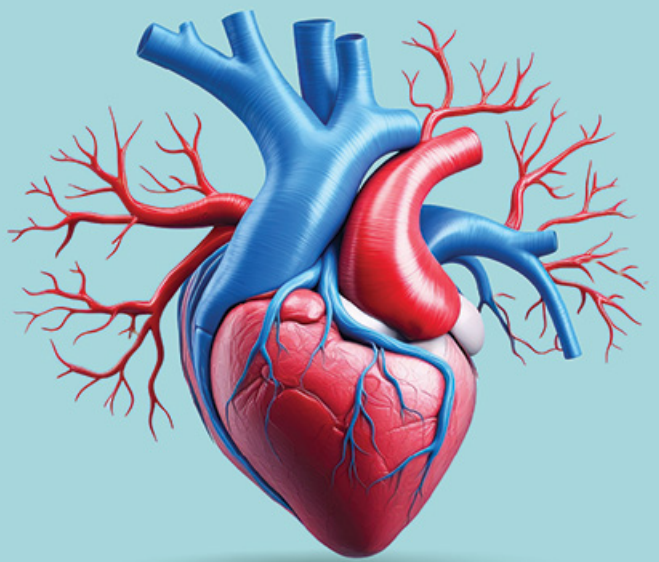
website www.ejaypee.com, for detailed information on cardiology books, visit our website www.ejaypee.com

CARDIOLOGICAL SOCIETY OF INDIA

Handbook on Cardiac Critical Care



Editor
Santanu Guha

Associate Editors
S Ramakrishnan
Rishi Sethi

JAYPEE

Contents

SECTION 1 Basics in ICU

1. Blood Gases in Coronary Care Unit 3
Anunay Gupta, SS Kothari

Is This Blood Gas Measurement Reliable? *3*
Is Venous Blood Comparable to Arterial Blood for Analysis? *4*
Is It Acute or Chronic Abnormality? *5*
Is It Only Respiratory or Metabolic (Mixed)? *5*
Use of Blood Gas Analysis in CCU *8*
Conditions in Which Blood Gas Helps in Management *10*

2. Electrolyte Abnormalities 15
Jacob Jose

Hyponatremia *15*; Treatment *16*
Vaptans in Hyponatremia *20*; Hypernatremia *20*
Hypokalemia *21*; Hyperkalemia *23*; Magnesium *24*

3. Hyponatremia in Heart Failure 26
Harikrishnan S

Drug-induced Hyponatremia *26*
Consequences of Hyponatremia in Heart failure *27*
Management of Hyponatremia in Heart Failure *28*
Treatment of Acute Symptomatic Hyponatremia in Patients with Heart Failure *29*

4. Ventilator Settings in Cardiac ICU 33
Chitra Mehta, Yatin Mehta

Hemodynamic Effects of PPV/PEEP *34*
High Frequency Oscillatory Ventilation *35*
Airway Pressure Release Ventilation *36*; Prone Ventilation *37*
Extracorporeal Membrane Oxygenation *37*
Noninvasive Ventilation *39*
Weaning from Mechanical Ventilation *39*

5. Noninvasive Ventilation 43
Anant Mohan, Saurabh Mittal

Equipment *44*; Indications of Noninvasive Ventilation *47*
Initiation of Noninvasive Ventilation *50*
Monitoring during Noninvasive Ventilation *51*
When to Terminate Noninvasive Ventilation? *51*
Contraindications of Noninvasive Ventilation *51*
Predictors of Outcome on Noninvasive Ventilation *52*
Complications of Noninvasive Ventilation *52*
Weaning from Noninvasive Ventilation *53*

6. Weaning in Patients with Cardiac Diseases 54
Mayank Singhal, Rohit MK, Parag Barwad

Mechanisms Contributing to the Development of Weaning Related Cardiogenic Pulmonary Edema *55*
Diagnosis of Weaning-Related Cardiogenic Pulmonary Edema *56*
Cardiac Catheterization *57*; Cardiac Biomarkers *58*
Transthoracic Echocardiography *58*
Weaning Associated Hemoconcentration *59*
Therapeutic Options *59*

7. Ventilator Associated Pneumonia 64
Randeep Guleria, Saurabh Mittal

Epidemiology and Risk Factors *64*; Treatment of VAP *70*

8. Hemodynamic Monitoring in Cardiac Critical Care Units 74
Anunay Gupta, Saurabh Kumar Gupta
Noninvasive Hemodynamic Monitoring *75*
Invasive Hemodynamic Monitoring *78*
Assessing the Fluid Responsiveness *92*
Recent Advances in Hemodynamic Monitoring *93*

SECTION 2 Heart Failure

9. Acute Decompensated Heart Failure 99
Sandeep Seth, Archana Saini
Background *99*; Definition *99*; Magnitude of Problem *99*
Causes *100*; Classification *100*; Management *101*

10. Inotropes 106
Ajay Bahl
Dobutamine *106*; Dopamine *107*; Noradrenaline *107*
Adrenaline *108*; Milrinone *108*; Levosimendan *109*
Which Drug? *109*

11. Value of IABP and Assist Devices in Critical Care Unit 111
Sundeep Mishra
Etiology *111*; Mechanism of Destabilization *112*;
High-Risk PCI *114*; Nonischemic Myocardial Injury *114*
Management of Ischemic and Hemodynamic Support in High-risk PCI *114*

12. Extracorporeal Membrane Oxygenation in Cardiac ICU 119
Poonam Malhotra Kapoor, Sameer Taneja
Decision to Institute ECMO *119*
Inclusion Guidelines for ECMO in Pediatric Patients *120*
Contraindications of ECMO *120*; Types of ECMO *121*
Central Versus Peripheral ECMO *125*
Uses of ECMO *126*; ECMO in Cardiogenic Shock *127*
ECMO In Heart Failure *127*; ECMO And Sepsis *127*
Noncardiac Indications for Extrcorporeal Membrane Oxygenation *127*; Complications *128*

SECTION 3 Arrhythmias and Resuscitation

13. Bradyarrhythmias in Critical Care 133
Gaurav Chaudhary, Rakesh Yadav, Rishi Sethi
Etiology *133*; Causes of Bradyarrhythmia *133*
Clinical features *134*; Diagnosis of Bradyarrhythmia *135*
Atrioventricular Blocks *135*; Medical Management *136*
Special Situations *138*
Transcutaneous External Cardiac Pacing *139*
Transvenous Pacing *139*; Epicardial Pacing *140*

14. Atrial Tachyarrhythmias 141
Anitha Sathishkumar, Ulhas M Pandurangi
Classification *141*; Management of Atrial Arrhythmias *148*
Drugs in Atrial Tachyarrhythmias *153*

15. Ventricular Arrhythmias 157
Zohaib Shaikh, Yash Lokhandwala
General Principles *157*
Approach to Ventricular Arrhythmias *158*
Premature Ventricular Complex *158*
Nonsustained Ventricular Tachycardia *158*
Sustained Monomorphic VT *158*

Sustained Polymorphic Ventricular Tachycardia/ Ventricular Fibrillation *158*
Ventricular Tachycardia/Ventricular Fibrillation Storm *161*
Management of VT/ VF Storm *161*
Defibrillator Basics *167*

16. Basic Life Support **170**
Anand P, Nitish Naik
Prevalence *170*; Recognition *170*
Maintaining Circulation *171*; Securing Airway *172*
Breathing *172*; Defibrillation *173*; Additional Care *173*

17. Advanced Life Support **175**
Satyendra Tewari, Deep Chandh Raja S
Advanced Cardiac Life Support 2010 Algorithms *175*
Ensuring Quality and Monitoring of CPR *177*
When to Interrupt Chest Compressions? *179*
Airway Management and Ventilation *180*
Drugs and Routes of Administration *181*
Common Resuscitation Drugs *181*
Termination of CPR *182*; Postcardiac Arrest Care *182*
Pediatric Advanced Life Support *183*
Role of Waveform Capnography in ACLS *183*
Key Messages for Healthcare Providers *183*

18. Cardiac Arrest Survivor **185**
Amit Vora
Step Care Management of Cardiac Arrest Survivor *185*
Neurologic Assessment and Management *186*
Maintain Hemodynamic Stability *187*
Adequate Oxygenation *187*; Maintain Internal Milieu *187*
Evaluate Cardiac Causes of Arrest *188*
Treatment of Cardiac Causes of Arrest *188*
Prognosis of Cardiac Arrest Survivor *192*

SECTION 4 Acute Coronary Syndrome

19. Caring for a Thrombolysed Patient **197**
SS Iyengar
Reassurance *197*; Close Observation *197*
Adverse Effects of Thrombolysis *198*
Result of Thrombolysis *200*; Complications of STEMI *201*
Adjuvant and Concomitant Therapy *202*; Transfer *203*
Secondary Prevention *203*
Summary of Steps to be Taken in the Care of a Thrombolysed Patient *204*

20. Nonthrombolysed STEMI **205**
Vishal Batra, Asit Das
Current Status of Treatment of STEMI in India *206*
Role of Thrombolysis in the Treatment of AMI *206*
Indications for Fibrinolysis *207*
Timing of Fibrinolytic Therapy *207*
Therapies for Non-reperfused Patients *208*

21. Practical Tips in Managing a Patient with USA/NSTEMI **213**
Vineet Bhatia, Upendra Kaul
Management of UA/NSTEMI *213*
Anti-Ischemic Therapy *215*
Antiplatelet Agents *217*

22. ECG Diagnosis of Myocardial Infarction in Challenging Situations **225**
G Justin Paul, G Ashok, P Anandharaj
Causes of Non Ischemic ST-Segment Elevation *226*
Benign Early Repolarization Syndrome *227*
Acute Pericarditis *228*
Left Ventricular Hypertrophy (LVH) *230*
Sgarbossa Criteria *233*
Left Ventricular Aneurysm *235*
Electrolyte Imbalance: Hyperkalemia *236*
Hypercalcemia and Hypocalcemia *237*
Brugada Syndrome *237*
Takotsubo Syndrome (Apical Ballooning Syndrome) *240*
Electrical Cardioversion *242*

23. Biomarkers in Acute Coronary Syndrome **245**
Ajay Kumar Sinha, Mridubhashini Sinha, Pramod Kumar, Manish Gunjan, Sanjeev Kumar
Biomarkers in Acute Coronary Syndrome *245*
Cardiac Troponin *246*; Creatine Kinase *247*
Myoglobin *248*; Natriuretic Peptides *248*
Emerging Diagnostic Markers of Acute Coronary Syndrome *248*
Limitations of Diagnostic Biomarkers in Acute Coronary Syndrome *250*

24. Postmyocardial Infarction Angina or Reinfarction: Always a Diagnostic Challenge in Critical Care Unit **254**
PC Mondal
Causes of Recurrent Chest Discomfort *254*
Causes as Per Time Frame *255*
Pathophysiologic Mechanisms *255*

25. Reinfarction **259**
Debasis Ghosh
Ischemic Complications Postmyocardial Infarction *259*

26. Cardiogenic Shock in Acute Myocardial Infarction **263**
Rajiv Ananthakrishna, Srinivas BC
Definition *263*; Etiology *263*; Pathophysiology *264*
Adverse Prognostic Factors *264*
Management *264*

27. Mechanical Complications of Acute Myocardial Infarction **274**
Bishav Mohan, Manbir Singh Khurana
Ventricular Septal Rupture *275*
Left Ventricular Free Wall Rupture *281*
Pseudoaneurysm *283*; Ventricular Aneurysm *284*

28. Stent Thrombosis in Drug-eluting Stents Era **288**
Nagendra S Chouhan, Praveen Chandra
Definition of Stent Thrombosis *288*
ARC Definition of Stent Thrombosis *289*
Pathogenesis of Stent Thrombosis *290*
Drug-related Thrombogenicity *291*
Device (Stent)-related Thrombogenicity *292*
Effect of Systemic Prothrombotic Environment and Plaque Characteristics (Acute Coronary Syndrome) *293*
Stent Malapposition and Role of Intravascular Imaging *294*

Acquired Stent Malapposition *294*
Endothelial and Neointimal Regeneration and Neoatherosclerosis *296*
Clinical Implications of Stent Thrombosis *297*
Incidence of Stent Thrombosis *298*
Management of Stent Thrombosis *300*
Improvement in Devices Technology *301*

29. Management of Postpercutaneous Coronary Intervention Complications **308**
G Sengottuvelu

Coronary Vascular Injury *308*
Noncoronary Vascular Injury *310*
Systemic Events *314*

30. Anticoagulants in Cardiac Critical Care **319**
Pritish Bagul, Swapnil Mate, Prafulla Kerkar

Unfractionated Heparin *319*
Newer Oral Anticoagulants *324*

SECTION 5 Cardiac Emergencies

31. Cardiac Tamponade **335**
Mullasari Ajit Sankardas, Aravind Sampath, Vijayakumar Subban

Anatomy and Physiology of the Pericardium *335*
Classification *336*; Pressure-Volume Relationship *337*
Clinical Features *339*; Clinical Signs *340*
Investigations *341*; Management *346*
ECHO Guided *347*; ECG Guided *348*
Blind Approach *349*; Fluoroscopy Guided *349*
Complications *349*; Contraindications *349*
Surgical Therapy *349*

32. Accelerated Hypertension **351**
Debasri Dasbiswas, Arindam Maitra, Arup Dasbiswas

Hypertensive Urgencies *351*
Hypertensive Emergency *352*

33. Management of Pulmonary Embolism **361**
Neil Bardoloi, Rituparna Baruah

Epidemiology *361*; Predisposing Factors *361*
Acquired Risk Factors *361*; Inherited Risk Factors *362*
Natural History *363*; Pathophysiology *363*
Clinical classification of Pulmonary Embolism Severity *363*
Diagnosis *363*; Clinical Presentation *364*
Thrombolytic Therapy *370*; Advanced Therapy *371*

34. Acute Dissection of Aorta **376**
NN Khanna, Suparna Rao

Epidemiology *376*; Pathophysiology *377*
Predispositions *377*; Genetic Predisposition *377*
Natural History *378*; Clinical Presentation *378*
Classification *380*; Diagnosis *382*
Biomarkers *384*; Management *385*
Medical Therapy *387*

35. Acute Limb Threatening Ischemia **393**
Sanjay Tyagi, Amit Mittal, Swati Tyagi

Common Etiologies *393*
Other Less Common Etiologies (10%) *394*
Evaluation *394*

Clinical Classification of Acute Limb Ischemia *396*
Diagnostic Studies *397*
Treatment of Acute Limb Ischemia *401*
Treatment Options: The Bottom Line *402*
Technique *403*

36. Unexplained Hypotension in ICU Setting **410**
Munna Das

Step 1: Heart Rate (HR) *411; Step 2*: Volume Status *411*
Step 3: Cardiac Performance *412*
Step 4: Systemic Vascular Resistance *413*
Ultrasound Triaging in Hypotensive Patient *416*

37. Acute Dyspnea **417**
Rajiv Ananthakrishna, Kapil Rangan, Srinivasa KH

Etiology of Acute Dyspnea *417*
Pathophysiology of Dyspnea *417*
Differentiating Cardiac from Respiratory Dyspnea *417*
Approach to a Patient with Acute Dyspnea *418*
Investigations *419*; Treatment *421*; Case Illustrations *422*

38. Emergency Cardiovascular Drugs **424**
SS Islam

Access to the ECVDs *424*; Scope for the use of ECVDs *425*
Adenosine *425*; Atropine *426*; Amiodarone *426*
Aspirin *426*; Digoxin (Lanoxin) *426*; Diltiazem *427*
Diuretics (Frusemide) *427*; Dopamine and Dobutamine *428*
Epinephrine (Adrenaline) *429*; Norepinephrine *429*
Ibutilide *429*; Isoprenaline *430*; Lidocaine *430*
Magnesium Sulphate *430*; Morphine *430*
Nitroglycerin or Glyceryl-trinitrate (NTG or GTN) *430*
Nesiritide *431*; Procainamide *431*
Sodium nitroprusside *431*
Verapamil *432*; Hydrocortisone *432*
Thrombolytic Agents *432*

39. Emergency Procedures in Cardiac Critical Care **434**
Jabir A, Anoop Mathew

Peripherally Placed Central Venous Access *434*
Pericardiocentesis *442*

40. Stress Cardiomyopathy: Recognition and Management **446**
Nair Anishkumar, Krishnan MN

Investigations *449;* Etiopathogenesis *446*
Clinical Profile *448;* Atypical Forms *448*
Complications *448;* Diagnosis *449;* Investigations *449*
Electrocardiogram *449;* Cardiac Biomarkers *450*
Echocardiography *451;* Cardiac Catheterization *451*
Imaging Studies *452;* Biopsy *453;* Treatment *453*
Prognosis and Recurrence *454*

Index *459*

CHAPTER 2

Electrolyte Abnormalities

Jacob Jose

HYPONATREMIA

Hyponatremia is defined as decrease in serum sodium *less than 135 mmol per liter*. Hyponatremia can be classified as follows:

- *Mild*: 130–134 mmol/L
- *Moderate*: 125–129 mmol/L
- *Profound* (*Severe)*: Less than 125 mmol/L.

The causes of hyponatremia can be categorized based on changes on the extracellular volume. They can be categorized into *3 broad types* as given below **(Table 2.1)**.

1. Hyponatremia with Hypovolemia
2. Hyponatremia with Euvolemia
3. Hyponatremia with Hypervolemia

Table 2.1 Causes of hyponatremia

Hypovolemic decreased ECF	*Euvolemic normal ECF*	*Hypervolemic increased ECF*
Renal sodium loss (Urine sodium >20 mmol/L) Diuretics Adrenal insufficiency Salt wasting nephropathy Bicarbonaturia	Hypothyroidism Adrenal insufficiency SIADH	Congestive heart failure Cirrhosis Nephrotic syndrome Renal failure Pregnancy
Extrarenal sodium loss (Urine sodium <20 mmol/L) Vomiting Diarrhea Blood loss Excessive sweating Fluid sequestration in 3rd space Peritonitis Pancreatitis Burns		

JAYPEE BROTHERS

Symptoms

They can be categorized into 3 groups:

1. *Severe*: Vomiting, altered sensorium, seizures, coma—Glasgow scale of 8 or less.
2. *Moderate*: Nausea without vomiting, headache or confusion
3. *Asymptomatic*: Many patients who are elderly with chronic hyponatremia are actually asymptomatic.

Work-up (Flow chart 2.1)

- Measure the *plasma osmolality*
  - *Hypotonic*: Most common one
  - *Hypertonic*: Excess of another osmole such as mannitol
  - *Isotonic*: Laboratory artifact due to lipids and hyperproteinemia
- Assess the *volume status*
  Look at the jugular venous pressure (JVP), skin turgor, peripheral edema, creatinine, etc. Based on volume status, we can classify as hypovolemic or euvolemic or hypervolemic hyponatremia. However, it has been found we as clinicians are not good at correctly classifying into these categories **(Table 2.2)**. Hence, the current recommendation is that we go to check the urine osmolality as step 2 and not to rely on the volume status clinically.
- *Urinary osmolality*: If less than 100 mOsm/kg consider primary polydipsia or beer potomania. If more than 100, then look at the urine spot sodium concentration. If it is less than 30 mmol/L it suggest low effective arterial blood volume. If more than 30 mmol/L, then we need to consider diuretics or kidney disease.

Problem in Fast Correction

If there is a rapid rise in plasma sodium more than 8 mmol/L in a day, can result in osmotic demyelination syndrome—ODS **(Figure 2.1)**. ODS is a rare but dramatic complication that occurs in chromic hyponatremia if the serum sodium increases too rapidly. ODS can be recognized in the MRI as hypointense lesions in T1 imaging and hyperintense in T2 imaging. The reason for these changes in the brain is due to blood brain barrier is functionally impaired allowing cytokines, lymphocytes to enter the brain and activate the microglial cells and cause demyelination.

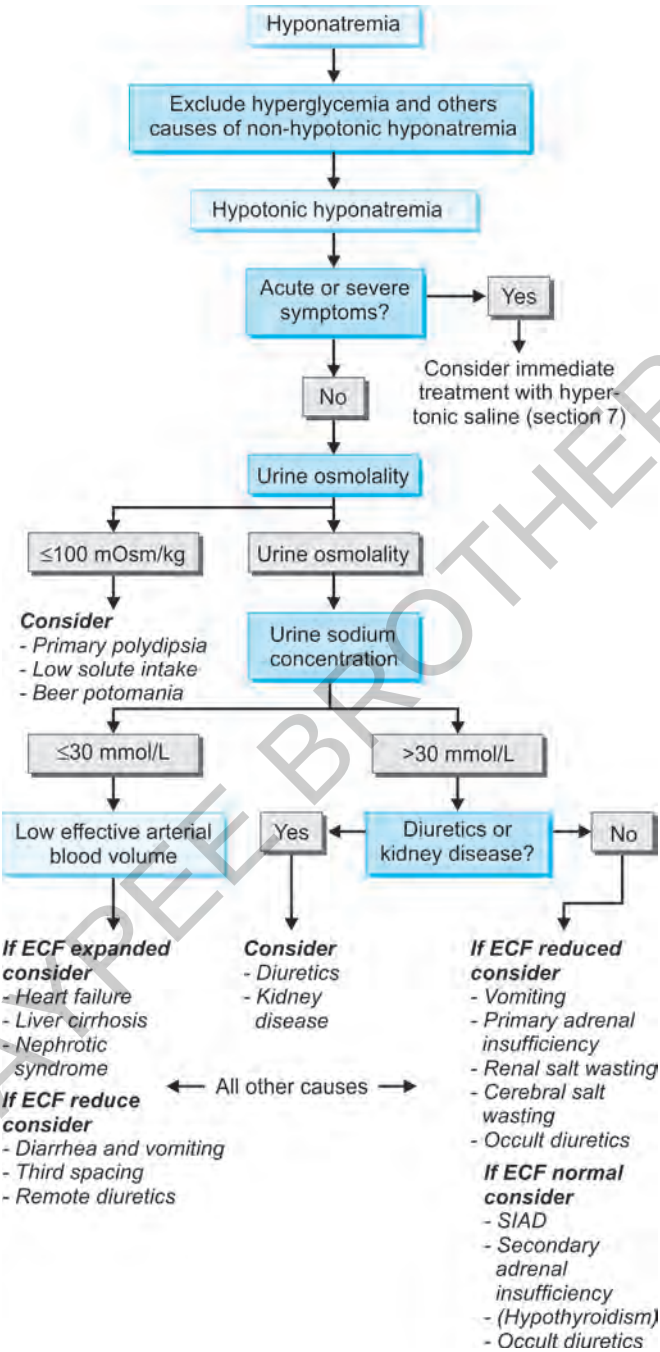
TREATMENT

How to Calculate and Give the 3% Saline Correction?

- *Calculate the Na deficit*
  Let us say a patient with 72 kg of body weight has serum Na of 108 Na in Infusate—Serum Na divided by 50% of body weight 3% saline has 513 of Na
  Na deficit = 513 – 108/36 = 10.9

JAYPEE BROTHERS

Flow chart 2.1 Evaluation of hyponatremia—2014 guidelines



- *Infusate required* = Goal/Infusate required
  Goal over time: Let us say we want to achieve 5 mmol/L over 12 hours
  5/10.9 = 0.46 liter in 12 hours

JAYPEE BROTHERS

Table 2.2 Types of hyponatremia algorithm simplified[1]

	Hypovolemic	*Euvolemic*	*Euvolemic*	*Hypervolemic*
Cause	Salt loss – GI, diuretics	SIADH Hypothyroidism Adrenal insufficiency	Primary polydipsia	Heart failure Cirrhosis
Physical Examination	Hypotension Tachycardia	No edema or hypotension		Edema, signs of underlying disease
Urine Osmo	>100	>100	<100	>100
Urine Sodium	<30	>30	Varies	<30

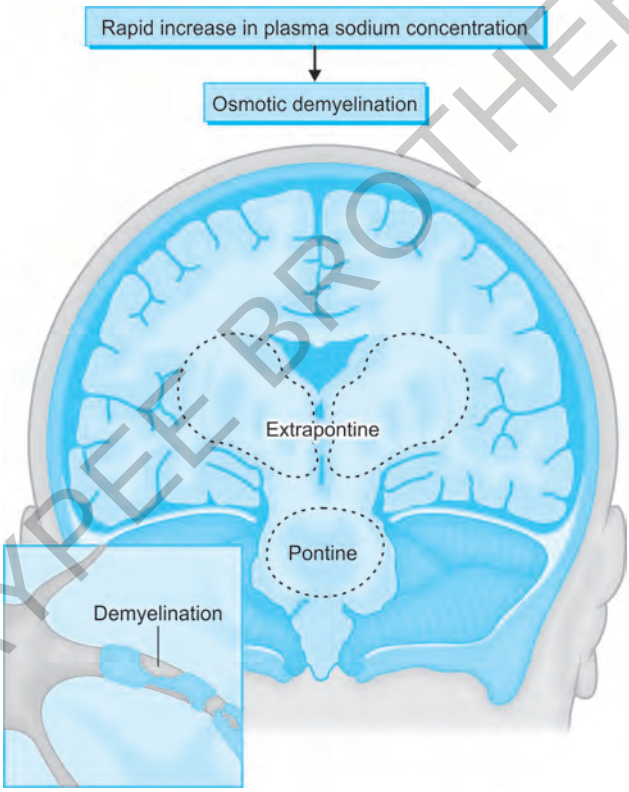


Figure 2.1 Rapid correction of chronic hyponatremia can lead to demyelination—ODS
Adapted from NEJM 2015; 372.

- *Calculate the per hour infusion*
  Infusate required/hours of infusion
  0.46/12 = 38 mL per hour
- *1 mL of 3% saline per kg body weight will increase plasma sodium by 1 mmol/L.*

JAYPEE BROTHERS

TREATMENT (TABLE 2.3)

Hyponatremia with Severe Symptoms

- Use 3% saline 150 mL over 20 min (2 mL/kg)
- Check serum sodium after 20 min
- Continue 3% saline till 5 mmol/L increase
- After the serum sodium has reached more than 5 mmol/L, stop the IV saline; keep 0.9% saline at the smallest possible volume of infusion
- Re-check sodium after 6 and 12 hours and then daily.

Hyponatremia with Moderate Symptoms

- 3% saline 2 mL/kg over 20 min
- Aim 5 mmol/L increase in plasma sodium.
- Check serum sodium after 1 hour, 6 hours and 12 hours

Hyponatremia with No Symptoms or Mild

Treatment should be cause specific. If syndrome of inappropriate antidiuretic hormone (SIADH) secretion, restrict the fluids, solute intake of urea or combination of low dose loop diuretics and oral sodium chloride tablets. If reduced circulating volume give IV saline 0.9%.

Hyponatremia–Dilutional[3]

- *Loop diuretics with or without hypertonic saline*: Loop diuretics remain the first line of therapy in acute

Table 2.3 Treatment of severe hyponatremia <120 mmol/L[2]

Duration	*Condition*	*Clinical*	*Goal*
Acute – over a few hours	Self-induced Use of ecstasy Marathon runners	Headache Vomiting Seizures Coma	100 mL of bolus 3% saline as needed for 3 times Increase sodium by 4 – 6 mmol/L in first 6 hours
1 – 2 days	Postoperative intracranial disease	Headache Vomiting Neurogenic Pulmonary edema	100 mL of bolus 3% saline three times as needed Increase sodium by 4–6 mmol/L in first 6 hours
More than 2 days or Unknown	High risk for demyelination syndrome	Fatigue Malaise Cramps Falls Seizures especially if Na is less than 110	Increase plasma Na by 4 – 6 mmol/L in first 24 hours Avoid increasing more than 8 mmol/L/day

JAYPEE BROTHERS

Table 2.4 Vaptans use in the treatment of hyponatremia—expert panel

Type of hyponatremia	*Recommendation for vaptan*
Hypovolemic	Not recommended
Euvolemic—asymptomatic	Vaptan is a treatment option
Euvolemic with symptoms	Not a treatment option
Hypervolemic—asymptomatic	Vaptan is a treatment option
Hypervolemic with moderate or severe CNS symptoms	Not a treatment option

decompensated heart failure with hyponatremia and volume overload. Many small studies have shown that combining with hypertonic saline in fact improved the outcome.

- *Acetazolamide*: A combination of Loop diuretic with acetazolamide which acts in the proximal tubule has also been shown to be useful.
- *Vaptans*: (AVP antagonists): These drugs directly promote the water excretion through the aquaporin 2 availability. Three of them have been tested in acute heart failure—Tolvaptan, conivaptan and satavaptan. The expert panel recommendations use of these agents are given in **Table 2.4**.[4]

VAPTANS IN HYPONATREMIA

- In a study published based on 20 randomized trials, it was noted that the mean increase in sodium level was 4.3 with a range of 3.51–4.95 mmol/L using vaptans.[5]
- The most important side effects have been polyuria, urinary frequency, thirst and mouth dryness especially with tolvaptan.
- FDA has recommended that 30 days as maximum for tolvaptan and the drug should not be used with liver disease.
- Vaptans can be given if the patient is euvolemic or hypervolemic and not much symptoms. After starting the vaptans plasma sodium level should be checked every 4 hours. When an increase of 6–8 mmol/L is achieved, water intake orally or IV dextrose should be started to match the urine output.
- For long-term use, the starting dose is 15 mg/day. As mentioned earlier maximum duration of use is *only for 30 days*. The dose can be increased after a week. In patients with decompensated heart failure, this drug acutely improves the congestion and dyspnea but is an adjunct to a natriuretic agent in the relief of fluid restriction.[6]

HYPERNATREMIA

Hypernatremia is defined as Na more than *145 mmol/L.* The causes are given in **Table 2.5**.

JAYPEE BROTHERS

Table 2.5 Causes of hypernatremia based on volume status

Volume status	*Causes*
Hypovolemic Na deficit with greater water deficit	Renal: Osmotic diuresis to hyperglycemia, urea, post-obstructive diuresis GI: Diarrhea
Euvolemic Water deficit alone	Diabetes insipidus
Hypervolemic Na retentions with relatively less water retention	IV or oral salt administration Chronic renal failure with salt restriction

Clinical Features

Mainly, it is neurological symptoms such as altered sensorium, to coma and seizures. Some patients can develop, rhabdomyolysis.

Work-up

- Urine osmolality and urine sodium, volume status (JVP, Skin turgor, orthostasis and vital signs)
- Urine osmolality >700–800: Extrarenal water loss or Na overload
- Urine osmolality <700 renal water loss.

Treatment

- Replace free water deficit
- Serum Na—140/140 × total body water
- *Example*: 50 kg man with plasma sodium of 160 mmol/L will have a water deficit of 160–140/140 × 40% of 50 kg = 2.9 L.
- Correct slowly the water deficit over 48–72 hours period.
- Water can be given by mouth or NG tube or IV 5% dextrose or half normal saline.

HYPOKALEMIA

The most common electrolyte abnormality encountered is hypokalemia in clinical practice. It is defined as value less than 3.5 mmol/L. Mild is defined as 3–3.5 mmol/L. Severe if the serum potassium is less than 2.5 mmol/L.

Causes

Though there could be multiple reasons for hypokalemia, the most common reason is potassium loss. The first step is to check the patient's drug record. A whole lot of drugs can lead to hypokalemia and they are given in **Table 2.6**.

- Reduced intake
- Loss

JAYPEE BROTHERS

Table 2.6 Drugs that cause hypokalemia

Transcellular shift	*Increased renal loss*	*Loss in stool*
Beta agonists: Epinephrine Bronchodilators: Terbutaline Tocolytics: Nylidrin Theophylline Caffeine Insulin overdose	Diuretics Mineralocorticoids High dose steroids Antibiotics: Penicillin, ampicillin Magnesium depletion: Cisplatin	Sodium polystyrene sulfonate

  - GI: Vomiting, diarrhea
  - Renal: Diuretics
  - Hyperaldosteronism
  - Renal tubular acidosis
- Redistribution
  - pH change: alkalosis
  - Drugs: Insulin, Beta 2 stimulants

Clinical Features

- Cardiac arrhythmias such as ventricular and atrial arrhythmias. ECG changes in the form of T becomes flat, ST depression and QT prolongation.
- Skeletal muscle: Weakness and paralysis.
- Intestinal smooth muscle weakness can lead to ileus.

Work-up[7]

- Obtain 24 hour urine potassium or spot potassium
- If 24 hour K is > 20 mmol/24 hour or spot K > 15 – renal loss is the reason
- If 24 hour K is < 20 mmol or spot K is less than 15 mmol/L, extrarenal loss
- If renal loss check blood pressure—if hypertensive, it is usually due to hyperaldosternosim or glucocorticosteroid excess. If normotensive, then it is due to diuretics or renal tubular acidosis.
- If non-renal loss of K, need to check on transcellular shifts such as drugs, acute cell proliferation due to acute leukemia or Andersen-Tawil syndrome or due to GI loss.

Treatment

- *Oral Potassium* supplementation is the main treatment. Potassium chloride is available in syrup and tablet forms. The syrup is cheaper but has an unpleasant taste. The tablets are of 2 types—wax matrix one and microencapsulated form. The tablets are well tolerated but can cause GI ulceration which is very rare. The microencapsulated form has less such a complication than the wax type.
- If magnesium deficiency is there, then this should be corrected.

JAYPEE BROTHERS

- *IV Potassium* can be given *in saline solution* if the potassium is less than 2.5 or patient cannot take orally. The maximum amount given should not exceed 20 mmol in 100 mL of normal saline. This is one situation peripheral injection is preferable rather than subclavian or internal jugular because in later type of infusions, cardiac potassium is increased and conduction of the heart can be affected.
- Using a *potassium sparing diuretic* such as amiloride, triamterene or spironolactone can be additional step in the treatment.
- Encouraging the patient to take foods that contain high content of potassium is also helpful. The potassium in the food products is coupled with phosphate not chloride. Hence, it is not useful in correcting chloride depletion that may be seen with diuretics or vomiting **(Table 2.7)**.

HYPERKALEMIA

Hyperkalemia is defined as a plasma potassium level of >5.5 mmol/L. The cut points for severity are: Mild hyperkalemia (>5.0–5.9 mmol/L), moderate–potassium levels of 6.0–7.0 mmol/L and severe—>7.0 mmol/L.

The most serious effect of hyperkalemia is cardiac toxicity. The earliest feature is the tall T waves in the ECG. As the K levels increase, the QRS and PR intervals widen. The wave will resemble sine wave and the terminal event is ventricular fibrillation or asystole.

Work-up

The most common cause for hyperkalemia is due to impaired K excretion **(Flow chart 2.2)**. If the creatinine is more than 5.67 mg/dL then it is due to renal failure. If the plasma creatinine is less than 5.6 mg/dL then we need to look for causes of tubular secretary failure. Next step is to measure the plasma aldosterone values. If the aldosterone is low with hyperkalemia, then Addison's has to be thought of.

Treatment (Table 2.8)

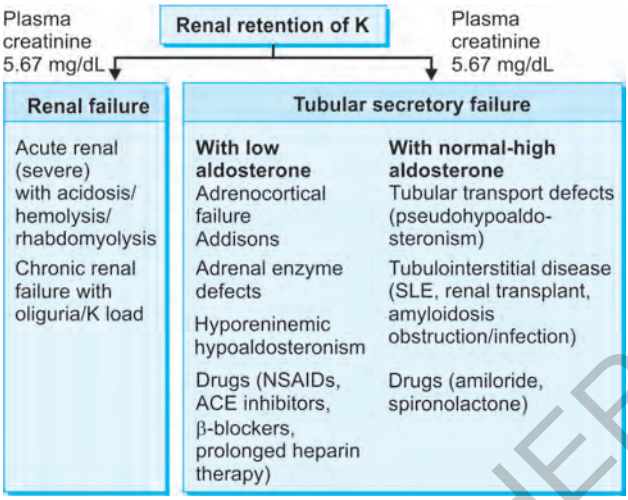
1. Stabilize the membranes: Administration of Calcium gluconate 10 mL of 10% solutions over 2–3 minutes is the first step.

Table 2.7 Foods containing high potassium content[8]

Highest *>1000 mg/100 g*	*Very high* *> 500 mg /100 g*	*High content* *> 250 mg /100 g*
Seaweed Dried figs Molasses	Dried fruits – dates Nuts Avocados Wheat cereal	Banana Kiwis Oranges Mangoes

JAYPEE BROTHERS

Flow chart 2.2 Evaluation of hyperkalemia



2. Shift K into cells: Insulin 5 units of actrapid in 50 mL of 50% glucose.
3. Removal of K from the body is done by furosemide or sodium polystyrene sulfonate orally or rectally (Kayxalate). Patiromer – oral, is new drug that has not yet approved by FDA, exchanges potassium for calcium in the colon and the drug itself is not absorbed. It is 10 times more powerful than kayxalate.[9] Constipation was the main side effect observed with this drug.

MAGNESIUM

The normal Mg level is 1.7–2.4 mg%. Suspect magnesium abnormalities if the patient has refractory hypokalemia, hypocalcemia and ventricular arrhythmia. Hypomagnesemia is defined as plasma level less than 1.7 mg% or 0.7 mmol/L. It occurs in nearly 2/3rd of critically ill patients. The common causes for hypomagnesemia are:

- *GI causes*: Malabsorption
- *Renal*: Renal tubular acidosis
- *Endocrine*: Hyperthyroidism, hyperparathyroidism
- *Others*: Diuretics, cyclosporine, alcoholism.

Clinical Manifestations

- *Cardiac*: Prolonged PR and QT, wide QRS tachycardia
- *Biochemical*: Hypokalemia and hypocalcemia
- *Neurological*: Tetany, convulsions, confusion

Treatment

- Oral if mild symptoms
- IV 10 mmol over 5 min then 20 mmol over 24 hours. Then 4–8 mmol/day.

JAYPEE BROTHERS

Table 2.8 Treatment of hyperkalemia

Drug/ Procedure	*Dose*	*Onset*	*Comments*
Calcium gluconate	1 – 2 amp IV	< 3 min	Transient effect, Lasts for 30–60 min
3% saline	50 mL IV bolus	1 – 3 min	Volume overload, hypertonicity
Insulin	5 – 10 units	15–30 min	Hypoglycemia
Bicarbonate	1 – 3 amps	15 – 30 min	Metabolic alkalosis, volume overload. Useful if hyperkalemia with metabolic acidosis
Beta 2 agonists	Salbutamol IV 0.5 mg over 15 min or as Nebs Terbutaline 7 µg/kg SC	30–60 min	Tachycardia
Kayexate	30 g oral or rectally	2 hours or more	Intestinal obstruction if resin is not passed
Diuretics	Furosemide 40 mg	30 min	Volume depletion
Dialysis		Immediate	1 mEq/L fall in 60 min of dialysis 2 mEq /L in 180 minutes Can precipitate arrhythmia – so monitor when dialysis is done

REFERENCES

1. David MC, David HE. Evaluating hyponatremia. JAMA. 2015; 313:1260-1.
2. Sterns RH. Disorders of plasma sodium – causes and consequences. NEJM. 2015;372:55-65.
3. Frederick HV, Steels P, Grieten L, et al. Hyponatremia in acute decompensated heart failure. JACC. 2015;65:480-92.
4. Verbalis JG, et al. Diagnosis, evaluation and treatment of hyponatremia: Expert panel recommendations. Am J Med. 2013; 126: Suppl 1: s1-s42.
5. Spasovski G, Vanholdrr R, Allolio B, et al. Clinical practice guideline on diagnosis and treatment of hyponatremia. Nephrol Dial Transplant. 2014;29:Suppl 2: ii 1-39.
6. Ingelfinger JR. Disorders of fluids and electrolytes. NEJM. 2015;2207-16.
7. Eugene PR, James AS, Anand SD, Case 4 – 2012: A 37-year-old man with muscle pain, weakness and weight loss. NEJM. 2012; 366:533-60.
8. Gennari J. Hypokalemia. NEJM. 1998;339:451-8.
9. Weir MR, Bakris, Bushinsky, et al. Patirmor in patients with kidney disease and hyperkalemia and receiving RASS inhibitors. NEJM. 2015;372:211-22.

JAYPEE BROTHERS

for detailed information on cardiology books, visit our website www.ejaypee.com, for detailed information on c

Handbook on
Cardiac Critical Care

In the present scenario, voluminous literature is available in the field of cardiology. However, a limited number of books are there in the field of cardiac critical care. This focused handbook is an amalgamation of ideas and views from key opinion leaders in the field. It covers quite a few unique chapters in cardiac critical care, which are not of common knowledge. Mainly it focuses on specific issues faced by Indian physicians in managing Indian patients.



Santanu Guha MD DM FCSI FACC FESC is currently Professor and Head, Department of Cardiology, Government Medical College, Kolkata, West Bengal, India, the oldest premier medical college in Asia. He has a 28 year illustrious career of teaching, both undergraduates and postgraduates. He contributed chapters in reputed textbooks in Cardiology. He authored many well-acclaimed papers in national and international journals. He delivered many prestigious orations in his checkered career. He represented the Cardiological Society of India (CSI) in various international congresses. He is an astute clinician, a must-to-listen orator, an adorable teacher and a much-liked examiner.



S Ramakrishnan MD DM FACC is presently employed as an Additional Professor, Department of Cardiology, All India Institute of Medical Sciences (AIIMS), New Delhi, India. He is actively involved in clinical research, patient care, and educational activities. He has also organized several updates, conferences and pediatric cardiology workshops, and has edited various educational books. He has been awarded with a Fellowship of the American College of Cardiology (ACC). He is the Secretary, Cardiological Society of India (CSI), Delhi branch and a life member of CSI. He is an Executive Editor, Indian Heart Journal. He is also an Assistant Editor of Annals of Pediatric Cardiology, and Secretary of Pediatric Cardiac Society of India.



Rishi Sethi MD DM FACC FESC FAPSIC FSCAI MAMS FCSI is currently working as Professor, Department of Cardiology, King George's Medical University, Lucknow, Uttar Pradesh, India. He has a distinguished academic and professional career. He has edited and published various educational books in cardiology. He has vast experience in teaching, research and clinical works.

Buy from ejaypee
JAYPEE BROTHERS

Shelving Recommendation
CARDIOLOGY
ISBN 978-93-85891-07-6
9 789385 891076