- **Antibiotic therapy:** empiric use with early de-escalation and short duration (7 days)



II. Suggested management

Antibiotic therapy

- Empiric therapy based on local/institutional antibiotic susceptibility pattern

Risk factors for MDR pathogens: Prior intravenous antibiotic use within 90 days

- Septic shock at time of VAP
- ARDS preceding VAP
- Five or more days of hospitalization prior to the occurrence of VAP
- Acute renal replacement therapy prior to VAP

- For patients with ***no*** *risk factors*/ ***CAP:*** Use mono therapy * Ceftriaxone 2 g IV daily or * cefefpime 2g IV q8 hours or *piperacillin-tazobactam 4.5g IV q8 hours
- For those ***with risk factor***s, >10% of pseudomonas prevalence or >20%MRSA, use double anti -pseudomonas therapy and MRSA coverage i.e

Cefepime or ceftazidime *or* (meropenem) *or* a (piperacillin/tazobactam)

+

(ciprofloxacin preferred) *or* (gentamicin)

+

vancomycin

III. Management of complications

- **ARDS:** supportive care and manage as per ARDS guideline
- **Sepsis, metastatic infection and Septic shock/Multi organ failure**
- **Atelectasis:** early mobility, deep-breathing exercises (incentive spirometry) and suctioning
- **Infection with MRD organisms, cavitary lesions, abscess:** early detection and management, identifying risk groups, prevention and supportive care
- **Empyema and pneumothorax**: tube drainage and diagnosis directed therapy

IV. Monitoring and problem solving

- Reducing the risk of self extubation and re-intubation - proper sedation with interruption
- Timely weaning and extubation
- De-escalation of antibiotics according to culture and sensitivity result
- Duration of antibiotics should be reduced to 7 days unless indicated (severe cases with gram negative bacteremia, in selected cases the duration may extend 10-14 days), Procalcitonin can be used for stopping if available

1.5 SEVERE ACUTE EXACERBATION OF BRONCHIAL ASTHMA/COPD

I. Introduction and principle of care

Definition: recurrent obstructive airway disease due to bronchial constriction, mucosal edema, mucous plugging and /or alveolar destruction.

Table 1-3: Showing severe and life threatening asthma

Parameter	Severe	Life threatening
Breathless	At rest	
Talks in	Words	Unable to speak
Alertness	Always agitated	Drowsy or confused
Respiratory rate	Often > 30/min	
Accessory muscles use	Usually	Paradoxical thoracic and abdominal movements
Wheeze	loud	Absence of wheeze
Pulse rate	>120	Bradycardia

Principle of care

- Use the ABCDE approach, put patients on oxygen targeting a saturation of (88-90%) for COPD and >90% for asthma(above 94% for pregnant)
- Treat the respiratory distress, relief bronchial obstruction using medical therapy
- Identify and treat precipitating factors
- Try non-invasive ventilation unless contraindicated(better to minimize intubation as much as possible)

II. Suggested management

- Put on face mask oxygen

a. **Salbutamol**: 4–8 puffs every 20 minutes for 3 doses, then every 1–4 hours as needed / nebulized

b. Ipratroprium: 0.5 mg every 20 minutes for 3 doses, then every 2–4 hours as needed

c. Steroids:

Hydrocortisone: 200mg IV stat followed by. 100mg IV QID or

Prednisolone: 40-60mg po per day or

Dexamethasone: 0.6mg/Kg/day (18mg maxi dose)

d. Additional management for severe cases:

- **Adrenaline 0.5 cc IM/SC**, repeats after 20 min if no improvement, for anaphylactic asthma and severe cases
- **Magnesium Sulphat**e $MgSO_4$ 2gm IV in 100 ml 5% D/W over 20 min. Nebulized magnesium can also be used.
- **Aminophyllin**e. A loading dose of 5–6 mg/kg is followed by a continuous infusion
- **Ketamine** 0.2mg/kg iv bolus followed by 0.5mg/kg/hr can be considered

e. **Antibiotics:** given only when infection is a clear precipitating cause especially in severe COPD exacerbation (consider azithromycin)

f. Ventilatory considerations:

Non-invasive ventilation: CPAP (BiPAP) start with 10 IPAP and 5 EPAP and titrate to the patient's response unless contra indicated. Try to avoid invasive ventilation as much as possible

Invasive ventilation: If deteriorating mental status or coma, respiratory or cardiac arrest, cyanosis and hypoxemia on O_2,$PaCO_2$ greater than 50 mmHG and rising > 5mmHg/hr., minimal chest movement/air exchange and Pneumothorax.

- Experienced personnel should intubate using larger ET tube(preferably 7.5 or more)
- Ketamine is the good choice for intubation
- After proper suctioning Inhalational drugs(salbutamol..) can be given through ET tube

Table 1-4: Initial ventilatory settings

Parameters	Settings
Mode	AV /VC
Tidal volume	6-8ml/Kg
PEEP	5-8
Fio2	100%
Respiratory rate	10-12
I: E ratio	1: 3 or more
Plateau pressure should be ≤30	

III. Management of complications

- **Dynamic hyperinflation (DHI)/Auto PEEP:** give more time for expiration, shorten inspiration(increase flow rate, square wave form), decrease tidal volume
- **Hypotension:** disconnect from MV, manage increased PEEP give fluids
- **Pneumothorax:** Tube drainage

IV. Monitoring and problem solving

- Monitor vital signs and saturation, keep plateau pressure <30, do ABG if available
- Sedate using short acting non histamine releasing drugs like ketamine, fentanyl or propofol
- **Acidosis (PH<7.25):** increase tidal volume, decrease the amount of consumed carbohydrate
- **ET tube blockage:** frequent suctioning and give expectorants as indicated
- Consider anesthetic inhalation drugs and Extra corporal CO_2 removal as a last resort
- Early libration from mechanical ventilation and consider NIV post extubation

1.6 PULMONARY EMBOLISM

I. Introduction and principle of care

Pulmonary thromboembolism (PE) is occlusion of pulmonary arteries by embolus dislodged from thrombus at a distant site, mostly deep vein of the lower limb.

Massive PE is with a SBP of <90 mm Hg for >15 minutes, or <100 mm Hg in a patient with a history of hypertension, or a >40% reduction in baseline SBP.

Sub massive PE is characterized by a normal or near-normal BP with right ventricular dysfunction or myocardial necrosis

Diagnosis:

CXR -normal or can have unilateral basilar atelectasis, Pleural based opacity (Hampton's hump), dilated pulmonary artery (Pala's sign), unilateral lung oligemia (Westermark's sign)

ECG: tachycardia, non-specific ST and T wave changes in the anterior leads (V_1-V_4), $S_1Q_3T_3$ pattern, P Pulmonale and incomplete or complete right bundle branch block.

Echo: Severely dilated RV with severely reduced systolic function, akinesia of the mid free wall but normal motion at the apex of the right ventricle, Paradoxical RV septal systolic motion, Pulmonary artery systolic pressure >30 mmHg, Dilated IVC with lack of respiratory collapse, visualization of clot etc…

Spiral CT: is diagnostic in 98 percent of patients with PE.

Doppler ultrasound: to look for DVT as supportive evidence

Criteria	Points
Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
Other diagnosis less likely than pulmonary embolism	3.0
Heart rate >100	1.5
Immobilization (≥3 days) or surgery in the previous four weeks	1.5
Previous DVT/PE	1.5
Hemoptysis	1.0
Malignancy	1.0

Risk factor assessment: clinical assessment using modified Wells criteria *(PE likely if score > 4)*

Principle of management

- Patients usually die from cardio respiratory failure Early detection of nonspecific management and addressing hypoxia and hemodynamic instability is mandatory
- Restricted fluid challenge for patients with PE and early initiation of vasopressors (preferably Norepinephrine) for those with no response.
- Early anticoagulation should be initiated as soon as possible and thrombolytics should be initialed for those with massive PE and without contraindication

II. Suggested management

- **Administer oxygen for hypoxaemia**
- **Empiric anticoagulation:** UFH is 5000 IV bolus followed by 1000 hourly, or 10,000-18500 IU(SC) BID can be given (preferred for patients with renal failure) or **Enoxaparin** 1mg/kg SC Bid (or 1.5mg/kg daily) is preferred over UFH and **Warfarin** 2.5 to 5 mg or **Rivaroxaban** 15mg po bid followed by 20 mg po /day after 21days

Thrombolytics: Indicated if in cardiac arrest, hypotension, respiratory failure or RV dysfunction: **Streptokinase** 250,000 IU/30 min then 100,000 IU/hr for 24 hr (72 hrs if concurrent DVT is suspected)

III. Management of complications

- **Anaphylaxis:** from thrombolytic therapy
- **Bleeding:** intracranial hemorrhage, and or other severe bleeding
- **Over anticoagulation:** Manage with FFP, vitamin k....as directed clinically and by INR
- Reversal with protamine sulphate might be necessary

IV. Monitoring and problem solving

- Check aPTT every 1 hour for the first 6 hour followed by daily after giving heparin (Target aPTT, 1.5 to 2 times)
- Check INR every 3 days, target INR 2-3
- Monitor vital signs, look for hypotension, manage with modest fluid therapy and

1.7 MECHANICAL VENTILATOR

- **For detail Mechanical ventilator utilization see video on 'utilization of common ICU equipments'**

I. Introduction and principle of care

Role of Mechanical Ventilation is to provide oxygenation and ventilatory support during respiratory failure. It also improves gas exchange; unload respiratory muscles and "Buy time" for healing and lung recovery.

Why is mechanical ventilation required?

a. Impending or existing respiratory failure

- Failure to oxygenate (inadequate exchange of gases at the alveolar level, as seen in acute respiratory distress syndrome [ARDS])
- Failure to ventilate (decreased mental status or decreased lung compliance)
- Combination of both

b. Airway protection

II. Suggested management

Management includes selecting initial mode of ventilator that is going to be used. Here is to high lighten the different modes of ventilator.

Modes

1. ***Assist Control (A/C)***

- Ventilator will allow a patient to initiate a breath and then ventilator will deliver a pre-set tidal volume
- Machine set at a minimum rate so apnea will not occur if the patient does not initiate a breath

Disadvantages:

- Hyperventilation if patient has increased respiratory rate (can lead to respiratory

alkalosis)

- Ventilator dysynchrony

2. ***Synchronized Intermittent Ventilation (SIMV)***

- Similar to A/C, but patients can take own breaths with their own TV between mechanically assisted breaths
- Can be used as a primary mode or a weaning mode
- May lead to a low respiratory rate in a patient who does not initiate breaths if set rate is low

3. ***Pressure Support Ventilation (PSV)***

- Also called "spontaneous mode"
- Patient initiates breath & ventilator delivers a pre-set inspiratory pressure to help overcome airway resistance and keeps airways open
- Patient controls the rate, tidal volume, and minute ventilation
- Tidal volume is variable
- Can be used in conjunction with SIMV or CPAP settings

4. ***Continuous Positive Airway Pressure (CPAP)***

- Positive airway pressure provided during both inspiration and expiration
- Ventilator provides O2 and alarms, but no respirations
- Improves gas exchange and oxygenation in patients able to breathe on their own.
- Can also be used non-invasively via a face or nasal mask for patients with sleep apnea

5. ***Bi level Airway pressure (BiPAP)***

- Delivered by mask, not through an airway
- Similar to CPAP, but can be set at one pressure for inhalation (IPAP) and another for exhalation (EPAP)
- Used in sleep apnea, but also has been found to be useful in patients with CHF and type2 respiratory failure to avoid intubation

6. ***Non-conventional modes of ventilator***

Used in patients who are not responding for conventional modes of ventilation like A/C or SIMV mode and with refractory hypoxia; these include:

- Pressure regulated volume control(PRVC)
- Airway pressure release ventilation(APRV)
- High frequency oscillatory ventilation(HFOV)

III. Management of complications

- **Asynchrony**
  - Proper sedation and paralysis
- **Auto PEEP**
  - Patient doesn't expire full tidal volume and air becomes trapped Can cause increased alveolar damage
  - Prolong E time by ↓RR, Shorten I time, ↑E cycle, Bronchodilator for asthma or AECOPD
- **Barotrauma**
  - Damage to alveoli caused by increased pressure and volume
  - Stick to lung protective strategy
- **Hemodynamic compromise**
  - Don't increase PEEP too much once target O2 saturation achieved
- **Nosocomial infection**
  - Oral hygiene, head elevation 30-45degree, early weaning and extubation
- **Anxiety/ Stress/ Sleep deprivation**
  - Proper sedation with sedation vacation daily
- **Muscle deconditioning/Ventilator dependence**
  - Daily SBT trial, chest physiotherapy

IV. Monitoring and problem solving

- Monitor vital signs
- Monitor alarms
- ABG at least once daily

Suggested Reading

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CHAPTER 2: CARDIOVASCULAR SYSTEM

2.1 ACUTE CORONARY SYNDROME/ACS/

I. Introduction and principle of care

- Acute coronary syndrome is a spectrum of clinical presentations that is related to acute luminal thrombosis/ stenosis of a coronary artery by atheromatous plaque.
- It includes unstable angina, ST elevation myocardial infarction/STEMI/ and non ST elevation myocardial infarction/NSTEMI/
- Patients with ACS could be admitted to ICU for continuous monitoring, after acute complication or post intervention.
- IV line should be secured and pulse oxymetry should be attached to patient.
- 12 lead ECG should be performed and correlate with previous ECG.
- Cardiac markers, CXR and Echocardiography should be done for diagnosis and monitoring
- Glycemic control-goal is RBS of 140-180mg/dl
- Sedatives and laxatives should be considered based on patient condition.
- Pain management should also be given accordingly

II. Suggested management

- UA/NSTEMI requires medical management while STEMI or New LBBB requires revascularization therapy using PCI or thrombolytics.
- If PCI and thrombolytics are available, PCI is recommended as initial therapy.
- If Primary PCI not available, or is delayed >120 min, use thrombolytics after ruling out any contraindications (table 2-1).

- Streptokinase 1.5 million U in 50 mL of 5% dextrose in water (D5W) given IV over 60 minutes

- Medical Management includes antiplatlets, antischemic, anticoagulants

a. Antiplatelets

- ***Aspirin(ASA):*** loading:162–325 mg chewed, then 81 mg/d
- ***Clopidogrel -loading:*** 300mg- 600mg Po, then 75 mg/d for at least 1 yr

b. Anti-ischemics

- ***Nitroglycerin (NTG):*** sublingual 0.4 mg every 5 min as needed or for treating chest pain. IV NTG can be given for persistent ischemia C/I= low BP,Bradycardia, sildenafil use in 24hrs;cautious in inferior MI
- ***Morphine sulphate:*** 2–5 mg IV , may be repeated every 5–30 min as needed to relieve chest pain.
- ***Metoprolol:*** 25–50 mg PO every 6 h (IV metoprolol 5mg every 5 minutes is given only if there is HTN, ongoing pain,tachycardia).Atenolol, propranolol or carvedilol are the other alternative B blockers.C/I- CHF, bradycardia, asthma, HR<50 or 2° or 3° blocks.
- ***Statins:*** atorvastatin 80mg/d is preferred or
- **Simvastatin/lovastatin** 40mg Po/day

c. Anticoagulants

- ***Un Fractionated Heparin:*** Bolus 60–70 U/kg (up to 5000 U) IV then infusion of 12–15 U/kg/ h (initial maximum 1000 U/h) titrated to 1.5 to 2.5 X aPTT or 50–70s.If no perfuser,12,500U SC BID is possible; or LMWH (Enoxaparin): 1 mg/kg SC every 12 h.
- ***Warfarin:*** initially 2.5 mg titrated to INR goal of 2-3 (for extensive anterior STEMI with severe LV dysfunction, or LV thrombus

Table 2-1: Contraindications of thrombolytics

Absolute contraindication	Relative contradications
Major trauma, surgery, head trauma Within 3 weeks	Cancer Age >75-80
Prior hemorrhagic stroke	Transient ischemic attack within
Ischemic stroke within	6 month
Prior 6 month	Oral anticoagulant therapy
Cystementral nervous	Non compressible punctures
Neoplasm	Traumatic resuscitation
Gastrointestinal bleeding within one	Refactory hypertention
Month	Advanced liver disease
Active bleeding	Infective endocardities Active puptic ulcer Pregnancy or within one week postpartum

III. Management of complications

- Arrhythmia and Mechanical complications should be early identified and treated
- Mechanical complications of myocardial infarction include:
  - Cardiac tamponade secondary to left ventricular free wall rupture
  - Pseudoaneurysm formation
  - Rupture of the interventricular septum
  - Acute mitral regurgitation due to partial or full rupture of a papillary muscle.
- Arrhythmias should be managed according to the rhythm and patient's condition (check arrhythmia on section 2.2)
- Cardiogenic shock, Left Ventricle failure should be managed medically (check shock and HF portion)

- In patients with inferior MI and cardiogenic shock consider RV infarction and treat initially with IV fluids
- Post MI Pericarditis should be treated with Aspirin 650 mg every 4 to 6 hours.
- Mechanical complications require early surgical consultations.

IV. Monitoring and problem solving

- All patients with ACS should be on continuous ECG monitoring with arrhythmia high priority alarm setup.
- Monitor patient's condition & assess treatment response (pain, ECG, cardiac enzymes)
  - Vital signs: Every 15 minutes until stabilization
  - Cardiac markers: every 6 hours if not confirmed
  - Manage chest pain: administer nitroglycerine, if no response or nitroglycerine is contraindicated give morphine
- Watch for mechanical complications, and manage accordingly.
- Blood sugar should be maintained at 140- 180mg/dl

2.2 ARRHYTHMIA

I. Introduction and principle of care

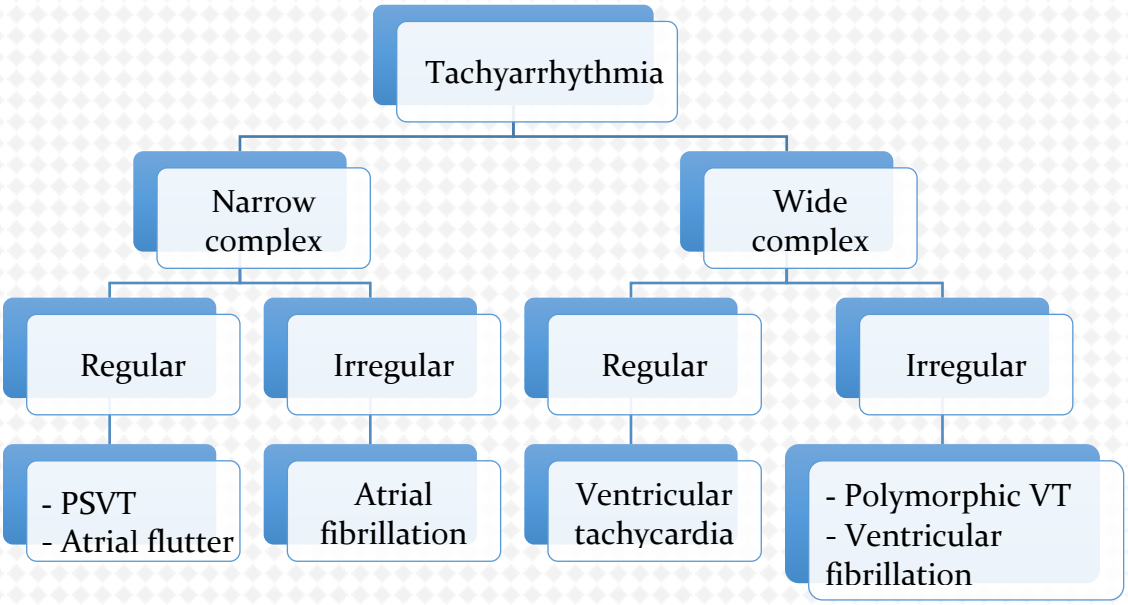
Arrhythmia in critically ill patient will be the cause for patient clinical deterioration. Therefore continuous ECG monitoring should be done in every patient admitted to ICU to early recognize and treat unstable arrhythmias.

Signs of Unstable arrhythmia;

- Ischemic chest pain
- Hypotension and poor perfusion
- Signs of respiratory distress
- Altered mental status

- Unstable arrhythmias require electrical therapy.
- Tachyarrhythmias with instability sign requires synchronized cardioversion if there is central pulse.
- Arrhythmias could be symptomatic and requires medical therapy if tachyarrhythmia of Rate>150 or bradyarrythmia of Rate<50.
- Tachyarrhythmia is classified according to QRS duration and regularity of RR interval.(Figure 2-1)
- If the patient is stable, 12 lead ECG should be done to identify the type of arrhythmia. If possible compare with old ECGs.
- Always correlate the ECG finding with patient clinical condition.
- Always conduct focused history and physical examination.
- Bradycardia in ICU think of 'DIE-Drugs, ischemia and electrolytes', hypoxia, hypothermia, hypothyroidism, head injury with increased ICP.

Figure 2-1: Tachyarrhythmia classification



II. Management of specific arrhythmias

1. Management of narrow complex tachyarrhythmias

Unstable patients: synchronized cardioversion

- Cardioversion in patient with atrial fibrillation requires exclusion of thrombus if possible.
- Initial dose of cardiversion
  - Narrow regular:50-100J
  - Narrow irregular:120-200J

For stable patients:

- Perform vagal mauevers (like carotid massage,valsava,induced vomiting,eye ball massage etc)after excluding any contraindications for SVT
- Adenosine 6mg IV fast push with followed by rapid fluid flush.If no response repeat 6 mg followed by 12 mg.
- Metoprolol 5 mg IV over 2-5 min
- Digoxin 0.25 mg IV

- Amiodarone: 150 mg IV bolus over 10 min followed by an infusion of 1 mg/min over 6 h and then 0.5 mg/min over the next 18 h (maximum total dose of 2.2 g over 24 h) is the preferred agent
- If Dilitiazem available 0.25 mg/kg over 2 min or Verapamil

2. **Management of Wide complex tachyarrythmias**

Unstable patients require synchronized cardioversion if there is central pulse

- Initial dose of cardioversion wide complex regular with pulse:100J

For stable patients:

- Amiodarone: 150 mg IV bolus over 10 min followed by an infusion of 1 mg/min over 6 h and then 0.5 mg/min over the next 18 h (maximum total dose of 2.2 g over 24 h) is the preferred agent.
- Procainamide: 17 mg/kg IV loading dose (at 20 mg/min) followed by an infusion at 1 to 4 mg/min. The infusion should be stopped if the patient becomes hypotensive or the QRS widens by 50% above baseline
- Magnesium 1 to 2 g IV over 20 to 30 min if polymorphic ventricular tachycardia or torsade de pointes considered.

3. **Management of Bradyarrythmia**

- Symptomatic bradycardia with evidence of reduced end organ perfusion is initially treated with IV boluses of Atropine 1 mg (upto3 mg).
- If there is no response:
  - Dopamine 5–20 mcg/kg/minute or
  - Epinephrine 2–10 mcg/minute can be started.
  - Transcutaneous/transvenous pacing is also an option if available
  - Pacemaker if available

Indications for pacing are:

- Symptomatic bradycardia unresponsive for medical therapy

- Mobitz type II second-degree and third degree AV block.

III. Monitoring of arrhythmias

- Patient with arrhythmias should be on continous ECG monitor and high priority alarm should be set.
- Spo2 and BP should be monitored.
- After patient stabilization identifying the possible precipitants and correction should be done.

2.3 ACUTE HEART FAILURE

I. Introduction and principle of care

- AHF is an acute clinical syndrome of new or worsening signs and symptoms of heart failure (HF) because of impaired ability of the heart to supply adequate oxygen and meet the demands of the body's metabolizing tissues.
- Clinically AHF can manifest as congestive symptoms/signs or with symptoms of hypoperfusion or a combination, giving rise to four clinical profiles of patient's with AHF.
- Admission to ICU for patient with AHF will be for continuous monitoring, treatment of complications like respiratory failure or cardiogenic shock.
- CXR, ECG and ECHO should be done for monitoring, diagnosis and/ or intervention guide purpose.
- ABC assessment and management is always the priority.
- Treatment of precipitant and underlying illness should be done.

II. Suggested management

a. AHF with pulmonary Edema

- Oxygen therapy - SPO2 92- 94%.
- Noninvasive ventilation can be tried if the patient does not have contraindication.
- Diuresis: Furosemide (Lasix) 1mg/kg or 40 mg IV and escalate the dose of furosemide every hr. With 40 mg increment depending on response to achieve urine output ≥0.5ml/kg/hr.

or

- Continuous Lasix infusion at 10-40mg/hr. If the urine output remains below 1ml/kg/hr., the infusion rate can be increased each hour as necessary till a maximum dose of 80-160 mg/hr, not to exceed 600mg/day
- Vasodilator: If there is no hypotension; Nitroglycerin 0.4 mg sublingual 3 doses every 5 minutes or

- IV Nitroglycerin 5-10 microgram/min infusion, with rise 10-20micro/min, every 15 minutes, if no response

- Morphine to relieve dyspnea and anxiety.
- Invasive ventilation is recommended in AHF when respiratory failure cannot be managed noninvasively

b. AHF with Cardiogenic shock

- This is hypotension (SBP<90) and signs of tissue hypoperfusion in patients with AHF
- Fluid challenge with 250-300 mL crystalloid over 15–20 min using the principle of fluid challenges should be done with caution in subset of patients with hypoperfusion but no congestion.
- **Vassopressors/innotropes:**
  - Noradrenaline is the drug of choice 2-20 mcg/min(0.1-1 µg/kg/min), or
  - Adrenaline 2-20 mcg/min(0.1-1 µg/kg/min) or
  - Dopamine 2-20mcg/Kg/min and titrate accordingly monitoring BP and urine out.
  - Dobutamine can be considered for low ejection fraction and low normal blood pressure (SBP 80-90) in isolation or in combination with other drugs:2.5-15mcg/kg/min
  - For concomitant pulmonary edema, diuresis with furosemide 20-40mg IV bolus after inotropic support and low normal BP.
  - Invasive mechanical ventilation for patients with increased work of breathing.
  - Arrhythmia related shock refers to arrhythmia

III. Monitoring and problem solving

- Precipitating factors should be identified and treated accordingly These are summarized using the Pneumonic– HEART FAILES

- H- Hypertension, E-Endocarditis, A- Arrhythmia R- Rheumatic fever /Myocarditis, T- Thyrotoxicosis and pregnancy, F- Fever (infection), A-Anemia, I-Infarction, L-Lack of compliance, E-Embolism (Pulmonary), S-Stress (emotional, dietary, fluid excess, physical)

- Patient should be on continuous ECG monitoring
- Spo2, BP, and RR should be monitored frequently
- Urine output and balance should be measured every 1 hr
- Clinical symptoms and chest condition should be evaluated frequently
- Invasive arterial line BP monitoring can be done for patient with cardiogenic shock if available

2.4 HYPERTENSIVE EMERGENCY

I. Introduction and principle of care

Hypertensive emergency is an acute increase in BP associated with severe, potentially life-threatening target end-organ damage.

End organ damages are the following:

a. **Cardiovascular:** Acute coronary syndrome /ACS/, Left ventricular failure/CHF/, Aortic dissection
b. **Central Nervous System:** Cerebral edema (hypertensive encephalopathy) Intracerebral or subarachnoid bleeding Cerebral infarct or transient ischemic attack
c. **Renal:** Micro-hematuria, Proteinuria, Acute kidney injury
d. **Ophthalmologic:** Retinal hemorrhages or exudates, Papilledema

- They require immediate hospitalization in an intensive care unit (ICU) for prompt BP control within minutes or a few hours by intravenous administration of antihypertensive drugs.
- ABC assessment and stabilization should be made.
- Focused History and physical exam is required to identify end organ damage
- ECG, CXR, U/A, fundoscopy or optic sheath ultrasound measurement should be done as a first investigation.
- Prompt intravenous administration of short-acting and titratable drugs is preferred in the first few minutes of treatment.
- Treatment of complications depends on the end organ damage.
- Identify and treat underlying cause for the hypertensive crisis

II. Management of specific complications

Table 2-2: Management of specific complications of hypertensive crisis

Diagnosis	Target goals	Treatment
Acute Ischemic stroke not rTPA Candidate	Treat if ≥220/120 mmHg Reduce by 15% Over 24 hour	Labetalol- 20–80 mg bolus every 10 min or 0.5–2 mg/min IV infusion ,or Nifedipine 20 mg po
Acute Ischemic stroke rTPA Candidate	Treat if BP>185/110 mmHg	Labetalol- 20–80 mg bolus every 10 min or 0.5–2 mg/min IV infusion ,or Nifedipine 20 mg po
Acute hemorrhagic stroke	Reduce BP to<140/90	Labetalol- 20–80 mg bolus every 10 min or 0.5–2 mg/min IV infusion ,or Nifedipine 20 mg po
Acute aortic dissection	SBP to 110-120mg PR≤60 beats/min	Sodium nitroprusside(0.25–10 µg/kg/ min IV infusion) and Labetalol- 20–80 mg bolus every 10 min or 0.5–2 mg/min IV infusion, or Metoprolol 5mg IV
Acute coronary syndrome	Avoid ≤25%reduction in MAP	Nitroglycerin sublingual Metoprolol 5 mg Iv
Acute renal failure	Reduce not more than 20 % acutely	Sodium nitroprusside and labetalol Dialysis can be considered
Acute pulmonary edema	Reduce 20-30% <130/80	Nitroglycerin Diuresis Captopril 12.5-25 mg po

III. Monitoring and problem solving

- Patient should be on continuous ECG monitoring.
- Serial and frequent BP measurement
- If possible invasive arterial BP measurement can be done.
- Avoid aggressive BP lowering which leads to organ perfusion abnormality.

2.5 SHOCK

I. Introduction and principle of care

IS a multifactorial syndrome in which cardiovascular dysfunction results in a reduction in tissue oxygen delivery to a level below that required to meet metabolic demands.

In adults: Systolic BP < 90 mmHg, or DBP<60mmHG, or MAP (*DBP+1/3(SBP-DBP),*< /= 60 mmHg, or reduction of systolic BP > 40 mm Hg from the patient's baseline.

Treatment of shock is based on underlying cause which can be classified in to 4.

Classification of shock

a. **Hypovolemic:** eg. Bleeding, dehydration
b. **Distributive:** Sepsis, anaphylactic, neurogenic, adrenal insufficiency
c. **Obstructive:** Pneumothrax, PE, tamponade
d. **Cardiogenic:** MI, cardiomyopathy, arrhythmia

ICU admission is required for shock patient for continuous and /or invasive monitoring and treatment

ABC assessment and management should be done

II. Suggested management

- Large bore IV line should be secured and high flow oxygen should be administered

a. Hypovolemic shock

- Give 2-3 liters of isotonic N/S/ R/L over 20-30 minute (give N/S alternatively with R/L).
- In haemorrhagicshock transfuse blood product if patient is hemodynamically unstable after 2 litres of crystalloid.
- In severe traumatic bleeding in addition to PRBC transfuse FFP and platelets as well
- Provide immediate control of hemorrhage, when possible
- If the patient remains hypotensive in spite of fluids exclude other problems such as sepsis, tension pneumothorax, tamponade, and give pressors like dopamine.

b. Septic shock

Apply the six sepsis management bundles with in 1hr: appropriate fluid management, Oxygen delivery, antibiotics, sending specimen for culture and sensitivity, and monitoring of lactate and hourly urine out.

These are discussed below:

- Immediate aggressive volume expansion with IV fluids, give the first bolus of 30ml/kg over 3 hours, then subsequent boluses depending on hemoynamics, the lung congestion status, urine output and mental status.
- If hemodynamic response after fluid administration for one hour is poor use vasopressors .The vassopressor of choice is norepinephrine (NE) (2-30 µg/min/ (0.1-1 µg/kg/min) but epinephrine (2-30 µg/min, (0.1-1 µg/kg/min) and dopamine (2-20 µg/kg/min) can be used respectively. Titrate dose based on response
    - For refractory shock, after fluid and pressors, consider corticosteroid-hydrocortisone 50mg IV qid administration, and if patient respond to this drug continue it for 7 days.
    - Treatment of underlying infection and proper debridement is important.
    - Initiate broad spectrum antibiotic therapy parentrally early, usually based on the possible focus of infection/septic focus then specific antibiotic based on culture and sensitivity test result
    - Surgical drainage or debridement of an abscess or dead and necrotized tissue.
    - Blood transfusion if Hgb is ≤ 7mg/dl to keep adequate O_2 saturation

N.B. Cardiogenic shock (mentioned acute heart failure section)

c. Anaphylactic shock

- Consider early definitive airway control for those evidence of significant airway edema
- Administer Epinephrine 0.3 to 0.5 mg IM on anterior lateral thigh, every 5 minuses
- Salbutamol puff/ nebulized can be given for wheezing
- Secure bilateral wide bore needle IV lines and Administer 20ml/kg of crystalloids as fast as possible
- Then mix 5 mg of epinephrine in 500 ml of normal saline. Infuse at 10 cc/hr, and titrate to arterial BP response.

- Antihistamines- prometazine 25 mg /IM or Dimenhydramine 50mg IV
- Administer 5-10 mg/kg of hydrocortisone or 1-2 mg/kg of methylprednisolone for late control of symptom (Doesn't have any role in the control of acute symptoms)

d. Neurogenic shock

- Aggressive fluid resuscitation, followed by vasopressors are used as other types of shock.
- The primary neurological disorders have to be addressed
- Hypoadrenergic shock/adrenal crisis:basal hormonal test can be done but management should be started immediately.
- Fluid management is similar other distributive shock followed by vasopressors(see septic/anaphylactic shock above)
- Administer 100 mg hydrocortisone IV followed by 100 mg every 6 hrs for 24 hours. The dosage can be reduced to 50mg every 6 hours when patient is stable.
- If hypoglycemia is present, give 40% glucose IV stat can be given.
- Treat precipitating factors.

e. Obstructive shock

- Tension pneumothorax: Needle decompression, chest tube
- Cardiac tamponade: Fluid challenge, Pericardiocentesis
- Massive PTE: Thrombolytics: Anticoagulation

III. Monitoring and problem solving

- Patient should be on continuous ECG monitoring.
- Spo2, BP, and RR should be monitored frequently.
- Urine output and balance should be measured every 1 hr.
- Check for fluid responsiveness using leg raising test, Check for IVC status using bed side ultrasound and CVP

2.6 CARDIAC ARREST RESUSCITATION

I. Introduction and principle of care

Cardiac arrest is defined as abrupt cessation of cardiac pump function which may be reversible by prompt intervention but will lead to death in its absence. There will be no central pulse (carotid or femoral pulse)

Patients post cardiac arrest will be admitted to ICU for post cardiac arrest care or patients admitted in the ICU will have cardiac arrest and require cardiac arrest resuscitation.

Cardiac arrest chain of survival in the ICU includes the following:

Figure 2-2: Cardiac chain of survival

Early recognition, high quality cardiopulmonary resuscitation, and early defibrillation are **essential for cardiac arrest survival**.

High quality CPR includes: Push hard (depth at least 5 inch), Push fast (Rate 100 to 120 per min), minimize interruption, allow adequate chest recoil, avoid excessive ventilation and switch compressor every 2 min or earlier if fatigued.

II. Systematic Approach for Cardiac Arrest

The main components during advanced cardiac arrest resuscitation are the following:

- Early recognition of cardiac arrest: Check responsiveness, check for central pulse and breathing
- Call for help to get defibrillator and activate code blue team
- Start high quality CPR

- 30 chest compressions to 2 breath if no definitive airway
- Continuous chest compressions at rate of 100-120 per minute if there is definitive airway

- Make airway patent and administer adequate ventilation
  - Check airway patency and open airway with maneuvers and devices
  - If intubated, disconnect from mechanical ventilator and attach BVM
  - 30 chest compressions to 2 ventilations if no definitive airway
  - Ventilations at a rate of 1 breath every 6 to 8 seconds (8 to 10 ventilations per minute) should be performed if definitive airway
  - Deliver each rescue breath over 1 sec.
  - Give a sufficient tidal volume to produce *visible chest rise*
- Check pulse and rhythm every 2 minutes then administer medications and/ or defibrillation based on cardiac arrest algorithm(figure 2-3)
  - Shockable rhythms (Vfib and Pulseless Vtac)-
    - Defibrillate
    - Adrenaline 1 mg IV every other cycle
    - Amiodarone 300 mg IV loading after initial adrenaline followed by 150 mg IV
  - Non Shockable rhythms (Asystole and PEA)-
    - Continue CPR
    - Adrenaline 1 mg IV every other cycle
    - Treat reversible causes
- Check for SAMPLE History which includes sign and symptoms, allergy, medication, past medical history, last meal, and events to identify causes of cardiac arrest
- Look for 5 H'S and 5 T'S and treat the causes accordingly.

Table 2-3: Lists of 5H'S and 5T'S

5H 'S	5T'S
Hypoxia	Tension pneumothorax
Hypotension	Tamponade cardiac
Hypo/hyperkalemia	Thrombosis-coronary
Hydrogen ion/acidosis	Thrombosis-pulmonary
Hypothermia	Toxins

Special consideration in pregnant with cardiac arrest

- Provide uterine lateral displacement
- Prepare and perform perimortem c-section if no ROSC in5 minutes
- Prepare neonatal team to receive neonate

III. Post cardiac arrest care

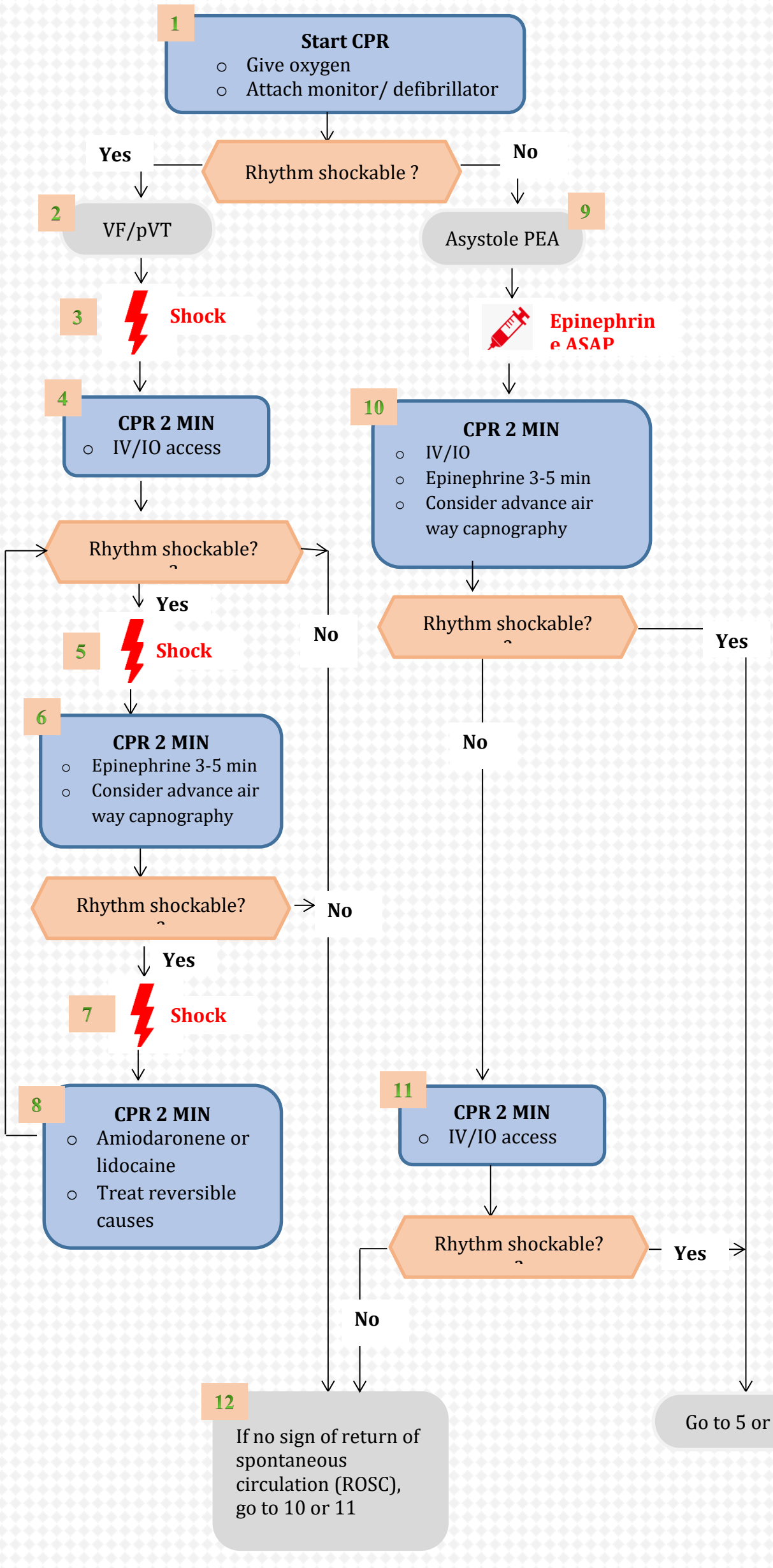
- If return of spontaneous circulation achieved, post cardiac arrest care should continue.
- Components of post cardiac arrest care includes
  - **Advanced air way and ventilation**
    - Early placement of definitive airway
    - Put on mechanical ventilator
      - ✦ Titrate FIO2 to maintain spo2-92-98%
      - ✦ Rate start 10 breath per minute
  - **Improve perfusion**
    - Systolic Blood pressure >90 mmHg ,MAP >65MMhg
      - ✦ Administer crystalloid and/or vasopressors
  - **Therapeutic hypothermia**
    - If patient unresponsive cool patient up to 2-36 for 24 hours
  - **Treat underlying cause**

- Perform 12 lead ECG
- Emergency cardiac intervention if STEMI

IV. Monitoring and problem solving

- Patient should be on continuous ECG monitor
- Monitor quality of CPR and ventilation during resuscitation
- Monitor SBP, PR, and SPO_2 post cardiac arrest
- Treat underlying cause for cardiac arrest
- Maintain glucose control as done for other critically ill patients
- Reduce the risk of multiorgan injury and support organ function if required

Figure 2-3: Cardiac arrest algorithm



CPR Quality

- Push hard (at least inches (5cm)) and fast (100-120/min)and allow complete chest recoll
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Change compressor every two 2min or sooner if fatigued
- If not advanced airway, 30:2 compression – ventilation ratio
- Quantitative wave formcapnography
  - If $PETCO_2$ is low or decreasing, reassess CPR quality

Shock energy for defibrillation

- Biphasic: manufacturer recommendation (e.g: initial dose of 120-200J): if unknown use maximum available.
  Second and subsequent doses should be equivalent and higher doses maybe considered
- **Monophasic:** 360J

Drug therapy

- **Epinephrine IV/IO dose:**
  1mg every 3-5 minutes
- Amiodarone IV/IO dose:
  First dose: 300mg 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

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway inplace, give e1 breath every 6 seconds (10 breaths/min) with continuous chest compression

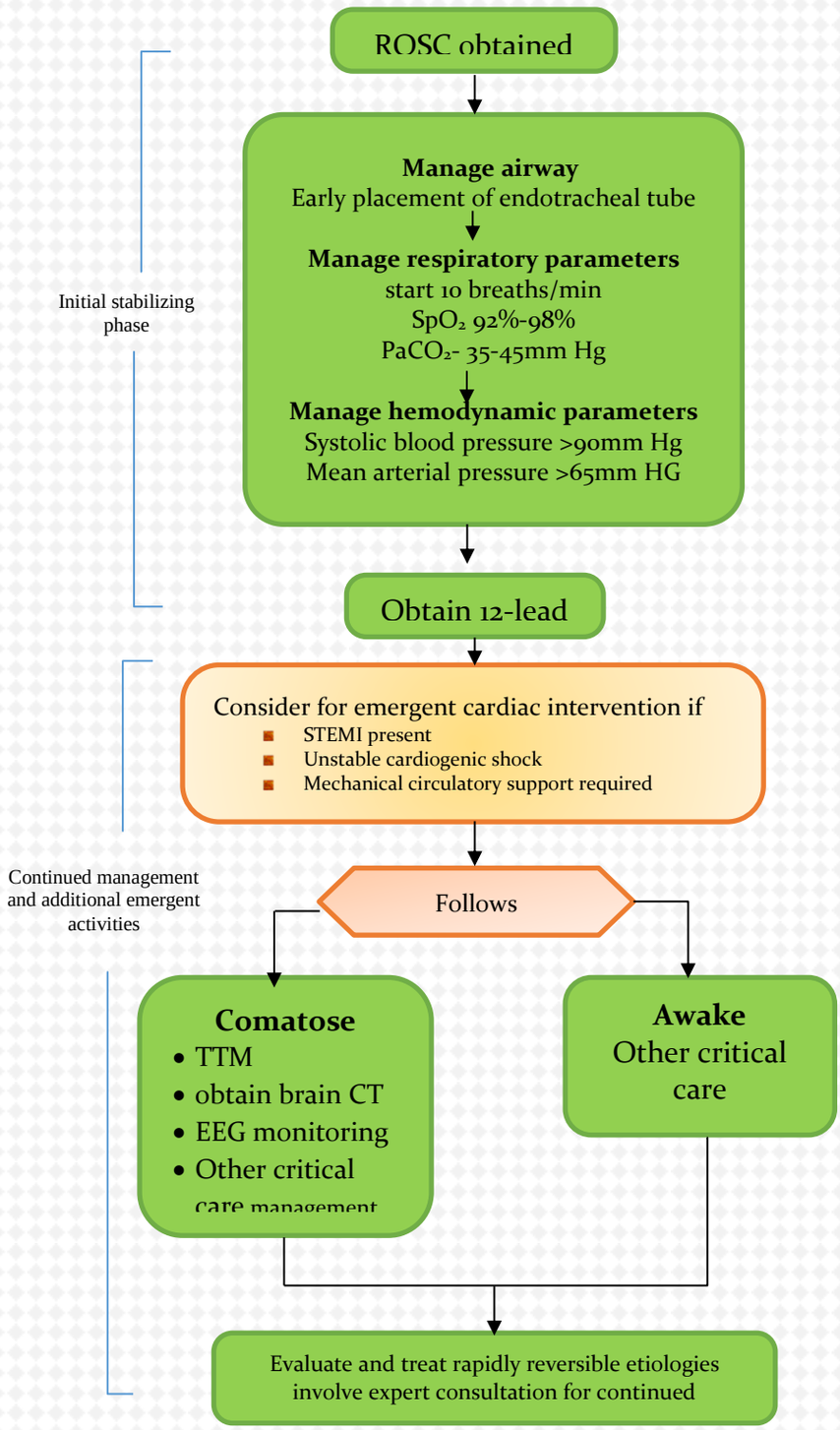
Return of spontaneous circulation (ROSC)

- Pulse and BP
- Abrupt sustained increase in $PETCO_2$ (Typically $\geq$ 40 mm Hg)
- Spontaneous arterial pressure waves with intra- arterial monitoring

Reversible causes

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo/hyperkalemia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, Pulmonary
- Thrombosis, coronary

Figure 2-4: Post Cardiac arrest care algorithm



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 FIO2 for Spo 2 920/o- 980/o; start at 10 breaths/min: titrate to PaCO2 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 32-36O 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/hyperkalemia
Hypothermia
Tension pneumothorax
Tamponade, cardiac
Toxins
Thrombosis, pulmonary
Thrombosis, coronary

2.7 HEMODYNAMIC MONITORING

I. Introduction and principle of management

The goal of hemodynamic monitoring in intensive care is to assess the adequacy of perfusion, specifically with regard to maintaining sufficient perfusion pressures and oxygen delivery. Precise volume management in critical care patients is crucial as under or over resuscitation is associated with adverse outcomes.

Types of hemodynamic assessment and monitoring:

- Non-invasive
- Invasive

a. Non-Invasive Hemodynamic Monitors

Clinical Assessment

Vital signs: heart rate, respiratory rate, blood pressure and body temperature

Physical examination: Mental status, capillary refill, jugular vein distension

Others: SaO_2, $EtCO_2$

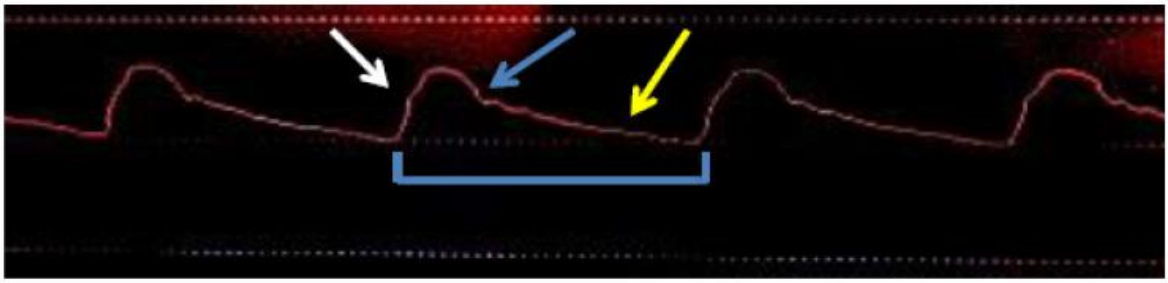
b. Invasive Hemodynamic Monitors

An invasive hemodynamic monitor utilizes an indwelling catheter placed in the vascular system. These catheters can be in arteries (typically the radial, brachial, femoral or infrequently dorsalis pedis) or veins (most typically internal jugular, subclavian and occasionally femoral). These include:

1. Invasive Blood pressure

- Measures BP with every heart beat and helps to intervene earlier. The line also uses for collecting arterial blood gas sample. Here is the normal arterial BP tracing:

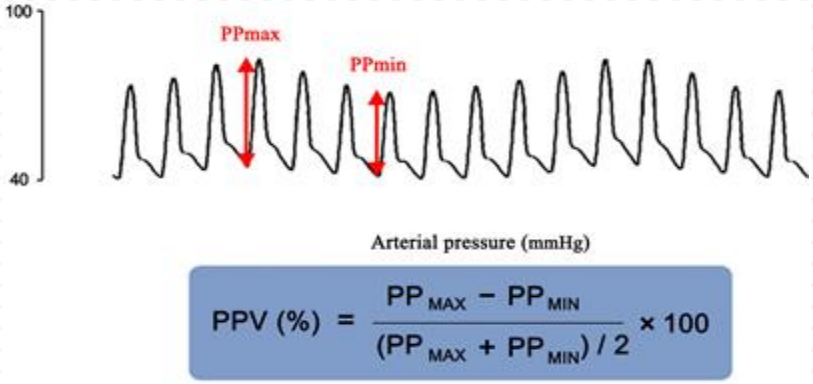
Figure 2-5: Arterial tracing



Description: White arrow signifies the systolic upstroke followed by the dicrotic notch of aortic valve closure (blue arrow) and the diastolic downslope (yellow arrow). The area under the curve of the enter best (bracket) represents the mean arterial pressure

- The transducer is zeroed to atmospheric pressure commonly at the level of the right atrium, phlebostatic axis.
- Phlebostatic axis: 4th intercostal space, on mid axillary line at supine position and up to 60 degrees of head-up tilt. It is the level where the transducer is fixed.
- Arterial line tracing also allows calculating pulse pressure variation (PPV), one of important parameters to assess fluid responsiveness.

Figure 2-6 : Blood Pressure wave form



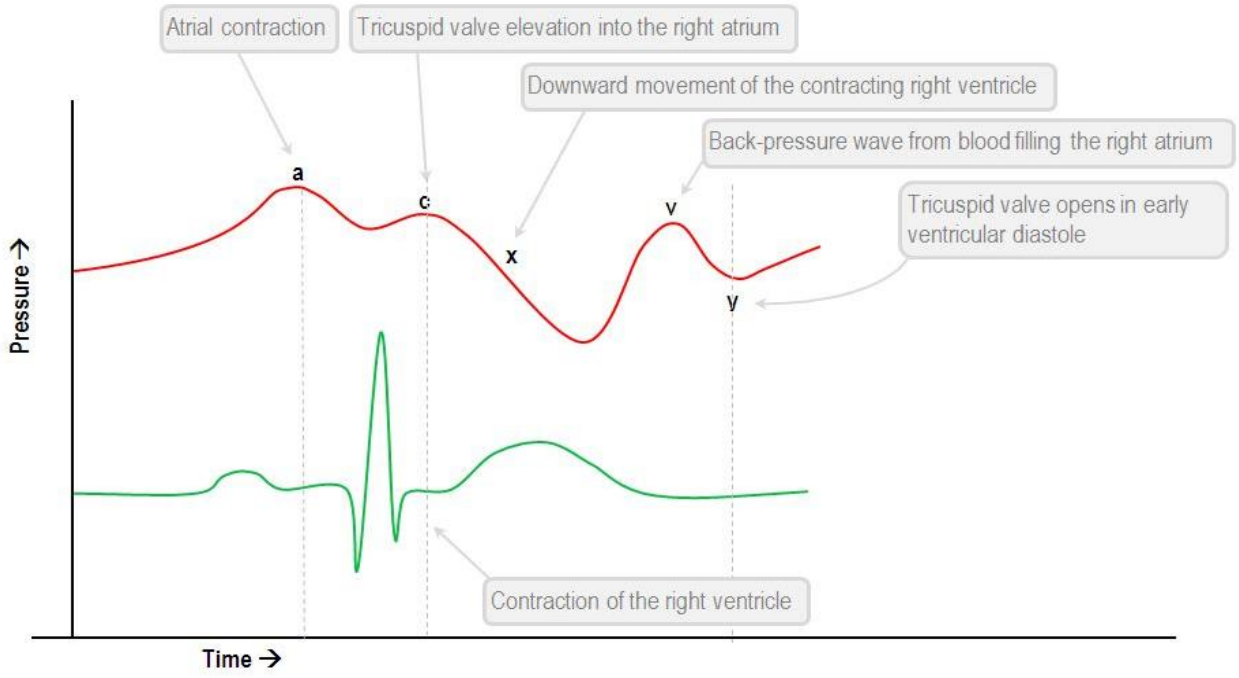
Interpretation: PPV< 12-15% -not fluid responsive

: PPV> 12-15% - preload responsive

2. ***Central Venous Catheters and Central Venous Pressure (CVP)***

- A CVC can be used to monitor both pressure and central venous oxygen saturation ScvO2. Most commonly, internal jugular or subclavian veins are used for a central venous catheter. Waveform analysis is also possible.
- CVP should be measured in the supine position, at the base of C wave, in end expiration without positive end expiratory pressure.
- The normal CVP in a spontaneously breathing patient is 0-5mmHg and in mechanically ventilated patients, up to 10mmHg is considered the upper limit. So that, CVP= measured CVP-PEEP for patients on mechanical ventilator.

Figure 2-7: Central venous pressure wave form



3. ***Cardiac Output Monitoring***

- Is essential in the management of critically ill patients and assessment of oxygen delivery
- Methods for cardiac output measurement which determines stroke volume:
  - **Direct**: cardiac catheterization

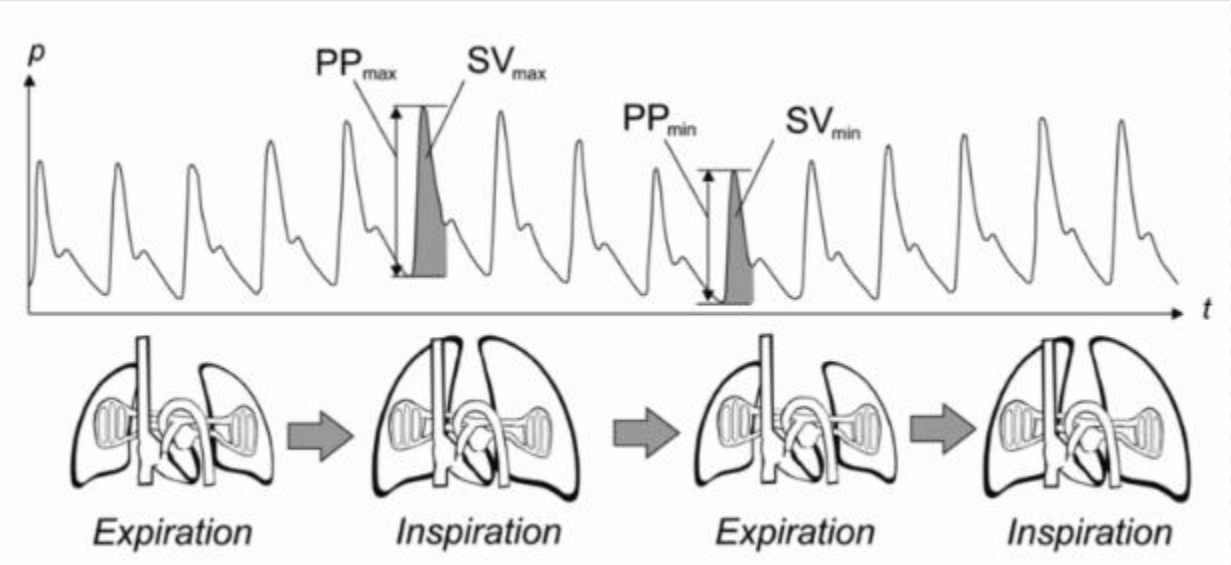
- **Indirect**: Fick principle (using CO_2) and indicator dilution (thermodilution, dye).

- Stroke volume can be measured by Ultrasound/Echocardiography, pulse wave contour analysis and by thoracic bioimpedance.

4. ***Pulse Contour Analysis***

- These systems can be divided into two categories.
- The first is based on measuring area under the systolic portion of arterial waveform and calculating the stroke volume mathematically.
- The second calculates stroke volume by looking at standard deviation of pulse pressure and effect of vascular tone on pulse morphology and comparing it to a proprietary hemodynamic database.
- Arterial pressure based cardiac output systems provide valuable information regarding fluid responsiveness using variables such as SVV (stroke volume variation).

Figure 2-8: Wage pressure



$$SVV = \frac{SVmax - SVmin}{\frac{SVmax + SVmin}{2}}$$

Interpretation: SVV <13% -not fluid responsive

: SVV > 13% -fluid responsive

5. ***Pulmonary artery catheter (Swan-Ganz Catheters)***

- PA catheter is a multiport system that allows for measurement of multiple **pressures** such as CVP, RAP, RVEDP, PAP and PCWP; **flow** CI, CO and SV in addition to blood sampling for mixed venous oxygen and central venous oxygen saturation and provides continuous core temperature measurement.
- PA catheter can serve as a resource for evaluation of cardio-pulmonary function and oxygen utilization.
- The risk of infection with any indwelling line, damage to cardiac and vascular structures and potential for inducing a right bundle branch conduction block limit its use in modern critical care practice.

II. Imaging in Hemodynamic Monitoring

Ultrasound

- Bedside scanning provides the clinician with rapid and real time determinants of volume status, cardiac function and may identify issues such as pleural or pericardial effusions.
- Ultrasound of the inferior vena cava has been used as a measure of volume status and predictor of volume responsiveness.

The IVC can be examined at the bedside for width and respiratory collapsibility. A sub costal view with or without M –mode is used to obtain cyclic changes in IVC diameter.

IVC diameter: 1.5-2.5cm –normal

: < 1.5cm- flat and >2.5cm –plethoric

Spontaneously/passively breathing patient, use collapsibility index

- Inspiratory collapse <50% -adequate volume/less fluid responsive

- Inspiratory collapse >50% -volume depletion/fluid responsive

For patients on mechanical ventilator, use distensibility index

- Distensibility Index(DI): (IVCmax – IVCmin)/(IVCmax) x100

Interpretation: DI <18% -not fluid responsive

: DI > 18% -fluid responsive

III. Assessment of fluid responsiveness

There are two types of parameters for fluid responsiveness assessment.

Static parameters: vital signs, CVP, PAC

Dynamic parameters: PPV, SVV, IVC indices, passive leg raise

IV. Monitoring and problem solving

- Close monitoring, the reading (every 10min to 1hour based on the condition of the patient)
- Proper interpretation
- Heparinise the line or flush the line with saline
- Calibration/Zeroing whenever wav form is deviated from the normal tracing

Suggested reading

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CHAPTER 3: NERVOUS SYSTEM DISEASE

3.1 STROKE

3.1.1 Acute ischemic stroke

I. Principle of care ICU

- Early identification and ruling out stroke mimics including hypoglycemia is important.
- Early signs includes: neurologic deficit, *A*rm drift, and abnormal speech.
- Stroke mimics includes seizure with post ictal paralysis, functional deficit (conversion reaction), hypertensive encephalopathy and syncope.
- Establish time when the patient was last known to be neurologically normal Avoid

II. Suggested management

- Assess the airway, breathing and circulation.
- Monitor patient with pulse oximetry. Begin oxygen therapy on any patient with Spo2 < 94%.
- Do brain CT scan to rull out intracranial bleeding.
- Early signs pf stroke on CT includes hyper dense vessel sign, Hypodensity, Loss of Grey, Sulcal effacement, Insular ribbon sign
- Fluid management: In patients with large strokes in danger of developing brain edema, fluid administration should be titrated carefully, and free water should be limited
- Control of glucose level: maintenance of blood glucose between 140 milligrams/dL (7.77 mmol/L) and 180 milligrams/dL (9.99 mmol/L).
- Antiplatelet Therapy: aspirin within 24 to 48 hours after stroke onset unless thrombolytics have been given within the prior 24 hours.

- No antiplatelet agent (including aspirin) should be given within 24 hours of receiving thrombolytic therapy
- Statin Therapy: Statins reduce the incidence of strokes among patients who are at increased risk for cardiovascular disease.
- Treatment of Fever: Fever has been associated with increased morbidity and mortality in stroke.
- Thrombolysis: Assess patients eligible for IV rtPA which is up to 4.5 h from onset.
- Give tissue plasminogen activator (tPA) after ruling out contraindications and counseling the patient and family Within 4.5 h ((if exact time of stroke onset is unclear, it is defined as the time when the patient Was last noted to be normal). Consider it when
    - 18 years or more
    - With measurable persistent neurological deficit consistent with ischemic stroke
    - BP controlled to <185/110
    - Glucose >50 mg/dL
- Exclusion criteria: ICH/SAH, Aortic arch dissection, Severe head trauma or ischemic stroke <3 month, Intracranial/spinal surgery <3 month, History of ICH, Intracranial neoplasm, Acute Bleeding diathesis
- Recombinant tissue plasminogen activator (tPA) is given at a dose of 0.9 mg/Kg up to a maximum of 90 mg (10% of dose given as a bolus over 1 min and the remainder infused over 1 h)
- If the patient is not a candidate for fibrinolytic therapy, give the patient aspirin. (First-dose 325 mg subsequent doses 100 mg–150 mg.)

III. Manage complications

Intracerebral hemorrhage: Stop thrombolytic infusion, anticoagulation and antiplatelet. CBC, PT (INR), aPTT, fibrinogen level, and type and cross match Emergent non-enhanced head CT Cryoprecipitate (Includes factor Vlll: 10 U infused over 10–30 min (onset in 1 h, peaks in 12 h); administer additional dose for fibrinogen level of <200 mg/dL or FFP.

- Tranexamic acid 1000 mg IV infused over 10 min OR c-aminocaproic acid 4–5 g over 1 h, followed by 1 g IV until bleeding is controlled (peak onset in 3 h) Hematology and neurosurgery consultations

Hypertension

- Hemorrhagic transformation occurs in 6–7% of patients.
- Stroke expansion
- Raised ICP
- Unable to protect air way and aspiration

Management of cerebral edema

- A large acute ischemic stroke typically causes cytotoxic edema. Simple measures such as keeping the head elevated at 30 or more degrees, avoiding hypotonic solutions (which lead to hyponatremia and worsening cerebral edema) and careful monitoring may be sufficient.
- In more advanced cases, osmotic therapy, external ventricular drainage, or decompressive posterior craniotomy may be necessary.

Hemorrhagic transformation

Raised ICP

Progression of the stroke: Avoid by preserving ischemic penumbra with preventing rapid lowering of blood pressure.

IV. Monitoring

- Worsening neurological examination—discontinue the infusion and obtain urgent head CT scan
- Measure BP and perform neurological assessments every 15 min during and alteplase infusion for 2 h, then every 30 min for 6 h, then hourly until 24 h after IV alteplase
- Increase the frequency of BP measurements if SBP is >180 mm Hg or if DBP is >105 mm Hg
- Delay placement of nasogastric tubes, indwelling bladder catheters, or intra-arterial pressure catheters if the patient can be safely managed without them

- Obtain a follow-up CT or MRI scan at 24 h later before starting anticoagulants or antiplatelet agents

3.1.2 Hemorrhagic stroke

I. Suggested management

Supportive care prevention of compilation is the main stay of treatment.

- Low molecular weight heparin or UFH are safe even in cerebral hemorrhage in prophylactic doses after the initial few days.
- Antiulcer prophylaxis should be started with H2 blocker.
- Proper skin and eye care should be provided.
- Aspiration precaution should be taken. Oral food should only be started after proper swallow assessment.
- Proper nutrition should be provided. The general rule of 25-30 kcal/kg and 1.2– 1.5 g protein /kg need to be provided. Nasogastric tube needs to be inserted if swallowing ability is under doubt.
- Bowel and bladder functions should be taken care of.
- Contractures should be prevented by supervised physiotherapy and appropriate use of splints.
- Fever worsens neurological outcome.
    - Fever needs to be suppressed with physical cooling and use of antipyretics. The cause of fever should be investigated.
- Glycemic control Keep blood sugar between 140 and 180 mg/dL. Hypoglycemia should be avoided.
- Full aggressive care for a day or two is appropriate in most cases of ICH.
    - Spontaneous ICH patients who present within 6 h of the acute event and who have an SBP between 150 mm Hg and 220 mm Hg, immediate lowering of SBP to less than 140 mm Hg can be potentially harmful and is not of benefit to reduce death or severe disability.

- IV labetalol is the drug of choice, preferably by IV infusion. Initial bolus dose of 10 mg IV followed by infusion of 2–8 mg/min, or intermittent boluses of 10 mg every half an hour.
  - ✦ Maximum daily dose of IV labetalol is 300 mg/day.

- **Reversal of Anticoagulation for Intracerebral Hemorrhage**
- **Neurosurgical intervention after ICH**
  - Supratentorial lobar hemorrhage >30 mL and within 1 cm of the surface , Posterior fossa or temporal lobe hemorrhage >3 cm, ICH causing hydrocephalus or brainstem compression, Hydrocephalus or IVH requiring EVD, Complicated cases requiring ICP monitoring

II. Manage Complication

Treatment of Cerebral edema

- Bolus therapy of 20% mannitol (1 mg/kg) IV given over 10–20 min repeated as frequently as needed or hypertonic saline.
- Maintain neck in neutral position to prevent impedance of jugular venous outflow.
- Elevate head of bed at 30°, mild sedation.
- Use isotonic saline for maintenance fluid, Avoid hypotonic fluids.
- In extreme case pharmacological paralysis, barbiturate coma is needed.
- External ventricular drainage is used when there is intraventricular bleed with raised ICP resulting in neurological deterioration and when there is hydrocephalus.

III. Monitor

- Neurologic deterioration

3.2 SUBARACHNOID HEMORRHAGE

I. Introduction

- Mostly caused by rupture of intracranial saccular (berry) aneurysms.

- Approximately 30–50% of patients report a history of a sudden and severe headache (the sentinel headache) that precedes a major aneurysmal bleed by 6 to 20 days which represents a warning leak.
- Examination might reveal neck stiffness and focal neurological
- **Grade the patient for prognosis purpose**
    - Grade 1 GCS score 15, no motor deficit
    - Grade 2 GCS score 13 to 14, no motor deficit
    - Grade 3 GCS score 13 to 14, with motor deficit
    - Grade 4 GCS score 7 to 12, with or without motor deficit
    - Grade 5 GCS score 3 to 6, with or without motor deficit
- CT of the head without contrast enhancement is the first essential step in the diagnosis of subarachnoid hemorrhage.
- If onset time is more than 6 hrs, lumbar puncture can be performed which detects blood or xanthochromia
- An elevated opening pressure and an elevated red blood cell (RBC) count that does not diminish from cerebrospinal fluid (CSF) tube 1 to tube 4 is the hallmark of SAH.

II. Recommended treatment

- Strict bed rest in a quiet environment to diminish hemodynamic fluctuations and pain control with titrated doses of opioid analgesics should be provided
- Deep venous thrombosis (DVT) prophylaxis should be provided with pneumatic compression stockings.
- GI ulcer prophylaxis and stool softeners are useful adjuncts.
- Hyperglycemia has been associated with a poor outcome after SAH and hence should be aggressively managed with insulin.
- Fever should be managed with antipyretics and adequate source control.
- Neuro surgical consultation for possible surgical clipping of the neck of the aneurysm and endovascular aneurysmal obliteration by metal coils are the options available for aneurysmal repair

III. Manage Complications

a. Rebleeding

- Rebreeding occurs in 8–23% of patients. The maximum risk of rebreeding is within the first 24 h after SAH, particularly within 6 h.

b. Delayed cerebral /Vaso spasm

- Generally starts 3 to 4 days after aneurysm rupture, peaks at 7 to 10 days, and resolves by 14 to 21 days.
- More than a third of the SAH patients, typically 4 to 14 days after aneurysm rupture develop focal neurologic deficits attributable to cerebral ischemia.
- In order to mitigate the effect of vasospasm, all patients with a SAH should receive Nimodipine at 60 mg fourth hourly should be administered orally to all patients from the time of presentation to 21 days after the occurrence of aSAH.
- Blood pressure to <160 mmHg
- Short-acting continuous infusion agents like Labetalol, Nicardipine, Esmolol or Enalapril are preferable for managing blood pressure.

c. Raised Intracranial Pressures and Hydrocephalus

- It appears within 3 days of the aSAH and is seen in 20% of patients. Delayed ventricular dilatation usually occurs after the tenth day.
- Acute hydrocephalus associated with aSAH can be managed by external ventricular drainage (EVD) or lumbar drainage which results in neurological improvement.

d. Prevent and Treat Seizures

- Seizures may occur in up to 20% of patients after a SAH, most commonly in the first 24 h.
- Routine prophylaxis with anti convulsant medication is not recommended in aSAH.

e. Management of Hyponatremia

- The incidence of hyponatremia following aSAH approaches nearly 50% syndrome of inappropriate secretion of anti-diuretic hormone (SIADH) and cerebral salt-wasting.

f. Manage Neurogenic Pulmonary Edema

- Characteristically presents within minutes to hours of a severe central nervous system insult such as subarachnoid hemorrhage.
- Treatment should focus on definitive treatment for aSAH while NPE management is predominantly supportive and usually resolves in 48–72 h.

IV. Monitoring

- Electrolytes specially sodium level poor prognosis necessitating daily Sodium monitoring
- Worsening neurologic status with re bleeding or hydrocephalus.

3.3 STATUS EPILEPTICS

I. Introduction and principle of care

Status epilepticus (CSE) for adults and older children (>5 years old) is "a continuous, generalized, convulsive seizure lasting more than 5 min, or two or more seizures during which the patient does not return to baseline consciousness." Initial assessment and treatment of status epilepticus should proceed simultaneously. For the purpose of standardization, initial pharmacotherapy of seizure has been divided into five

1. Stabilization phase (0–5 min)
2. Initial therapy phase (5–20 min)
3. Second therapy phase (20–40 min)
4. Third therapy phase (40–60 min)
5. Refractory status epilepticus (RSE) (>60 min)

II. Treatment algorithm for convulsive status epilepticus

1. Stabilization phase (0–5 min)

- Perform ABCD, IV access, finger stick glucose
- Consider IV Thiamine + IV Glucose

2. Initial therapy phase (5–20 min)

- IV Lorazepam (0.1 mg/kg/dose; max: 4 mg/dose, may repeat dose once,)
- OR IM Midazolam (10 mg for >40 kg; 5 mg for 13-40 kg, single dose)
- OR IV Diazepam (0.15-0.2 mg/kg/dose; max: 10 mg/dose, may repeat dose once,)
- If no IV access: Midazolam 10 mg IM can be given

3. Secondary therapy phase (20–40 min)

- IV Fosphenytoin 20 mg phenytoin equivalent (PE) IV mg/kg at 100 to150mg PE/minute, max: 1500 mg PE/dose, single dose, OR IV Phenytoin 20 mg/kg at 25-50 mg/min

OR

- IV Valproic acid (40 mg/kg, at 10 mg/kg/minute, max: 3000 mg/dose, single dose,

OR

- IV Levetiracetam (40 to 60 mg/kg, max: 4500 mg/dose, single dose,) over 15 min If none of the above is available, then:
- IV Phenobarbital (15 mg/kg, single dose)

4. Third therapy phase (40–60 min)

- No clear evidence to guide therapy
- Intubate and start mechanical ventilation
- Repeat second line therapy repeat fosphenytoin if given previously (5 mg/kgPE),
- OR Anesthetic doses of thiopental, midazolam, pentobarbital, propofol (with continuous EEG monitoring)

5. Refractory status epilepticus (>60 min)

Adults and children: Midazolam 0.2 mg/kg IV (maximum 10 mg) bolus over 2 min followed; 2by 0.05–0.5 mg/kg/h cIV or propofol 2–5 mg/kg IV bolus followed by 5–10 mg/ kg/h cIV or thiopental 10–20 mg/kg IV bolus followed by 0.5–1 mg/kg/h cIV or pentobarbital bolus 10 mg/kg at <25 mg/min followed by cIV 0.5–2 mg/kg/h

If seizures continue, consider the following emerging therapies

Inhalational anesthetic agents: Isoflurane at 0.8–2 vol.%, titrated to obtain the EEG burst suppression pattern

Ketamine: 1.5 mg/kg bolus, cIV 0.01–0.05 mg/kg/h

IV intravenous, *cIV* continuous intravenous infusion, *NGT* nasogastric tube, *GCSE* generalized convulsive status epileptics

III. Manage complication

- Air way and breezing compromise
- Electrolyte imbalance
- Rhambdo myolisis and renal failure
- Raised ICP
- Injury with fracture and dislocation

IV. Monitoring

- Clinical monitoring for complications

3.4 GUILLAIN-BARRE SYNDROME (GBS)

I. Introduction

Causes of admission to the ICU

- Inability to cough out secretions
- Inability to swallow and at risk of aspirating orogastric secretions
- Impaired respiratory muscle function leading to respiratory failure
- Secondary complications of primary disease e.g. sepsis
- Dyspnea might not be a predominant symptom in patients with respiratory failure due to neuromuscular weakness
- Impaired forced expiration results in ineffective cough causing accumulation of secretions
- Retention of secretions results in segmental collapse and History of acute gastrointestinal or respiratory tract infection 1–3 weeks

II. Treatment

- Supportive care and prevention of complication
- Intravenous Immunoglobulin (IV IG) 400 mg/kg/day for 5 days OR
- Plasmapheresis 40–50 mL/kg/exchange (total 200–250 mL/kg) in three to five exchanges over 7–14 days

III. Management of Complications

- Respiratory failure
- Sepsis
- Air way and breathing compromise

IV. Monitoring

- In non-intubated patient single breath count and or spirometer And ABG for monitoring of PH and carbon dioxide level.

3.5 TETANUS

I. Principle of care

Tetanus severity depend on onset time of less than 7 days, onset time shorter than 48 hrs, tetanus acquired from burn, surgery abortion or injections, narcotic addiction, fever more than 40 degree, tachy cardia more than 120. Moderate tetanus (more than 2 score) or above or complications may mandate admission to ICU

II. Management

- ABC of life
- Prophylactic tracheostomy shall be considered early in the course
- Environmental control: Isolation, dim light, reduce noise…
- Wound debridement
- Antibiotic: Metronidazole (preferred), penicillin G (alternative), 2nd or 3rd generation cephalosporins (mixed infection).
- Antitoxin to neutralize unbound toxin:
- Human Tetanus Immunoglobulin (HTIG), 3000 to 6000 units IM, is preferred and is associated with less anaphylactic reaction.
- Equine Tetanus antitoxin (TAT), 10,000 – 20,000 units IM
- Control of muscle spasms
    - Benzodiazepines- for rigidity and spasms.
    - Diazepam, with starting dose of 10 to 30 mg IV, although doses as high as 120 mg/kg/day can be used via infusion or divided doses.
    - Chlorpromazine can be used alternating with diazepam (if higher doses of the latter are required while patient is not intubated), magnesium sulphate
    - Anesthetic drugs like propofol can be considered if the spasm is not controlled with the above drugs
    - Neuromuscular blocking agents such as atracurium are used when sedation alone is inadequate.

- Active immunization with a total of three doses of tetanus toxoid (tt) vaccine spaced at least two weeks apart.

III. Manage complications

- Cardiovascular **instability and dysoutonomia**
  - Increase sedation with IV magnesium sulfate, morphine, or other sedatives.
  - Cardiovascular selective drugs (e.g., labetalol, esmolol, calcium antagonists, and inotropes.) Short-acting drugs that allow rapid titration are preferred
- Trauma and fracture
- Malnutrition
- Rhambdo myolysis
- Electrolyte imbalance

IV. Monitoring

- Continues ECG monitoring
- Electrolyte and RFT daily
- Air way

3.6 MENINGITIS AND MENINGOECEPHALITIS

I. Introduction

- **Meningitis** is defined as inflammation of the membranes of the brain or spinal cord and is also called arachnoiditis or leptomeningitis. Encephalitis denotes inflammation of the brain itself.
- **Bacterial meningitis** is a life-threatening emergency that affects about 1.2 millions individuals worldwide each year.
- The incidence and major pathogens vary by region, country, age group, and immunization status.
- In direct contiguous spread, organisms gain entry into the CSF from adjacent infections such as sinusitis, brain abscess, or otitis media. Organisms can also enter directly with penetrating traumatic injury, through congenital defects, or during neurosurgical procedures.
- **Risk factors** includes Sinusitis, Immunosuppression/splenectomy, Alcoholism, Pneumonia, Diabetes mellitus, Neurosurgical procedure/head injury, Indwelling neurosurgical device/cochlear implant, Unvaccinated to Haemophilus influenzae type b, Neisseria meningitidis, or Streptococcus pneumonia

II. Treatment

- After addressing airway, breathing, and circulation status, immediately initiate empiric antibiotic therapy if bacterial meningitis is clinically suspected.
- Obtain blood cultures to assist in identification of the organism and to help guide inpatient therapy if it will not delay time to antibiotics.
- Antibiotic selection based on the clinical scenario including age, immunization status, living conditions, and past medical history.

Table 3-1: Guidelines for Treatment of Meningitis Based on Patient Characteristics

Patient Characteristic	Primary Empiric Therapy	Presumed Bacteria
Immunocompetent	Cefotaxime 2 grams IV or ceftriaxone 2 grams IV, plus vancomycin 15–20 milligrams/kg IV If severe penicillin allergy, can replace ceftriaxone with meropenem 2 grams IV or moxifloxacin 400 milligrams IV	*S. pneumoniae* and *N. meningitides*
Age >50 y **Immunocompromised**	Cefotaxime 2 grams IV or ceftriaxone 2 grams IV, plus vancomycin 15–20 milligrams/kg IV PLUS ampicillin 2 grams IV If severe penicillin allergy, can replace ampicillin with trimethoprim-sulfamethoxazole 15–20 milligrams/kg/d ivided 4 times daily	*L. monocytogenes*

Administration of dexamethasone to patients with presumptive pneumococcal meningitis before or with the first dose of antibiotics can reduce CSF inflammation, reduce the risk of morbidity and mortality in adults, and reduce hearing loss and other neurologic sequelae in children.

- The recommended dosage of dexamethasone is 10 milligrams IV every 6 hours for 3 days for adults

3.7 VIRAL ENCEPHALITIS

I. Introduction and principle of care

- Viral CNS infections can also cause viral encephalitis, which is an infection and inflammation of the brain parenchyma.
- Viral encephalitis is clinically distinguished from viral meningitis with presence of neurologic findings such as altered level of consciousness, focal weakness, or seizures, although the two often coexist.
- The causes of viral encephalitis vary year to year and across geographical locations, with an incidence of 3.5 to 7.5 per 100,000 people.
- HSV accounts for 40% to 50% of cases where a cause is determined.
- HSV-1 is responsible for most cases of HSV encephalitis; HSV-2 frequently causes aseptic meningitis but is not usually associated with development of encephalitis.
- Consider encephalitis in patients exhibiting behavioral changes, new psychiatric symptoms, cognitive deficits, or seizures.
- Rash or skin vesicles suggest herpes zoster, and skin vesicle culture may be useful for diagnosis. Lymphadenopathy or splenomegaly points to Epstein-Barr virus, which can be picked up on serologic testing.

II. Treatment

- High-dose acyclovir at 10 milligrams/kg per dose IV every 8 hours.
- Initiate treatment as soon as possible because the prognosis of HSV encephalitis is correlated with neurologic condition at the time antivirals are initiated.
- Treat varicella-zoster virus with acyclovir, 10 to 15 milligrams/kg per dose IV every 8 hours. Patients with herpes zoster virus encephalitis may also benefit from acyclovir therapy.
- Treat patients with cytomegalovirus encephalitis with ganciclovir, 5 milligrams/kg per dose IV every 12 hours.

III. Mange complications

- Raised ICP
- Electrolyte disturbance

IV. Monitoring

- Electrolyte, neurologic status and air way

Suggested reading

1. R. Chawla and S. Todi (eds.), ICU Protocols: A stepwise approach, DOI 10.1007/978-81-322-0535- 7_76, © Springer India 2012.
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CHAPTER 4: RENAL CRITICAL CARE

4.1 ACUTE KIDNEY INJURY (AKI) IN ICU

I. Introduction and principle of care

- Abrupt (less than 48hrs) increase in creatinine (Cr) ≥0.3mg/dl, increase in Cr >50% or urine output <0.5ml/kg/hr for greater than 6hrs. Ischaemia (prerenal and acute tubular necrosis (ATN)) accounts for >50% of in-hospital AKI.
- Oliguria is less than 0.5 mL/kg/h urine output for at least 2 h. Perform supra-pubic percussion or ultrasound for bladder fullness in all cases of low urine output to exclude retention of urine.
- Sudden drop of urine output, no urine or fluctuating levels of urine output in a catheterized patient who is otherwise stable, may indicate a partial or complete catheter block with clot or debris or pericatheter leak.
- Patients with AKI are frequently classified as being nonoliguric (urine output >400 mL/day), oliguric (urine output <400 mL/day), or anuric (urine output <100 mL/day).
- Etiologies can be intrinsic, prerenal and post renal

Laboratory evaluation

- Serum creatinine, hyperkalemia, and, on urinalysis, albuminuria and/or an abnormal urine sediment
- Serum and urine protein electrophoresis should be performed in patients with bone pain, hypercalcemia, and hyperglobulinemia where paraproteinemia is suspected.

Specific urine analysis findings:

- **Acute interstitial nephrits (AIN):** WBCs, WBC casts with or without RBCs
- **Glomerulonephritis:** dysmorphic RBCs, RBC casts
- **Serologies-if indicated:** ANA, anti-dsDNA, C_3, ANCA

Renal ultrasound or CT: To rule out obstruction and estimate chronicity

Renal biopsy: if cause remains unclear

II. Treatment

- Maintain mean arterial pressure (MAP) of more than 65 mmHg by adequate volume loading and with vasopressors if necessary. MAP may have to be kept higher if the patient is hypertensive or has a high IAP.
- Always suspect sepsis
- Avoid nephrotoxic insult
- A Furosemide challenge test, after a reasonable correction of volume status, by administering 1–1.5 mg/kg of Furosemide bolus may help in identifying AKI. Urine output of <200 ml in 2 h of the diuretic challenge is Indications for urgent dialysis (when condition refractory to conventional therapy):
    - **A**cid-base disturbance-acidemia
    - **E**lectrolyte disorder-hyperkalemia, hypercalcemia(tumor lysis)
    - **I**ntoxication-methanol, ethyleneglycol, salicylates
    - **O**verload of volume
    - **U**remia-pericarditis, encephalopathy, bleeding

Mange specific causes

Rhabdomyolysis: Treat with vigorous hydration (may require up to 10 L of normal saline during 24 hr) to replete volume and increase urinary flow rate

Ischemia: Results from prolonged prerenal state (hypovolemia, sepsis, cardiogenic shock). Hence treat underlying illness

Intravascular iodinated contrast: Characterized by both renal vasoconstriction and tubular injury Risk factors: preexisting renal dysfunction, heart failure, diabetes, volume depletion, multiple myeloma, large volume of contrast, and high-osmolarity contrast. Hydration with intravenous sodium chloride or sodium bicarbonate shown to be beneficial N-acetylcysteine orally or intravenously may help in prevention

Immune complex: Hypocomplementemia seen with postinfectious GN, MPGN, SLE, endocarditis, and cryoglobulinemia. Treat the underlying cause

Pauci-immune and anti-GBM disease: pulmonary hemorrhage syndromes

- May have positive serum c-ANCA or p-ANCA or anti-GBM antibody (Goodpasture's)

  Dysmorphic RBCs in urine suggest glomerular origin
- Supportive therapy for pulmonary compromise; Corticosteroids and cytotoxic agents
- Plasma exchange for Good pasture's or vasculitis with pulmonary hemorrhage or advanced renal failure

Microvascular HUS and TTP: low platelets, hemolytic anemia, and schistocytes on perip TTP requires emergent plasma exchange, Supportive care for HUS, Refractory TTP may benefit, from immunosuppression, with prednisone

III. Monitoring

- Monitoring of urine output, by placing an indwelling urinary catheter.
- Continuous monitoring of hemodynamic parameters (CVP and arterial line) is also mandatory.
- Intra-abdominal pressure if abdominal compartment syndrome is suspected
- Electrolyte specially potassium
- Optimize hemodynamics, monitor Watch for and correct complications: volume overload, electrolyte abnormalities, acid/base status
- After relieved obstruction **watch for:**
  - ✦ hypotonic diuresis-treat with IVF,
  - ✦ hemorrhagic cystitis-avoid by decompressing slowly

4.2RENAL REPLACEMENT THERAPY

I. Principle of care

Conventional indications for RRT include metabolic acidosis, hyperkalemia, and volume overload, and severe uremic symptoms refractory to medical management. Other indications include certain intoxications of certain substances (ethylene glycol, methanol, lithium, 2, 4 D etc.), where either the substance or the toxic metabolite will be cleared with dialysis. Once the decision has been made to initiate RRT, one needs to select a modality. The available modalities are IHD, CRRT, sustained low-efficiency dialysis (SLED), or peritoneal dialysis. The choice depends on the availability of therapies at the institution, physician preference, the patient's hemodynamic status, and the presence of comorbid conditions.

II. Complications of renal replacement therapy

Hypotension

Volume-depleted and septic patients are at heightened risk, and careful attention to the physical examination and invasive hemodynamic monitoring when indicated can help ensure adequate volume resuscitation prior to initiating the dialysis session.

Arrhythmias

Cardiac arrhythmias can occur in the setting of rapid electrolyte shifts in acute hemodialysis

Dialyzer Reactions

Dialyzer reactions are rare during hemodialysis.

Disequilibrium syndrome

Dialysis Catheter-Related Problems

Infection and bacteremia occur, prompt catheter removal is generally recommended unless vascular access is especially difficult. Thrombus or fibrin sheaths can form around or inside the catheters causing inadequate blood flows for dialysis.

III. Monitoring

- Care full hemodynamic monitoring
- Electrolyte after dialysis
- Urea drop after dialysis

4.3 ELECTROLYTE ABNORMALITIES

4.3.1 Hyponatremia

I. Introduction

It is generally defined as a serum level of less than 135 mEq/L (**Normal range**: 135 - 145 mol/L).

Emphasis should be given to treatment of the underlying causes in an asymptomatic patient with chronic illnesses.

Mild: serum sodium = 130-135 mEq/L,

Moderate: serum sodium = 125-129 mEq/L,

Severe: serum sodium <125 mEq/L.

Time of onset

- **Acute hyponatremia:** Hyponatremia that is documented to have occurred over <48 hours. This usually results in cerebral edema and significant symptoms.
- **Chronic hyponatremia:** Hyponatremia that is documented to have occurred over ≥48 hours.
- **Unknown duration:** When it cannot be classified by time of onset. Treatment should be based on symptoms.

Serum tonicity

The most common type of hyponatremia is **hypotonic hyponatremia**, while hypertonic and isotonic hyponatremia are less common.

II. Treatment of hyponatremia

General issues to be addressed before specific treatment:

- Is it acute or chronic or unknown duration?
- Is it symptomatic or not?
- Optimal rate of correction?
- Fluid status?

a. **Treatment based on Onset of hyponatrimea**

1. ***Acute and symptomatic:*** Aim to increase plasma Na+ by 1-2meq/hr to a total of 4-6meq/ 24 hrs which is enough to relieve acute symptoms (usually within 4hrs)

   After this, treatment as protocol of chronic hyponatremia applies

   Use 3% hypertonic saline (= 513meq/L of sodium) or table salt

   Na deficit = TBW (Na desired-Na measured)

   Give usually over 4hrs

2. ***Chronic:*** Correct at a rate of <10-12meq/24hrs or <18meq/ 48hrs

Treat underlying causes

- Hypovolemic hyponatremia- isotonic saline
- Treat causes of hypervolemia
- IV saline and resumption of normal diet for low solute intake
- Hypokalemic patients-correction of K corrects Na
- Water restriction <1 liter per day

3. ***Unknown Duration:*** Treatment should be dependent on the presence or absence of symptoms. It should aim to relieve symptoms with the acute treatment protocol above. If patient is asymptomatic, treatment should rely on treatment of the causes.

b. **Treatment based on volume of hyponatrimea**

Hypovolemic hyponatremia

Isotonic intravenous fluids (e.g., normal saline 0.9% or a balanced solution such as lactated Ringer solution) should be administered in 250-1000 mL boluses to maintain blood pressure

Hypervolemic or euvolemic hyponatremia

All patients should be fluid restricted (including all oral and intravenous fluids) of 1 L/day.

Rate and Degree of Correction

- Calculate Na Deficit (for men) = Wt. X 0.6 (target Na – current Na)
- Na Deficit (for women) = Wt. X 0.5 (target Na – current Na)
- Volume of Hypertonic Saline in ml = Na deficit/512 X 1000

- Infusion Rate (L/h) = Volume of hypertonic saline in ml/ target Na – current Na/desired correction rate
- In chronic hyponatraemia correction should not exceed 0.5 mmol/L/h in the first 24 h and 0.3mmol/L/h thereafter.
- In acute hyponatraemia the ideal rate of correction is 1.5 – 2 mEq/h for 4 hours then slow correction 12 mEq/L over 24 hours.
- Plasma Na+ of 125–130 mmol/L is a reasonable target for initial correction of both acute and chronic states.
- Treat complication
- The rate of correction of serum sodium should be kept to <8 mEq/L/day to prevent myelinolysis.
- Attempts to achieve normo- or hypernatraemia rapidly should be avoided.
- Neurological complications, e.g. Central Pontine Myelinolysis, are related to the degree of correction and the rate. Premenopausal women are more prone to these complications.

4.3.2 Hypernatremia (>145meq/L)

It results from water loss, hypothalamic lesions impairing thirst (osmoreceptor function) or sodium overload.

Administration of desmopressin increases urine osmolality by 50% in central diabetes inspidus (CDI) but no or little response in nephrogenic diabetes inspidus (NDI)

Treatment

- Slowly correct over 48-72hrs by no more than 10meq/day
- Rarely, Na can be corrected by 1meq/hr in acute hypernatremia (<48hrs) due to Na loading
- Calculate water deficit – replace it, ideally orally
- Water deficit= TBW x (Na measured- Na desired)/Na desired Plus ongoing loss(insensible, others)
- D_5W IV can cause hyperglycemia which precipitates hypernatremia

- ¼ or ½ NS if hypotensive or low BP
- NS only if severe hyponatremia of frank hypotension

If central diabetes insipidus (CDI):

- **Restrict salt:** give loop diuretics (furosemide).
- **Complete CDI:** desmopressin (DDAVP) (10µg BD intra-nasally or 1–2µg BD IV)
- **Partial CDI:** desmopressin, but often responds to drugs that increase the rate of ADH secretion or end-organ responsiveness to ADH, e.g. chlorpropamide, hydrochlorothiazide

Nephrogenic DI:

- Low salt diet and thiazides.
- High dose desmopressin may be effective.
- Consider removal of causative agents, e.g. lithium, demeclocycline.

4.3.3 Hypokalemia (<3.5 meq/l)

I. Introduction and principle of care

The causes could be spurious (in vitro uptake by profound leukocytosis), inadequate intake, redistribution (from insulin, alkalosis, B_2 adrenergic agonists, theophylline, caffeine, hypokalemic paralysis with thyrotoxicosis), extrarenal losses, renal loss (diuretics, high doses of penicillin related antibiotics, steroids, theophylline, renal tubular toxins, DKA, Conn's syndrome, Secondary hyperaldosteronism, Cushing's syndrome, metabolic alkalosis, hypomagnesemia) and other causes like hypoaldosteronism, metabolic acidosis and hypomagnesemia.

Clinical manifestation

Symptoms include respiratory muscle weakness, ileus, cardiac arrhythmias and ECG changes.

ECG: changes prolongation of the PR interval, T wave flattening and inversion, ST depression, Prominent U waves

II. Treatment

- Estimate potassium deficit (100meq/L fall in total K+ for 0.27meq/l decrease in plasma K+, this may not apply in redistributive causes)
- Potassium bicarbonate and its precursors –in metabolic acidosis and diarrhea
- Potassium phosphate- rarely used except in concomitant severe hypophosphatemia
- KCl in all other clinical situations
    - IV (via central line) with ECG monitoring when there is a clinically significant arrhythmia (20 mmol over 30 min, repeated according to levels)
    - Slower intravenous replacement (20 mmol over 1 h) should be used where there are clinical features without arrhythmias.
    - Oral supplementation (a total of 80–120 mmol/day) where there are no clinical features.
    - HSeck Mg++ level as adequate Mg++ is necessary for correction.

a. Treatment of mild to moderate hypokalemia (3-3.4meq/l)

- Po supplementation (20-80meq/d in 2-4 divided doses)
- Potassium sparing diuretics for renal wasting and primary aldosteronism
- Non-selective B agonists like propranolol for hypokalemia due to increased sympathetic activity

b. Treatment of severe (<2-3meq/l) or symptomatic hypokalemia

- Rapid administration of K (PO or IV)
- PO KCl 40meq 3-4x/day or IV 20 meq every 2-3hrs
- Monitor serum level every 2-4hrs
- Treat as mild to moderate hypokalemia once K level is persistently >3-3.4meq/l and asymptomatic

III. Prevent complications

- Pain and phlebitis occurs at a rate greater than 10meq/hr or concentration more than 3meq/l
- If large infusions are needed for severe hyperkalemia, use central vein or multiple peripheral veins

- 1000ml fluid- maximum 60meq/l of K
- 100-200ml in peripheral vein-maximum 10meq/l
- 100ml in central vein- maximum 40meq/l

4.3.4 Hyperkalemia (>5.5meq/L)

I. Introduction and principle of care

The cause could be Pseudohyperkalemia (fast clenching during venipuncture, marked thrombocytosis, leukocytosis, and/or erythtrocytosis, cooling of blood after venipuncture), redistribution hyperkalemia (resulting from acidosis, beta adrenergic blockers, digitalis overdose, hypertonic states, familial hyperkalemic periodic paralysis, fluoride, succinylcholine), impaired K excretion, aldosterone deficiency or tubular non responsiveness to aldosterone, renal failure(GFR<20%), medications (like ACEis, ARBs, renin inhibitors, cyclosporine, tacrolimus, NSAIDs, COX-2 inhibitors, K sparing diuretics).

Clinical features

Include arrhythmias, ECG changes, ascending paralysis, metabolic acidosis

ECG: A tall peaked and symmetrical T wave is the first change seen on the ECG in a patient with hyperkalemia

II. Treatment

a. **Treat reversible causes first e.g. stop medications that increase serum K**

b. **Antagonism of cardiac effects:** Calcium gluconate 10ml of 10% (contains 90mg of elemental calcium) or 3-4ml of 10% calcium chloride (10ml of 10% contains 270mg of elemental calcium) IV over 3-4min without cardiac monitoring

- Onset is over 1-3min and lasts for 30-60min
- Repeat dose if no ECG change or symptoms recur
- Same amount can be given in 100ml of D_5W over 20-30min to avoid acute hyperkalemia

c. **Redistribution into cells**

1. ***10IU of Regular Insulin IV + 50ml of 50% dextrose (25g glucose)***

- Onset in 10-20min, peaks at 30-60min and lasts for 4-6hr
- Should be followed by infusion of 10% dextrose at 50-75ml/hr, but no need if glucose >200-250mg/dl
- Frequent blood glucose monitoring

2. ***B2 agonists***

- Albuterol 10-20mg via nebulizer in 4ml of NS over 10min
- Onset in 30min, peak at 90min and lasts 2-6hrs

d. Removal of potassium (potassium exchange resins, diuretics, dialysis)

- Sodium polystyrene sulfonate (SPS): 15-30g + 33% sorbitol
- Dialysis effective means of removal of k

4.4 ACID- BASE DISORDERS & ABG INTERPRETATION

I. Introduction and approach to Acid–Base Disorders

Step 1: See the pH: To determine whether the abnormality is acidosis or alkalosis. Use 7.40 as absolute normal. Acidosis < 7.40 or alkalosis >7.40

Step 2: determines whether primary disorder is a respiratory or metabolic process.

Step 3: Expected compensation is calculated.

Step 4: **Calculate anion gap**

Anion Gap = [Serum Sodium (Na+) – (Serum Chloride [Cl–] + Serum HCO_3–)]

Normal values are between 8 and 12 mEq/L.

Step 5: **Calculate the delta-delta:** it is used in anion gap metabolic acidosis. This would reveal additional metabolic disorder.

Step 6: Finally, determine the likely cause of the disorder and whether or not urgent intervention is necessary.

Table 4-1: Expected Compensation to a Primary Acid-Base Disorder

Primary Disorder	Expected Compensation	PH
Metabolic Acidosis	Winter's Formula $pCO_2 = 1.5 [HCO_3] + 8 +/- 2$	
Metabolic Alkalosis	Summer's Formula $pCO_2 = 0.7 [HCO_3] + 20 +/- 5$	
Respiratory Acidosis Acute	$[HCO_3]$ rises by 1 for every 10 mm Hg increase in pCO_2	Expect pH decrease 0.08 for every 10 mmHg increase in PCO_2
Respiratory Acidosis Chronic	$[HCO_3]$ rises by 4 for every 10 mm Hg increase in pCO_2	Expected pH do decrease 0.03 for every 10 mmHg increase in PCO_2
Respiratory Alkalosis Acute	$[HCO_3]$ decreases by 2 for every 10 mm Hg decrease in pCO_2	Expect pH increase 0.08 for every 10 mmHg increase in PCO_2
Respiratory Alkalosis Chronic	$[HCO_3]$ decreases by 5 for every 10 mm Hg decrease in pCO_2	Expected pH to increase 0.02 for every 10 mmHg decrease in pCO_2

II. Identify the Cause and treat

a. Cause and treatment of Metabolic Acidosis

Causes of metabolic acidosis

If there is an **elevated anion gap**, consider what these sources might be with the mnemonic **MUDPILES**: **M**ethanol, **U**remia, **D**iabetic ketoacidosis (other types of ketoacidosis as well as alcohol, starvation), **P**araldehyde, **I**soniazid (**&I**ron), **L**actic acidosis, **E**thylene glycol, **S**alicylates.

Causes of Normal Anion Gap Acidosis

GI losses**:** Diarrhea**,** Pancreatic, biliary, intestinal fistulas**,** ostomy.

Renal losses: Treatment phase of ketoacidosis, acute& chronic renal failure.

1. Correction of the underlying cause

The first goal of therapy is to identify and correct the underlying cause.

Reversal of these underlying processes is of great importance and can often be sufficient to improve the acidemia.

A common example of this can be illustrated in patients with DKA, who have prompt improvement with the administration of insulin and fluids.

2. Sodium bicarbonate therapy

Administration of bicarbonate is usually favored in life-threatening acidemia with a pH less than

3. Hemodialysis

Continuous or intermittent hemodialysis may also be used in refractory cases.

b. Cause and treatment of Metabolic Alkalosis

Causes of Metabolic Alkalosis

Most cases of metabolic alkalosis encountered in the intensive care unit are induced by diuretics or the loss of gastric secretions due to vomiting or nasogastric suctioning.

Metabolic alkalosis can be broadly categorized into a chloride-responsive or chloride-resistant process.

- Intravenous sodium chloride fluid administration will reverse chloride-responsive metabolic alkalosis.
- Oral potassium chloride 40 mEq every 4 to 6 hours
- If the patient is unable to take medication enterally, intravenous infusion of potassium chloride at a rate of 10 mEq/hr
- Acetazolamide can be considered in non-chloride responsive metabolic alkalosis

c. Cause and management of respiratory acidosis

Causes of respiratory acidosis

Alveolar hypoventilation: neuromuscular diseases, central nervous system (CNS) medication/drug effects, asthma and COPD, pulmonary edema.

- Correcting alveolar hypoventilation is the main stay of treatment and requires treating the underlying cause of hypoventilation.
- This may include bronchodilators (for patients with asthma and COPD), reversal of medication/drug effects, treatment of pulmonary edema, treatment of neuromuscular diseases, and mechanical ventilation (either via NIPPV or via endotracheal intubation).
- Small doses of sodium bicarbonate can be considered in cases of intractable hypercapnia with severe acidosis (pH <7.1).

d. Cause and management of respiratory alkalosis

Causes of Respiratory Alkalosis

Causes include: Hyperventilation (due to heart disorder or other, including improper mechanical ventilation), stress, pulmonary disorder resulting in hypoxemia, high altitude, and salicylate poisoning.

- Treatment of the underlying etiology
- Causes of hypoxia should be identified and treated

- In mechanically ventilated patients, minute ventilation should be decreased by decreasing the respiratory rate and/or tidal volume
- For psychogenic hyperventilation, reassurance and anxiolytics can be used
- Acetazolamide can be used to induce a metabolic acidosis to compensate for the respiratory alkalosis caused by high altitudes

Suggested reading

1. R. Chawla and S. Todi (eds.), ICU Protocols: A stepwise approach, DOI 10.1007/978-81-322-0535- 7_76, © Springer India 2012.
2. Brian Brown, the Washington Manual Of Critical Care. Lippincott Williams & Wilkins, A Wolters Kluwer, 2012

CHAPTER 5: ENDOCRINE DISEASE

5.1 DIABETIC KETOACIDOSIS

I. Introduction and Principle of care

- Diabetic ketoacidosis (DKA) is an acute, life-threatening complication of diabetes mellitus.
- DKA occurs predominantly in patients with type 1 diabetes mellitus, but 10% to 30% of cases occur in newly diagnosed type 2 diabetes mellitus.
- The clinical manifestations of DKA are related directly to hyperglycemia, volume depletion, and acidosis.
- Meeting the goals of safely replacing deficits, supplying missing insulin, and correcting acidosis and electrolyte disturbance requires ICU admission and frequent monitoring specially for severe DKA.
- The principle of management in DKA includes Correction of hypovolemia with intravenous fluids, Correction of hyperglycemia with insulin, Correction of electrolyte disturbances, particularly potassium loss, Correction of acid-base balance and Treatment of precipitants.

II. Management

a. Fluid resuscitation

- Fluid management depends on patient presentation
- Patients without extreme volume depletion can be managed safely with a more modest fluid replacement regimen such as 250 to 500 mL/h NS for 4 hours.
- The estimated fluid loss in patient with extreme fluid depletion is around 5-10L and it should be replaced over 24hrs.
- In general, the first 2 L are administered rapidly over 0 to 2 hours, the next 2 L over 2 to 6 hours, and then an additional 2 L over 6 to 12 hours. This replaces approximately 50% of the total water deficit over the first 12 hours, with the

remaining 50% water deficit to be replaced over the subsequent 12 hours depending on the degree of dehydration and central venous pressure readings (IVC size)

- When the patient becomes euvolemic, the physician may switch to half the isotonic sodium chloride solution, particularly if hypernatremia exists.
- When glucose approaches 250 (11 mmol/L) change IV to D5 0.5 NS
- If the patient is in shock approach and manage according to shock management guideline

b. Correcting electrolyte disturbances

- If initial [K+] >5.2 do not administer potassium
- If initial [K+] is >3.3 and <5.2 and the patient has adequate urine output add 20 of K+ to each liter of fluid.
- If initial [K+] is <3.3 hold insulin drip and give K+ at 40 mEq/h in 500ml N/S through IJ/EJ vein access with rate of 20meq/hr.; until [K+] is >3.3 then initiate insulin drip.

c. Glucose management

- Insulin should be started about an hour after IV fluid replacement is started
- Administer insulin at a rate of 0.1 to 0.14 unit/kg/h with no insulin bolus once hypokalemia ([K+] <3.3 mEq/L) is excluded.
- Alternatively, if there is no infuser administer bolus of 10IU RI/IV and IM the same dose; and 5IU RI IV every 1hr
- If blood glucose does not decrease by 10% (50-75 milligrams/dL/h) after 1 hour of insulin therapy, give 0.14 units'/kg bolus then resume previous rate
- If blood glucose decreasing faster than 50-75 milligrams/dL/h, decrease insulin drip. Check glucose hourly.
- When glucose approaches 250 (11 mmol/L) change IV to D5 0.5 NS and decrease insulin rate to 0.02-0.05 units/kg/h
- Maintain serum glucose 180-200 (10-11 mmol/L) and continue insulin drip for at least 12 hours or until DKA resolves:
- The goals of management are: glucose <200 (11 mmol/L), Normal AG, pH >7.3 and HCO_3 >15

- Patient able to eat: give SC short- and long acting insulin, feed patient, discontinue IV insulin 1-2 hours AFTER SC insulin

d. Management of acidosis

- Sodium bicarbonate only is infused if decompensated acidosis starts to threaten the patient's life, especially when associated with either sepsis or lactic acidosis.
- Give bicarbonate only if the initial pH is ≤6.9
- Adults with a pH <6.9 can be given 100 mEq of sodium bicarbonate in 400 mL of water with 20 mEq KCl at 200 mL/h for 2 hours until the venous pH >7.0.

e. Manage precipitants

- Manage precipitant factors (Infection (particularly sepsis), Myocardial ischemia or infarction, Medication non-compliance)

III. Management of complication

Cerebral edema: remains the most common and feared cause of mortality

- Deterioration of the level of consciousness in spite of improved metabolic state usually indicates the occurrence of cerebral edema.
- 0.5-1 g/kg intravenous mannitol may be given over the course of 20 minutes and repeated if no response is seen in 30-120 minutes; Hypertonic saline (3%), 5 to 10 mL/kg over 30 minutes, may be an alternative to mannitol. Intubation and fluid restriction are generally necessary if patient GCS is worsening

IV. Monitoring

Table 5-1: Follow up (DKA follow up chart)

Parameters	**VS(RR, PR, B/P, SPo2)**	**Mental status (GCS)**	**Serum glucose**	**Urinalysis Ketone**	**electrolyte**	**Blood gas**	**ECG**	**BUN/Cr**
Frequency	Continues monitoring	Continues monitoring	Every 1hr	Every 2hrs	Every day	Every 6hrs	Daily	daily

5.2 MYXEDEMA CRISIS

I. Principle of management

- Is a state of metabolic and multi organ decompensation characterized by uncorrected hypothyroidism, mental status changes or coma, and hypothermia usually <35.5°C /95.9°F
- Precipitated by a number of conditions, including infection, anesthetic agents, cold exposure, trauma, myocardial infarction or congestive heart failure, cerebrovascular accident, GI hemorrhage, metabolic conditions, hypoxia, hypercapnia, hyponatremia, hypoglycemia, surgery, burns, medications (e.g., β-blockers, sedatives, narcotics, phenothiazine, amiodarone), or thyroid medication noncompliance
- Evident clinical features are: bradycardia, hypotension, hypothermia, hypoventilation, and altered mental status or coma. Pleural effusions are frequently demonstrable. Other potential respiratory problems include upper airway obstruction from glottic edema, vocal cord edema, and macro glossia.
- Comatose patient, patient with symptomatic bradycardia and patient with airway problem should be admitted to the ICU, in order to manage air way, ventilation and start on vasopressor after replacing thyroid hormone

II. Principles of management in ICU are

a. Supportive care

- Airway, breathing, and circulation support: ensure airway control, oxygen, IV access, and cardiac monitor
- IV therapy: dextrose for hypoglycemia; water restriction for hyponatremia
- Vasopressors: if indicated (ineffective without thyroid hormone replacement)
- Hypothermia: treated with passive rewarming
- Steroids: hydrocortisone (due to increased metabolic stress; 100–200 milligrams IV)

b. Thyroid replacement therapy

- IV thyroxine (levothyroxine) at 4 micrograms/kg (typically between 200 and 500 micrograms as initial dose),
- Followed in 24 h by 100 micrograms IV, then 50 micrograms IV until oral medication is tolerated. Thyroxine is readily available and preferred in the elderly and those with cardiac disease.
- Starting dose in the elderly is 100 micrograms IV.

c. Stress dose steroid

- Hypothyroidism may associate with adrenal insufficiency, either due to pituitary disease or as a multifocal autoimmune disorder.
- Hydrocortisone 100 mg IV q8hr is the standard therapy here.
- Patients may be weaned off steroid fairly rapidly, once they are hemodynamically stable and improving.

III. Management of complications

Cardiovascular

- Bradycardia
- Pericardial effusion & pleural effusions, which can impair diastolic filling of the heart.
- Cardiogenic shock (late finding)

✦ **Replacement of thyroid hormone and vasopressor**

Neurologic

- Delirium or coma is a core feature.
- Seizures can occur, including status epilepticus (potentially exacerbated by hyponatremia).

✦ **Replacement of thyroid hormone, control active seizure and correct hyponatremia with hypertonic fluid**

Pulmonary

- Hypoventilation may result from central reduction in respiratory drive, as well as from muscular weakness.
- Pleural effusions may promote hypoxemia.
- Edema of the tongue can complicate intubation.

✦ **Put on mechanical ventilator, therapeutic thoracentesis if massive effusion, ready for surgical airway if difficult intubation**

Hyponatremia

- Severe hyponatremia may be a contributory factor towards seizure and delirium.
- Mild hyponatremia will resolve with thyroid supplementation.

✦ **Replace sodium with 3%NS and restrict free fluid intake**

Management of precipitants

- **Infections:** Manage sepsis
- **Drugs:** discontinue sedatives, beta blockers, antipsychotic, amiodarone (after discussion with senior physician)
- **Congestive heart failure:** manage heart failure according to the guideline
- **Gastrointestinal hemorrhage:** correct coagulopathy and GI acid secretion

IV. Follow up

Parameters	**V/S, GCS**	**TFT(free T3/T4/TSH**	**CBC**	**Electrolyte**	**RBS**	**ECG**	**Cr/BUN**	**S/E, occult blood**	**Bedside cardiac U/S**
Frequency	continues	Daily	Daily	daily	Q6hrs	Q12hrs	daily	once	daily

5.3 THYROID STORM

I. Principle of care

- Thyroid storm is the extreme manifestation of thyrotoxicosis.
- This is an acute, severe, life-threatening hyper metabolic state of thyrotoxicosis caused either by excessive release of thyroid hormones causing adrenergic hyperactivity or altered peripheral response to thyroid hormone
- Involve adrenergic hyperactivity either by increased release of thyroid hormones (with or without increased synthesis) or increased receptor sensitivity.
- Many of the signs and symptoms are related to adrenergic hyperactivity and patients may present with decompensated heart failure, uncontrolled arrhythmia, seizure and come.
- Patients need ICU admission for airway protection and ventilation, management of uncontrolled arrhythmia, heart failure and coma care.

II. Principle of management

a. Supportive care

- General: oxygen, cardiac monitoring
- Fever: external cooling; acetaminophen 325–650 milligrams PO/PR every 4–6 h (aspirin is contraindicated because it may increase free thyroid hormone)
- Dehydration: IV fluids, IV isotonic saline with 5% dextrose may be used to replace glycogen depletion if blood sugar is low
- Nutrition: glucose, multivitamins, thiamine, including folate can be considered (deficient secondary to hypermetabolism)
- Cardiac decompensation (atrial fibrillation, congestive heart failure): rate control and inotropic agent, diuretics, sympatholytics as required.

b. Inhibition of new thyroid hormone synthesis with thionamides

- Methimazole 40 milligrams given PO as loading dose and followed by 25 milligrams every 4 h.

- Total daily dose should be given: 120 milligrams/d.
- If given PR, 40 milligrams should be crushed in aqueous solution.
- (Avoid methimazole for pregnant women in first trimester as it can cause teratogenic effect.)

OR

- PTU, a loading dose of 600–1000 milligrams given PO and followed by 200–250 milligrams every 4 h. Total daily dose should be given: 1200–1500 milligrams/d.

c. Inhibition of thyroid hormone release (at least 1 h after step 2)

- Lugol solution 8–10 drops PO every 6–8 h /Potassium iodide (SSKI) five drops PO every 6 h/

d. β-Adrenergic receptor blockade

- Propranolol IV in slow 1- to 2-milligram boluses, which may be repeated every 10 to 15 min until the desired effect is achieved. For less toxic patient, PO dose of 20 to 120 milligrams per dose or 160 to 320 milligrams/d in divided doses (contraindicated in bronchospastic disease and congestive heart failure)

OR

- Esmolol 500 micrograms/kg IV bolus, then 50–200 micrograms/kg/min maintenance

e. Preventing peripheral conversion of thyroxine to triiodothyronine

- Hydrocortisone 100 milligrams IV initially, then 100 milligrams three times/d until stable (also for adrenal replacement due to hypermetabolism)

OR

- Dexamethasone 2 milligrams IV every 6 h

III. Management of complications

Cardiovascular

- Hypovolemia (from diaphoresis, vomiting, diarrhea)

- Systolic heart failure, including cardiogenic shock
- Distributive shock (increased tissue oxygenation causes systemic vasodilation)
- Tachycardia (either sinus tachycardia or atrial fibrillation).

✦ **Manage shock and heart failure according to the guideline; if there is atrial fibrillation with rapid ventricular response manage according to tachyarrhythmia guideline; and avoid beta blocker in patients with heart failure.**

Hyperthermia

- Paracetamol 1gm q6hr.
- Use of cooling blankets is recommended for high fever.
- Avoid salicylates or NSAIDs, since these may increase free thyroid hormone levels.

Agitation management

- Agitation may worsen hyperthermia and impede ability to provide care.
- Olanzapine is a drug of choice 2.5-5mgQ4hrs, either by PO, IM, or IV routes.

Table 5-2: Burch and Wartofsky's Diagnostic Parameters and Scoring points for thyroid storm

Score more than 45 is thyroid storm 25 to 45 is impending score and less than 25 is unlikely.

Diagnostic Parameter	Scoring Points
1. Thermoregulatory dysfunction **Temperature °C (°F)** 37.2-37.7 (99-99.9) 37.7-38.3 (100-100.9) 38.3-38.8 (101-101.9) 38.9-39.4 (102-102.9) 39.4-39.9 (103-103.9) ≥ 40 (≥ 104.0)	 5 10 15 20 25 30

2. CNS effects Absent Mild (agitation) Moderate (delirium, psychosis, extreme lethargy) Severe (seizure, comma	 **0** 10 20 30
3. GI-hepatic dysfunction Absent Moderate (diarrhea, nausea/vomiting, abdominal pain) Severe (unexplained jaundice)	 0 10 20
4. CV dysfunction **Tachycardia (beats/min)** 90-109 110-119 120-129 ≥140	 5 10 15 25
5. Congestive heart failure Absent Mild (pedal edema) Moderate (bibasilar rales) Severe (pulmonary edema)	 0 5 10 15
6. Atrial fibrillation Absent Present	 0 10

Treat precipitating event

- **Infection:** start broad spectrum antibiotics
- **Myocardial infarct:** manage AMI according to the guideline

IV. Follow-up

Parameters	**V/S, GCS**	**TFT(free T3/T4/TSH**	**CBC**	**Electrolyte**	**RBS**	**ECG**	**Cr/BUN**	**Liver function test**
Frequency	continues	Daily	daily	daily	Q6hrs	Q12hr	daily	once

5.4 ADRENAL CRISIS

I. Introduction and principle of management

- Adrenal crisis is shock refractory to volume resuscitation and pressors.
- It could be primary (autoimmune disease, adrenal hemorrhage, infection) or secondary (prolonged use of steroid, pituitary gland disorder)
- should be suspected in any patient in the emergency/ICU who develops shock of unclear etiology, or refractory to fluid resuscitation and vasopressors
- Other features of adrenal insufficiency: hyponatremia and hyperkalemia (primary) and either hypernatremia /hyponatremia and hypokalemia (secondary)
- Patient should be admitted to the ICU for rapid correction of shock and replacement of steroid and management of other metabolic complications.

II. Management

a. Manage hypotension: IV Fluid and vasopressor
b. Replace steroid; Optimize maintenance dosage of steroid
c. Determine and manage underlying cause

a. Management of shock

- Manage shock with Normal saline according to shock management guideline
- Use dextrose-containing saline if the patient is hypoglycemic.

b. Steroids

- Hydrocortisone 100-mg IV bolus: is the drug of choice for cases of adrenal crisis or insufficiency, especially for underlying primary insufficiency (provides both glucocorticoid and mineralocorticoid effects). or
- Dexamethasone, 4-mg IV bolus: (preferred if rapid adrenocorticotropic hormone stimulation test is contemplated).
- Patients may require lifelong glucocorticoids ± mineralocorticoid ± androgen supplementation.

- Optimize Maintenance Dosage of Steroids; During periods of stress, increase maintenance dose of chronic steroids to three times the daily dose, to satisfy increased physiologic need for cortisol

c. Vasopressors

- Administer only after steroid therapy in patients unresponsive to aggressive fluid resuscitation (choice of norepinephrine, dopamine, or phenylephrine).

d. Determine risk factors and Underlying Cause:

- Investigate as appropriate for underlying risk factors
- Sepsis, adrenal hemorrhage, CNS abnormality, prolonged steroid use.
- Serum cortisol >18 micrograms/dL generally rule out adrenal insufficiency
- The ACTH stimulation test measures serum cortisol after stimulation by synthetic ACTH; Serum cortisol rises significantly in secondary insufficiency.
- Serum ACTH: A high ACTH level is seen in primary insufficiency, and low in secondary adrenal insufficiency.
- An abdominal CT scan can identify adrenal gland hemorrhage or infarction.

III. Management of complications

- **Hyponatremia:** 3%N/S and/or free fluid restriction
- **Hyperkalemia:** regular insulin with dextrose, calcium gluconate, sodium bicarbonate depending on the severity of hyperkalemia and ECG changes
- **Hypernatremia:** hypotonic fluid depending on the amount of water deficit
- **Hypokalemia:** IV or PO KCl depending on the severity of hypokalemia
- **Hypoglycemia:** dextrose/ dextrose in NS
- **Manage Coma:** Repeat ABC assessment and intubate if the patient failed to protect the air way

IV. Follow-up

- Follow blood pressure during correction of shock
- Neuro sign chart for comatose patients
- Serum glucose every 6hrs

- Update Serum electrolyte daily
- ECG: related to potassium imbalances (prolonged QT, peaked T waves, and heart block in hyperkalemia in primary adrenal insufficiency, or inverted T waves and presence of U wave in hypokalemia in secondary adrenal insufficiency).

Suggested reading

1. Tintinalis principle of emergency medicine 8th edition
2. Rosen's emergency medicine 8th edition volume 2
3. Basic Medical Endocrinology Fourth Edition
4. M aurice Goodman Basic Medical Endocrinology 4th Edition
5. Rosens principle of emergency medicine 8th edition volume 2
6. Harrison principle of internal medicine 20th edition

CHAPTER 6: INFECTIOUS DISEASE

6.1 MALARIA

I. Principle of care

- Malaria caused by 5 parasite species plasmodium parasites that cause malaria in humans. Severe and complicated malaria is most commonly caused by infection with Plasmodium falciparum.
- Unless patients early resuscitated death from severe malaria often occurs within hours of admission.
- Management of severe malaria comprises mainly clinical assessment and stabilization specific antimalarial treatment, additional treatment and supportive care.

II. Suggested management

a. Initial Assessment and Resuscitation

- Assess for the airway, breathing and circulation.
- Fluid resuscitation with close monitoring for volume overload and pulmonary edema should also be done, as capillary leak syndrome is a known complication during fluid administration.
- Patients presenting with encephalopathy syndromes need closer airway assessment and assisted ventilation.

b. Treatment of severe malaria

- Intravenous or intramuscular artesunate for at least 24 h and until the patient can tolerate oral medication.
- Once a patient has received at least 24h of parenteral therapy and can tolerate oral therapy, complete treatment with full course artemether + lumefantrine (AL).

Recommended Dosage for injectable artesunate:

- Children less than 20kg: artesunate 3.0 mg/kg bw
- Older children and adults: Artesunate 2.4 mg/kg body wait

Dosage regimen: Give 3 parenteral doses of injection artesunate in the first 24 hours

- First dose on admission (time zero),
- Second dose 8 hours after the first dose and
- Third dose at 24 hours after the first dose.
- Thereafter every 24 hours until patient is able to tolerate oral medication to complete the treatment with in three days. Artisunate can be continued for 7 days

III. Management of complications

It is assumed that appropriate antimalarial treatment will have been started in all cases.

Table 6-1: Immediate clinical management of severe manifestations and complications of P. falciparum malaria

Manifestation or complication	**Immediate management[a]**
Coma (cerebral malaria)	Maintain airway, place patient on his or her side, exclude other treatable causes of coma (e.g. hypoglycemia, bacterial meningitis); avoid harmful ancillary treatments, intubate if necessary.
Hyperpyrexia	Administer tepid sponging, fanning, a cooling blanket and paracetamol.
Convulsions	Maintain airways; treat promptly with intravenous or rectal diazepam, lorazepam, midazolam or intramuscular paraldehyde. Check blood glucose.
Hypoglycemia	Check blood glucose, correct hypoglycemia and maintain with glucose-containing infusion. Although hypoglycemia is defined as glucose < 2.2 mmol/L, the threshold for intervention is < 3 mmol/L for children < 5 years and < 2.2 mmol/L for older children and adults.

Severe anemia	Transfuse with screened fresh whole blood.
Acute pulmonary edema	Prop patient up at an angle of 45^0, give oxygen, give a diuretic, stop intravenous fluids, intubate and add positive end-expiratory pressure or continuous positive airway pressure in life-threatening hypoxemia.
Acute kidney injury	Exclude pre-renal causes, check fluid balance and urinary sodium; if in established renal failure, add hemofiltration or hemodialysis, or, if not available, peritoneal dialysis.
Spontaneous bleeding and coagulopathy	Transfuse with screened fresh whole blood (cryoprecipitate, fresh frozen plasma and platelets, if available); give vitamin K injection.
Metabolic acidosis	Exclude or treat hypoglycemia, hypervolemia and septicemia. If severe, add hemofiltration or hemodialysis.
Shock	Suspect septicemia, take blood for cultures; give parenteral broad-spectrum antimicrobials, correct hemodynamic disturbances.

IV. Monitoring

- Hemodynamic monitoring
- Continues ECG and neurologic monitoring
- Organ function and electrolyte monitoring

6.2 SEPSIS AND SEPTIC-SHOCK

I. Principle of care

Sepsis: Is diagnosed when there is evidence of proven or suspected infection source and organ dysfunction as evidenced by increase in sequential organ failure assessment (SOFA) score by two or more from baseline.

Septic shock: Sepsis with persistent hypotension/ hypo perfusion requiring vasopressor support to maintain MAP ≥ 65mmHg and initial blood lactate level 4mmol/l or >2mmol/l despite adequate fluid resuscitation.

Table 6-2: The Criteria for Assessment of Sequential Organ Failure Assessment (SOFA) Score

SOFA score	1	2	3	4
Neurological system				
Glasgow Coma Score	13-14	10-12	6-9	<6
Respiratory system				
PaO2/FIO2 ratio	<400	<300	<200 with respiratory support	<100 With respiratory support
SaO2/FIO2 ratio	221-301	142-220	67-141	<67
Cardiovascular system				
Hypotension	MAP<70	Dopamine ≤ 5 or Dobutamine (any dose)	Dopamine >5 or Epinephrine ≤0.1 or Norepinephrine≤0.1	Dopamine>15 or Epinephrine>0.1 or Norepinephrine>0.1
Renovascular system				
Creatinine mg/dl (µmol/L) or urine output	1.2-1.9 (110-170)	2.0-3.4 (171-299)	3.5-4.9 (300-440) or <500 ml/day	>5.0 (>440) or <200 ml/day
Coagulation				
Platelets x 10^3/mm^3	<150	<100	<50	<20
Liver				
Bilirubin mg/dl (µmol/L)	1.2-1.9 (20-32)	2.0-5.9 (33-101)	6.0-11.9 (102-204)	>12.0 (>204)

II. Treatment

The following **'Hour- 1 Bundle'** is adopted from the Surviving Sepsis campaign 2018 guideline.

- Measure lactate level, Re-measure if initial lactate is>2 mmol/L
- Obtain blood cultures prior to administration of antibiotics
- Administer broad-spectrum antibiotics
- Rapidly administer 30 mL/kg crystalloid for hypotension or lactate ≥4 mmol/L
- Apply vasopressors if patient is hypotensive during or after fluid resuscitation to maintain MAP ≥65 mm Hg

a. Fluid treatment

- Start balanced crystalloids (Ringer`s lactate/acetate), plasmalyte or normal saline (NS) 20-30ml/kg over 30-60minutes or as needed to target in 6 hours; targeting MAP >65-70mmHg or >80mmhg in chronic hypertension, urine output >0.5ml/kg/hr, capillary refill time<2-3 second, strong and full peripheral pulses, skin warmth and color, and improvement in mental status
- Albumin is as effective as crystalloids in expanding intravascular volume when a substantial amount is needed.
- Avoid hypotonic fluid or semisynthetic colloids
- Packed RBC if Hct <21% unless patient has myocardial ischemia, severe hypoxemia, acute hemorrhage and cyanotic heart disease. Targeting Hct 27-30% doesn`t improve outcome.
- Platelet transfusion if the platelet is <5000/microliter or if there is significant bleeding.

b. Vasopressor and inotrope treatment

1. Nor-epinephrine

- Is the first choice and titrate to desired effect
- Is potent vasoconstrictor with less increase in HR

2. ***Epinephrine***

- Can be used as an alternative to norepinephrine or as additional to achieve desired effect
- Is potent vasoconstrictor, and also with inotropic effect
- Titrate to desired effect

3. ***Vasopressin***

- Can be used to reduce norepinephrine dose
- Caution if not euvolumic
- Fixed dose at 0.03 U/min

4. ***Dopamine***

- Restrict as it may be associated with increase mortality and increase in tachyarrhythmia

5. ***Dobutamine***

- First choice inotrope in warm shock with borderline BP
- Start at 2.5µg/kg/min and titrate to desired effect maximum 20 µg/kg/min
- Risks include tachyarrhythmia and hypotension

c. Antibiotics

- Modify as culture results dictate
- Source control (abscess drainage, tissue debridement and removal of lines)

Table 6-3: Antibiotic Selection for Community Acquired and Immunocompetent patients

Syndrome	**Treatment**
No source identified	Ceftriaxone 1 – 2g IV q24 hours
Pneumonia	Ceftriaxone 1 – 2g IV q24 hours + (azithromycin 500mg day 1 and 250mg for 4 days or doxycycline 100mg po q12 hours)
Urinary tract infection	Ciprofloxacin 400mg IV q12hours
Intra-abdominal infection	Ceftriaxone 1 – 2g IV 24 hours + Metronidazole 500mg IV q8hours

Skin/Skin structure infection pure cellulitis	Cefazolin 1 – 2g IV q8 hours (Add vancomycin if necrotizing abscesses or high risk of MRSA)
Skin/skin structure infection with special risks (Immersion injuries, animal bites, diabetic foot ulcer)	Piperacilin Tazobactam 4.5gm IV q8 hours OR Meropenem 1g IV q 8hours (Add vancomycin if necrotizing abscesses or high risk of MRSA)
Bacterial meningitis (Spontaneous)	Ceftriaxone 1 – 2g IV q 12 hours (add vancomycin if gram positive cocci seen in CSF gram stain or add Ampicillin if age <1 month and >50 years or immunocompromised).

Duration of antimicrobials: 7–10 days is usually adequate. Longer courses may be required in patients who have a slow clinical response, undrain able foci of infection, bacteremia with *S. aureus*, some fungal and viral infections, or immunologic deficiencies, including neutropenia

d. **Steroids:** Hydrocortisone (200mg/day) divided every 6h for 5 days if refractory shock despite fluid and vasopressors, or if corticosteroid history warrants it. Titrate down to twice daily the next 5-8 days, then daily on the next 9-11 days before discontinuation.

e. **Mechanical ventilation:** Is indicated if ARDS or depressed mental status or respiratory failure develops.

f. **Renal replacement therapy:** Hemodialysis only if there are clear indications.

g. **Ulcer and venous thromboembolic prophylaxis** with proton pump inhibitors/H2 blockers & unfractionated heparin.

h. **Glycemic control:** Target a blood sugar of 140 – 180mg/dl.

III. Monitoring

Hemodynamic monitoring: BP, mean arterial pressure (MAP), HR, Urine output, cardiothoracic ultrasound, lactate clearance.

6.3INVASIVE FUNGAL INFECTIONS

I. Introduction

- Invasive candidiasis is now the fourth leading cause of nosocomial bloodstream infection with mortality as high as 40% to 50%.
- The strongest risk factor for invasive candidiasis is length of ICU stay, with most studies revealing peak incidence at approximately day 10.
- None of the currently available tests have adequate sensitivity and specificity for reliable diagnosis. For this reason, empiric therapy for invasive candidiasis is often appropriate for the critically ill patient with risk factors and not responding to appropriate antibacterial agents.
- All patients with suspected candidemia should have an ophthalmological examination, cardiac examination, abdominal examination, neurological examination, catheters and skin.

Differentiate Colonization from Infection with Candida

- Growth of candida in the blood should be considered infection
- Growth of Candida from respiratory secretions usually represents colonization, and therefore, it should not be treated.
- In the absence of systemic features, growth of Candida in the urine often represents colonization.
- Candida colonization is frequent in surgical drains which have been in situ for some time and from superficial wound swab.

II. Suggested management

a. Antifungal regimens for suspected or proven fungal infections

Choice of antifungal agents in candidemia is guided by many factors:-

- Histories of recent azole exposure- avoid azoles.
- Local epidemiological data from the ICU- choose most effective antifungal agents initially pending culture results.

- Severely of illness- use fungicidal drugs.
- Comorbidity- check renal and hepatic function and avoid amphotericin deoxycholate and voriconazole respectively.
- Involvement of CNS, cardiac valves, and eyes- choose antifungal agents with good penetration at the infected site.
- History of intolerance to any antifungal agent or drug interactions.
- Antifungal agents used for candidemia are triazoles, echinocandins, and amphotericin B.

b. Manage Persistent Candiduria in a Catheterized Patient

- Avoid treating with antifungal drugs in an asymptomatic, afebrile, stable patient.
- Remove the catheter if possible.
- Echhinocandins are not recommended for candida urosepsis
- In high-risk patients consider fluconazole therapy if the species is susceptible.

c. Consider Central Nervous System Candidiasis in Patients with an Intraventricular Device

- Consider liposomal amphotericin B at a dosage of 3–5 mg/kg/day with or without flucytosine at a dosage of 25 mg/kg/dose four times daily.
- After initial response, deescalate to fluconazole 400–800 mg daily.
- Remove the infected ventricular device.

d. Consider Azole Prophylaxis in the Selected Group of Patients

- Prophylactic antifungal therapy should be avoided in medical/surgical ICU patients.
- For high-risk patients; fluconazole 400 mg (6 mg/kg) daily, posaconazole 200 mg three times a day, or an echinocandin is recommended during the period of neutropenia.

e. Invasive Aspergillosis

- Identify risk factors and consider empiric antifungal in severe cases
- Look for involvement of lungs and paranasal sinuses by CT scan, which may show a "halo sign."

- Voriconazole is considered a first-line agent.

f. Consider Zygomycosis (Mucormycosis/mold infection) in Some Specific Situations

- Uncontrolled diabetes presenting with rhino-cerebral disease.
- Immunosuppressed patients and mainly involves the lung.
- Diagnosis is based mainly on tissue biopsy.
- Therapy is usually given for months.
- Surgical intervention is usually required in this angioinvasive disease.
- Mucormycosis carries extremely poor prognosis in the absence of early aggressive treatment.

6.4 TB AND HIV PATIENT IN ICU

The most common reason for ICU admission remains late stage AIDS-related OIs on a background of poor nutritional status and advanced immunosuppression.

Pneumocystis jirovecii pneumonia (PCP), cerebral toxoplasmosis and tuberculosis are the leading diagnoses during ICU admissions.

Suggested management and monitoring

a. General principle

- If assisted respiration is required, initially High Flow Nasal Oxygen (HFNC) or noninvasive ventilation should be tried.
- The patient should be closely monitored, and if no improvement or deterioration occurs in 2 h, invasive ventilation should be initiated.
- Urinary catheters and central lines should be avoided as these patients are coagulopathic and neutropenic and have a high risk of line sepsis.
- If absolutely necessary, invasive catheters and lines should be placed with utmost aseptic precautions by an experienced person.
- Management of OIs: standard diagnostic methods and therapies for the most common severe opportunistic infections in HIV-infected patients should be followed as national protocol.

b. ART in ICU

There are no prospective evaluations of the safety, efficacy and timing of cART administration in the ICU.

cART initiation at 2 weeks after the start of specific OI treatment, except for those with cryptococcosis or CNS tuberculosis due to the risk of severe IRIS that may outweigh potential benefits from rapid immune recovery. In these situations, cART initiation must be deferred until at least 4 weeks and proven disease control.

c. Mycobacterial infections

Treatment of tuberculosis is complicated by the drug interactions of ART agents and antituberculous agents and the occurrence of IRIS. Rifampin, in particular, has complex interactions with the protease inhibitors and nonnucleoside reverse transcriptase inhibitors.

Suggested readings

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CHAPTER 7: LIVER AND GASTRO INTESTINAL SYSTEM

7.1 ACUTE LIVER FAILURE (ALF)

I. Introduction and principle of care

Acute liver failure refers to the development of severe acute liver injury with encephalopathy and impaired synthetic function (INR of ≥1.5) in a patient without cirrhosis or preexisting liver disease. A commonly used cutoff is an illness duration of <26 weeks.

Etiology: includes drugs acetaminophine, alcohol, viral hepatitis, autoimmune, ischemic, sepsis, acute fatty liver of pregnancy/HELLP syndrome

Liver test abnormalities with signs of hepatic encephalopathy and a prolonged prothrombin time (INR ≥1.5). Other clinical manifestations may include jaundice, hepatomegaly, and right upper quadrant tenderness.

Lab findings includes elevated aminotransferase levels (often markedly elevated), Elevated bilirubin level, Low platelet count (≤150,000/mm3)

Diagnosis

- Acute liver failure is diagnosed by demonstrating all of the following:
- Elevated aminotransferases (often with abnormal bilirubin and alkaline phosphatase levels)
- Hepatic encephalopathy
- Prolonged prothrombin time (INR ≥1.5)

II. Suggested Management

a. Supportive measures

- Hemodynamic management with fluids
- N-acetylcysteine — acetaminophen toxicity, but it may be beneficial in other forms of acute liver failure

b. Bleeding Prevention

- Stress ulcer prophylaxis with an H2 blocker or proton pump inhibitor.
- Fresh frozen plasma (FFP) or platelet transfusion should be reserved for active bleeding or prevention of bleeding during invasive procedures.

c. Infection Surveillance and prevention

- Cultures of blood, sputum, and urine.
- A low threshold for the use of empiric antibacterial and/or antifungal therapy

d. Nutrition

- Early feeding with a daily intake of 60 grams of protein for most patients.

e. Management of underlying causes:

- Pregnancy-induced ALF—treat with delivery of child/ termination of pregnancy
- Autoimmune hepatitis-induced ALF—treat with steroids
- HSV-induced ALF—treat with acyclovir
- Budd-Chiari-induced ALF—treat with anticoagulation and transjugular intrahepatic portosystemic shunt (TIPS).

f. Liver transplantation whenever feasible

III. Manage complication and precipitating cause

Hepatic encephalopathy:

- Nursing with head of bed at >30 degree elevation.
- Regular lactulose 20-30mL qds PO, to achieve at least 2-3 bowel motions/day
- Correct/avoid potential aggravating factors, e.g. gut haemorrhage, oversedation, hypoxia, hypoglycaemia, infection, electrolyte imbalance.

Hypoglycemia:

Frequent glucose monitoring and infusions of 10% to 50% dextrose solutions may be required.

IV. Monitoring

- Neurologic status
- Hemodynamic status
- Bleeding and liver function test daily

7.2 ACUTE PANCREATITIS

I. Introduction and principles of care

- **Acute pancreatitis** should be suspected in patients with severe acute upper abdominal pain but requires biochemical or radiologic evidence to establish the diagnosis.
- **Moderately severe acute pancreatitis,** which is characterized by transient organ failure (resolves within 48 hours) and/or local or systemic complications without persistent organ failure (>48 hours)
- **Severe acute pancreatitis,** which is characterized by persistent organ failure that may involve one or multiple organs

Common causes: Gallstones, alcohol, Obstructive: tumors, primary or metastatic, annular pancrease, ascaris. Less common causes: drug, metabolic, infections, autoimmune, ischemia, post ERCP, post trauma.

Clinical manifestation: can be **epigastric** abdominal pain, radiating to back, constant, some relief with leaning forward nausea, vomiting, fever, tachycardia, hypotension with or without shock

Signs of retroperitoneal hemorrhage (Cullen's=periumblical; Grey Turner's=flank): rare

Laboratory

- Elevated amylase: levels >3 x ULN suggestive of pancreatitis, level is not equal to severity
- Elevated lipase: more specific than amylase
- ALT>3 X ULN: suggests gallstone pancreatitis
- Others: elevated WBC, high or low Hct, Elevated BUN, low calcium, elevated glucose, elevated CRP, triglyceride

Imaging studies

- Abdominal CT: excludes other diagnosis, stage severity, rule out complications
- Abdominal ultrasound: to investgate biliary etiology,

Indications for intensive care: Admission to an intensive care unit setting is indicated in the following patients:

- Patients with severe acute pancreatitis
- Patients with acute pancreatitis with hemodynamic instability, electroly imbalance, hypoxia, Serum glucose >800 mg/dL, Serum calcium >15 mg/dL
- Anuria, Coma

II. Suggested management

Supportive care with fluid resuscitation, pain control, and nutritional support

a. Fluid resuscitation

Provide aggressive hydration at a rate of 5 to 10 mL/kg per hour of isotonic crystalloid solution (eg, normal saline or lactated Ringer's solution). Provide more rapid repletion with 20 mL/kg of intravenous fluid given over 30 minutes followed by 3 mL/kg/hour for 8 to 12 hours.

b. Nutrition: NPO, early 4hrs decreases infectious complications and severity

if NPO for > 7days expected parenteral nutrition

c. ERCP with sphincterotomy: reserved for severe cholangitis/sepsis and total bilirubin>5

d. Prophylactic antibiotics are not recommended

e. Debridement: if necrotic tissue gets infected

III. Management of complications

A pseudocyst is a collection of pancreatic juice, which is enclosed by a wall of granulation tissue. This takes at least 4 weeks to form.

- Drainage—percutaneous ultrasound-guided endoscopic or surgical—is required in those with large and/or symptomatic pseudocysts.
- The common complications of the pseudocyst include compression of adjacent structures, rupture, infection, and bleeding in 5% of cases.

Infected collection CT: guided drainage of multiple drains may be required.

Walled Off Pancreatic Necrosis (WOPN): Endoscopy is used for drainage

Infected pancreatic necrosis: Surgical Pancreatic necrosectomy. Effort should be made to delay intervention to the fourth week as the results before that are not good.

Pseudoaneurysms and massive intra-abdominal hemorrhage: is best managed by angiographic embolization.

Abdominal compartment syndrome: in which percutaneous/other drainage techniques are not successful.

IV. Monitoring

- Hemodynamic status and input out put
- Respiratory condition
- Electrolyte and renal status
- Glucose level

7.3 UPPER GASTROINTESTINAL BLEEDING (UGIB)

Common causes are duodenal and gastric ulcers and erosions, oesophageal and gastric varices, esophagitis, Mallory – Weiss tears

Uncommon causes: Angiodysplasia, cancer, portal gastropathy, aortoenteric fistula, coagulopathy all hemodynamically unstable patients should be admitted to ICU. Assess drop in BP or postural drop.

I. Suggested Management

a. Resuscitation of patients with hemodynamic instability

Intravenous access: Adequate peripheral access should be attained with either two 18 gauge or larger intravenous catheters and/or a large-bore, single-lumen central.

Fluid resuscitation: Fluid resuscitation should begin immediately with crystalloids (NS or RL)

Transfusion: Patients with active bleeding and hypovolemia may require red blood cell transfusion despite apparently normal hemoglobin.

- Patients without active bleeding who become hemodynamically stable with fluid resuscitation should receive a blood transfusion if the hemoglobin is <9 g/dL (90 g/L) for high-risk patients and if it is <7 g/dL (70 g/L) in low-risk patients.
- Endotracheal intubation for airway protection before endoscopy if massive or continuous hematemesis.
- Any coagulopathy found needs to be corrected by appropriate blood products.
- Somatostatin intravenous infusion or octreotide subcutaneous may also be tried
- If there is history of active alcohol abuse, thiamine replacement should be started.
- Prophylactic antibiotic with 1 g of ceftriaxone

b. Treatment of specific causes of UGIB

Management of variceal bleeding:

- ***Vasoactive medications:*** such as vasopressin, somatostatin, and their analogs (Octreotide - usually administered as 25 – 100 µg IV bolus followed by a continuous infusion at 25-50 µg/hour for 48 to 120 hours)
- ***Prophylactic antibiotics*** (e.g., quinolone or cephalosporin) in cirrhotic patients result in reduction of infectious complications and risk of recurrent esophageal variceal bleeding.
- ***Endoscopic therapy***

II. Mange complication

- Anemia
- shock
- Encephalopathy

7.4 DIARRHEA IN ICU

I. Introduction and principle of care

Severe diarrhea in ICU is usually of infective origin. However, other etiologies should be sought including, Inflammatory bowel disease, Ischemic colitis, Diverticulitis and various medications.

Detailed investigations should be done if the patient has any one of the following:

- Profuse diarrhea with dehydration
- Grossly bloody stools
- Fever of more than 38 °C
- Duration of more than 48 h without improvement
- Recent antibiotic use
- New community outbreak
- Associated severe abdominal pain in the patient older than 50 years
- Elderly (>70 years), Immunocompromised patients

Investigations

Stool tests *for WBCC, difficile* toxin A and B /GDH/PCR for *C. difficile,* Aerobic culture—for bacteria (Enteropathogenic *E. Coli*.)

II. Suggested management

a. **Fluid and electrolyte management** is the main modality of care

b. **Specific Pharmacotherapy** may be given empirically in all severe diarrheas

Usually Metronidazole, Rifaximin, Trimethoprim/sulfamethoxazole, or Doxycycline

If Giardiasis or Amebiasis suspected—Metronidazole

If *C. difficile* suspected—Metronidazole or oral Vancomycin

Antivirals (Acyclovir, Ganciclovir)—herpes simplex virus, cytomegalovirus

c. **Empirical antibiotics**

Are usually recommended especially in:

- ✦ Elderly
- ✦ Immunocompromized
- ✦ Mechanical heart valves
- ✦ Vascular grafts

d. Opiate derivatives: loperamide 2–4 mg four times a day

e. Anticholinergics: diphenoxylate 2.5–5 mg four times a day

f. Oral microbiota daily is as a sachet or preparation with Yoghurt is recommended for suspected antibiotic related diarrhea.

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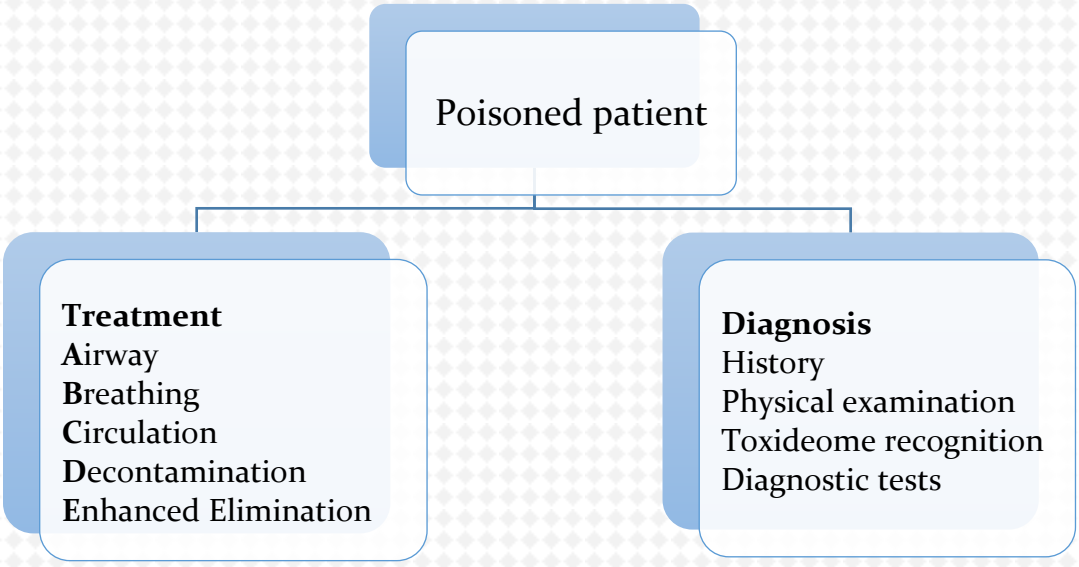
CHAPTER 8: POISONING

8.1 GENERAL APPROACH TO POISONING

I. Introduction and principle of care

- Consider the dose of the substance ingested, time since ingestion, clinical features, patient factors, geographical location, and available medical facilities.
- Basic emergency medical care-the ABCs (airway, breathing, circulation).
- Rapid blood glucose measurement should be obtained and managed
- Supplemental oxygen, naloxone and thiamine should be considered in the appropriate cases and situations.
- The various methods of decontamination should be considered in any poisoned patient based on each individual clinical situation.
- Obtain patient history and focused physical examination with attention to toxidrome recognition, poison identification and deciding on the appropriate diagnostic tests to be performed.



- The most common toxidromes are the anticholinergic syndrome, Cholinergic syndrome, sympathomimetic syndrome, opioids, and serotonin syndrome.

II. General Suggested management

a. Resuscitation

- The initial priorities for a poisoned will be securing the airway and breathing and stabilizing the circulation.
- Adequate ventilation and intubation with mechanical ventilation must be done early in the intoxicated patients with depressed mental status, except in cases of easy reversible causes of coma like hypoglycemia.
- If the patient hypotension, first-line treatment is IV crystalloid with bolus of (10 to20 mL/kg); if hypotension is not responding to fluid, it may be necessary to add vasopressors such norepinephrine.
- The treatment of hypertension and agitated patients starts with sedatives such as benzodiazepines; if not responding for initial treatment and there is evidence of endorgan dysfunction, calcium-channel blocker is preferred treatment.
- The use of betablockers is not recommended in the case of sympathetic hyperactivity because it may cause unopposed alpha-adrenergic stimulation and intensified vasoconstriction.
- Ventricular tachycardia occurs because of tricyclic antidepressant toxicity and sodium bicarbonate is first line therapy.
- Magnesium sulfate can also be used in the case of drug-induced torsade de pointes and prolonged QT intervals on ECG.
- Treatment of bradyarrhythmias with hypotension starts with atropine and/or temporary pacing. Calcium, glucagon, or high-dose insulin are used in the case of calcium channel blocker or beta blocker intoxication.
- The best treatment of intoxicated patients with seizures is benzodiazepines; and barbiturates may be added if necessary. Phenytoin is not recommended to control seizures in poisoned patients.
- Treatment of hyperthermia includes active cooling like ice water immersion; if active cooling is ineffective, the patient may need sedation with benzodiazepines.

b. Decontamination

Decontamination of severely poisoned patient must only be performed after careful consideration of the potential risks and benefits of the decontamination procedure.

1. Skin decontamination

- Corrosive agents rapidly injure the skin and must be removed immediately.
- Remove contaminated clothing and flush exposed areas with copious quantities of tepid (lukewarm) water or saline. Wash carefully behind ears, under nails, and in skin folds. Use soap and shampoo for oily substances.
- Health care providers should always wear personal protective equipments to prevent secondary contamination to themselves.

2. Ocular decontamination

- Eye exposures need prolonged irrigation with copious amount of water or saline
- Alkalis produce greater injury than acids due to deep tissue penetration via liquefaction so that prolonged irrigation (1 to 2 h) may be required.
- Ophthalmologic consultation is indicated for all ocular alkali injuries.

3. Gastro-intestinal decontamination

3.1 Gastric lavage

- Is the process of irrigating the gastric cavity to remove ingested material within one to two hours post ingestion.
- In certain circumstances, such as delayed gastric emptying accompanying intoxication with anticholinergic drugs and phenobarbitone, benefit may be noted longer after ingestion.
    - ✦ Ensure a protected airway if consciousness level is reduced.
    - ✦ Use a 36 to 40F-gauge orogastric tube (22 to 24F in children).
    - ✦ Position the patient on the left side with the head down 20 degrees.
    - ✦ Gently lavage with 200 mL (10 mL/kg in children) of warm tap water.

- ✦ Continue until returned fluid is clear.
- ✦ Consider administration of activated charcoal via orogastric tube before removal.

3.2 Activated charcoal

- It should be administer within 1-2 hrs of hazardeous material
- It should be given 0.5-1gm/kg Po and may be repeated very 4-6 hrs

c. Enhance elimination

Urine alkalization

- It may be considered for agents that are excreted as weak acids in the urine
- Urine alkalinization may be considered for, 2, 4-dichlorophenoxyacetic acid (2,4 D), methotrexate, phenobarbital and salicylates.

d. Dialysis

- Drugs which may be dialyzed are Theophylline, Carbamazepine (multidose activated charcoal or high-efficiency hemodialysis also effective) and Paraquat (theoretical benefit only if instituted early after exposure).

e. Antidotes

Although most poisonings are managed primarily with appropriate supportive care, several specific antidote agents may be employed.

The **common antidotes** include:

- **Oxygen:** for any cause of hypoxia
- **Dextrose:** insulin, hypoglycemics, coma, or protracted vomiting causing hypoglycemia
- **Atropine: Organophosphate poisoning**
- **Naloxone:** Narcotics
- **Thiamine**: Wernicke`s or chronic alcohol abuse and in Malnourished patients.
- **Actyl cysteine: Acetaminophen poisoning**

8.2 ORGANOPHOSPHATE POISONING

I. Introduction and principle of care

Admission to the intensive care unit is necessary for significant organophosphate poisonings in patients who need continuous monitoring, mechanical ventilator support and hemodynamic unstable patients

II. Suggested management for organophosphate poisoning

- Treatment consists of airway control, intensive respiratory support, general supportive measures, decontamination, prevention of absorption, and the administration of antidotes.
- Decontamination and initial resuscitation

a. Administration of antidote

- **Atropine:** Initial bolus of 1.2-3.0 mg Iv or 0.05mg/kg IV and then double the dose of IV every 5 minute to achieve adequate atropinzation; clear chest on auscultation(most important), heart rate>80 beats/minute and systolic blood pressure>80mmHg.
- Follow with continuous infusion of 10%-20% per hour of the intial dose of atropine that was required to achieve adequate atropinization (typical infusion rates vary from 0.4-4mg/hr IV in adult. Adjust infusion rate to maintain adequate atropinization and avoid atropine toxicity (absent bowel sounds, hyperthermia, delirium).
- **Pralidoxime** (2- PAM) in patients with neurologic symptom secondary to atropine

b. Supportive measures

- Fluid resuscitation is very important in addition to administration of antidotes in hypotensive patients
- Seizures can be managed with Benzodiazepines

III. Common/Management complications

- Acute complication with organophosphate poisoning which results death is a respiratory failure due to respiratory suppression / respiratory distress or CNS depression.
- Bronchorrhea, seizure, weakness
- Neuropathy as a delayed complication should be managed supportively
- Atropine toxicity can happen if the patient take excess amount of atropine which is characterized by absent bowel sounds, hyperthermia, delirium

IV. Monitoring

- Patient should be monitor on cardiac monitor, Spo2, respiratory pattern, response to the management(atropinization) and Vital signs

8.3 ALUMINUM PHOSPHIDE POISONINGS

I. Introduction and principle of care

- It is now one of the most common causes of poisoning among agricultural pesticides.
- This poisoning has a high mortality (30–100%) and survival is unlikely if more than 1.5 g is ingested and the lethal dose is 150–500 mg for an adult,
- It liberates lethal phosphine gas when it comes in contact either with atmospheric moisture or with hydrochloric acid in the stomach.
- Human toxicity occurs either due to the ingestion of AlP (commonest mode) after exposure and injury from phosphine inhalation (uncommon) or even after absorption through the skin (rare).
- After ingestion, AlP releases phosphine gas in the presence of HCl in the stomach, which is rapidly absorbed throughout the gastrointestinal tract, leading to systemic toxic effects involving the heart, lung, kidney, liver with manifestation of serious cardiac arrhythmias, intractable shock, acidosis and pulmonary edema.
- The toxicity of AlP particularly affects the cardiac and vascular tissues, which manifest as profound and refractory hypotension, congestive heart failure and electrocardiographic abnormalities.

II. Suggested management

a. Initial Evaluation and resuscitation

- The health care provider must take personal protection measures, including full face mask and rubber gloves during decontamination
- As phosphine is absorbed through the cutaneous route, decontamination of skin and eyes must be carried out thoroughly with plain water as early as possible
- Supplementary oxygen, check for pulse and establish intravenous access, preferably central venous, to start normal saline and vasopressor therapy as appropriate.

b. GI decontamination

- Potassium permanganate (1:10,000) is used for gastric lavage through a nasogastric tube as it oxidises phosphine to nontoxic phosphate.
- 100 g of activated charcoal to reduce absorption if the patient arrives within 1 hr

c. Hemodynamic support

- Early resuscitation with fluid and vasoactive agents. norepinephrine or phenylephrine could be used
- Intravenous sodium bicarbonate could be considered for mild to moderate metabolic acidosis or as a rescue therapy in severe acidosis.
- Magnesium sulfate has a membrane stabilization and anti-peroxident effect and it combats free radical stress due to phosphine. The dose is 3 g as infusion over 3 h, followed by 6 g per 24 h for 3–5 days,

d. Hemodialysis

- Is helpful when renal failure, severe metabolic acidosis or fluid overload is present.

III. Complications/Management complications

- Common complications dute to ALP are refractory, metabolic acidosis, renal failure, Hyperglycemia(poor prognostic sign) and arrhythmias

IV. Monitoring and problem solving.

- Monitoring of vitals should be performed very closely.
- Blood glucose, arterial blood gas, electrolytes including magnesium, routine hemogram, liver function test and renal function test should be determined
- Continuous ECG should be done to see any arrhythmias

8.4 DICHLOROPHENXY ACETIC ACID (2, 4-D) POISONING

I. Introduction and principle of care

- Human toxicity can occur after dermal contact, inhalation or ingestion and the mechanism is unknown. Patients with 2,4-D poisoning presented with mucosal irritation after direct contact. Vomiting and nausea after ingestion; and Tachypnea, dypsnea and Pulmonary edema after inhalation exposure.
- Systemic toxicity(If large exposure) result in Neurologic toxicity (Mental status changes and seizures); Cardiac toxicity (hypotension, tachycardia, and dysrhythmias); Skeletal toxicity (muscle tenderness, fasciculation, rhabdomyolysis)

II. Suggested management

a. **Initial evaluation and resuscitation:** Initial resuscitation with ABC focus

b. **GI decontamination:** Administer activated charcoal if the patient comes within 1-2 hrs

c. **Urinary alkalinization:** Will increase the elimination & is recommended for severely poisoned patients

d. **Hemodialysis :** Can also be used to enhance chlorophenoxy herbicide clearance in severe poisoning

III. Complications/ Management of complications

- Patients may develop respiratory failure, rhabdomyolysis and CNS depression should be followed and treated as necessary.

IV. Monitoring

- Patients should be put on continuous monitor and vitals are should be followed
- Patients mental status, respiratory condition should be followed
- The urine output should be followed every 4-6 hrs
- Serum RFT, serum electrolyte should be followed

8.5 ACETAMINOPHEN POISONING

I. Introduction and principle of care

- In an overdose, production of the toxic metabolite exceeds glutathione capacity and the metabolite reacts directly with hepatic macromolecules, causing liver injury and renal injury

II. Suggested management

a. Emergency and supportive measures

- Provide general supportive care for hepatic or renal failure if it occurs. Encephalopathy, metabolic acidosis, hypoglycemia, and progressive rise in the prothrombin time are indications of severe liver injury.
- Treatment for acetaminophen-induced fulminant hepatic failure includes acetylcysteine therapy, correction of coagulopathy and acidosis, monitoring for and aggressive treatment of cerebral edema, and early patient referral to a liver specialty/transplant center.

b. Decontamination

- Administer activated charcoal if the patient has a stable mental and clinical status, patent airway, and presents to the hospital within 1 hour of ingestion.

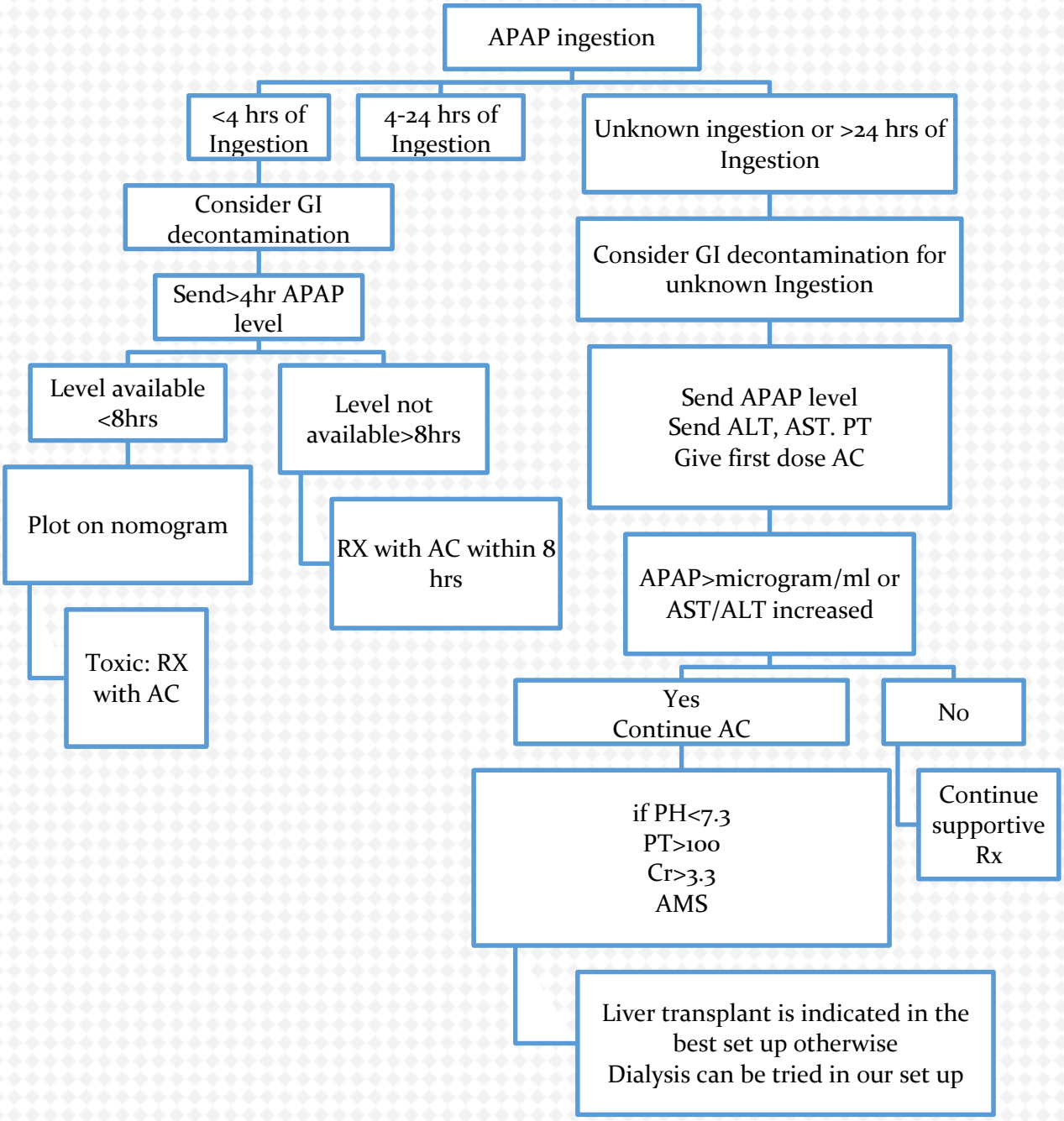
c. N- Acetylcysteine (NAC)

- Start acetylcysteine, if serum levels are not immediately available
- Initiate antidotal therapy with NAC, with a loading dose of 140 mg/kg orally, followed by 70 mg/kg every 4 h for 17 more doses over 72 h (a total of 18 doses). If the patient vomits, repeat the dose. If vomiting interferes with oral acetylcysteine administration, give it by gastric tube and use metoclopramide or ondansetron or give the NAC IV if necessary.
- Continuous IV infusion is recommended for acute ingestion, as follows:

- Loading dose:150 mg/kg IV; mix in 200 mL of D5W and infuse over 1 hr
- Dose 2: 50 mg/kg IV in 500 mL D5W over 4 hrs
- Dose 3: 100 mg/kg IV in 1000 mL D5W over 16 hrs

- Intermittent IV infusion may be considered for late-presenting or chronic ingestion. A loading dose of 140 mg/kg iv (diluted in 500 mL D5W) is infused over 1 hr. Maintenance doses of 70 mg/kg IV are given every 4 hours for at least 12 doses (dilute each dose in 250 mL of D5W and infuse over a minimum of 1 hr.).
- The duration of NAC treatmentis17 doses of oral NAC given over approximately 72 hrs. If IV NAC is needed, give for only 20 hrs.

Figure 8-1: Treatment guidelines for acetaminophen (APAP) ingestion



Abbreviation: AC = acetylcysteineALT = alanine aminotransferase; AMS = altered mental status; AST = aspartate aminotransferase; Cr = creatinine; LFTs = liver function tests; PT = prothrombin time; Rx = treatment.

III. Common complications/Management complications

- Hepatic failure, coaglopathy and acute renal failures are common complication
- Complication with treatment with NAC is rare specially with infusion therapy but few patients may develop anaphylactic reaction

IV. Monitoring

- Patients should be put continous monitor, vital signs should be followed, UoP
- Measuring ALT and INR every 12 hrs is very important to look for any hepatic injury
- Serum Creatinine, bicarbonate, glucose should be also monitored every 12 hrs

8.6 BARBITURATES POISONING

I. Introduction and principle of care

- Barbiturates are addictive, producing physical dependence and a withdrawal syndrome that can be life-threatening.
- The main action of barbiturates is to depress activity in the nervous and musculoskeletal systems. The blockade of the calcium channel may contribute to the cardiac contractility impairment seen with barbiturate overdoses.
- Patients with severe CNS depression, Respiratory failure and Hemodynamically unstable should be admitted to ICU for strict management and follow-up

II. Management

a. ABCDE assessment and Initial Stabilization

Barbiturate toxicity also results in decreased cardiac output and vascular tone, often resulting in profound hypotension. Volume expansion is the mainstay of circulatory support in the absence of cardiac failure.

b. Decontamination

- With very large overdoses, antecedent gastric lavage may be considered if is not done before ICU admission.
- A single dose of activated charcoal should be given to cooperative, clinically stable patients who present with in 1 hour of acute oral overdose. For patients who have a compromised airway, endotracheal intubation is advised prior to giving charcoal
- Multi-dose activated charcoal is considered if a patient has ingested a life-threatening amount of phenobarbital.
- A typical adult regimen for multi-dose activated charcoal is an initial dose of 50 to 100 grams PO followed by 12.5 to 25 grams PO every 4 hours.
- The dose for pediatrics is of 1g/kg for a child (for adolescents give 50g).

c. Urinary Alkalization

- Sodium bicarbonate as an IV bolus of 1 to 2 mEq/kg or an IV infusion of 3 to 4 mEq/kg over an hour. Urinary pH should be monitored frequently (every 15 to 30 minutes) until the urine pH is 7.5 to 8.5.
- Urinary alkalinization is sustained by either intermittent bolus or continuous infusion of bicarbonate. Serum pH should not be allowed to rise above 7.5 to 7.55. Adequacy of therapy should be monitored frequently, typically every 2 to 4 hours.
- Thus serum potassium must remain above 4 mEq/L to reliably achieve continuous urinary alkalization.

d. Extracorporeal Elimination

- Hemodialysis should be considered for patients who are deteriorating despite aggressive supportive care.

III. Management complication

- Risks associated with urinary alkalinization include volume overload (heart failure and pulmonary edema), pH shifts, and hypokalemia.

IV. Monitoring and problem solving

- Continuous monitoring of Respiratory condition and mental status should be done to look for respiratory and deterioration mental status.
- Serial ABG analysis, electrolyte determination should be done especially in patients taking Sodium bicarbonate as Urinary alkalinization treatment.

8.7 TRICYCLIC ANTIDEPRESSANT (TCA) POISONING

I. Introduction and principle of care

- Clinical toxicity in intentional and pediatrics age group occurs in >2.5 mg/Kg of drug ingestion, especially in geriatric, underlying heart or neurologic comorbidities, concomitant ingestion of cardiotoxic or sedative –hypnotic drug.
- Life threatening symptoms in drug ingestion 10-20 mg/Kg, Fatal dose > 1 gm
- Severe toxicity usually occurs within 6 hours if ingestion, consist of Coma, cardiac conduction delays, supraventricular and ventricular tachycardia, hypotension, Respiratory depression, seizure.
- The classic ECG with cyclic antidepressant toxicity shows sinus tachycardia, right axis deviation, and prolongation of the PR, QRS, and QT intervals and less commonly a brugada pattern (incomplete RBBB with ST elevation in V1-3 leads) and various block type.

II. Suggested management

a. Initial resuscitation

- Evaluate patients for alterations of consciousness, hemodynamic instability, and respiratory impairment.
- Establish an IV line, Initiate continuous cardiac rhythm monitoring, and obtain serial ECGs.
- Manage Air-way as indicated; many patients require tracheal intubation
- Supplemental oxygen provision
- Urinary catheterization to prevent urinary retention and a nasogastric tube may be needed if ileus is present.
- Laboratory studies include serum electrolytes, creatinine, and glucose.

- **Hypotension** treated initially with isotonic saline fluids in IV boluses in increments of 10 mL/kg to a maximum of 30 mL/kg.
    - Hypotension that does not improve with appropriate fluid challenges should be treated with sodium bicarbonate (regardless of QRS complex duration).
    - Vasopressors (Norepinephrine and epinephrine)should be used when hypotension is unresponsive to fluids and sodium bicarbonate therapy.
    - Start the IV infusion at 1 microgram/min and titrate according to blood pressure.
    - Vasopressin can be tried if there is no response to norepinephrine or epinephrine.

b. GI decontamination

- After the airway, breathing and circulation have been secured; attention may be turned to gastrointestinal decontamination.
- A single 1 gm/kg (up to 50gm) dose of activated charcoal PO if patients are awake, have a patent airway/ intubated, and arrive within 2-3 hour of ingestion.

c. Sodium bicarbonate

- Used to treat cardiac conduction abnormalities (QRS interval >100 msec), ventricular dysrhythmias, or hypotension refractory to IV fluid; LD: 1-2mEq/Kg of 8.4 % of sodium bicarbonate through a wide bore needle,
- Monitor with 12 lead ECG for narrowing of QRS, R wave amplitude decrement in AVR, or resolution of arrhythmia.
- Mix 125 to 150 mEq of sodium bicarbonate in 1 liter of 5 percent dextrose (D5W), and infuse at 250 mL/hour ; reduce the infusion by about 25 percent per hour over four hours if patient respond
- Monitor pH (urine/ plasma) goal: >7.50-7.55 and clinical response.
- Also watch for volume overload, hypokalemia, hypernatremia.
- Torsades de pointes should be treated initially with 2 grams of IV magnesium sulfate.

d. Treatment of Seizures or agitation

- Benzodiazepines for seizures or agitation
- Phenobarbital 10–15 milligrams/kg for seizures refractory to benzodiazepines; Do not give physostigmine, flumazenil, or phenytoin

III. Management of complications/Complications

- Aspiration pneumonia, pulmonary edema, anoxic encephalopathy, hyperthermia, rhabdomyolysis can contribute to morbidity and mortality.
- Death from TCA overdose may result from ventricular fibrillation, intractable cardiac shock, or status epileptics and from secondary complications.
- Sodium Bicarbonate administration causes metabolic alkalosis, Hypernatremia, hypokalemia and fluid overload.

IV. Monitoring and problem solving

- Hemodynamic monitoring including Vital signs and UoP
- Continous ECG monitoring to see any arrythmias
- Mental status should be assessed frequently in a patient having change in mentation
- ABG should be determined if it is available
- Serial Electrolyte determination and ECG should be done in a patient to see complications or response to the treatment in a patient taking Sodium Bicarbonate.

Suggested readings

1. Tintinallis Emergency medicine. A comprehensive Guide, 10th edition
2. Rosen's Emeregency Medicine, 8th edition
3. Goldfranks Toxicological Emergency, 9th edition

CHAPTER 9: SURGICAL ISSUES IN ICU

9.1 PERIOPERATIVE PREPARATION OF THE ICU PATIENT

I. Introduction and principle of care

Thorough clinical examination of the patient presenting for an operative procedure is important for using the clinical findings to guide the request for special investigations and make the patients safe during the perioperative period.

Advantages Preoperative preparation

- Decrease surgical morbidity
- Reduce anxiety
- For optimum pain management
- Help to have proper intraoperative monitoring tools
- Minimize expensive delays and cancellations on the day of surgery
- Increase perioperative efficiency

Components of Preoperative Preparation

- History and Physical examination
- Doing risk assessment
- Developing a plan of medical intervention and optimization
- Reducing the patient's (and family's) anxiety and fears through education
- Discuss perioperative care and options for postoperative pain control
- Determine the appropriate laboratory tests and diagnostic studies
- Discuss anesthesia risks and
- Obtain informed consent.

Indications for ICU admission (Preop)

- Unstable hemodynamics
- Respiratory failure

- Low GCS (GCS<8)
- Delayed awakening
- Pain management
- Massive transfusion
- For 24hours observation after major surgeries (sedation, paralysis….)

II. Suggested management

a. Preoperative period

1. Continue all medications for chronic medical illness

Hold the following medications or replace with safer drugs;

Drug	When to discontinue preoperative/prior to surgery?	Adverse effect if not held/Intraoperative
Loop diuretics	24hours	Hypotension
ACE inhibitors	24hours	Hypotension
OHA(Metformin)	24hours	Lactic acidosis
Warfarin	5days	Bleeding
Aspirin	4days	Bleeding
Clopidogrel	7days	Bleeding
Unfractionated Heparin **IV UFH**	24hrs 6hours	Bleeding
LMWH	72hours	Bleeding
Thrombolytics	5days	Bleeding

2. Prophylactic antibiotics

- Cefazolin 1gm or ceftriaxone 1gm or 2gm (for brain surgery) single dose and repeat the dose if surgery lasts >6hours

3. **Premedication**

- Anxiolytics: low dose diazepam (at bed time)
- Antacids: Non particulate antacid suspension or IV Cimetidine or IV omeprazole
- Antiemetics: metoclopramide or ondansetron

b. Intraoperative period

Have these medications at hand:

- Antibiotics: for redosing if surgery lasts longer
- Antihypertensives: IV labetalol or nitroglycerin
- Anti-hypotension: Noradrenaline, Phenylephrine or ephedrine
- Antiarrythmics: IV lidocaine, amiodarone , adenosine

c. Postoperative period

Continue medications which were started for intraoperative complications till patients get stabilized.

Example:

- Noradrenaline for hypotension
- Oxytocin infusion: for possible atonic uterus
- Sedation
    - Use ketamine or propofol accordingly
- Paralysis
    - Intermittent vecuronium
- Analgesics
    - Morphine or Fentanyl
    - Don't use two analgesics which have the same mechanism of action

III. Management of complications

Unstable hemodynamics

- Fluid 20ml/kg bolus, assess IVC and clinical parameters
- If not improved, start vasopresors preferably, noradrenaline

Pain

- Use systemic analgesics (opioids, paracetamol,dexmedetomidine, ketamine....) as first line
- If not improved, add regional block (epidural, peripheral nerve block, plane/ganglionic block.....) on top of systemic analgesics

Bleeding/Hematoma

- Secure hemostasis with reexploration or with medical therapy (blood and blood products transfusion, adding tranexamic acid...)
- Drain hematoma

↑ICP

- Hyperventilation,head elevation, mannitol,Hematoma evacuation, EVD,decompressive craniotomy,shunt,lumbar drain.....please see under head injury

Hypothermia

- Use warm fluid, blood with line warmer
- Cover the whole body with blanket
- Use heater with target body temperature >35°C (axillary)

Fever (postop)

- Antipyretics, cold compression
- Do septic workup if it is >38.3°C

IV. Monitoring and problem solving

- Follow vital signs

Discontinue medications accordingly

- Glucose monitoring

Target glucose: 140-180mg/dl

- Follow GCS

Titrate sedative drugs accordingly/look for raised ICP

- Assess pain
- Check the functionality of every drainage devices in place

9.2 POSTOPERATIVE PERIOD AFTER ABDOMINAL SURGERY

I. Introduction and principle of care

Major abdominal surgery is when there is either a large incision in over the abdomen or complex keyhole surgery on over the bowel or abdominal wall for different pathologies arising extra or intraperitoneal.

The incidence of postoperative complications and death is low overall, but the sub-group of high-risk patients accounts for over 80% of postoperative deaths, even though these high-risk patients account for fewer than 15% of the in-patient procedures. Advanced age, comorbid disease and major and urgent surgery are the key factors associated with increased risk. Patients undergoing gastrointestinal surgery for malignancy are typical representatives of such high-risk patients.

The complication rate after major surgery for gastrointestinal, hepatobiliary and pancreatic malignancies is high. These postoperative complications significantly increase the hospital stay and mortality.

Table 9-1: Complications after major abdominal Surgery

Infection	**Pneumonia:** Confirmed chest X-ray, marked in case history **Abdominal:** Confirmed abdominal CT, marked in case history **Urinary tract:** Clinical diagnosis, UTI marked in case history **Wound:** Clinical diagnosis, marked in case history **Septic shock:** ACCP consensus criteria
Respiratory	**Mechanical ventilation >24 hr** **Reintubation regardless of the reason**
Cardiovascular	**Acute Myocardial Infarction:** ECG signs of ischemia troponin T > 0.03 ng/ml; diagnosis marked in case history **Cardiac arrest:** **Cardiac arrhythmia:** atrial fibrillation, ventricular fibrillation, marked in case history, use of iv antidysrhythmics (amiodarone ≥ 150mg/day; metoprolol ≥ 5mg; propafenone ≥ 70 mg)

Neurological	**Transient confusion:** Need intravenous therapy with haloperidol and/or clonidine, marked In case history **Stroke**: Clinical diagnosis confirmed with CT, marked In case history
Abdominal	**Anastomotic leak:** Need drainage or reoperation, marked In case history **Ileus:** Requiring nasogastric aspiration or surgery, marked In case history
Renal	**Urine output < 5 mg/kg/hr for more than 12 hr or increased creatinine** **Required dialysis for acute renal failure**
Other	**Postoperative massive hemorrhage:** need for therapeutic endoscopy or re-operation, marked In case history **Re-operation for other reasons than listed above**

II. Prevention of complications

Encourage early mobilization:

- Deep breathing and coughing
- Active daily exercise
- Joint range of motion
- Muscular strengthening
- Make walking aids such as canes, crutches and walkers available and provide instructions for their use

Ensure adequate nutrition

Prevent skin breakdown and pressure sores:

- Turn the patient frequently
- Keep urine and feces off skin

Provide adequate pain control

III. Suggested management

a. Fluid management

- Target is euvolemia
- Replace ongoing loss including third space loss and maintenance with crystalloid +/- dextrose

b. Pain management

- IV opioids: Morphine bolus/Fentanyl infusion +/- Paracetamol
- Combine it with Continuous epidural
- Assess pain severity using pain assessment score

c. DVT prophylaxis

- UFH 7500IU Sc BID or LMWH daily dose
- After 24hours in communication with surgeon

d. Stress prophylaxis

- IV omeprazole or esomeprazole
- Those kept NPO for several hours to days
- Start immediately during the post op period

e. NPO time

- Consider TPN if patient is going to be keep NPO for >72hours

f. Examine abdomen

Look for signs of abdominal compartment syndrome/Peritonitis

Communicate surgeons as soon as possible for possible re-exploration

Abdominal compartment syndrome

- Decompression (upper GI with NG tube and lower GI with rectal tube) as far as no contraindication
- Sepsis management with potent and broad spectrum antibiotics
- Paracentesis if it is due to massive intraperitoneal collection
- Tumor excision if operable
- Laparastomy

g. Examine the wound

- Any bleeding, dehiscence etc...
- Communicate surgeons as soon as possible

h. Take care of drainage devices

i. Deep breath exercise after communication with surgeons

j. Early immobilization

9.3MAJOR BURN

I. Introduction and principle of care

Major Burns cause massive tissue destruction and activation of an inflammatory response that leads to dramatic system wide physiologic derangements

Burn shock: there will be the release of circulating mediators such as tumor necrosis factor and interleukins that result in a systemic inflammatory response syndrome. Within 6 to 8 hours of injury, increased micro vascular permeability, vasodilatation, vascular stasis, decreased cardiac contractility, and reduced cardiac Output.

Hypermetabolic phase: A massive surge in catecholamines and corticosteroids, 10 to 50 times greater than non-burned plasma levels, drives the hyper metabolic response causing increased myocardial oxygen consumption and cardiac work.

Table 9-2: Pathophysiologic changes during early and late phases of major burn injury

	Early phase	Late phase
Cardiovascular	Hypovolemia ↓ cardiac output, ↑ Systemic vascular resistance (SVR)	↑ Cardiac output, tachycardia, systemic Hyoertension
Pulmonary	Airway obstruction and edema, pulmonary edema Carbon monoxide poisoning	Chest wall restriction due to scar formation Trachea stenosis due to repeat/prolonged intubation
Renal	↓ Glomerular filtiration rate, myoglobinuria	↑Glomerular filtration rate, ↑ tubular dysfuncction
Endocrine and metabolic		↑Metabolic rate, ↑ core body temperature, ↑ muscle catabolism ↑ Insulin resistance, ↑ lipolysis, ↑ glucolysis ↓ Thyroid and parathyroid hormones
Hepatic	↓ Perfusion	↑ Perfusion, ↑ metabolism
Hematologic	Hemoconcentration, hemolysis, thrombocytopenia	Anemia
Gastrointestinal	↓ Perfusion with mucosal damage	Stress ulcers, adynamic ileus
Neurologic	↑ Cereral edema, ↑ intracranial pressure	Hallucination, personality change, delirium, seizure, coma

Table 9-3: American burn association burn injury severity grading system

Minor burn	Moderate burn	Major burn
Criteria < 10% TBS (Total body surface area) burnin adults < 5 % TBSA (Total body surface area) burn in young or old	10% - 20% TBSA (Total body surface area) burn in adults 5% - 10% TBSA (Total body surface area) burn in young or old	>20 % TBSA (Total body surface area) burn in adults >10% TBSA (Total body surface area) burn in young or old >5% full-thickness burn
< 2 % full- thickness burn	2% - 5% full-thickness burn High voltage burn Suspected inhalation injury Circumferential burn Medical problem predisposing to infection (e.g: DM, sickle cell disease	High voltage injury Known inhalation injury Significant burn to face, eye, ears, genitals, hands, feet or joints. Significant associated injuries (e.g: Fructure or other major trauma)
Despositon Out patient	Admission to hospital	Refer to burn center

Table 9-4: Classification of burn based on depth

Classification	Burn depth	Appearance	Sensation	Outcome
Superficial **First degree**	Confined to epidermis	Dry and red, blanches	Painful	Heals spontaneously
Partial thickness **Second degree**				
Superficial dermal	Epidermis and upper dermis	Blisters, moist, red and weeping, blanches	Painful to air and temperature	Heals spontaneously
Deep dermal	Epidermis and deep dermis	Blisters, wet or waxy dry, patchy to cheesy white to red, does not blanch	Pressure only	Requires excision and grafting for return of function

Full thickness Third degree	Destruction of epidermis and dermis	Waxy white, leathery gray or charred and black, dry and inelastic, does not blanch	Deep pressure only	Requires complete excision, limited function
Fourth degree	Muscle, fascia, bone		Deep pressure only	Requires excision and grafting, limited function

II. Suggested management

a. **Airway Assessment:** Immediate airway assessment is always the first priority because of the potential for massive airway edema that can result in acute obstruction and death.

- Look for preexisting airway abnormality, current airway injury (i.e, inhalation injury), and signs of airway obstruction.
- Inhalation injury is the most frequent cause of death in burn patients, occurring in 10% to 25% of burn injured patients and increasing mortality by up to 25%.

Prophylactic intubation may be required.

b. **Inhalational injury:** depends on the chemical composition and particulate size of inhaled smoke, the duration of exposure, and the patient's tidal volume during inhalation.

Results in 3 types of injury which includes:

- Thermal injury- mostly restricted to the upper airway
- Chemical irritation- the respiratory tract, and
- Systemic toxicity- due to the absorption of toxic gases such as carbon monoxide.

Management: intubation and high FIO_2 administration

c. **Carbon Monoxide Poisoning:** Carbon monoxide poisoning should be suspected in patients with inhalation injuries and is diagnosed by elevated carboxyhemoglobin (COHb) levels.

- A COHb level greater than 30% requires a high concentration of inspired oxygen to quickly reduce COHb half-life because carbon monoxide binds to hemoglobin, myoglobin, and cytochromes with an affinity 200 times stronger than that of oxygen, and elimination of COHb is dependent on alveolar oxygen pressure rather than alveolar ventilation.

Management: administer 100% FIO2 to reduce the COHb half life

e. **IV Access:** two large-caliber peripheral intravenous catheters through unburned tissue or central line should be secured

f. **Fluid Resuscitation:** The Parkland Formula is the most widely used resuscitation formula in the world, though only the initial 24-hour crystalloid portion tends to be followed closely.

- According to the formula, patients receive 4 ml/kg per percentage of TBSA burned of lactated Ringer's solution within the first 24 hours after burn injury, with half of that volume administered within the first 8 hours.
- Actual fluid rates should be continuously adjusted to maintain urine output of 0.5 to 1.0 ml/kg/h in adults and 1.0 to 1.5 ml/kg/h in children.
- The use of colloids (eg, albumin) for volume replacement remains controversial despite theoretical benefits over crystalloids including replenishment of plasma proteins and greater longevity in the intravascular space.
- **After 24hours:** ongoing loss (third space loss) + maintenance +fluid deficit if patient is NPO will be replaced. To decrease the large volume of crystalloid, albumin 5% infusion can be considered.

g. **Hematology:** The combined effects of burn injury and fluid resuscitation during the acute phase results in anemia and thrombocytopenia.
   - DIC may occur and patient may require Platelet and FFP transfusion.

h. **Renal Effects:** Patients with major burn injuries are susceptible to acute renal failure (ARF) due to the combined effects of hypovolemia and increased levels of catecholamines, angiotensin, vasopressin, and aldosterone, which cause systemic vasoconstriction and can lead to renal insufficiency.

Prevention: fluid resuscitation that is adjusted to urine output, early wound excision, and the prevention of sepsis.

i. **Hepatic Effects**: result from hypovolemia-related hypoperfusion during the initial stages of burn injury, and subsequently from sepsis, drug toxicity, or blood transfusion.

j. **Temperature Regulation:** Patients with major burn injuries are at great risk for heat loss due to loss of skin barriers. **Apply heat conserving strategies which include:**

- Placing the patient on warming blankets or other insulated surfaces
- Covering the patient with plastic or forced-air blankets
- Using warming fluids and blood products.

k. **Pain management:** aggressive pain management with IV paracetamol and opioids plus or ketamine infusion for intubated patients

- Avoid NSAIDs

l. **Glucose monitoring**: avoid hypoglycemia

m. **Sepsis management:** Patients should be on potent broad spectrum antibiotics covering gram –ve, gram+ves and anaerobes

Suggested reading

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CHAPTER 10: TRAUMA INTENSIVE CARE

10.1 PRINCIPLE OF TRAUMA INTENSIVE CARE

Trauma can be defined as physical injury sustained from an interaction between various forms of energy that exceed the physiological tolerance threshold or result in acute failure of vital functions worsening of existing injuries can happen in certain altered physiological conditions that may result from or as a consequence of primary injury.

The secondary damage is the major cause of late mortality and it should be prevented by appropriate monitoring and early correction of the deranged physiology.

A structured approach is vital during an acute early phase of trauma. One such protocol that is widely used is the Advanced Trauma Life Support (ATLS).

A: Airway maintenance and Cervical Spine

Airway obstruction requires immediate intervention, C- Colar should be applied untile CT rules out otherwise.

B: Breathing and Ventilation

Tension pneumothorax, Massive hemothorax, Flail chest and open chests should be identified in primary survey and managed.

Oxygen administration should be initiated via facemask with the flow titrated or with mechanical ventilator if intubated, to achieve target Spo2.

C: Circulation and Hemorrhage control

Hypovolemia: Stop bleeding, IV loading including blood transfusion,

Obstructive Shock: Tension pneumothorax and pericardial tamponade should be identified and manage.

D: Disability (Neurological Evaluation)

E: Exposure/ Environmental control

Emergency Diagnostics and Resuscitation:

IV, Labs, ECG, Pulse Oximetry, Catheters (Gastric, Urinary), Chest X ray, Pelvic X ray and eFAST

Secondary Survey: Head to Toe exam, AMPLE history (Allergies, Medications, PMH, Last Meal intake, Events leading to presentation) Further imaging

Comprehensive Plan of Care: Surgery, Splinting, Consultation,

10.2 CHEST INJURIES

Chest injuries that should be identified during the primary survey include tension pneumothorax, open pneumothorax, flail chest, massive hemothorax and cardiac tamponade.

Management

- Patients are usually managed with chest physiotherapy and oxygen supplementation as needed.
- Oxygen may be given via conventional techniques or high flow therapy.
- Use of NIMV with IPAP and PEEP can be considered in patients with hypoxia who are spontaneously breathing, but a careful assessment should be carried out in ruling out pneumothorax (drain it if necessary).
- If there is no clinical or gas exchange improvement, invasive mechanical ventilation is recommended. Protective ventilation should be practiced.

10.2.1 Flialchest

- Segmental fractures of three or more adjacent ribs anteriorly or laterally often result in an unstable chest wall physiology
- These patients may fatigue rapidly as a vicious cycle of decreasing ventilation, increased work of breathing, and hypoxemia may develop, resulting ultimately in sudden respiratory arrest.

Management

- Mechanical Ventilation should be instituted if necessary.
- Almost 50 % of patients with flail chest eventually require mechanical ventilation.
- Indications for early ventilatory support include: shock, severe head injury, comorbid pulmonary disease, fracture of eight or more ribs, other associated injuries, age >65 years, or arterial partial pressure of oxygen (Po2) <80 mm Hg despite supplemental oxygen

- Effective analgesia is paramount: This may involve multi modal techniques that include both intravenous medications and regional analgesia.
- Regional analgesia should be strongly considered for: Patients with four or more rib fractures, Age > 65 years, Significant cardio pulmonary disease

10.2.2 Pneumothorax

- Tension pneumothorax that causes circulatory distress must be emergently drained to relieve pleural hyper pressure, while occult pneumothorax may be missed on chest X ray, which may be detected by eFAST.
- The latter may then be confirmed by a CT scan. Indications for drainage of occult pneumothorax should be done on case by case basis (for example the need for mechanical ventilation or air transport) and associated injuries.

10.2.3 Hemothorax

Hemothorax requires larger drains of 28-36 French. Drainage of hemothorax allows quantification of blood loss that may dictate further management.

Surgical chest exploration is usually considered in the following settings:

- Blood drain > 1500 ml + > 200ml / hr. for next hour after drainage
- Blood drain < 1500 ml + > 200ml / hr. for next 3 hours.

- If massive hemothorax with active bleeding is suspected, it is reasonable to stop aspiration and consider clamping.
- Size of Chest tube is dependent on the indication and patient size: Pneumothorax: 8-14F, Hemothorax: 28-32F for adult patient.

10.3 SHOCK RESUSCITATION

Trauma management can be described in three hemodynamic phases.

Hemodynamic phases in trauma include:

1. **Phase of hemorrhagic shock:**

The goal in this phase is fundamentally to save life by minimizing bleeding and to preserve hemostasis.

2. **Phase of stabilization:**

Frequently encountered in the OR/ICU setting; the primary goal of resuscitation during this phase is to rescue of organ perfusion and return of oxygen debt

3. **Phase of maintenance:**

This phase typically occurs in ICU setting. The patient is stable hemodynamically with fluid balance mostly neutral or slightly negative.

Targets of hemodynamic resuscitation:

- Macro circulatory stability and recovery of normal blood volume
- Normalization of microcirculatory perfusion indicators
- Normalization of hemostasis
- Normothermia
- Normalization of blood gas exchange

Transfusion strategies

The most important factor in the post-acute phase is the correction of any coagulopathy as it prevents rebleeding. Also excessive bleeding may be associated with dilutional coagulopathy, acidosis and hypothermia which forms the lethal triad.

Damage control resuscitation (DCR protocols) during the early resuscitation phase, which typically involves maintaining moderately hypotensive targets, limiting crystalloids with the goal to limit bleeding and to preserve body hemostatic capacity.

Goal directed correction of trauma induced coagulopathy:

- **Platelet transfusion:** Target platelets should be conservatively maintained around 50 * 10^9. This target may be higher in traumatic brain injury (> 100 * 10^9)
- **Target hemoglobin:** Target hemoglobin varies with the type of injuries, various co-morbid conditions etc. In general, it is advisable to have a target of 7-9 g/dl.
- **Targets of hemostasis:** Correct hypothermia, acidosis and hypocalcemia, Hemoglobin of 7-9 g/dl, Platelets > 50 * 10^9/l; If Traumatic brain injury – TBI: > 100 * 10^9/l 5, PT and aPTT < 1.5 times normal

10.4 TRAUMATIC BRAIN INJURY

I. Introduction (common forms)

Cerebral contusions: areas of 'bruising' within the brain tissue with relatively localized cellular damage, hemorrhage and edema

Diffuse axonal injury: is caused by widespread shearing forces that occur as the brain undergoes stresses such as rapid deceleration.

Traumatic subarachnoid hemorrhage: is bleeding associated with tearing of an intracranial vessel by the shaking of brain tissue in a traumatic situation

Epidural or subdural hematoma: Epidural hematomata may have relatively little underlying associated 'brain damage'; although if of sufficient size, brain compression and ischemia may occur. Subdural hematomata, because of the involvement of brain tissue, have a much worse prognosis.

II. Management

Golden rules

- **Positioning:** The patient should be nursed in a head-up 30-degree position to improve venous drainage and reduce ICP
- **If signs of cerebral herniation on primary assessment:** give hypertonic osmotic solutions E.g.: hypertonic saline or mannitol urgently, and sedation then bridge to surgery
- In extreme life threatening herniation, immediate intubation and mechanical ventilation to protect airway and optimize gas exchange shortly before surgery. **Typical targets:** avoid hypoxia and hypercarbia with ETCO2 of 35-38 mm HG
- Secondary brain injury should be avoided at all costs by preventing cerebral hypo perfusion. Typical target systolic blood pressure is around 120 mm HG in case of severe TBI.
- **Seizure control:** Both clinical and subclinical seizures may have dramatic effects on cerebral metabolism and ICP; they should be prevented

- Temperature control and induced hypothermia: an increase in body temperature to more than 37 should be actively avoided
- Urgent CT brain imaging should be performed.

There **are five key principles** that should guide the ongoing management of the head-injured patient on the ICU:

a. **Normotension:** a single episode of systolic pressure below 90 mm Hg has a direct negative effect on outcome, maintain a mean arterial pressure (MAP) of at least 70 mm Hg,

b. **Normoxia:** hypoxaemia (defined as SpO_2 < 90%) is associated with worsened outcome. Maintenance of oxygenation needs to be balanced against the cardiovascular effects of additional PEEP.

c. **Normocapnia:** Hyperventilation ($PaCO_2$ < 25 mm Hg) should be specifically avoided in the first 24 h and should not be a target for prolonged ventilation beyond this time period.

d. **Normothermia:** avoidance of hyperthermia should be one of the mainstays of head-injury management; it may require the use of pharmacological antipyretics and surface cooling measures.

e. **Normoglycaemia:** Blood sugar concentrations need to be controlled tightly with insulin infusions; administration of dextrose infusions should be avoided. maintain blood glucose between 4--8 mmol/l in these patients.

10.5 TRAUMA COMPLICATIONS

10.5.1 Rhabdomyolysis

Defined as a condition with **necrosis of skeletal muscles**

- This muscle damage in trauma patient often caused by the Crush injury, Muscular compartment syndrome and Prolonged immobilization
- Most patients report myalgias and muscular swelling. Fever, tachycardia, malaise and GI symptoms may be present.
- Reddish brown discoloration of urine may be present.
- Confirmation is made by increased levels of serum myoglobin (first) and creatinine kinase (CK).

Increased risk of kidney injury:

- Serum myoglobin > 1000 mg/ml
- Serum CK > 5000 – 10,000 U/L

Management

1. Ongoing cause of rhabdomyolysis should be addressed such as ischemia, hyperthermia and muscular compartment syndrome.
2. Liberal intravenous hydration with isotonic saline should be considered with an infusion rate of 200 – 500 mls/hr. in order to achieve urine output of approximately 2 ml/kg/hour.
3. Clinical studies have demonstrated that renal replacement therapy has limited role in preventing kidney damage in rhabdomyolysis. Therefore, it should be employed in established acute kidney injury with the traditional indications

10.5.2 Muscular compartment syndrome

- Is an abnormal increase in pressure within confines of a fascial compartment with adverse consequences of irreversible damage to muscles, vessels and nerves.

- Muscular compartment pressure of 30 mm HG or more is considered a compartment syndrome
- Clinical scenarios where muscle compartment syndrome should be considered include: Fracture Crush injury and Tight cast.
- The most prominent clinical sign in awake patient is burning pain in the muscle out of proportion to the clinical findings.
- Findings related to functions of nerve and arteries occur late.

Management

- It is considered as a surgical emergency and hence immediate surgical consultation should be sought.
- External pressure if any on the compartment should be relieved. The affected limb should be kept at a level with the torso. Analgesics, fluids and oxygen should be supplemented as necessary.
- Surgical fasciotomy is the definitive treatment for muscular compartment syndrome and should be performed within 6 hours before the irreversible injury sets in.

Suggested reading

1. Trauma Intensive Care – Principles and Practice - An enchiridion; Ramakanth Pata, MD Ramlingam Pata, MS (Surg), Joanna Kristeva, LCSW
2. Tintinalli's Emergency Medicine A Comprehensive Study Guide Eighth Edition

CHAPTER 11: CRITICAL CARE OF COMMON OBSTETRICS CONDITION

Introduction

ICU admission is indicated with:

- Obstetric hemorrhage, Severe preeclampsia/eclampsia, Hemolysis, elevated liver enzymes, and low platelet, (HELLP) syndrome, Chorioamnionitis, Acute pulmonary edema, Respiratory failure, Acute respiratory distress syndrome, Acute renal failure.
- Considering physiologic changes during pregnancy such as increased heart rate, cardiac output, lowering of platelets, respiratory and air way changes is important.

PRE-ECLAMPSIA AND ECLAMPSIA

I. Introduction and principle of care

Preeclampsia is characterized by hypertension and proteinuria that typically occurs after 20weeks of gestation.

Some of the risk factors include:

- History of preeclampsia in a prior pregnancy,
- Primigravida,
- Women younger than 20 years or older than 35 years,
- Multifetal gestations,
- Obesity,
- Underlying chronic hypertension or chronic renal insufficiency,
- Connective tissue disorders.

Table 11-1: Definitions of Preeclampsia/Eclampsia

Hypertension	Proteinuria
≥140 mm Hg systolic or ≥90 mm Hg diastolic	≥300 mg in 24 h
Previously normotensive patient >20 wk gestation	Protein/creatinine ratio ≥0.3 mg/dl
BP measured two times at least 4 hours aparta	Dipstick ≥1+ (only if other methods not available)

Severe features of preeclampsia

- Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥110 mm Hg on 2 occasions at least 4 hours apart while the patient is on bed rest.
- Thrombocytopenia (<100,000 platelets/mL).
- Impaired liver function (liver enzymes levels increased to twice normal) or persistent right upper quadrant/epigastric pain unresponsive to medication and not accounted for by a different diagnosis)
- Progressive renal insufficiency (serum creatinine >1.1 mg/dL or doubling of creatinine level without other renal disease)
- Pulmonary edema
- Cerebral or visual disturbances
- Fetal growth restriction
- Proteinuria of ≥5 g in a 24-hour urine specimen (or 3+ on random urine dip on two occasions at least 4 hours apart)

Eclampsia

- Eclampsia is the development of new-onset seizures, superimposed upon preeclampsia, in a woman between 20 weeks of gestation and 4 weeks postpartum.
- Occasionally, eclampsia can present with seizure in the absence of blood pressure elevation and proteinuria.

II. Suggested management of Preeclampsia/Eclampsia

a. General principles

- Stabilization of air way, breathing and circulation is the initial step.
- Always anticipate difficult airway in pregnant patients.
- Two large-bore intravenous cannula (14G or 16G) should be placed to administer fluids.
- The Foley catheter should be placed to monitor urine output.
- Nurse in the left lateral position (30° wedge to the right hip) to prevent supine hypotension syndrome.

b. BP control

- Arterial pressure greater than 160/110 mmHg in preeclampsia can increase the risk of complication, and it should be controlled.
- Goal of BP control is 15–25% reduction in the mean arterial pressure, and reduction of pressure to normal levels (<140/90 mmHg) should be avoided as it may compromise placental perfusion.

Table 11-2: Antihypertensive Drugs for Treatment of Acute, Severe Hypertension in Preeclampsia and Eclampsia

Generic Name	Onset of Action	Dosage	Comment
Labetalol	5 min	20 milligrams IV, then 40–80 milligrams IV every 10 min as needed (maximum, 300 milligrams); IV infusion 1–2 milligrams/min titrated	Less hypotension and reflex tachycardia than hydralazine. Higher doses cause neonatal hypoglycemia. Longer use associated with fetal growth restriction.
Hydralazine	20 min	5 milligrams IV or 10 milligrams IM, repeat at 20-min intervals; consider other drug if no response at maximum of 20 milligrams IV or 30 milligrams IM	Maternal hypotension, fetal distress; must wait 20 min for response between IV doses.
Nifedipine	10–20 min	10 milligrams PO, repeat in 30 min if necessary	

c. Seizure control

- The initial bolus of magnesium (4 g over 20 min) is followed by an infusion of 1–2 g/h.
- Monitor toxicity—loss of deep tendon reflexes; loss of patellar reflex occurs when the plasma magnesium level is more than 10 mg%.
- If seizures recur while the patient is receiving magnesium, a repeat (2g) bolus of magnesium may be given.
- Infusion dose should be reduced in case of renal dysfunction. Serum magnesium level should be monitored
- Discontinue magnesium sulfate 24 h after delivery or after last seizer.
- Other alternatives include intravenous administration of benzodiazepines (lorazepam or diazepam).

d. Delivery

- Women with severe preeclampsia and eclampsia who are managed expectantly need deliver, hence need consultation to obstetrician.

III. Manage complications

a. HELLP syndrome

- HELLP syndrome can complicate 4–12% of patients with severe preeclampsia.
- Delivery is the definitive treatment for HELLP syndrome.
- Delivery is indicated for women with HELLP syndrome at greater than 34 weeks' gestation. During labor and for 24-h postpartum, patients should receive intravenous magnesium sulfate for seizure prophylaxis.
- If gestation is less than 34 weeks, delivery may be delayed for a steroid with delivery 24 h after the last dose.
- Platelets are generally transfused when the platelet count is less than 20000 or 50000 if labor or surgery is expected.

b. Acute pulmonary edema

Management is similar as in non-pregnant patients.

c. PRES and intra cranial bleeding

d. Fetal complications of severe preeclampsia

Fatal complication including Intrauterine growth retardation, Premature delivery, Abruptio placentae, Fetal distress/fetal demise should be managed accordingly.

e. Other complication: to manage accordingly are disseminated intravascular coagulopathy, hemorrhage, Acute renal failure.

IV. Monitoring and problem solving

Repeated clinical assessment including

- Neurological examination (deep tendon reflexes for magnesium toxicity).
- Continues ECG and BP and oxygenation monitoring.
- Foley catheterization—urine output monitoring.
- Blood gas monitoring.
- Watching for increased intra-abdominal pressure during resuscitation,
- Serum magnesium levels and deep tendon reflexes
- Fetal monitoring. Avoid sudden fall in BP as it can result in fetal distress.

Suggested reading

1. R. Chawla and S. Todi (eds.), ICU Protocols: A stepwise approach, DOI 10.1007/978-81-322-0535- 7_76, © Springer India 2012.
2. Brian Brown, the Washington Manual Of Critical Care. Lippincott Williams & Wilkins, A Wolters Kluwer, 2012

CHAPTER 12: COMMON ICU DRUG DOSAGES AND SIDE EFFECT

Table 12-1: Common ICU drugs dosage and side effects

Drugs	Dosing	Reconstitution	Common side effect
Shock			
Norepinephrine	0.02–3 μg/kg/min		Hyperglycemia
Epinephrine	0.01–0.1 μg/kg/min	Cardiac arrest • IV bolus: 10 ml 1 in 10 000 solution (1 mg). Anaphylaxis (p. 200) **IV bolus:** 0.5–1.0 ml 1 in 10 000 solution (50–100 mcg), may be repeated PRN, according to BP	Tachycardia, hyperglycemia
Phenylephrine	0.5–10 μg/kg/min	**Phenylephrine** Unimedic 10 mg/ml will be administered as an intravenous injection or infusion after dilution in sodium chloride 9 mg/ml (or glucose 50 mg/m	----
Dopamine	5–20 μg/kg/min	200mg made up to 50ml 5% dextrose or 0.9% saline (4000microgram/ml)	Tachycardia, arrhythmias
Vasopressin	0.01–0.04 U/min		
Dobutamine		250mg made up to 50ml 5% dextrose or 0.9% saline (5000microgram/ml)	Tachycardia
Milrinone	50 μg/kg bolus, then 0.25–0.75 μg/kg/min		Hypotension

Corticosteroids Hydrocortisone	Septic Shock: 200–300 mg/ day in 3–4 divided dose		**Short-term:** hyperglycemia, mood changes, insomnia, gastrointestinal irritation, increased appetite **Long-term:** Osteoporosis, acne, thin skin, fat redistribution, muscle wasting, cataracts HPA axis suppression, increased blood pressure, infection
Mineralocorticoids Fludrocortisone	Septic shock: 50–100 μg PO q24h		Increased blood pressure, edema, hypernatremia, hypokalemia
Respiratory Disorders			
Corticosteroids Methylprednisolone ▪ **ARDS,** ▪ **Status asthmaticus** ▪ **COPD exacerbation**	1–2 mg/kg/day in 3–4 divided doses, tapering schedule dependent on disease process		**Short term** Hyperglycemia, mood changes, insomnia, gastrointestinal irritation, increased appetite **Long term** Osteoporosis, acne, thin skin, fat redistribution, muscle wasting, cataracts, HPA axis suppression, increased blood pressure, infection
Beta-agonists			
Albuterol	2–4 puffs BID to QID		
Levalbuterol	0.63–0.125 mg TID		

Anticholinergics			
Ipratropium	2–4 puffs BID to QID		
Pulmonary hypertension			
Calcium channel blockers			Peripheral edema flushing, headache
Diltiazem	up to 720 mg/day		hypotension
Nifedipine	up to 240 mg/day both in divided doses		
Warfarin	Target goal INR		Bleeding
Unfractionated heparin	80 U/kg bolus, then 18 U/kg/hr, adjust to aPTT 1.5–2.5 × control		Bleeding, type I heparin-induced thrombocytopenia bleeding
Alteplase	100 mg IV over 2 hr		Bleeding
Disorders Cardiovascular disorder			
Acute myocardial infarction			
Aspirin	160–325 mg PO daily		Bleeding, dyspepsia
Beta-blockers			
Metoprolol			
Esmolol			
Nitrates			
Nitroglycerin	10–200 μg/min		Headache, flushing, dizziness, hypotension, tachycardia

Isosorbide dinitrate	5–40 mg PO TID		
Isosorbide mononitrate	30–120 mg PO daily		
Morphine	1–4 mg IV q5min		
Fibrinolytics			
STEMI			
Streptokinase	1.5 million units over 2 hrs.		
Unfractionated heparin	60 U/kg bolus, then 12 U/kg/hr, adjust to aPTT 1.5–2.5 × control	IV: Loading dose of 5000 units followed by continuous infusion of 1000–2000 units/h 24 000 units heparin made up to 48ml with 0.9% saline (5000 units/ml). Check APTT 6h after loading dose and adjust rate to keep APTT between 1.5 and 2.5 times normal	Bleeding, type I heparin induced thrombocytopenia bleeding
Low-molecular weight Heparins **Enoxaparin**	1 mg/kg SC q12h		Bleeding
GPIIb/IIIa inhibitors	?		Bleeding
Clopidogrel	75 mg PO daily		Nausea, vomiting, diarrhea, bleeding
Arrhythmias and conduction abnormalities			
Atropine	1 mg IV q3–5min	Dilute 1ml of 600microgram/ml **Atropine** with 5 ml Sodium Chloride 0.9% to give 6ml of 100microgram/ml	Dry eyes, dry mouth, urinary retention, tachycardia
Epinephrine			
Vasopressin	40 units IV		

Procainamide	15–18 mg/kg bolus then 1–6 mg/min		Diarrhea, nausea, vomiting
Lidocaine	1–1.5 mg/kg bolus (may repeat doses 0.5–0.75 mg/kg in 5–10 min up to max 3 mg/kg), then 1–4 mg/min		----
Amiodarone	300 mg bolus, then 1 mg/min for 6 hr, then 0.5 mg/min for ≥18 hr		Bradycardia, hypotension, nausea
Calcium channel blockers	0.25 mg/kg bolus (may repeat 0.35 mg/kg bolus after		Bradycardia, hypotension, constipation
Diltiazem	15 min), then 5–15 mg/hr		(verapamil > diltiazem) headache, flushing, edema
Verapamil	5 mg bolus (may repeat up to total 20 mg), then 5–15 mg/hr		
Adenosine	6 mg IV, if not effective in 1–2 min can give 12 mg, may repeat 12 mg		Flushing, lightheadedness, headache, nervousness/ anxiety
Congestive heart failure			

Digoxin	Load: 10–15 μg/kg; give 50% of load in initial dose, then 25% at 6–12 hr intervals × 2 Maintenance 0.125–0.5 mg/day (Dose should be reduced by 20%–25% when changing from oral to IV)		Bradycardia
Captopril	6.25–50 mg PO TID		Cough, hyperkalemia, hypotension, renal insufficiency
Lisinopril	2.5–40 mg PO BID		Cough, hyperkalemia, hypotension, renal insufficiency
Enalapril	2.5–10 mg PO BID		Cough, hyperkalemia, hypotension, renal insufficiency
Spironolactone	12.5–50 mg PO daily		
Furosemide	20–80 mg/day IV/PO in 2–3 divided doses		Hypomagnesemia, Hypocalcemia
Hypertensive emergencies			
Nitroprusside	Usual, 0.25–3 μg/kg/min Max, 10 μg/kg/min		Nausea, vomiting, hypotension, tachycardia, thiocyanate and cyanide toxicity

Nicardipine	3–15 mg/hr		Hypotension, tachycardia, headache, flushing, peripheral edema
Labetalol	20–40 mg (max, 80 mg) as IV bolus at 10–20 min intervals, then 0.5–2 mg/min if needed		Hypotension, bradycardia, nausea, vomiting
Clonidine	0.1–0.3 mg PO BID–TID		Drowsiness, dizziness, hypotension, bradycardia, dry mouth
Hydralazine	1.25–5 mg IV q6h		Hypotension, hyperkalemia, renal insufficiency
Electrolyte Abnormalities			
		Hyponatremia	
Conivaptan	20 mg IV bolus, then 0.8–1.6 mg/hr IV continuous infusion		Diarrhea, hypokalemia
		Hyperkalemia	
Regular Humulin insulin	10–20 units IV (given with dextrose, every ~1 unit for 4–5 g dextrose)		Hypoglycemia, hypokalemia, weight gain
Sodium bicarbonate	1 mEq/kg IV		Metabolic alkalosis, hypernatremia, hypokalemia
Albuterol	10–20 mg nebulized over 30–60 min		------
Calcium gluconate	1 g IV over 2 min		Hypercalcemia, constipation (oral)
		Hypercalcemia	

Pamidronate	60–90 mg IV bolus		Fever, fatigue
Calcitonin	Initial 4 U/kg IM q12h, up to 8 U/kg IM q6h		Facial flushing, nausea, vomiting
		Hypophosphatemia	
Phosphate salts **Potassium phosphate** **Sodium phosphate**	0.08–0.16 mmol/kg IV over 6 hr.		Hyperphosphatemia Hypocalcemia Hypomagnesemia
Endocrine Disorders			
Levothyroxine	Myxedema coma: 50–100 μg IV q6–8h × 24 hr, then 100 μg IV q24h	-----	--------
Propylthiouracil	Initial 300–600 mg/day in 3 divided doses q8h, maintenance 50–300 mg per day		Rash, arthralgias, fever, leukopenia, nausea, vomiting
Methimazole	Initial 30–60 mg/day in 3 divided doses q8h, Maintenance 5–30 mg per day		
Propranolol	10–40 mg PO q6h		Bradycardia, hypotension, fatigue, malaise, cold extremities
Iodide	1–2 drops PO q12h		Metallic taste, nausea, stomach upset, diarrhea, salivary gland swelling

Adrenal insufficiency			
Corticosteroids **Hydrocortisone** **Dexamethasone**	100 mg IV q8h 10 mg IV prior to ACTH stimulation test		Short term: hyperglycemia, mood changes, insomnia, gastrointestinal irritation, increased appetite Long term: osteoporosis, acne, thin skin, fat redistribution, muscle wasting, cataracts HPA axis suppression, increased blood pressure, infection
Fludrocortisone	50–200 μg PO q24h		Increased blood pressure, edema, hypernatremia,
Insulin			Hypoglycemia, hypokalemia, weight gain
Oncologic Emergencies			
Allopurinol	600–800 mg/day in 2–3 divided doses		Rash
Toxicology			
Activated charcoal	25–100 g		Vomiting, constipation, fecal discoloration
Naloxone	0.4–2 mg IV q2min, up to 10 mg		
Flumazenil	0.2–0.5 mg IV q1min, up to 5 mg		Abrupt reversal may cause withdrawal symptoms (sweating, agitation, hypertension, tachycardia, nausea, vomiting, cardiovascular events, seizures)

***N*-Acetylcysteine**	Oral 140 mg/kg loading dose, then 70 mg/kg q4h × 17 doses IV 150 mg/kg bolus, then 12.5 mg/kg/hr × 4 hr, then 6.25 mg/kg/hr × 16 hr		Nausea, vomiting (oral), unpleasant odor (oral)
Deferoxamine	1 g IV bolus, then 500 mg IV q4h × 2 doses		Urine discoloration (orange-red)
Infectious Diseases			
Vancomycin	15 mg/kg IV q12h		
Amikacin	8 mg/kg IV q12h or 15 mg/kg extended interval		
Gentamicin	3 mg/kg bolus, then 2 mg/kg IV q8h or 5–7 mg/kg extended interval	IV infusion: 7 mg/kg in 50 ml 5% dextrose or 0.9% saline, given over 1 hour	Nephrotoxicity Ototoxicity
Erythromycin	250–500 mg PO qid or 0.5–1 g IV q6h	Reconstitute with 20ml WFI, shake well, then further dilute in 250ml 0.9% saline, given over 1 h	Nausea, vomiting, diarrhea, abnormal taste
Azithromycin	250–500 mg IV/PO daily		Nausea, vomiting, diarrhea, abnormal taste
Clarithromycin	250–500 mg PO BID	Reconstitute in 10ml WFI. Then make up to 250ml with 5% dextrose or 0.9% saline and give over 60min If creatinine clearance #30ml/min, give half the dose (250mg 12 hourly)	Nausea, vomiting, diarrhea, abdominal pain, rash

Metronidazole	500 mg IV/PO q8h		Nausea, vomiting, metallic taste, disulfiram-like reaction
Antifungal agents			
Fluconazole	100–800 mg PO/IV daily		Nausea, vomiting, diarrhea rash, visual disturbances, phototoxicity
Amphotericin B deoxycholate	0.3–1.5 mg/kg q24h	The liquid in each reconstituted vial will contain 5mg/ml amphotericin. This is further diluted to a final concentration of 0.625mg/ml by diluting 1 volume of the reconstituted amphocil with 7 volumes 5% dextrose Flush an existing intravenous line with 5% dextrose before infusion Although anaphylactic reactions rare, before starting treatment, an initial test dose of 2 mg should be given over 10 min, infusion stopped and patient observed for 30 min. Continue infusion if no signs of anaphylactic reaction.	Acute infusion-related reactions, hypokalemia, hypomagnesemia
Antiviral agents			
Acyclovir	400 mg PO TID or 5 mg/kg IV q8h	Available in 250 and 500 mg vials for reconstitution Reconstitute 250 mg vial with 10 ml WFI or 0.9% saline (25 mg/ml) Reconstitute 500 mg vial with 20 ml WFI or 0.9% saline (25 mg/ml) Take the reconstituted solution (25 mg/ml) and make up to 50 ml (for 250 mg vial) or 100 ml (for 500 mg vial) with 0.9% saline or 5% dextrose, and give over 1 h	Bone marrow suppression, headache, nausea, vomiting, diarrhea
Hepatic Disorders			

Lactulose	20–30 g (30–45 mL) PO q2h until initial stool, then adjust to maintain 2–3 soft stools/day		Diarrhea, flatulence, nausea
Neomycin	500–2000 mg PO q6–12h		Nausea, vomiting, diarrhea, irritation or soreness of mouth or rectal area
Neurologic Disorders			
Lorazepam	0.1 mg/kg at 2 mg/min up to 8 mg		CNS depression
Midazolam	0.2 mg/kg bolus, then 0.75–10 μg/kg/min		CNS depression
Phenytoin	20 mg/kg IV bolus, then 5–7 mg/kg day		Concentration dependent: nystagmus, diplopia, ataxia, sedation, lethargy, mood/behavior changes, coma, seizures
Phenobarbital	20 mg/kg IV bolus		Sedation, nystagmus, ataxia, nausea, vomiting IV form: hypotension, bradycardia, respiratory depression
Propofol	30–250 μg/kg/min		Hypotension, bradycardia, CNS depression, hypertriglyceridemia
Valproate	1000–2500 mg/day IV/PO in 2–4 divided doses		Somnolence, diplopia nausea, vomiting, diarrhea

Mannitol	1–1.5 g/kg IV bolus, then 0.25–1 g/kg q3–6h as needed		Hypotension, acute renal failure, fluid and electrolyte imbalances
Stroke			
Alteplase	Ischemic stroke: 0.9 mg/kg IV (NOT to exceed 90 mg), infused over 60 min with 10% of the dose given as an initial bolus over 1 min	IV infusion: 7 mg/kg in 50 ml 5% dextrose or 0.9% saline, given over 1 hour	Bleeding
Factor VII	Hemorrhagic stroke: 1.2–4.8 mg IV		Hypertension
Pregnancy			
Magnesium	4–6 g IV over 15–20 min, then 2 g/hr infusion		
Phenytoin	20 mg/kg IV bolus, then 5–7 mg/kg day		
Labetalol	100–800 mg PO q8–12h, max 2.4 g/day		Hypotension, bradycardia, nausea, vomiting
Hydralazine	10–40 mg IV q4–6h or 10–75 mg PO TID–QID		Hypotension, bradycardia, nausea, vomiting, hypotension, tachycardia, flushing, headache

CHAPTER 13: PEDIATRIC CRITICAL CARE

13.1 ASSESSMENT OF A CRITICALLY ILL CHILD

I. Introduction

- Sick child evaluation should be done rapidly and systematically with General, Primary, Secondary and Tertiary assessments.
- Life-threatening conditions should be intervened and child stabilized before proceeding to the next level of assessment.

a. General assessment

Includes evaluation of Appearance, Breathing and Circulation

- **Appearance:** Look tone, consciousness, gaze, and speech/cry
- **Breathing:** look for abnormal airway sounds, abnormal positioning, retractions
- **Circulation:** Pallor, mottling, cyanosis
- Allow the child to assume his position of comfort during the evaluation
- Put all children with life-threatening condition on oxygen (preferably facemask) and activate emergency system.

b. Primary survey

1. Airway Assessment

- Listen for abnormal sounds e.g., snoring, gurgling, stridor, wheeze or grunting
- Look for secretions, drooling, vomiting, bleeding, cyanosis, symmetry of chest rise
- Look for any sign of burn and trauma on head and neck

Management of Airway

- Open the airway: Head tilt-chin lift (non-trauma patient) or jaw thrust (suspected trauma)
- Put cervical collar in place for suspected trauma

- Suction secretion and remove foreign bodies UNDER direct inspection
- Start Face mask O2 at 6-8 l/min
- Apply air way adjuncts if airway not maintainable and advanced airway as needed

2. Breathing Assessment

- LOOK: RR , work of breathing and chest movement
- FEEL: Air movement
- LISTEN: Breath sounds
- MEASURE: SPO2

Breathing Management

- Start BMV if apnea or irregular breathing
- Start oxygen based on patient need initially with face mask
- Needle decompression for tension pneumothorax
- Salbutamol puff (nebulizer) for suspected asthma

3. Circulation Assessment

- **Pulse quality:** Palpate central and peripheral pulses .
  - Check radial pulse (older children) and brachial pulse (infants)

➢ If not palpable check central pulses (femoral for infant and carotid for older child).

- **Skin temperature, Mottling, Sunken eyes , and skin pinch**
- **Capillary refill** > 3 is sign of shock (affected by cold environment)
- **Blood pressure**:
  - Minimum systolic BP:
    - < 1 month = 60 mmHg
    - 1 month to 1 year = 70 mmHg and
    - 1-10 years BP = 70 + (2 X age in years)
    - Above 10 years BP= 90 mmHg

Circulation Management

- Start CPR if no pulse
- Rapid pulse, delayed capillary refill, cool skin+/- altered mention: Manage for shock

- If patient has signs of dehydration, manage based on **ETAT** protocol
- Cardioversion or defibrillation for patients with dysrhythmia

4. Disability Assessment

- AVPU: **Alert**, responds to **verbal** commands, responds to **pain**, **Unresponsive**
- Check bilateral pupil size and reaction
- Look for seizure (Beware of subtle seizure in children)
- Check glucose (RBS)

Disability Management

- Treat hypoglycemia
- Anticonvulsant for active seizure lasting > 5 minutes
- ICP management
- Recovery position

5. Exposure assessment and Management

- Remove cloths and see the back and extremities for trauma and rashes
- Maintain warm ambient environment and minimize heat loss.
- Monitor temperature

c. Secondary survey

- **History**: SAMPLE mnemonic: Signs/Symptoms, Allergies, Medications, Past medical problems, Last food or liquid, Events leading to injury or illness
- **Physical examinations** Focused on pertinent systems relevant to the child
- Continue to stabilize the child and consider specific management based on likely diagnosis

d. Tertiary survey

- Plan sequence of laboratory testing and imaging as indicated clinically

13.2 PEDIATRIC RESPIRATORY CRITICAL CARE

13.2.1 APPROACH TO A CHILD WITH RESPIRATORY FAILURE

Introduction

- As young infants are mainly nasal breathers, clearing noses can improve their breathing.
- Recognizant of the differences of pediatric airway with older children and adults
- Keep the child with respiratory distress in his/her position of comfortable.

a. Respiratory distress

- Refers to the use of compensatory mechanism to maintain oxygenation and ventilation.
  - Increased respiratory rate
  - Increased work of breathing (WOB)
  - Tachycardia, hypertensive, anxious looking

Respiratory failure

- Defined by inadequate oxygenation or ventilation with inability to meet metabolic demand of the body.
- Three types: hypoxic (room air PA02 </= 60mmhg), hypercapnic (PA02 >/= 50mmhg and acidosis PH <7.35) and mixed.
- Respiratory failure can occur with or without respiratory distress.

Clinical indicator of respiratory failure:

- Respiratory failure should be recognized early as it is a pre-arrest state
- Marked tachypnea, bradypnea, apnea
- Cyanosis (late sign)
- Worsening saturation and clinical condition despite appropriate interventions
- Poor or absent air movement

- Mental change: key in differentiating respiratory failure and distress
- Infants could maintain SPO2 while progressing to respiratory failure; don't solely rely on pulse oximeter.

Table 13-1: Causes and management of respiratory distress and failure

<table>
<tr><td colspan="3">General management of Respiratory Emergencies
Airway positioning
Oxygen
Pulse oximetry
ECG monitor (as indicated)
BLS as indicated</td></tr>
<tr><td colspan="3">Upper Airway Obstruction (Management for Selected Conditions)</td></tr>
<tr><td>Croup</td><td>Anaphylaxis</td><td>Foreign Body Aspiration</td></tr>
<tr><td>Nebulized epinephrine (0.5 ml/kg L epinephrine, max 5 ml)
Corticosteroids (Dexamethasone 0.6 mg/kg, max 16 mg IM stat)</td><td>IM epinephrine (auto-injector) Im
Salbutamol
Antihistamines
Corticosteroids
IV fluids</td><td>Allow position of comfort
Rigid bronchoscopy removal</td></tr>
<tr><td colspan="3">Lower Airway Obstruction (Management for Selected Conditions)</td></tr>
<tr><td colspan="2">Bronchiolitis</td><td>Asthma</td></tr>
<tr><td colspan="2">Nasal Suctioning
Bronchodilator trial in severe cases and older infants</td><td>Salbutamol+/- Ipratropium
Corticosteroids
SQ epinephrine
Magnesium sulfate</td></tr>
<tr><td colspan="3">Lung Tissue (Parenchymal) Disease (Management for Selected Conditions)</td></tr>
<tr><td colspan="2">Pneumonia/Pneumonitis
Infectious Chemical Aspiration</td><td>Pulmonary Edema Cardiogenic or Non cardiogenic (ARDS)</td></tr>
<tr><td colspan="2">Salbutamol
Antibiotics (as indicated)</td><td>Noninvasive or invasive ventilator support with PEEP</td></tr>
</table>

	▪ Consider vasoactive support ▪ Consider diuretic
Disordered Control of Breathing (Management for Selected conditions)	
Increased intracraneal pressure (ICP)	Poisoning /Overdose	Neuromuscular Disease
▪ **Avoid hypoxemia** ▪ **Avoid hypercarbia** ▪ **Avoid hyperthermia** ▪ **Mannitol/ HTS**	▪ Antidote (if available) ▪ Contact poison control	▪ Noninvasive or invasive ventilator support

13.2.2 UPPER AIRWAY OBSTRUCTION

13.2.2.1 CROUP (LARYNGO TRACHEA BRONCHITIS)

I. Introduction

- Croup is acute viral infection of upper airway mostly in children 6 months to 3 years.
- Manifest with barking cough, hoarseness, stridor, and increased work of breathing

a. Classification of croup

Grade 1: Stridor at rest, no retractions

Grade 2: Stridor at rest, retractions

Grade 3: Marked respiratory distress, irritability, pallor or cyanosis, exhaustion and 50% require intubation

b. Diagnosis: clinical diagnosis and does not require radiograph

- Be aware of important differentials however especially in atypical and severe cases.

Investigation

Radiography only in children with failed therapy or if need to exclude other diagnosis e.g. retropharyngeal abscess, or aspirated foreign body.

II. Management of croup

Mild illness is managed as out patient

Moderate: admitted to emergency or high dependency unit (HDU).

- Provide oxygen; epinephrine 1:1000 nebulization; repeat after 30 min as needed
- Dexamethasone (0.6mg/kg IM/IV stat, max 16 mg), OR prednisolone 1 mg/kg stat
- Observe for at least 3 hours' post-treatment

Sever croup

- Keep calm and in comfort position, avoid procedures (throat examination, lab test)
- Give 100% oxygen
- Nebulize adrenaline and Steroid as above
- Call anesthesiologist and ENT for intubation if altered mentation, and hypoxemic despite the appropriate treatment
  - If this takes time do cricothyidoitomy
- Intubate with smaller than normal endotracheal tube

13.2.2.2 EPIGLOTTITIS

I. Introduction

- Though rare it is a life-threatening bacterial infection in children above 12 months and characterized by inflammation and edema of the epiglottis
- Abrupt onset of high fever (toxic), respiratory distress, drooling, muffled voice, swallowing difficulty, and limitation of neck movement.
- Tripod position

Diagnosis

- Is clinical (if laryngoscope done it should be in operative or intensive care unit)
- Neck x-ray: thumb sign

II. Epiglottitis Management

- ABCD
- Minimize interventions if child is conscious and maintain his/her airway.
- Administer 100% O_2
- Intubation (call Anesthesiologist and ENT; consider needle cricothyrodotomy if intubation delayed); Tracheostomy if intubation is difficult.
  - Use small ETT 1 size lower than estimated.
  - Preferably intubate with inhalational anesthesia if possible

- Keep the patient on maintenance fluids after securing the airway.
- Give IV antibiotics active against H. Influenza, Streptococcus pneumoniae and Staphylococcus aureus

13.2.2.3 BACTERIAL TRACHEITIS

- A life-threatening Bacterial infection of the trachea with purulent exudates
- Presents as croup but patient are more toxic, with high fever and poor response to adrenaline
- Organisms include Staphylococcus aureus, H. influenzae and pneumococcus
- Bronchoscopy can confirm diagnosis and help suction debris and exudate
- Broad-spectrum antibiotics covering staph aureus and Hemophilus
- Intubation if severe airway obstruction

13.2.3 LOWER AIRWAY OBSTRUCTION

13.2.3.1 ASTHMA

I. Introduction

Status Asthmaticus is defined as severe asthma that fails to respond to inhaled B2 agonists, steroids, and oxygen, and that require admission for continued treatment.

NB: Consider other differentials if history and examination are not consistent with asthma.

II. Managing severe asthma exacerbation

Give high flow Oxygen, Steroids and B-agonist simultaneously.

Short acting B agonist

- Salbutamol pMDI with spacer for 3 doses every 20 minute(make more frequent based on how sever patient is) followed by 1-2 hourly or use continuous nebulization (0.3 mg/kg/hr)
  - 6 puff for < 6 year and 8-12 puff for older child
- Nebulizer driving flow: minimum of 5–10 L/min and as specified by manufacturer
  - Ipratropium Nebulization
    - 250 mg Ipratropium with salbutamol in same nebulizer every 20 minutes for 3 doses, then every 4 hours.

Steroid

- Prednisolone 2 mg/kg/d (max 60 mg daily) po or IV/IM dexamethasone 0.6 mg/kg/d (max 20 mg) or IV hydrocortisone 4 mg/kg stat, then 2 mg/kg Q6 hourly.

Intravenous Fluid

- Give 20 ml/kg NS for shock then maintenance 5%DNS with 40 meq/liter KCL
- Restrict maintenance to 2/3rd if sodium is <138 meq/L

Magnesium Sulfate

- 50%, 0.1 ml/kg (50 mg/kg) IV dilute with 100- 200 ml saline over 20-30 minutes. Watch for drug side effect hypotention, neurolgic sideffect (eg deep tenden reflex)
- Repeat as needed or give continuously at 10-20mg/kg /hr.

Aminophylline

- Reserved for non-responders with beta- agonist and magnesium sulphate.
- Loading 5-6 mg/kg over 30-60 min then 0.5 -1.0mg /kg/hr continuously (max 300 mg) or 3 to 5 mg/kg every 6-8 hours, dilute in equal saline and give over 20 – 60 min.

Side effect: arrhythmia, hypotension, disorientation, fits, GI symptoms. Stop immediately if child vomits, has a pulse rate >180/min, headache or convulsion.

Subcutaneous Beta –Agonist

Epinephrine

- children with poor air flow, cannot cooperate with nebulization, or didn't improve with continuous nebulization or no response with all the above management
- 1:1000 solution (max dose 0.4mg), 0.01mg/kg/dose, repeat every 15 -20 min x 3 doses.

Other Considerations

- CXR only if no response or if concern for pneumothorax or other differentials
- Consider noninvasive ventilation like BIPAP early in the course
- Antibiotics only if there are evidences for bacterial infection

Intubation and ventilation

Intubation is best avoided unless mandatory, try your best early as above to avoid it.;[

Indications for intubation:

  - Cardiopulmonary arrest
  - Rapid change in mentation
  - No improvement despite maximum therapy and patient is exhausted
- Bag slowly if patient arrests to give exhalation time and use smaller tidal volume
- Use ketamine as sedative, or midazolam +/-fentanyl.
- Avoid muscle relaxant if possible and intubate rapidly with a cuffed tube.
- Anticipate hypotension and give NS bolus during intubation.
- Ventilator setting:

- Lower RR and Lower tidal volume
- Lower PEEP =3-5, I: E ratio 1:3 or more,
- Tolerate permissive hypercapnia and hypoxia.

Discharge

- Discharge with prednisolone for 3-5 days or dexamethasone for a 2-days and continue salbutamol puff.
- Consider controller initiation before discharge and arrange follow up in 1-2 weeks

III. Monitor

- Monitor vital signs, work of breathing air entry, ability to talk and mental status

Laboratory:

- Blood gases and electrolyte in particular potassium.
- Lactic acidosis results from dehydration and /or respiratory muscle fatigue

IV. Complication

- Hypotension and pneumothorax should be anticipated and managed accordingly

13.2.3.2 BRONCHIOLITIS

Definition: is characterized by a prodrome of viral infection followed by lower respiratory symptoms including wheeze, crackles and variable degree of increased work of breathing in infants less than 2 years of age.

Diagnosis of Bronchiolitis: Clinical

Investigation: No routine investigation is important or necessary

Management of bronchiolitis

- Manipulate infant as little as possible
- Suction nose and oropharynx as needed
- Oxygen (CPAP or HFNC for severe cases with marked increase in WOB)
- Hydration (if infant is intolerant for oral feed use nasogastric tube or IV fluids)

- Adrenaline nebulization can be tried in very severe cases
- B2 agonists and steroid: no proven benefit and use should be individualized

Monitoring: Close monitoring of vital sign, work of breathing, hydration status and mentation

13.2.4 PEDIATRIC ACUTE RESPIRATORY DISTRESS SYNDROME

I. Introduction

Definition: Pediatric Acute respiratory distress syndrome (pARDS) represents the nonspecific lung injury that results from an aggressive inflammatory process caused by severe pulmonary or extra pulmonary pathologies.

Table 13-2: Pediatric Acute respiratory distress syndrome

<table>
<tr><td>Age</td><td colspan="5">Exclude patients with perinatal related lung disease</td></tr>
<tr><td>Timing</td><td colspan="5">Within 7 days of known clinical insult</td></tr>
<tr><td>Origin of Edema</td><td colspan="5">Respiratory failure not fully explained by cardiac failure or fluid overload</td></tr>
<tr><td>Chest Imaging</td><td colspan="5">Chest imaging findings of new infiltrate (s) consistent with acute pulmonary parenchymal disease</td></tr>
<tr><td colspan="6">Severity classification</td></tr>
<tr><td rowspan="4">Oxygenation</td><td colspan="2">Non Invasive mechanical ventilation</td><td colspan="3">Invasive mechanical ventilation</td></tr>
<tr><td colspan="2">No severity classification</td><td>Mild
4≤ OI < 8
5≤ OSI< 7.5</td><td>Moderate
8≤ OI <16
7.5≤OSI<12.3</td><td>Severity
OI >16
OSI>12.3</td></tr>
<tr><td>Nasal mask CPAP or BiPAP</td><td>Oxygen via mask, nasal cannula or High Flow</td><td colspan="3" rowspan="2">Oxygen Supplementation to maintain SpO_2> 88% but OI< 4 or OSI <5[1]</td></tr>
<tr><td>FiO2 > 40% to attain SPO2 88- 97 %</td><td>SPO2 88-97% with oxygen supplementation at minimum flow [2]:
▪ <1 year: 2 L/min
▪ 1-5 years: 4 L/min
▪ 5-10 years: 6 L/min
▪ >10 years: 8 L/min</td></tr>
</table>

***N.B**: OI=oxygenation index= (FiO2*mean airway pressure*100)/PaO2. OSI=oxygen saturation index= (FiO2*mean airway pressure*100)/SatO2.*

Causes and clinical features of pARDS

Causes of ARDS are many and may be local (pulmonary) or remote (systemic)

Clinical pictures may vary according to the causes of ARDS

- Increased work of breathing, tachypnea and hypoxemia are universal findings

II. Management of ARDS

- **Noninvasive positive pressure ventilation (NPPV):** use for hemodynamically stable less severe patients.
- **Invasive ventilation**: **Lung protective mechanical ventilation**:
  - Low tidal volume: 4-8 mL/kg predicted body weight (PBW) with plateau pressure ≤30 cmH20
  - Permissive hypercapnia
  - Relatively high respiratory rate
  - PEEP =10-15 sometimes up to 15 cmH20 PEEP may benefit in severe ARDS
- Reduce instrumental dead space and use heated humidifier
- Wean Fio2 to <0.6 by optimizing PEEP and permissive hypoxemia
- Prone positioning is not recommended as routine therapy
- Suction cautiously only as indicated
- Use fluid and inotrope to maintain normal cardiac output
- Keep lungs dry (restrict fluid) without compromising organ perfusion
- Adequate analgesia and sedation
- Elevate bed 30 degrees to prevent aspiration and ventilator associated pneumonia
- **Nutrition**: Early enteral feeding
- **Corticosteroids:** use should be individualized
- Treatment of predisposing factor and inter current infection.
- **Recruitment maneuvers**– Recruitment maneuvers should not be used routinely

- In cases of clear derecruitment: (endotracheal aspiration, accidental or planned disconnection), careful recruitment maneuver can be used.
- Brief application of a high CPAP such as 35 to 40 cm H_2O for 40 seconds

- **Early and short neuromuscular blockade**
  - Neuromuscular block should be considered if PaO_2/FiO_2 ratio<150 mmHg.
  - Neuromuscular block should be administered early for no more than 48 hr.

13.2.5 SEVERE PNEUMONIA

Definition: is an inflammation of the lung parenchyma caused by bacterial, viral or fungal infection and characterized by reduction in total capacity and compliance.

Clinical manifestation: Fever, cough, dyspnea, crackles, or presence of an acute infiltrate on the chest radiograph.

Management of Pneumonia

- Provide oxygen if saturation < 90% and increased work of breathing .
- Maintain euvolemia and normal electrolyte balance.
- Invasive and non-invasive ventilation as required.
- Antibiotics: use Augmentin / ampicillin-sulbactam or second generation cephalosporin as first line drugs

13.3 PEDIATRIC CARDIO VASCULAR DISEASES

13.3.1 COMMON DYSRHYTHMIAS

a. **Sinus Bradycardia**: is a rate lower for age of the child (*Table 13-3*).

Most common rhythm seen in pediatric arrest, and leads to asystole if left untreated

Table 13-3: Sinus Bradycardia in Children

Age	Rate (in beats per minute)
Infant - 2 years	<90
Children 2-6 year	<80
Children 6-11 year	<70
Children older than 11 year	<60

Causes: drugs, hypotension, hypoxia, airway suctioning, elevated intracranial pressure, hypoglycemia, hypercalcemia, and acidosis

Management:

- Optimize oxygenation and ventilation.
- Begin CPR if heart rate <60 beats/min with signs of poor perfusion.
- If vagal block or AV blocks is the reason, give atropine 0.02mg/kg Iv (minimum 0.1mg and max 1mg); repeat as needed and consider cardiac pacing.

b. **Sinus tachycardia**: sinus rhythm with a rate greater than normal (age-specific)

Causes: many e.g. fever, stress, anxiety, pain, hypotension, and hypoxia.

Management: treatment of the underlying pathology .

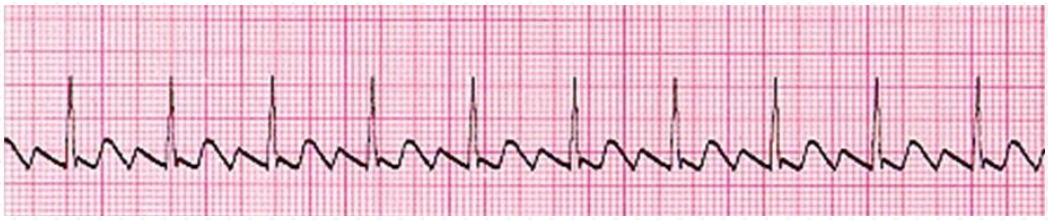
Rhythms Originating from the Atria

a. **Atrial Flutter**

- A rapid, **regular** atrial tachycardia with atrial rates of 200 - 500 / minute. ECG shows saw tooth configuration.

Causes: Sign of significant cardiac pathology except in neonates (could be normal), structural heart disease, myocarditis / pericarditis, atria surgery, digitalis toxicity and thyrotoxicosis.

Figure 13-1: Atrial Flutter

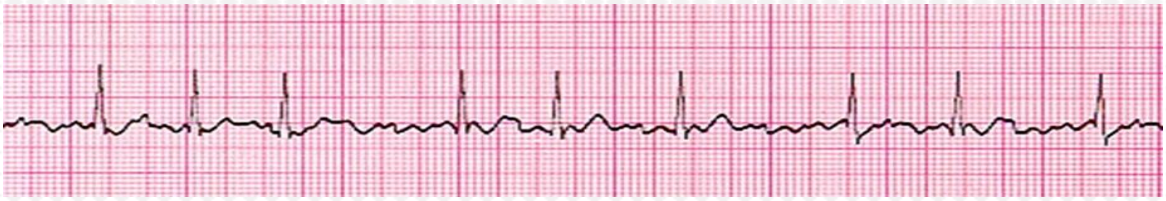


b. Atrial Fibrillation

- A rapid, **irregularly irregular** rhythm with atrial rates of 350 - 600/minute.
- Ventricular response is irregular and significantly slower than atrial rate
- Much less common than atrial flutter except in teenagers.

Causes: e.g. Congenital heart diseases, rheumatic heart diseases, thyrotoxicosis

Figure 13-2: Atrial fibrillation



Management of Atrial Flutter and Atrial Fibrillation

- **Acute conversion**: synchronized conversion as for supra ventricular tachycardia
- **Chronic suppression**: after 48 hours, risk of thrombus; so do echocardiography and start warfarin for 4 weeks. Then cardioversion after 2-3 week.
- **Ventricular rate control** by B blocker or calcium channel blockers.
- Other options; amiodarone, procainamide or surgical ablation.

c. Supraventricular Tachycardia (SVT)

Causes: half don't have heart disease; some have congenital heart disease, Post surgery.

Clinical Presentation: may be asymptomatic, variable presentations. Narrow QRS rhythm.

Differentiating SVT from sinus tachycardia:

- Heart rate is >220 bpm for infants in SVT and> 180 bpm in older children
- P wave difficult to define, 1:1 with QRS

Management of Supraventricular Tachycardia

- Check if patient is stability (hemodynamic unstable and neurologic manifestations)

a. Stable patients:

- **Vagal maneuvers**, ice pack to side of face, rectal stimulation, squatting, turning patient upside down (rarely), Valsalva maneuver; Don't use eye ball massage
- **Adenosine**
  - A dose of 0.1mg/kg (max 6 mg) if no response repeat 0.2mg/kg (max 12mg)
  - If peripheral IV, need to be closest to the central circulation
  - Use 2 syringes with 3 –way stop cock. One syringe for adenosine, 10 ml saline in 2nd syringe to rapidly flush after adenosine administered.

NB: monitor the vital sign and clinical condition.

b. Unstable patients:

- IV adenosine if already IV in-situ and does not waste time.
- Synchronized cardioversion 1 J/kg (with sedation if it doesn't delay procedure); repeat with 2J/kg followed by 4j/kg if no response.
- If unsuccessful or recurrent tachycardia, consider Amiodarone
  - 5mg/kg IV/IO; repeat up to 15mg/kg: Max 300 mg in 30 -60 minutes (slowly if perfusing rhythm).
- If all the above does not work, obtain expert consultation and Procainamide 15mg/kg over 30 - 60minutes or atrial pacing. Don't use Amiodarone and procainamide together.

Complication

- Decreased cardiac output / shock
- Thrombus formation and embolic events

Rhythm originating from the ventricle

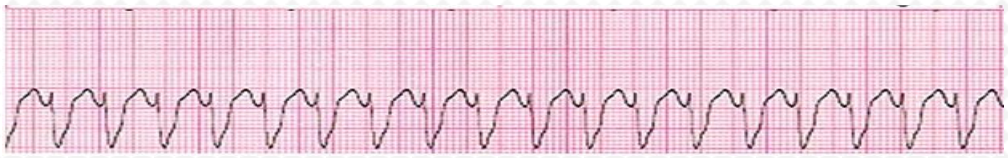
a. **Ventricular tachycardia** is a potentially life-threatening arrhythmia originating below the bundle of His.

- Wide QRS except in infants and small children, in whom the QRS may be narrow but distinct from the sinus rhythm.
- If polymorphic VT in the setting of a prolonged QT interval, the rhythm is "torsade de pointes"

Clinical manifestation: dizziness, syncope, palpitation or chest pain.

Causes: drugs, structural heart diseases

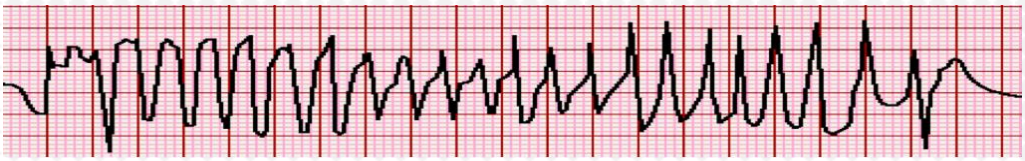
Figure 13-3 : Monomorphic ventricular tachycardia



b. **Torsade's de Pointe: (twisting of the points):**

Is a paroxysm of VT with progressive changes in the amplitude and polarity; **a form of polymorphic VT.**

Figure 13-4: Torsade de pointes



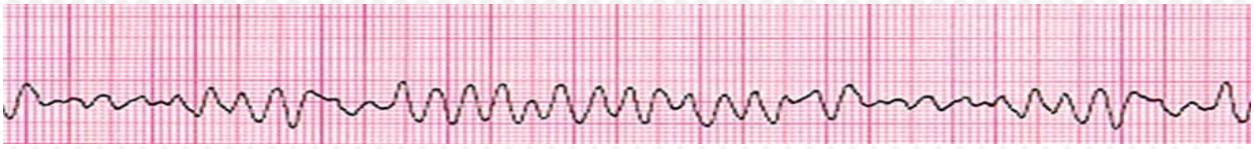
Management

- If hemodynamically unstable and/or altered consciousness, do immediate **cardioversion**.
- Monomorphic VT and Torsade's de Pointe with pulse, manage like unstable SVT.
  - If NO pulse start CPR and give defibrillation 2j/kg; if no response 4J/kg.
  - If this does not work, start Amiodarone or procainamide as above.
- Give magnesium sulphate for Torsade's de Pointe

- Reversible causes of VT, such as hypoxia or hyperkalemia, must be treated promptly

Ventricular fibrilation: is unusual presenting rhythm in infants and children

Figure 13-5: Ventricular fibrillation



Management: start CPR with epinephrine and give defibrillation 2J/kg if no response 4J/kg

Monitoring

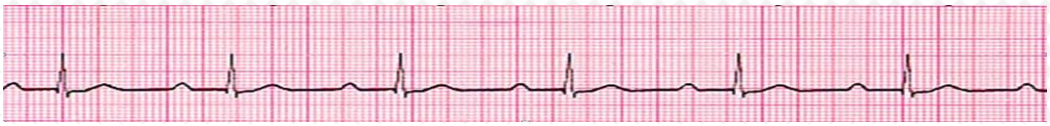
- Though **supraventricular tachycardias** are better tolerated than **ventricular tachycardias**, every patient **should be** carefully evaluated for end-organ perfusion: blood pressure, mentation, and urine output.

Rhythms with Conduction Delays

a. First Degree AV Block

Prolonged PR caused mainly by drugs and no treatment need except removing the cause.

Figure 13-6: First degree AV block



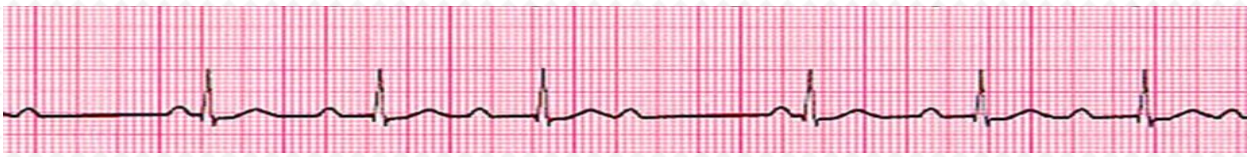
b. Second Degree: All impulses don't transmit to the ventricle that there are escaped beats.

Two types

Second Degree Type I (Mobitz Type I block /Wenckebach phenomenon)

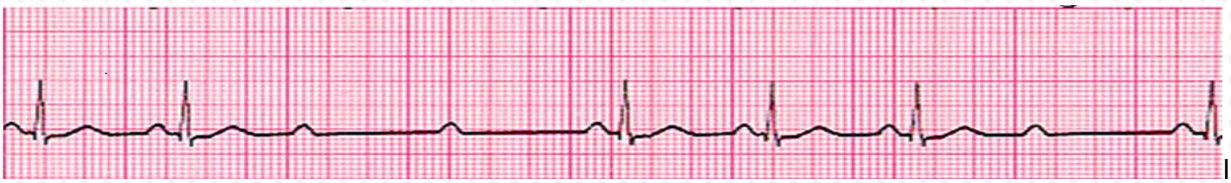
PR interval progressively prolonged until there is an impulse which is not conducted.

Figure 13-7: Second degree Av block type I



Second Degree Type II: Sudden drop of an atrial impulse without increasing length of PR interval. This may progress to complete AV block.

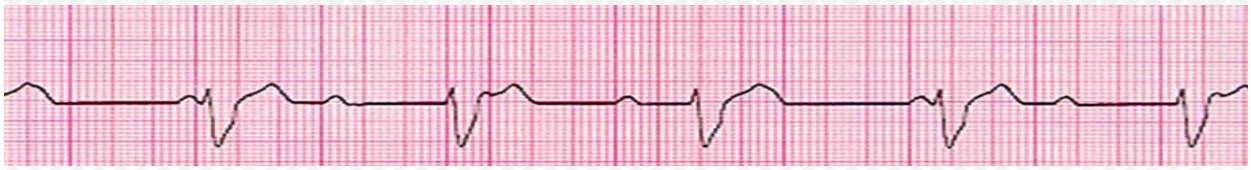
Figure 13-8: Second-degree AV block Type II



c. Third Degree AV Block (complete heart block)

Complete dissociation between the atria and ventricle.

Figure 13-9: Third degree AV block



Management: acute treatment: isoproterenol infusion, atropine and pacemaker is definitive solution.

Asystole: no electrical activity, start CPR with epinephrine

Pulseless electrical activity

- Electrical waves but the patient doesn't have pulse. Start CPR with epinephrine.

13.3.2 CONGESTIVE HEART FAILURE (CHF)

Definition: Failure of the heart to pump blood enough to meet the body's metabolic demand as a result of ventricular dysfunction, volume or pressure overload.

Clinical features:

- Respiratory distress, Tachycardia, Poor appetite, feeding difficulty and growth failure
- Dyspnea, Profuse perspiration, Palpitation, exercise intolerance, orthopnea, raised JVP , hepatomegaly, and edema

Diagnosis

Is based on combination of clinical, imagining and laboratory findings

Investigations

Chest radiograph (Cardiomegaly - cardiothoracic ratio > 0.60 in neonates, > 0.55 in infants, and > 0.50 in older child). Electrocardiogram and Echocardiography

Management principles

a. General measures

- ABCDE: put patient on monitor
- Positive pressure ventilation may be required for pulmonary edema
- Intubation may be required for refractory heart failure.
- Bed rest- recommended but absolute restriction rarely necessary.
- Place patient in semi upright position
- Fluids and Diet- 2/3 of maintenance through NG tube or IV fluid while increasing calorie
- Low sodium formula not recommended as it may exacerbate diuretics induced hyponatremia; Breast milk is an ideal low salt diet.

b. Control of heart failure states

1. Decrease congestion

Diuretics: first line to decrease preload.

Furosemide: IV 1 mg/kg up to 2 mg/kg/dose. 1 mg/kg Po dose BID up to max 6 mg/kg/day

- Check urine output after 2 hours of administration
- Change to IV infusion if there is inadequate response at 0.1 mg/kg/hr titrate progressively to 1 mg/kg/hr.
- Spirolactone: - 1- 2mg/kg/day
- If no improvement adds:
  - Hydrochlorothiazide: 1 mg/kg/hr. oral every 12 hourly

2. Decrease afterload and prevention of cardiac remodeling

- **ACE inhibitor:**
  - Monitor BP and renal function
  - Indications: Cardiomyopathies, mitral and aortic insufficiency, left -right shunt lesions
  - Contraindicated in obstructive lesions
  - Captopril, oral, initial dose: 0.5 – 1 mg/kg/24 hrs in 3 doses (8 hourly) for 24–48 hours. Increase by 0.5 mg/kg/24 hours every 24–48 hours until maintenance of 3–5 mg/kg/24.
  - Enalapril oral 0.2-1 mg/kg/day single dose or two divided doses. Start low and increase by 0.2 mg/kg/day at 1-2-day interval.
- **B blockers:**
  - Generally, not used in acute decomposition
  - use especially in ischemic heart disease
    - Metoprolol: 0.1 mg/kg dose BID up to max 1 mg/kg do
    - Carvedilol: 0.025 mg/kg/dose BID up to max 0.5 mg/kg/dose BID

3. Increase contractility

Digoxin- add for persistent symptoms despite treatment with other agents (diuretics and ACE I).

Dose: 3-5 microgram/kg/dose oral 12-24 hourly

c. Treatment of precipitating factors

- Blood transfusion for heart failure with hemoglobin < 8 mg/dl
- Treatment of infections: infective endocarditis, pneumonia, malaria etc.
- Treatment of arrhythmias and other precipitants

d. Elimination of underlying cause

- Surgical or catheter based interventions to correct underlying structural defect.

13.3.3 HYPERTENSION IN CHILDREN

Definition

Table 13-4: Stage of Blood pressure

For Children Aged 1-13 years	For Children Aged ≥13
Normal BP:<90 percentile	Normal BP: <120/80mmhg
Elevated BP: ≥90th percentile to <95th percentile Or 120/80 mmhg to <95th percentile (whichever is lower)	Elevated BP: 120/<80 to 129/<80mmhg
Stage 1: ≥95th percentile to <95th percentile +12 mmhg Or 130/80 to 139/89 mmhg (whichever is lower)	Stage 1:130/80 to 139/89 mmhg
Stage 2: ≥95th percentile + 12 mmhg Or ≥140/90mmhg (whichever is lower)	Stage 2: ≥140/90 mmhg

Causes:

- **Primary HTN**: No underlying cause identified.
- **Secondary HTN**: Identifiable cause present. 98% pediatric hypertension is secondary
  - **Renal /renovascular:** the most common cause
  - **Endocrinologic disease**: thyroid, parathyroid, and adrenal diseases
  - **Cardiovascular:** Coarctation of the aorta
  - **Nervous system:** increased intracranial pressure, Autonomic dysfunction (eg. tetanus, Guillian Barrie syndrome).
  - **Tumors:** wilms tumor , neuroblastma , pheocromocytoma
  - **Miscellaneous:** Chronic upper airway obstruction, hypercalcemia, drugs e.g. steroid

Clinical manifestation:

- Seizure, altered mentation, headache, dizziness, visual disturbance, tachycardia/palpitations, congestive heart failure

Hypertensive crisis: presentation with hypertensive urgencies and emergencies

- **Hypertensive emergency:** rare in children; hypertensive encephalopathy, renal insufficiency, eyes (papilledema, retinal hemorrhages, exudates), and heart failure.
- **Hypertensive urgency:** severe elevation in BP without severe symptoms

Management

- Treatment goals are to manage hypertensive emergency and achieve BP level that targets to reduces risk for target organ damage.
- Indications for pharmacologic therapy include:
  - Severe symptomatic hypertension with crisis
  - Stage 2 hypertension without a modifiable risk factor
  - Hypertension in patients with comorbidities such as diabetes (types 1 and 2) or CKD

Treatment

- Admit hypertensive emergency to PICU ; ABCDE
- Continious BP monitoring (eg, automatic BP measurements every minute).
- Medications that increase BP (eg, ketamine) should be avoided during intubation.
- Treat seizure with benzodiazepam
- A stepwise reduction in BP, reduced by no more than 25% in the first 6 to 8 hours ; plan to normalize BP over 3 to 4 days.
- Except in children with intracranial hemorrhage, ischemic or traumatic brain injury, manage hypertensive emergency by initial IV bolus of labetalol or hydralazine.
- Subsequently, continuous infusion of labetalol with strict monitoring of BP
- In hypertensive urgency, oral agents such as nifedipine, unless oral medications are not tolerated

Table 13-5: Drugs for management of hypertensive emergency in children 1–17 year's old

Drug	Class	Dose	Route	Comment
Esmolol	b-blocker	100–500 mcg/kg/min	iv infusion	Very short-acting—constant infusion preferred. May cause profound bradycardia. Produced modest reductions in BP in a pediatric clinical trial.
Hydralazine	Vasodilator	0.2–0.6 mg/kg/dose	iv, im	Should be given every 4 hours when given iv bolus.
Labetalol	α- and b- blocker	bolus: 0.2–1.0 mg/kg/dose up to 40 mg/dose infusion: 0.25–3.0 mg/kg/hr	iv bolus or infusion	Asthma and overt heart failure are relative contra- indications

Table 13-6: Drugs for hypertensive urgency

Drug	Dose	Onset of action Maximum effect duration of action	Side effect
Nifedipine	0.25-0.5mg/kg PO Maximum dose: 10 mg /dose. 3 times every 30 min. Max.dose:3mg/kg/dia–120mg/day	Onset 5min Max.effect: 30-60min Duration: 4-6h	Headache, tachycardia, hypotension, rash. Contraindication: Shock, advanced aortic stenosis, Encephalopathy/cranial hypertension
Enalapril	5-10 mcg Kg IV in 5 min. Maximum: 0.5 mg dose Maintenance: Every 6-8 hour. 0.08 mg/kg/dose 24h. PO Max. dose 5 mg	Onset:1 min Max. Effect: 4h Duration: 6 h	Hypotension
Hydralazine	0.1-0.2mg kg dose IV, IM Max. dose: 20mg dose	Onset: 10-20min Max. Effect: 20-40min Duration 3-8 hr	Hypotension, headache tachycardia

13.3.4 SHOCK IN PEDIATRICS

Introduction

Shock is the inability of the circulatory system to provide adequate oxygen and nutrient to meet the metabolic demand.

Categories of shock

i. **Severity class:** compensated and hypotensive shock

Compensated shock: BP maintained by compensatory mechanism and normal mentation

Hypotensive shock: late phase of shock when physiological compensation fails.

***NB**: there is hours before patient goes to decompensated shock; once the patient is in decompensated shock the patient will go to cardiac arrest soon.*

ii. **Based on causes;** four types: Hypovolemic, distributive, cardiogenic, and obstructive shock

a. **Hypovolemic Shock**

- External fluid loss like hemorrhage, diarrhea, vomiting, Diabetic, burn.
- Internal third space loss: malnutrition, nephrotic syndrome, chronic liver diseases

Clinical manifestation:

- Signs of shock +/- sign of dehydration e.g. sunken eye, delayed skin pinch

Management of hypovolemic shock

- Recognize early, maintain airway, breathing

For well-nourished child:

- 20 ml/kg NS/RL fast then reassess and if no improvement repeat 20ml/kg and reassess; Give third bolus if no improvement until signs of adequate perfusion. If hypovolemic shock due to diarrhea may need more bolus eg in cae of cholera
- Consider septic shock if no response after adequate bolus

NB: After patient out of shock assess for dehydration and manage with 70ml/kg of NS/RL over 5 hours (less than 1 year) or 2 and ½ hour (age 1 year).

For sever acute malnutrition:

- 5% dextrose in NS or 5% dextrose in ringer lactate 15 ml/kg over 1 hour with close monitoring of vital sign.
- Replace ongoing loss with ORS (10ml/kg for each loss) if well-nourished or with ReSoMal if the child has malnutrition.
- If the shock is from bleeding, control bleeding by direct pressure, give 20ml/kg of NS/RL, repeat 2 -3 as needed and prepare blood for possible transfusion.

b. Cardiogenic shock

Cardiogenic shock is failure of the heart as a pump, resulting in decreased cardiac output.

Clinical manifestation:

- Lethargy, Poor feeding, Tachycardia, and tachypnea,
- Pale, cold extremities and barely palpable pulses
- Can be **difficult to differentiate cardiogenic and septic shock**.
    - Findings specific to cardiogenic shock: - gallop, Rales, Jugular venous distension, hepatomegaly, cardiomegaly and pulmonary congestion.

Management of Cardiogenic shock

- Follow bradycardia /tachycardia algorism if cause is rhythm problem
- 5 to 10ml/kg NS/LR bolus, repeat PRN for structural causes
- Inotrope infusion (adrenaline or dopamine or Miliron)
- consult expert early

c. Distributive shock

Due to capillary leak, vasodilation or maldistribution of blood flow. Occurs in neurogenic, septic and anaphylactic shock.

1. **Neurogenic shock:** result of loss of vascular tone from cervical cord or upper thoracic spinal injury.

Classic picture of neurogenic shock is hypotension without tachycardia or cutaneous vasoconstriction and respiratory muscle weakness.

Management of neurogenic shock

Fluid as for hypovolemic shock and if fail to improve raise BP by vasopressors (Dopamine, epinephrine) infusion.

2. **Anaphylaxis shock**

It is severe allergic reaction, which may cause airway obstruction and shock. This can occur in the ICU following blood and drug administration.

Clinical manifestation: rash, wheeze, airway obstruction, stridor, swelling of lips and tongue

Management Anaphylaxis shock

- Adrenaline 0.01 mg/kg IM (1:1000)/0.01 ml/kg (max 0.5 mg) in the thigh; repeat every 5–15 min for persistent or recurrent symptoms.
- If complete airway obstruction call anesthesiologist for immediate intubation or tracheostomy in parallel to the rapid IM administration of epinephrine.
- Do emergency cricothyroidotomy if intubation delayed.
- If patient is not breathing or pulseless, start CPR
- Administer 100% oxygen with bag-valve-mask ventilation as indicated.
- Salbutamol using a MDI and spacer or nebulization if wheezing.
- Diphenhydramine 1 mg/kg IM or IV (max 50mg). Preferred orally if patient stable. Consider cimetidine (5 mg/kg PO; maximum 300 mg), may work synergistically.
- 20ml/kg of iv /IO NS if the child in shock and repeat as needed
- Systemic steroid: dexamethasone 2 mg/kg PO/IV (max 50 mg) or prednisolone 1-2 mg/kg PO (max 80 mg).

3. **Septic Shock**

It is sepsis + (cardiovascular dysfunction) evidenced by persistent hypotension or need for vasopressors despite adequate fluid resuscitation.

Clinical manifestation

- Hypotension or need vasoactive medication to maintain BP
- Unexplained metabolic acidosis, base excess (BE) >-5 or lactate >twice upper limit
- Decreased urine output <0.5ml/kg/hr.
- Capillary refill >5 sec
- Core to peripheral temperature gradient >3℃. Hypo or hyperthermia; altered mental status, cold (vasoconstriction) or warm (vasodilation).

Diagnosis of Septic shock: is clinical and laboratory has limited utility.

Management of septic shock

- Manage ABCDE
- Assess if child has malnutrition (Manage malnourished child as above)
- 20 ml/kg RL or NS and assess response: If no response repeat up to 20ml/kg up to total of 60ml/kg
- If the patient hypotensive give fluid rapidly. If patient in compensated state give fluid over 30 minutes.
- Inotrope if no response to 3 boluses of fluid. Preferred is adrenaline / Noradrenaline. Use dopamine if adrenaline not available.
- Blood transfusion: consider in patients with refractory shock.
- Broad spectrum antibiotic within first hour of diagnosis after taking blood culture.
- Narrow antibiotic based on culture result. Identify focus and control source e.g. abscess drainage.

Metabolic and electrolyte correction:

- Frequently check and correct hypoglycemia. If persistent add steroid beside 10 % dextrose maintenance.

- If persistent hyperglycemia with dextrose free fluid, give low dose insulin 0.05-0.1 units /kg/hr with hourly monitoring of blood glucose
- Correct hypocalcaemia < 0.8mmol/l

Steroids

- Only in inotrope refractory shock and /or persistent hypoglycemia and patients who are on steroid previously
- Dose hydrocortisone 1-2mg/kg then 1 mg/kg QID

d. Obstructive shock

Caused by obstruction of blood flow either into or out of the heart.

- Cardiac Tamponade
- Tension pneumothorax
- Duct dependent lesions (e.g. coarctation of aorta, aortic stenosis)

Management of obstructive shock

Table 13-7: Obstructive shock management

Duct Dependant (LV outflow obstruction)	Tension Pneumothorax	Cardiac Tamponade	Pulmonary Embolism
Prostaglandin E1 Expert consultation	Needle decompression Tube thoracostomy	Pericardiocentesis 20 ml/kg NS/LR bolus	20 ml/kg NS/LR bolus, repeat as needed Consider thrombolytics, anticoagulant

Monitoring of shock

- **Pulse**: assess the rate, volume, rhythm:
- **Blood pressure**
- Check capillary refill
- Level of consciousness, and urine output
- Signs of fluid overload (pulmonary edema or new or worsening hepatomegaly that should limit further fluid bolus therapy)

13.4 FLUID THERAPY IN INFANTS AND CHILDREN

I. Introduction and Principles of Management

Maintenance fluid (MF) represents the quantity of IV fluids required to maintain hydration in an euvolumic child where the child is not experiencing ongoing lose e.g. diarrhea, burn etc.

Maintenance intravenous (IV) fluids are used in a child who cannot be fed enterally.

Goals of Maintenance Fluids:

- Prevent dehydration
- Prevent electrolyte disorders
- Prevent protein degradation
- Prevent ketoacidosis

Glucose in MF provides approximately 20% of the normal caloric needs

II. Suggested Management

Table 13-8: Fluid therapy

Weight	Fluid therapy /day	Hourly fluid rate	Remark
0-10kg	100ml/kg	4ml/kg/hr	
11-20kg	1000ml +50ml/kg for each kg >10kg	40ml/hr+ 2ml/kg/hrx(Wt-10kg)	
>20kg	1500ml +20ml/kg for each kg >20kg	60ml/hr+ 1ml/kg/hrx(wt-20kg)	Maximum fluid per day =2.4 Liter

For example: A 26 kg child who is NPO waiting for surgery: 60ml/hr +1ml/kg/hrx(wt-20kg) : 60ml/hr+1ml(26-20)=66ml/hr of iv fluid is needed.

The type of fluid used for maintains fluid:

- It should be a solution with 0.45% saline with 5% dextrose or normal saline with 5% dextrose (DNS). A solution less than 0.45% NS should not be used except for the neonates. Potassium in replacement therapy should wait until the patient has adequate urine output
- Resuscitation fluid: is crystalloid (NS, RL) 20ml/kg after 60ml/kg consider colloid see section 13.3 for pediatric shock

NB: the neonates' fluid requirement is not included in this guideline.

- Maintenance may be increased or decreased depending on the clinical situation.
  - Excessive sweating ,profuse diarrhea, vomiting , radiant warmer, phototherapy, fever ,surgical drain /third spacing need increase MF
  - Humidified mechanical ventilator decreases insensible loses

In children receiving more than 50% MF receiving through IV electrolytes should be determined at least daily.

III. Management of Complications

- Fluid overload: restrict fluid intake and consider diuretics
- Extravasation: examine IV site daily and change the site if there is any swelling, limb elevation
- Compartment syndrome (limb): elevate the limb, remove IV line on the limb, rarely fasciotomy
- Electrolyte and acid base disturbance: correct accordingly (see electrolyte imbalance and acid base disturbance on chapter 4 section 4.3)
- Hypo/hyperglycemia: give glucose/correctional insulin

IV. Monitoring and Problem Solving

- Vital signs and urine output-every 1hourly
- Examine IV sites-atleast daily
- Monitor RBS every 4-6hourly
- IVC assessment with ultrasound-every time the child require bolus of fluid and at least once during every shift

13.5 ELECTROLYTE THERAPY IN INFANTS AND CHILDREN

I. Introduction and Principles of Management

Estimates of the normal requirements for major electrolytes are several times greater than actual minimum requirements; however, vigorously anabolic children may have even greater requirements.

Normal Daily Electrolytes

- Na+ and cl- =2-3 meq /100ml of Maintenance fluid /day
- K+=1-2meq/100ml of Maintenance fluid /day
- Ca+ = 700–1300 mg/day
- Mg++ = 0.3–0.5 mEq/kg/day

II. Suggested Management

a. Sodium imbalance

1. Hyponatermia

- It is a serum sodium level <135 meq/L (<135mmol/L).
- Initiate treatment required immediately if neurologic symptoms and sodium is <120.Give 3 % saline preferably through central line. The administration is over 15 to 20minutes to gain rapid correction.The target being 120-125meQ/L or until seizure stops.1.2ml/kg of 3 %saline will raise the level by 1mEq/L.
- Correct hyponatremia slowly. Rapid correction of sodium more than 12 mEq/L in the first 24 hours or an average of 0.5 mEq per L/hr. Except in neurologic symptoms or typically with sodium level <120 mmol/L where rapid correction required.
- Formula to get required sodium: 0.6 x(weight in kg)x(target Na-measured Na) and use NS or D5NS fluid to correct.
- If **hypovolemic** and hemodynamically unstable: correct instability with NS boluses (20 mL/kg) over 5 min followed by reassessment after each bolus. For **euvolemic**

hyponatremia, after correction of serum sodium level, institute water restriction and treat the underlying disorder. For **hypervolemic** hyponatremia (edema) institute sodium and water restriction, and administer diuretics if needed to treat the clinical condition (e.g., congestive heart failure).

Example: A 7 week old child (4kg) is symptomatic with Na=114

Step 1: This child needs urgent correction since Na<120 amount of Nacl required is:

0.6x (wt in kg) x (target Na-measured Na)

0.6x4x (120-114) = 14.4mEq of Na needed.

1 ml 3% saline is 0.5 mEq/L or in order to get 14.4meq we need 28 ml of 3 % saline

Or NB: 0.9% NS =0.15meqNa/L but 3 % saline= 0.5mEqNa/L.

If 3% saline not available give NS fluid bolus 20ml/kg

Step 2: we need to raise the serum sodium level at additional 12 meq over the next 24 hours from the current 120meq/L

= 0.6 x 4 kg (132 desired Na-120 Na)= 29Meq/L of additional sodium needed over the next 24hurs.

NB: 1teaspoon of table salt (NaCl) has 2.3gm of Na+.

1mg NaCl has 17mEqNa+. 1mgNa+ has 43mEqNa+. Normal saline has 0.9gm of NaCl/100ml.

3%NaCl has3gmNaCl/100ml

Table 13-9: Summary of management of hyponatremia

Symptoms	Treatment
If hypovolemic and hemodynamic ally unstable	Correct instability with NS boluses(20ml/kg over 5min followed after reassessment in each bolus)
Symptomatic	Correct deficit to normal over 48h mEq Na required=[(Na $^{+}$desired)-Na+)]x(0.6xweight in kg)
Neurologic symptoms (altered mental status, seizures)	1 - 2 Ml/kg/h of3% sodium chloride until asymptomatic or Na level > 120mEq/Ml ,then increase Na level 0.5mEq/Ml/h (not to exceed increase of 12 m Eq/ml in first 24h or 18mEq/m in first 48 h)

2. Hypernatermia

Goals of treatment:

i. To quickly correct underlying hypovolumia
ii. To treat the underlying cause of hypernatremia eg diabetic insipidus
iii. Third to carefully lower the serum sodium level, usually by replacement of the body's total water deficit)

Treatment of hypernatremia

- Serum sodium levels of >160 mEq/L require immediate attention .
- Correct serum sodium gradually to avoid cerebral edema and associated central pontinemyelinolysis. Therefore, closely monitor serum sodium levels every hour initially to ensure that the level is reduced no faster than 1 mEq/L/h and no more than12 mEq/L in the first 24 hours. This may require more than 48 hours for complete correction.
- Resuscitate with NS if the patient has shock 20ml/kg till it is corrected. Avoid Ringer lactate because it is more hypotonic than NS and may cause to rapid correction decrease in serum sodium concentration .
- Estimate free water deficit by one of the following methods and correct dehydration over 48-72 hrs:
  - 0.6x patient weight in kg x (patient sodium/140 -1) or 3-4mlx desired change in serum sodium x body weight (kg). If serum sodium is greater than 170 mEq / L: 3 mL of water / kg is needed to decrease serum sodium by 1 mEq / L. and, for sodium less than or equal to 170 mEq / L: 4 mL of water / kg of weight is required.
  - Use maintenance + deficit of fluid + replace also ongoing lose.
  - Type of fluid: correction of shock with isotonic saline should be followed by ½ normal saline (0.45%) with 5% dextrose (with potassium) in order to replace the remaining free water deficit. Typical fluid: 5% dextrose + half-normal saline (with 20 mEq/L KCl unless contraindicated)
  - Typical rate: 1.25-1.5 times maintenance
- Frequent measurement, initially every 2-3 hours. If the correction rate is not adequate, the rate or type of fluid should be changed

- The maximum rate of sodium fall: 10 to 12 mEq/L/day. Monitor serum sodium every 2 to 4 hours, later every 6-8 hours if the correction speed is adequate if possible (if Na falls≥ 0.6 -1 mEq /hr. slow IV rate.
    - Sodium decreases too rapidly; either: Increase sodium concentration of intravenous fluid or decrease rate of intravenous fluid. If Na falls < 0.5 mEq/hr increase the flow IV rate or are imminent, consider a short infusion of 3% Nacl (2-3ml/kg) over 1-2 hours.
    - Sodium decreases too slowly; either: decrease sodium concentration of intravenous fluid or increase rate of intravenous fluid.
    - Replace ongoing losses as they occur in the chronic conditions, without exceeding in both cases a correction> 10-12 mEq / l / day.

      NB: 1L of D5 0.45% normal saline will provide 400ml of free water .To prepare D5 0.45% normal saline (Use 1/2 DW and 1/2 DNS fluid in our setting)

b. Potassium imbalance

1. Hypokalemia

Classification: mild (k=3-3.5mEq/l); Moderate (k= 2.5 -2.9 mEq/l); sever (k<2.5 mEq/l)

Management

2. Check renal function test as we need to be careful of potassium administration in renal failure.
3. Treat underlying causes eg reduce dose of diuretic
4. Use oral or IV route depending on the severity
5. Oral is safe and preferred for asymptomatic and normal ECG. Oral replacement with potassium, 2 to 5 mEq/kg/day ever 4 to 6 hour
6. If symptomatic or ECG abnormal If K<2.5 and life treating complication arrhythmias or respiratory muscle weakness use IV route 0.5 -1 mEq kg(maximum 20 mEq/dose) in 1 to 2 hour with adequate solution dilution with normal saline preferably with central line . If concentration is more than 60 meq/L use central line as it is vein irritant. Concentrated IV potassium infustion is required unless patient is in renal

failure. Usually Potassium 0.2 to 0.3 mEq/kg/h is generally adequate. Administered, with continuous ECG monitoring.

7. Add on maintenance fluid Kcl 40 to 60 meq/L in such patient
8. In diabetic ketoacidosis, potassium repletion should begin early in the course of therapy, (see section 13.7 for DKA)
9. *When hypokalemia is resistant or refractory to K+ replacement, magnesium deficiency is the likely treat hypomagnismia.*
10. **Hyperkalemia**

- Hyperkalemia is a serum potassium level of >5.5 mEq/L.

Management

- Any patient with ECG abnormality, K>6.0-6.5 meq/L, associated acidosis, hypocalcaemia, hyponatermia need emergent therapy.
- Asymptomatic patients with normal ECG findings usually do well with therapy to enhance potassium excretion.
- Sodium polystyrene sulfonate is a resin that exchanges sodium for potassium administered PO or by enema. A dose of 1 gram/kg lowers the serum potassium level by up to 1.2 mEq/L. when administered PO it is usually given with a cathartic to speed transit time through the GI tract. Hypernatremia and volume overload are potential complications.
- In patients metabolic acidosis, normalization of serum pH usually restores serum potassium to normal levels.

Table 13-10: Summery of Treatment of hyperkalemia

	Agent	**Dose**
Increase cardiac stability	Calcium gluconate, 10%*	100milligrams/kg(1ml/kg/dose) Iv at rate not to exceed 100 milligrams /min; onset of action, minutes; duration of action, 60 min maximum 3 grams / dose. Can be administered peripherally.

Decrease Potassium	Salbutamol	Nebulization or meter dose onset of action is in minutes
	Sodium Bicarbonate	1-2 mEq/kg/IV/Io; onset of action is in minutes ; duration of action in hours
	Regular insulin with glucose	0.1 units /kg Iv in 5ml/kg of 10 % dextrose in water , 0.5 gram /kg Iv over 30 min; check glucose level every 30 min ; onset of action 30 min .May e repeated every 30 to 60 min
	Sodium polystyrene sulfonate	1-2 grams /kg Po , via nasogastric tube , or pere rectum

c. Hypocalcemia

Hypocalcaemia is a serum calcium level <8 mg/dL (2 mmol/L) or ionized calcium level <1 mmol/L. Total calcium can be affected by the level of abumin therefore better to follow ionized calcium.

Treatment

- Asymptomatic can be treated with po calcium 50mg/kg /day in 3-4 doses
- For ICU patient preferred dose of Iv calcium 0.5ml/kg of 10% calcium gluconate diluted in an equal amount of saline over 10 minute or calcium gluconate 10% in a dose of 100 milligrams/kg at a rate not to exceed 100 milligrams/min, Stop if the ECG or if the heart rate drop by 20 from base line
- Correct abnormal magnesium, potassium and PH. Hypomagensemia causes refractory hypocalcemia.
- Treat underlying cause eg treat rickets

Table 13-11: Treatment of hypo and hyper calcium and magnesium

	Treatment	**Comments**
Hypocalecemia	10% calcium gluconate Iv ,100milligrams /kg at a rate <100milligrams/min	Continues ECG monitoring
Hypercalcemia	Hydrate with twice maintenance fluids , furosemide 1-2 milligrams/kg iv to a maximum of 40 milligrams	Treat underlying cause

Hypomagnesemia	10% magnisum sulfate ,25-50milligrams /kg over 30 mints	
Hypermagnesium	Hydration ,diuresis 1-2 milligrams/kg Iv furesamide to maximum 40 milligrams ;or 10% calcium gluconate iv ,0.5 ml/kg	

III. Management of Complications

Further electrolyte imbalance

- Stop administration of electrolyte once the imbalance is corrected or for over correction

Neurologic squeale

- Continue supportive care and consider mannitol if cerebral edema is suspected
- Avoid extra administration of electrolyte

Extravasation and tissue necrosis

- Stop and ministration and change IV site with possible bigger cannula or central line if available

Arrhythmia

- Stop further administration
- Give binder/membrane stabilizer if indicate (eg: Ca gluconate for hyperkalemia and hypermagnesemia, Phostat for hyperphosphatemia)
- Manage arrhythmia with AHA guideline

Acid base disturbance

- Correct accordingly (see under acid base disturbance in Chapter 4 section 4.4)

IV. Monitoring and problem Solving

- Vital signs and urine output- every 1hour
- ABG-every 6hourly if available
- ECG -continuous

13.6 PEDIATRIC NEURO-CRITICAL CARE

13.6.1 STATUS EPILEPTICS IN INFANTS AND CHILDREN

I. Introduction

Status epileptics are continuous or recurrent seizure activity without regaining consciousness and lasting for more than 5 min.

Causes

Electrolyte abnormality, hypoglycemia, head trauma, brain infection; stroke, hypoxic–ischemic insult, hypertensive, renal or hepatic encephalopathy; brain tumors etc.

II. Treatment

Initial treatment Algorithm

- Manage ABCD
- suction if vomiting / excess secretions ,put collar if trauma
- Do not insert anything in the mouth unless child cyanosed.
- Avoid long acting muscle relaxant if intubation is required

Termination of status epileptics

- Diazepam 10mg /ml IV 0.25mg/kg (0.05 ml/kg max 20 mg) IV, or per rectum as 0.5mg/kg (0.1 ml/kg). (Use IV cannula without needle, NG tube and insert 4 to 5 cm in the rectum).
- Check blood glucose; If hypoglycemia or not able to determine give 5ml/kg D10% IV stat.
- If still convulsing after 5-10min, give second dose of diazepam; If still seize consider third dose of diazepam and Phenytoin 20mg/kg IV/IO over 20 min (1mg/kg/min) max of 50 mg / minute
    - In patients taking phenytoin chronically, use initial dose of 5-10 mg/ kg unless serum level is very low.
    - If child still seizing give additional 10 mg / kg of phenytoin IV.

- Add Phenobarbital 20 mg/kg over 20 minutes (max 300mg) if phenytoin is not effective or contraindicated (allergy, known therapeutic level).

Refractory status epileptics

- IV bolus followed by continuous infusion of midazolam, propofol, thiopental or ketamine can be used.
- Thiopental 3 to 5 mg/kg bolus, followed by boluses of 1 to 2 mg/kg every 3 to 5 minutes until a clinical response is achieved, maximum total dose of 10 mg/kg. Thereafter, infuse at a rate of 3 to 5 mg/kg/h.
- Propofol at 1-2mg/kg loading and 20mcg/kg/minute titrated to 5-10mcg/kg/min; be caution as it may lead to propofol infusion syndrome.
- Ketamine is an alternative with 0.5-2 mg/kg loading then continuous infusion. Often, therapies are maintained for 1 or more days before it is gradually tapered, usually over a few days. orNon convulsive status epilepticus may occurs after refractory seizure; suspect if subtle muscle jerks, eye deviation, or abnormal eye movements.

Laboratory studies

- Glucose, electrolyte, CBC, blood and spinal fluid cultures, toxic screens, tests for inborn errors of metabolism, anti-epileptic drug levels, Lumbar puncture, toxicologic screen, CT or MRI based on clinical scenario
- **EEG**: type of status epilepticus and monitoring response; detect non-convulsive seizure.

III. Monitoring and Complication

- Phenytoin rapid infusion may lead to hypotension and cardiac dysrhythmias that cardiac monitoring is required.
- Phenobarbital may cause significant sedation and hypotension and respiratory depression that may need intubation.

13.6.2 MANAGEMENT OF INCREASED INTRACRANIAL PRESSURE (ICP)

I. Introduction

Increased ICP occurs in many serious neurological conditions.

Causes of ICP: Abnormal increase (e.g. infections, blood, tumor) in CSF, blood flow or brain

Clinical manifestation

- Headache, vomiting, depressed level of consciousness, hypertension +/- bradycardia.

Neuroimaging

If no obvious metabolic /toxic cause for coma, child should get a CT +/- MRI.

NB: Normal CT scan doesn't rule out raised ICP

II. Management of ICP

Goal is to maintain appropriate cerebral perfusion pressure (CPP): Anything which affects ICP or MAP will affect CPP.

a. Address ABCDE

- Treatment of hypoxia, hypercarbia, hyperthermia, and hypotension
- Bed elevation to 30 degrees with neutral position, not > 40 degrees

Airway

Indications for endotracheal intubation include:

- GSC of ≤ 8 or rapid decrement of GCS by 3 or more from baseline
- Acute herniation requiring controlled hyperventilation

Medications for Rapid sequence intubation during ICP

- **Lidocaine IV**, or as local anesthetic prevents ICP surges.
- **Etomidate** generally favored sedative for its rapid onset action and minimal side effects

- **Thiopental** can be used for hemodynamically stable patients.
- **Benzodiazepines** may provide cerebral protection, use in hemodynamically stable seizing patients
- **Ketamine** may increase MAP and ICP but could be considered in trauma patients.
- **Rocuronium** is preferred for paralysis in brain tumor as succinylcholine may increase ICP.

For intubated patient

- Avoid high PEEP, may increase intrathoracic pressure and impede venous drainage
- Deep sedation with continuous infusion of diazepam/midazolam with Morphine/Fentanyl.
- Neuromuscular blockade if ICP remains elevated despite adequate sedation
- **Lidocaine** (1 mg/kg IV) or fentanyl (1-2 mcg/kg) before ETT suction (be careful of cumulative dose to avoid toxicity)

Breathing

- Maintain $PaCO_2$ between 35 - 40 mmHg unless signs of impending herniation.
- Aggressive hyperventilation is reserved for acute brain herniation or ICP that fail to respond to other management

Circulation

- Treated hypovolemia with isotonic fluids and avoid hypotonic fluids
- Don't treat hypertension with anti-hypertensive medications.

Disability **and E**xposure

- Treat all seizures; **prophylactic phenytoin** to high risk patients (parenchymal abnormalities, depressed skull fractures, or severe traumatic brain injuries).
- Avoid high temperature.
- Maintaining adequate analgesia and sedation.

b. Specific measures

Mannitol

- 0.25 to 1 g/kg IV bolus then TID or QID.
- Monitor electrolytes and renal function

Hypertonic saline (HTS)

- 3% HTS initial bolus 2 to 6 mL/kg then continuous infusion at rate of 0.1 to 1 mL/kg/hr. Unlike mannitol, HTS does not cause profound osmotic diuresis, and the risk of hypovolemia as a complication is decreased. It maintain efficacy with repeat dosing even in patients who have stopped to respond to mannitol .
- **Complications:** osmotic demyelination syndrome and heart failure have not been reported
  - Renal insufficiency is associated with serum osmolality >320 mOsm/L
  - 1ml/kg of HTS will increase serum sodium by 0.8 meq/l. Maintain Serum Na b/n 145-160 meq/l and serum osmolality 300- 320 mosm/L

c. Surgical management

CSF drainage: Neurosurgical consult in uncontrolled ICP as intracranial drain can be placed to remove CSF.

d. Corticosteroids

- Indicated for vasogenic edema with mass (tumors and abscesses); (**not** in ICP from infarction, hemorrhage, or head trauma)
- **Dexamethasone** 0.25 - 0.5 mg/kg QID, maximum dose 16 mg / day.

III. Monitoring

- Invasive cardiopulmonary monitoring, EEG, and ICP monitoring

13.6.3 TETANUS

I. Introduction

Tetanus is an acute, spastic paralytic illness caused by tetanospasmin neurotoxin produced by Clostridium Tetani.

Clinical Manifestation

Incubation period is 2-14 days and manifests with one of four patterns: Generalized, localized, cephalic and neonatal tetanus.

Sign and symptoms

- Trismus (masseter muscle spasm)
- Risus Sardonicus(sardonic smile of tetanus)
- Spasm and patient remains conscious: Opisthotonos that lasts seconds to minutes and aggravated by sight, sound, touch, manipulation, Laryngeal spasm and asphyxiation.
- Autonomic effects: Tachycardia, dysrhythmias, labile hypertension, diaphoresis and cutaneous vasoconstriction. Fever may reach to-40 °C.

Diagnosis

- It is clinical diagnosis; leukocytosis from secondary bacterial infection or stress. CSF, EEG and EMG are normal.

II. Treatment

a. Supportive care and Spasm control

- Keep Patient in dark quiet room, and decrease stimulus that precipitate spasm
- Cardiorespiratory monitoring, gentle suctioning as required, and maintenance of the fluid, electrolyte, and caloric needs
- **Diazepam** 0.1- 0.2 mg/kg every 3 - 6 hour I.V; subsequently titrated to control tetany, effective dose is sustained for 2 - 5 weeks + chlorpromazine.
- **Neuromuscular blocking agents** — used when sedation alone is inadequate.

b. Halting toxin production

- Optimal wound management to eradicate spores and necrotic tissue
- Antimicrobial- Metronidazole 30mg/kg/day every 6-8 hours (preferred) or Penicillin G 100,000 U/kg/day, at 4-6 hr. intervals, for 7-10 days.

c. Neutralization of unbound toxin

- Single injection of 500 U of Tetanus immune globulin (TIG)I.M to; Doses as high as 3000-6000 U can be used.
- If TIG is not available, I.V human immunoglobulin or tetanus antitoxin (TAT): 1,500-3,000 U to be administered after skin testing.

d. Management of autonomic dysfunction

- Magnesium sulfate is the drug choice and labetalol is an alternative
- Morphine sulfate (0.5-1 mg/kg/hr by continuous IV infusion) to control autonomic dysfunction and to induce sedation.

NB: don't insert NG tube unless the patient is fully sedated. Careful nursing attention to mouth, skin, bladder, and bowel function is needed.

e. Active immunization

- As tetanus infection does not confer immunity, the patient should receive active immunization (a total of three doses of tetanus and diphtheria toxoid spaced at least two weeks apart), commencing immediately upon diagnosis.

III. Complication

- Aspiration of secretion and risk of pneumonia
- The spasm may result in laceration, hematoma, rhabdomyolysis with myoglobinuria and renal failure, Long bone or spinal fracture
- Venous thrombosis and rarely pulmonary embolism
- Gastric ulceration and paralytic ileus
- Decubitus ulceration
- Iatrogenic complication from medications and ventilation

13.7 PEDIATRIC DIABETIC KETOACIDOSIS

I. Introduction

DKA presents with polysymptoms and combinations of nausea, vomiting, abdominal pain, deep fast breathing, altered consciousness and dehydration based on its severity.

Diagnostic Criteria for DKA

Clinical presentation plus: Blood glucose >200 mg/dL, Bicarbonate <15 mEq/L, and pH <7.3 with ketonuria or ketonemia

Classification

Severe: PH<7.15 Bicarbonate <10, acidotic breath and altered mentation

Moderate: PH 7.15-7.25, Bicarbonate 10-15 and acidotic breathing

Mild: PH of 7.25-7.35, Bicarbonate 15-18 with no acidotic breathing and normal mentation

Investigation

RBS, urine analysis, Serum electrolyte, BUN, creatinine, osmolality and blood gas if available. Blood cultures and other laboratory tests only as clinically indicated.

ICU Admission

Severe DKA patients, patient younger than 2 year of age, GCS < 12, [Na+] > 150 mmol / L, [K+] < 2.5 mmol / L and cerebral edema.

II. Management

- ABCDE
- Severe and moderate DKA with no shock, 0.9% saline 10 ml/kg over 1 hour.
- Patients with shock, NS 20 ml/kg bolus as quickly as possible. Repeat as necessary, with careful reassessment after each bolus.
- If septic shock, rapid fluid boluses, broad spectrum antibiotic and vasopressors

- Subsequent fluid management
- Calculate fluid deficit and maintenance fluid
    - Deficit is assumed 5% for mild, 7% for moderate and 10% for severe DKA. Eg for weight 10 kg and mild it will be 5%x 10kg =500ml
    - For weight 10 kg and sever will be 10% 10kg = 1000ml
    - (Mainetance fluid MF48 hours) + Deficit fluid -Minus Bolus fluid) = -------- ml/ 48hr. which is similar to 1 and ½ mainatence fluid

The type of fluid

- Use NS in the first one-hour and for at least 4-6 hours for severe DKA.Change fluids to 0.45% NS once corrected sodium is normal.
- After 4- hours of fluid and when RBS reaches 270 mg/dl, change the fluid to ½ NS in 5% DW for infant and DNS for older children. (to make this in our setting use half the fluid DNS and half DW)
- When RBS <150 change fluid to 10 % dextrose in NS (to prepare this make 15% of the fluid 40% dextrose and 85% DNS) or 10 dextrose in ½ NS ..
- Oral fluids should be introduced only when clinical improvement has occurred.

Potassium replacement therapy

Potassium 40 meq / L after K level known or adequate urine passed K < 3 mmol per L after the initial bolus: K+ of 60 mEq per L or greater may be necessary.

[K+] = 3 to 4.5 mmol: after bolus fluid and child passed urine, 40 mEq / L of K.

[K+] = 4.6 to 5.0, only 20mEq per L of potassium /L

[K+] > 5.0, withheld K in the initial fluids

Insulin therapy

- Started after 1 hour of fluid resuscitation as IV infusion 0.1 IU /kg / hr regular insulin (0.05U/kg/hr for age less than 2 years)
    - Dilute 50 u regular insulin in 50 ml NS to make 1 u=1ml
    - Infuse insulin via a dedicated Iv line .Ensure that the infusion is clearly labeled and cares a << don't Flush >> order.

- If continuous infusion not possible, use (0.3-0.4U/kg every 3-4 hour SC administration of regular insulin with half this does for children lessthan 2 years ; Consider the first dose ½ IV and ½ IM
- If again this is not visible use subcutaneous insulin dose: 0.5 unit/kg every 6 hours (but 0.25IU/kg every 6 hour for under five children) except the first dose ½ iv and ½ IM (because SC will not be absorbed right away on dehydrated child in the first dose).

  NB: if your setting don't have perfuser it is better to refer children under 2 years to a setting where there is perfuser
- Don't omit insulin but decrease by 50% if
  - If rapid decline of glucose (100mg/hr), do the RBS every one hour
  - If child become hypoglycemic <70mg/dl
  - Unresponsive hypokalemia despite treatment
- Maintain the blood glucose between 150 and 250 mg per dl
- If IV perfusion used, give subcutaneous insulin 30 minutes before infusion stopped.
- Continue iv insulin until blood glucose <200mg/dl and metabolically stable (PH>7.3 and Hco3>15)
- Child ketone free and stable can start combination NPH and regular insulin.

III. Complication

- Cerebral edema
- Electrolyte abnormality (Hypokalemia , Hyperkalemia, Hypophosphatemia)
- Hypoglycemia
- Profound metabolic acidosis

Treat complications

Cerebral edema: If this signs appear give the child mannitol 1 g per kg IV over 10 minute

Hypoglycemia: change fluid to 10% glucose in NS and decrease insulin

IV. Monitoring

- Use the DKA flow sheet for monitoring the child
- Serum [K+] every 2 to 4 hours until the acidosis and hyperglycemia are normalized
  - More frequently if hypokalemia is encountered.

13.8 ONCOLOGIC EMERGENCIES IN PEDIATRIC INTENSIVE CARE UNIT

13.8.1 HYPER LEUKOCYTOSIS

I. Introduction and principle of care

Definition: Peripheral Leukocyte > 100,000 cells/ml which lead to increased viscosity and microvascular stasis and thrombosis to organs.

Clinically significant if > 200,000cells/ml in AML and > 300,000cells/ml in ALL.

Table 13-12: Organs involved and clinical features

End organ involved	Clinical features and imaging
Lung	Tachypnea, dyspnea, hypoxemia, pulmonary infiltrate, acidosis CXR: may reveal varying degree of diffuse interstitial or alveolar infiltrate
CNS	Headache, lethargy, tinnitus, blurred vision, seizure, stroke, ataxia, retinal haemorrhage, CT: haemorrhage or leukemic infiltrate
Others	Renal failure, priapism, dactylitis, Tumer lysis sysndrom(TLS), consumptive coagulopathy, Miocardial ishemia , acute limb ischemia , bowel infraction

Laboratory

- CBC, platelet count, lactate, PT, aPTT, fibrinogen, Electrolyte, organ function tests, Uric acid, Chest x ray, CT and /or MRI as indicated
- PaO_2 should be relied on than SPO_2 as methemoglobin may be elevated.

II. Management

Goals of management

- Prevent/manage tumor lysis syndrome(TLS)
- Prevent/manage Leukostasis
- Manage disseminate intravascular coagulation(DIC)

Supportive care

- Vigorous hydration (3L/m² /24 hr) to reduce risk of TLS and increase GFR
- Transfuse only in severe anaemia with CHF (whole blood 5-10 ml/kg over 3-4hrs)
- FFP and vitamin K to correct coagulopathy.
- Maintain platelet above 50,000/mm³

Cytoreduction

Asymptomatic: Hydroxyurea: 50-100 mg/kg given PO 3-4 doses daily

Symptomatic

- Induction chemotherapy, e.g. for ALL, cytarabine with anthracycline can be started
- Consider low dose prednisolone reduction phase prior to induction chemotherapy in patients with ALL and WBC>300-500, 000/mm3.

13.8.2 TUMOR LYSIS SYNDROME (TLS)

I. Introduction

TLS is a metabolic abnormality due to rapid release of intracellular metabolites from dying cells that exceed excretory capacity of the kidneys. TLS can happen spontaneously or following chemotherapy.

Table 13-13: Cairo-Bishop Definition of Laboratory and clinical Tumor Lysis Syndrome

Laboratory Tumor Lysis Syndrome	
Metabolite or electrolyte	**Criterion for diagnosis**
Uric acid	≥ 8mg/dL or 25% increase from the baseline
Potassium	≥ 6mEg/L or 25% increase from the baseline
Phosphorus	≥ 6.5 mg/dl (children), ≥ 4.5 mg/dl (adult), or 25% increase from the baseline
Calcium	≥ 25% increase from the baseline
Clinical Tumor Lysis Syndrome	
LTLS (Laboratory Tumor Lysis Syndrome) and one or more of the following: 1. Creatinine X ≥ 1.5 ULN-Upper Limit of Normal (age > 12 years of age or adjusted); 2. Cardiac arrhythmia or sudden death; 3. Seizure	

***N.B. Laboratory TLS** is defined as two or more of the above present within three days before treatment or seven days after chemotherapy.*

Laboratory: CBC, electrolytes, urea, creatinine, uric acid

II. Treatment and Prevention

- Intermediate and high risk patients
  - IV fluid (Preferred NS) 2-3 L/m²/day (or 200ml/kg/day in a child </=10kg). Target urine output of 3-5mlkg/hr.

- Begin 24-48hr before chemotherapy and continue 48-72hours after
- Mannitol or furosemide if adequate diuresis not achieved and for fluid overload.
- Dialysis for AKI and medically refractory and dangerous metabolic derangements
- Allopurinol 50-100mg/m^2 or 10mg/kg/day TID
- Hypocalcemia: Give calcium only for symptomatic hypocalcemia.
- Aluminum containing antacids to prevent gut absorption of phosphate.

III. Monitoring

- Electrolytes every 8-12 hrs in the acute phase. Careful input /output and vital sign monitoring.

13.8.3 SUPERIOR MEDIASTINAL SYNDROME (SMS) AND SUPERIOR VENACAVAL SYNDROME (SVCS)

I. Introduction

Definition: SVCS refers to signs and symptoms resulting from compression, obstruction, or thrombosis of the superior vena cava.

- SMS = SVCS + tracheal compression

Superior vena cava syndrome	Superior mediastinal syndrome
• Swelling, plethora, and cyanosis of the face, neck, and upper extremities. • Suffusion of the conjunctiva. • Altered mental status	• Cough hoarseness, dyspnea, orthopnea, wheezing, Chest pain, Dysphagia, and stridor. • Supine position worsens symptoms. • Altered mental status and syncope

NB: distal trachea can still collapse after intubation by external pressure and ventilation difficulty.

Laboratory

- Radiograph and CT (if tolerated). CBC, LDH, uric acid, α-fetoprotein, and β-hCG, pleural fluid analysis
- Echocardiogram: cardiac function and possible intravascular thrombus
- If high risk, perform least invasive tests with local anesthesia (e.g bone marrow aspirate).

II. Management

- Positioning the patient **upright;** *DON'T PUT PATIENT IN RECUMBENT POSITION.*
- Noninvasive ventilationtemporarily reduce degree of airway obstruction and improve airflow.
- IV access preferably in lower extremities

- If intubation couldn't be avoided anesthesiologists and ENT surgeons should be present and done with spontaneous breath without deep sedation and paralysis.
- Empiric treatment with radiation, chemotherapy, or both when diagnostic tests are not safe; biopsy as soon as mass shrinks and patient stable.
- Prednisolone 60 mg/m2/day (2 mg/kg/day) or dexamethasone 6 mg/m^2/day in two divided doses or methyl prednisolone 48 mg/m^2/day (1.6 mg/kg/day) should be employed.
- Anticoagulation for thrombosis with no evidence of hemorrhage.

13.8.4 FEBRILE NEUTROPENIA

I. Introduction

Definition: A single oral temperature >38.3C or > 38.0C sustained for >1 h or occurs twice within 24-h in a child with ANC < 500 or ANC <1000 expected to decrease to <500 in 48hr.

Etiologies

- Gram positive and negative rods, gram positive cocci, viruses, fungi and anaerobes

Clinical manifestation

- Unexplained fever without evident focus
- Examine thoroughly every system e.g., anal area (*Avoid PR exam though!*), skin

Investigations

- CBC, Blood cultures, urine cultures, Gram stain and culture from suspicious sites

II. Management

- **Empiric antibiotic with in 1 hour of triage** and response assessed in 3-5 days
- Broad spectrum antibiotics with pseudomonal coverage, 4th generation cephalosporins, carbapenem, or piperacillin tazobactam for uncomplicated patients
- Modify antibiotic based on blood culture or if patient deteriorates consider vancomycin at a dose of 60 mg/kg/day IV divided q8h (max 4 g/day) in the following situations:
    - Patients with AML receiving high-dose cytarabine
    - Presentation with hypotension or other evidence of shock and Mucositis
    - Prior history of alpha-hemolytic Streptococcus infection
    - Skin breakdown or catheter site infection
    - Colonization with resistant organisms treated only with vancomycin
    - Vegetation on echocardiogram

- Stop antibiotics if culture is negative, patient afebrile, ANC >500, or ANC>200 & rising, and a total duration of not more than 7-10 days in gram positive and 10-14 days in gram negative infections.
- Addition of antifungal 3-5 days after presentation, with amphotericin B.

13.8.5 TYPHILITIS

I. Introduction

Definition and clinical features

- Necrotizing colitis mostly in neutropenic patients with hematologic malignancies with breakdown of gut mucosal integrity as a result cytotoxic chemotherapy
- Cecum is most commonly affected site; mostly a polymicrobial infection
- Should be considered in neutropenic patients with fever and abdominal pain
- Abdominal distension, nausea, vomiting, watery or bloody diarrhea, Paralytic ileus
- signs of peritonitis and shock

Investigations

- Blood and stool culture, Imaging including plain abdominal film and CT scan

II. Management

- NPO, NG suction; Packed red cell and platelet transfusions, as indicated
- Broad-spectrum antibiotics: Piperacillin-tazobactam **or** cefipime/ceftazidime plus metronidazole or Meropenem, or Ciprofloxacin plus metronidazole or Augmentin
- Fungal coverage is needed if fever is protracted >72 hr.

Indications for surgical intervention

- Persistent GI bleeding despite resolution of neutropenia and thrombocytopenia.
- Evidence of free air in the abdomen on abdominal radiograph and clinical deterioration

Suggested reading

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13. Fuhrman and Zimmerman Pediatric critical care, 5th edition, 2016
14. SCCM: Pediatric Fundamental Critical Care Support (PFCCS) 2018
15. Lanzkowsky P, management of oncologic emergencies from: Manual of pediatric hematology and oncology Philip Lanzwosky 5th edition. Academic Press, 2011.
16. Tintinal's 9th edition Fluid and electrolyte mangment in pediatrics page 851-856
17. Fundamental of critical care second edition chapter 8 Fluid, electrolyte and neuroendocrine metabolic derangements page 8-1-8-16

ANNEXES

ANNEX 1: HISTORY AND PHYSICAL EXAMINATION FOR INTENSIVE CARE UNIT

Name:	Age:	Sex:
Marital Status:	Occupation:	
Spouse / Parent / Guardians Name :		Address:
INFORMANT : Name	RELATIONSHIP: (OF INFORMANT):	
Reason for Transfer to Intensive Care:		Date & Time
Background information, Level of activity (Family & personal history)		
Existing co – morbidities & allergies :		
Medications:		
Chief complaints:		
History of presenting illness & Treatment history : (Chronological order)		
General Physical Examination		Transferred in with(tubes/Lines)
Pulse :		ETT/TT/Airway
Blood Pressure :		Peripheral Line
Weight :		Central Line
Temperature :		Arterial Line
Respiration :		Foleys Catheter
Pallor:	Cyanosis	HDA/ AV Fistula
Icterus:	Clubbing	Collars/Splints
Pedal Edema	JVP	ICD/Epidural Catheter
Lymphadenopathy:	Hydration	Dentures/Caps/Contact lenses
Scars :		NG Tube
Surgical Scars:		Metallic implants
Deformities (congenital/ acquired):		Pace maker (permanent / temporary)

CVS: - S1 S2	Rhythm	peripheral pulses
Murmurs		Added sounds
RS : Signs of Respiratory Failure – Flaring alae nasi / Tracheal tug / Accessory muscle use		

Diaphragmatic paradox / Intercostal indrawing / Flail chest						
Air entry			Added sounds			
Abdomen: Hepatomegaly	Yes	No	Splenomegaly	Yes	No	
Umbilicus	Yes	No	Engorged veins	Yes	No	
Guarding Rigidity	Yes	No	Bowel sounds	Yes	No	
Ascites	Yes	No	Scars	Yes	No	
Rectal Examination:						
CNS/Musculoskeletal:						
Pupils						
Limb movements:						
DTRs :			Plantars			
Specific / Local Examination:						
Provisional Diagnosis:						
Plan: Investigation & Treatment						
Discussion :						

ANNEX 2: MEDICATION CHART FOR ICU

DRUGS IV Fluids & STAT ORDERS

Attach sticker

Drug chart number

Admission diagnosis:
Allergies:

Date:

Special Investigations

Peripheral Smear: _____

_____

Retic : _____ PCV:_____ MCV: _____ MCHC:_____

Bone Marrow : _____

FDP / Dimer : _____ LDH: _____ Sr. Cortisol: _____

Uric Acid : _____ TSH: _____ T: _____ T: _____

ESR:_____ CRP: _____ ANA: _____ ANCA: _____

Lipid Profile } Cholesterol: _____ HDL: _____ LDL: _____ TG _____

Urine Myoglobin: _____ ABG:_____

EEG:_____

ENMG: _____

ANNEX 3: DRUG CHART

Strike through entire row, initial when stopping drug

1		**DATE**																					
DRUG Name	**Dosage**																						
		TIME																					
Drs Name:	Dr sign	**Nurse sign**																					
2		**DATE**																					
DRUG Name	**Dosage**																						
		TIME																					
Drs Name:	Dr sign	**Nurse sign**																					
3		**DATE**																					
DRUG Name	**Dosage**																						
		TIME																					
Drs Name:	Dr sign	**Nurse sign**																					
4		**DATE**																					
DRUG Name	**Dosage**																						
		TIME																					
Drs Name:	Dr sign	**Nurse sign**																					
5		**DATE**																					
DRUG Name	**Dosage**																						
		TIME																					
Drs Name:	Dr sign	**Nurse sign**																					

ANNEX 4: INVESTIGATION CHART

PATIENT'S NAME: **Bl Group/ Type** **OP/IP:**

DATE							
Hemoglobin							
Total Leukocyte Count							
Differential Count							
Platelet Count							
Prothrombin Time							
INR							
APTT							
Malarial Parasite-QBC							
Blood Sugar							
Blood Urea							
Creatinine							
Serum Sodium							
Serum Potassium							
Chloride							
Serum Magnesium							
Serum Calcium							
Phosphorus							
Total Protein							
Albumin							
A:G							
Total Bilirubin							
Conj. Bilirubin							
AST							
ALT							
Alkaline Phosphatase							
Gamma GT							
Amylase							
Lipase							
Plasma Ammonia							
TROPONIN							
CPK / MB							
CPK							
Blood Widal							
Weil-felix							
Leptospira-IgM							
Dengue-IgM							
HbsAg/HCV							
HRV							
Body Fluid—TC/DC							
Protein/Albumin							
Sugar							
Chloride							
Peripheral smear							

ANNEX 5: GRAPHIC (T.P.R) CHART (AXILLARY TEMPERATURE)

Month .... Year ....

Name	Age	Sex	Marital Status	Hospital Number
Service	Ward	Bed	MRD No.	Occupation Status

DATE

NO. OF HOSPITAL DAYS

DAYS POST-OP

TIME: 2 6 10 2 6 10 2 6 10 2 6 10 2 6 10 2 6 10 2 6 10 2 6 10 2 6 10 2 6 10 2 6 10 2 6 10 2 6 10 2 6 10

PULSE	TEMP C°	TEMP F°
210	41.1	106
200	40.6	105
190	40.0	104
180	39.4	103
170	38.9	102
160	38.3	101
150	37.8	100
140	37.2	99
130	36.7	98
120	36.1	97^{N}
110	35.6	96
100	35.0	95
90	RESP 60	
80	50	
70	40	
60	30	
50	20	
40	10	

BP

STOOLS

URINE

WEIGHT

BATH

ANNEX 6: ISBAR COMMUNICATION TOOL

I Identify	➢ Yourself: ▯ name, ▯ position, ▯ location ➢ Receiver: Confirm who you are talking to ➢ Patient: name, age, sex, location
S Situation	➢ State purpose "The reason I am calling is……." ➢ If urgent – SAY SO, Make it clear from the start ➢ May represent a summary of Assessment and Requirement
B Background	➢ Tell the story ➢ Relevant information only: ▯ history, ▯ examination, ▯ test results, ▯ management ➢ If urgent: Relevant vital signs, current management
A Assessment	➢ State what you think is going on, your interpretation ➢ Use ABCDE approach ▯ Airway ▯ Breathing ▯ Circulation ▯ Disability ▯ Exposure ➢ State any interventions e.g applied oxygen
R Requirement	➢ What you want from them – BE CLEAR ➢ State your request or requirement ▯ Urgent review (state time frame) ▯ Give approval / recommendation for further course of action while awaiting attendance eg. ECG, bloods ▯ Give opinion on appropriate management

S	**Situation**	I am (name), (X) nurse on ward (X)
		I am calling about patient (X). I am concerned that...
B	**Background**	Patient (X) was admitted on (XX) date with...
		They have had (X operation/ procedure/investigation)
A	**Assessment**	I think the problem is (XXXX) and I have... *or*
		I am not sure what the problem is but patient (X) is deteriorating
R	**Recommendation**	I need you to come and see the patient in the next (XX mins)
		Is there anything I need to do in the mean time?

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