information on medicine books, visit our website **www.jaypeebrothers.com,** for detailed information on medicine books, visit our website **www.jaypeebrothers.com**

CRITICAL CARE



Chief Editors
Narendra Rungta
Rajesh Pande

Editors
Manish Munjal
Sudhir Khunteta

JAYPEE

Foreword
Shirish Prayag

Contents

Section 1: General

1. **Assessment of Critically Ill Patients** 3
   Kapil Zirpe, Sushma Gurav
2. **Airway Management in ICU** 9
   Anamika Chaudhary, Manish Munjal, Neeru Gaur, Neena Rungta, Narendra Rungta
3. **Hemodynamic Monitoring in ICU** 23
   Vijaya P Patil, Nayana Amin, Vandana Agarwal
4. **Fluid Balance in Critically Ill Patients** 37
   Shikha Panwar
5. **Vasopressors and Inotropes** 46
   Poonam Malhotra Kapoor
6. **Multiple Organ Dysfunction Syndrome** 57
   R Pratheema, R Senthilkumar, N Ramakrishnan
7. **Cardiopulmonary Resuscitation: A Paradigm Shift** 64
   Khusrav Bajan
8. **Therapeutic Hypothermia** 70
   Pinak Ashok Shrikhande
9. **Management of Organ Donor** 75
   Rajesh Pande
10. **Scoring Systems in ICU** 79
    Avijatri Datta, Arindam Kar, Sauren Panja
11. **Guidelines for ICU Planning and Designing in India** 87
    Narendra Rungta, Neena Rungta, Manish Munjal, Neeru Gaur, Anamika Chaudhary
12. **Guidelines and Protocols in ICU** 94
    Manoj Singh, Mehul Solanki
13. **Clinical Audit and Handoff in ICU** 98
    Mohammad Omar Faruq
14. **Critical Care Nursing in India** 104
    Arindam Kar, Sati Chakrabarti, V Ruth Adora Rao, Manish Munjal, Neena Rungta, Narendra Rungta

Section 2: Cardiac Care

15. **Acute Coronary Syndrome** 111
    Suresh Ramasubban

16. **Heart Failure** 117
Yatin Mehta, Mukesh Kumar Gupta

17. **Cardiac Arrhythmias in ICU** 129
Supradip Ghosh

18. **Hypertensive Emergency** 145
Yatin Mehta, Mukesh Kumar Gupta

19. **Pacing in the ICU Setting** 154
Balbir Singh, Lakshmi N Kottu

20. **Pulmonary Embolism** 160
Rajesh Chawla, Manish Bharti

21. **Intensive Care Unit Management of Patients with Right Heart Failure** 169
Harjit Dumra

Section 3: Respiratory Care

22. **Community Acquired Pneumonia** 183
Rajesh Pande, Mir Faisal Mazid

23. **Ventilator-Associated Pneumonia** 190
Manoj K Goel

24. **Acute Respiratory Distress Syndrome** 195
Rajesh Pande, Vipin Kauts

Section 4: Liver and Digestive System

25. **Acute Liver Failure** 203
Utpal Sarma, Vaibhav Kumar Nasa, Amit Kumar Singhal

26. **Acute Pancreatitis** 216
Yogesh Batra, Ashish Garg

27. **Hepatorenal and Hepatopulmonary Syndromes** 226
Rajesh Pande

28. **Anesthesia for Liver Transplantation** 233
Vaibhav Kumar Nasa, Utpal Sarma, Amit Kumar Singhal

29. **Critical Care Aspects in Adult Liver Transplantation** 253
Shrikanth Srinivasan, Deepak Govil

Section 5: Renal Care

30. **Diagnosis of Acute Kidney Injury** 263
Vinay Malhotra

31. **Renal Replacement Therapy** 267
Rajesh Pande

32. Critical Care Management of Renal Transplant Recipients 273
Sachin Gupta, Deepak Govil

33. Acid-Base Disorders in Critical Care 276
Palepu B Gopal, Krishna Prasad Mulavisala

34. Disorders of Potassium 289
Yogendra Pratap Singh, Sanjay Kumar Nihlani, Rahul Kumar Anand

35. Sodium Disorders 296
Yogendra Pratap Singh, Manish Gupta, Akhil Taneja

36. Disorders of Calcium and Magnesium 306
Khalid Khatib, Subhal Dixit, Kapil Borawake

Section 6: Neurological Care

37. Management of Critically Ill Trauma Patients 313
Babita Gupta

38. Management of Spinal Injury 329
Samir Sahu

39. Neurocritical Care Management of Subarachnoid Hemorrhage 337
Mukesh M Gupta, Vimal K Agarwal, Girish Rajpal

40. Intensive Care Management of Postoperative Neurosurgical Patients 346
Mukesh M Gupta

Section 7: Obstetric Critical Care

41. Physiology of Pregnancy 357
Rajesh Chandra Mishra, Pratibha Dileep, Srinivas Samvedam, Sunil P Pandya

42. Respiratory Disorders During Pregnancy 364
Rajesh Chandra Mishra, Pratibha Dileep, Srinivas Samvedam, Sunil P Pandya

43. Liver Disease Complicating Pregnancy 369
Rajesh Chandra Mishra, Pratibha Dileep, Srinivas Samvedam, Sunil P Pandya

44. Peripartum and Postpartum Intensive Care in Pregnancy 372
Rajesh Chandra Mishra, Pratibha Dileep, Srinivas Samvedam, Sunil P Pandya

Section 8: Pediatric Critical Care

45. Recognition and Assessment of Critically Ill Child 381
Praveen Khilnani

46. Pediatric Septic Shock 387
Saptharishi LG, Sunit Singhi

47. Status Epilepticus 398
Mullai Baalaaji AR, Sunit Singhi

48. **Raised Intracranial Pressure in Children with an Acute Brain Injury: Monitoring and Management** 406
R Ramesh Kumar, Sunit Singhi

Section 9: Infections

49. **Extended Spectrum Beta Lactam Producing Infections in Intensive Care Unit** 421
Afzal Azim, AK Baronia

50. **Infections in Immunocompromised Patients in ICU** 427
AK Baronia, Afzal Azim, RK Singh

51. **Invasive Fungal Infections in Critically Ill Patients** 434
AK Baronia, Armin Ahmed, Afzal Azim, Mohan Gurjar, Rungmei Marak

52. **Febrile Neutropenia** 443
Rajesh Pande

53. **Fever in the ICU** 448
Rajesh Pande

54. **Cytomegalovirus Infection in Critically Ill Patients** 454
Armin Ahmed, Afzal Azim, Mohan Gurjar, AK Baronia

55. **Tropical Infections in ICU** 456
Prashant Nasa, Dhruva Chaudhry

56. **Tropical Fever—Management Guidelines ISCCM Tropical Fever Group** 465
Sunit Singhi, Dhruva Chaudhary, George M Verghese, Ashish Bhalla, SP Kalantri, JV Peter, Rajesh Chandra Mishra, Rajesh Bhagchandani, TD Chugh, Narendra Rungta, Manish Munjal

Section 10: Ethics and End-of-Life Care Issues

57. **Bioethical Considerations** 475
RK Mani, Prashant Saxena

58. **End-of-Life Care Practices in the World** 478
RK Mani, Prashant Saxena

Section 11: Miscellaneous

59. **Burns, Inhalation and Electrical Injury** 483
Diptimala Agarwal

60. **Diabetic Ketoacidosis** 494
DK Singh

61. **Oncological Emergencies** 498
Sheila Nainan Myatra, Amol Kothekar

62. **Post-cardiac Arrest Syndrome** 508
Yash Javeri

63. Intra-abdominal Hypertension and Abdominal Compartment Syndrome 516
Rajesh Pande, Puneet Khanna

64. Nutrition in a Critically Ill Patients 521
Pravin Amin

65. Approach to an Unknown Poisoning 525
Vikas Loomba, Ashish Bhalla

66. Specific Intoxications 531
Ajeet Singh, Dhruva Chaudhry

67. Fatal Envenomations 543
Prashant Kumar, Dhruva Chaudhry

68. Care of Obese Patient in ICU 550
Gaurav Kochhar, Abhinav Gupta

69. Imaging in Intensive Care Unit 555
Chander Mohan, Rajul Rastogi

Section 12: Mechanical Ventilation

70. Respiratory Mechanics: Basics 569
Rajesh Pande, Vipin Kauts

71. Principles of Mechanical Ventilation 573
Rajesh Pande, Maitree Pande

72. Basic Modes of Ventilation 576
Maitree Pande, Rohini Arora

73. Ventilator Graphics 580
Rajesh Pande, Puneet Khanna

74. Newer Modes of Ventilation 587
Rajesh Pande, Vipin Kauts

75. Weaning/Liberation from Mechanical Ventilation 592
Rajesh Pande, Monish Nakra

76. Non-Invasive Ventilation 596
Rajesh Pande, Rakesh Gupta

77. Ventilation Strategy in Obstructive Airway Disease 602
Rajesh Pande, Atul Pathak

78. Ventilation Strategy in Trauma 605
Vikas Maurya

79. Rescue Strategies in ARDS—Recruitment Maneuvers 610
Rajesh Pande, Anupam Basumatari

80. Rescue Therapy in ARDS: Prone Ventilation and High Frequency Oscillation Ventilation (HFOV) 613
Rajesh Pande, Abhishek Vishnu

CHAPTER

4

Fluid Balance in Critically Ill Patients

Shikha Panwar

INTRODUCTION

Fluid balance is closely related to acid base balance which is maintained by kidneys and various neuroendocrine and autonomic mechanisms. The normal fluid and acid base balance can be disturbed by various diseases. Disease is accompanied not only by changes in the balance of water and electrolytes between the body and its environment (external balance) but also by changes in the relationship between the fluid compartments within the body and the fluxes between them (internal balance). Disease also influences the cardiovascular and renal responses to fluid and electrolyte intake through autonomic and neuroendocrine mechanisms. Intake of fluid and electrolytes versus the output from the kidneys, the gastrointestinal tract, and the skin and lungs (insensible loss) all are important for an integrated balance of fluids.

In the human body the total body water is around 60% of total body weight. It is then further divided as per its distribution to 20% extracellular and 40% intracellular compartment which are again subdivided into the intravascular 6% (within the circulation) and the interstitial 14% fluid (extravascular fluid surrounding the cells) (Fig. 1).

The volume of fluid (water) within a compartment is determined by cell membrane permeability, osmolarity and electroneutrality. Cell membrane permeability refers to the ability of a cell membrane to allow certain substances such as water and urea to pass freely, while charged ions such as sodium cannot cross the membrane and are trapped on one side of it. Osmolarity is a property

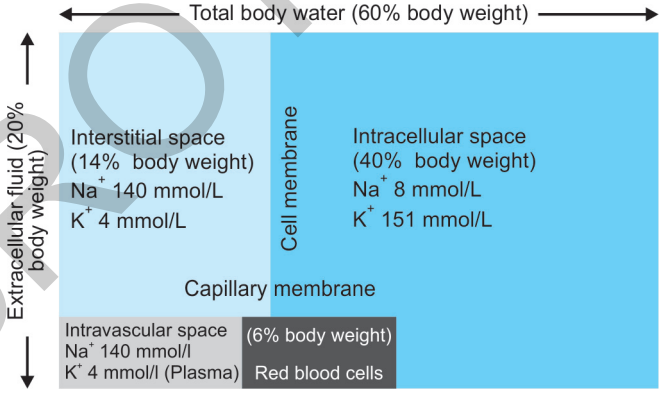


Fig.1: Body fluid compartments with approximate electrolyte concentrations. Red blood cells (hematocrit) account for approximately 45% of total intravascular volume.

of particles in solution. If a substance can dissociate in solution, it may contribute more than one equivalent to the osmolarity of the solution. For instance, NaCl will dissociate into two osmotically active ions: Na^+ and Cl^-. One millimolar NaCl yields a 2 milliosmolar solution. Electroneutrality means that the overall number of positive and negative charges balances. For instance, in conditions like renal tubular acidosis where carbonic acid is lost, chloride is retained leading to a hyperchloremic state. Expected osmolarity of plasma can be calculated according to the following formula:

Plasma osmolarity = 2(Na)+(Glucose/18)+(BUN/2.8) {Glucose and BUN are measured in mg/dl).

Concentration of sodium is the major determinant of serum osmolarity. Normal serum osmolarity ranges from about 280 mOsm to 295 mOsm.

JAYPEE BROTHERS

Components of the extracellular fluid (ECF) are separated by the capillary membrane, with its micropores, which allow only a slow escape rate of albumin (5% per hour), which is then returned to the circulation via the lymphatics at the same rate, thereby maintaining a steady state of equilibrium. Osmotic forces of "trapped" solutes govern water distribution between compartments and volume. While the hydrostatic pressure within the circulation tends to drive fluid out, the oncotic pressure of the plasma proteins, e.g. albumin, draws fluid in and maintains the relative constancy of the plasma volume as a proportion of the ECF (Starling effect).

There is also a clinically important flux of fluid and electrolytes between the ECF and the gastrointestinal tract involving active secretion and reabsorption of digestive juices. In health there is a mechanism to ensure a constant relationship between them, with a constant flux between these various spaces to maintain the internal fluid balance.

Organs, which sense the changes in osmolality of plasma (osmoreceptors), are located in the hypothalamus and signal the posterior pituitary gland to increase or decrease its secretion of vasopressin or antidiuretic hormone (ADH). Dilution of the ECF, including plasma, by intake of water or fluid of osmolality lower than plasma, causes ADH secretion to fall, so that the distal tubules of the renal glomeruli excrete more water and produce a dilute urine (this dilution requires the permissive effect of glucocorticoid upon the distal tubules and is, therefore, lost in adrenal insufficiency—one of the reasons for the hyponatremia of Addison's disease). Conversely, dehydration causes the ECF to become more concentrated, ADH secretion rises and the renal tubules reabsorb more water, producing concentrated urine. In response to dehydration, the normal kidney can concentrate urea in the urine up to a hundredfold, so that the normal daily production of urea during protein metabolism can be excreted in as little as 500 mL of urine (Table 1).

Table 1: Distribution of various solutes in intracellular fluid (ICF) and extracellular fluid (ECF).

	ICF (mEq/L)	*ECF (mEq/L)*
Cations	Potassium (150–154)	Sodium (135–140)
	Sodium (6–10)	Calcium (5)
	Magnesium (40)	Potassium (4–5)
		Magnesium (3)
Anions	Organic phosphate (100–06)	Chloride (103–105)
	Protein (40–60)	Bicarbonate (24–27)
	Bicarbonate (10–13)	Phosphate (3–5)
	Organic acids (4)	Organic acids (2–5)
	Sulfate (17)	Sulfate (4)

SODIUM

Since the integrity of the ECF volume and its proportion of the total body water are largely dependent on the osmotic effect of Na^+ and its accompanying anions, it is important that the kidneys maintain Na^+ balance within narrow limits. If salt depletion occurs, the ECF along with the plasma volume decreases. Pressure sensors in the circulation are then stimulated and these excite renin secretion by the kidney. This, in turn, stimulates aldosterone secretion by the adrenal gland, which acts on the renal tubules, causing them to reabsorb and conserve Na^+. Conversely, if the intake of Na^+ is excessive, the renin-aldosterone system switches off, allowing more Na^+ to be excreted, until normal balance is restored. The mechanism for salt conservation is extremely efficient and the kidney can reduce the concentration of Na^+ in the urine to less than 5 mmol/L. The mechanism for maintaining sodium balance may become disturbed in disease, leading to Na^+ deficiency or, more commonly, to excessive sodium retention, with consequent edema and adverse clinical outcome.

POTASSIUM

Although only a small proportion of the body's K^+ is in the extracellular space, its concentration has to be maintained within narrow limits (3.5–5.3 mmol/L) to avoid the risk of muscular dysfunction or potentially fatal cardiac events. This is achieved by exchange of K^+ in the renal tubules for Na^+ or H^+, allowing more or less K^+ to be excreted. In the presence of K^+ deficiency, H^+ ion reabsorption is impaired, leading to hyperchloremic acidosis.

Pathophysiology

In critically ill patients the normal homeostatic mechanisms are deranged and basic life preserving senses, such as thirst and hunger, may be abolished by disease itself or may be hindered by sedation or other treatment modalities. As a result, survival of the critically ill patient depends on the administration of appropriate volume of fluid and quantities of electrolytes and nutrition. Basal requirement apart from the ongoing disease process has to be met by the intravenous (IV) fluid. So the fluid requirement by a patient can be assessed by dividing it into maintenance,

JAYPEE BROTHERS

replacement and resuscitation fluid. If the patient is clinically stable and does not have excess losses above insensible loss he needs only maintenance fluid which is approximately 30mL/kg/24 hours. Patients having to fast for over 8–12 hours should be started on IV maintenance fluid. Replacement of ongoing losses, either previous or current has to be added to the maintenance fluids according to the disease pathology. If losses are predicted, it is best to replace these later rather than give extra fluid in anticipation of losses which may not occur. This fluid is in addition to maintenance fluid. If the patient is hypovolemic as a result of dehydration, blood loss or sepsis he requires urgent correction of intravascular fluid depletion so he has to be given rapid resuscitation fluids according to guidelines.

Intravenous fluids are chemically prepared solutions that are administered to the patient. They are tailored to the body's needs and used to replace lost fluid and/or aid in the delivery of IV medications venous fluids. The choice of IV fluid will depend upon a number of factors. The most important is the type of fluid that has been lost, the electrolyte status and the adverse effects of each IV fluid.

Intravenous fluids come in two different forms: (1) colloids and (2) crystalloids.

Colloid solutions are IV fluids that contain solutes in the form of large proteins or other similarly sized molecules. The proteins and molecules are so large that they cannot pass through the walls of the capillaries and onto the cells. Accordingly, colloids remain in the blood vessels for long periods of time and can significantly restore the intravascular volume faster than crystalloids and with lesser quantity. The proteins also have the ability to attract water from the cells into the blood vessels. However, although the movement of water from the cells into the bloodstream may be beneficial in the short-term, continual movement in this direction can cause the cells to lose too much water and become dehydrated.

During sepsis, the membrane permeability is altered resulting in greater movement of colloids into the interstitium. Colloids are expensive, have specific storage requirements, and have a short shelf life.

The clinical relevance of the theoretical advantages of colloids (with regards to effects on the microvasculature) was questioned as early as the late 1970s. The saline versus albumin fluid evaluation (SAFE) trial compared the effects of 4% albumin versus normal saline[1] in 6,997 critically ill patients and found no difference in the primary outcome (all-cause mortality at 28 days). Subgroup analysis revealed a possible association between the use of albumin and increased mortality in patients with traumatic brain injury. It should be noted that this post hoc analysis was performed on only 492 patients or 7% of the original study population. In a follow-up report of 460 traumatic brain injury patients from the original SAFE trial, the association of albumin with higher mortality rates in this group persisted at 2 years following randomization. The current consensus statement of the European Society of Intensive Care Medicine already discourages the use of hydroxyethyl starches (HES) in patients with severe sepsis or acute kidney disease.

Resuscitation with colloid solutions was associated with an absolute increase in the risk of mortality of 4% (95% confidence interval 0–8%), or four extra deaths for every 100 patients resuscitated. As colloids are not associated with improved survival and are considerably more expensive than crystalloids, it is hard to see how their continued use outside randomized controlled trials in subsets of patients of particular concern can be justified.[2]

- Commonly used colloid solutions include plasma protein fraction, salt poor albumin, dextran and hetastarch.

Crystalloid solutions are composed of small particles dissolved in water, which pass easily through the endothelial barrier. Crystalloids contain electrolytes (e.g. sodium, potassium, calcium, chloride) but lack the large proteins and molecules found in colloids. Crystalloids come in many preparations and are classified according to their "tonicity". A crystalloid's tonicity describes the concentration of electrolytes (solutes) dissolved in the water, as compared with that of body *plasma* (fluid surrounding the cells).

Isotonic crystalloids have a tonicity *equal* to the body plasma. When administered to a normally hydrated patient, isotonic crystalloids do not cause a significant shift of water between the compartments.

Hypertonic crystalloids have a tonicity *higher* than the body plasma. The administration of a hypertonic crystalloid causes water to shift from the extravascular spaces into the bloodstream, increasing the intravascular volume. This osmotic shift occurs as the body attempts to dilute the higher concentration of electrolytes contained within the IV fluid by moving water into the intravascular space.

Hypotonic crystalloids have a tonicity *lower* than the body plasma. The administration of a hypotonic crystalloid causes water to shift from the intravascular space to the extravascular space, and eventually into the tissue cells. Because the IV solution being administered is hypotonic, it creates an environment where the extravascular

JAYPEE BROTHERS

Table 2: Composition of common intravenous fluids.

Electrolyte	*Plasma*	*0.9% NaCl*	*Lactated Ringer's*	*Dextrose 5%*	*Human albumin*	*Hetastarch*
Sodium	140	154	131	0	145	154
Potassium	5	0	5	0	0	0
Chloride	100	154	111	0	95	154
Calcium	2.2	0	2	0	0	0
Magnesium	1	0	1	0	0	0
Bicarbonate	24	0	0	0	0	0
Lactate	1		28	0	0	0
mOsm/L		308	273	278	300	310

Table 3: Volume of infusion required to expand the plasma volume by 1 litre.[4]

	Infused Volume (mL)	*Interstitial Volume (mL)*	*Intracellular Volume (mL)*
0.9% normal saline	4,700	+3,700	
5% dextrose	14,000	+3,700	+9,300
5% albumin	1,000		
25% albumin	250	–750	

spaces have higher concentrations of electrolytes. The osmotic change results in the body moving water from the intravascular space to the cells in an attempt to dilute the electrolytes.

Of the different types of IV solutions, crystalloids are the mainstay of resuscitation (Table 2). The most common isotonic solutions used are:

- *Lactated Ringer's* (LR) is an isotonic crystalloid that contains NaCl, potassium chloride, calcium chloride, and sodium lactate in sterile water.
- *Normal saline solution* (NS) is an isotonic crystalloid that contains 0.9% NaCl (salt) in sterile water.
- *Dextrose 5% in water* (D5W) is packaged as an isotonic carbohydrate (sugar solution) that contains glucose (sugar) as the solute.

Lactated Ringer's and NS are commonly used for fluid replacement because of their immediate ability to expand the volume of circulating blood. However, over the course of about 1 hour, approximately two-thirds of these IV fluids eventually leave the blood vessels and move into the cells. The ability of a solution to expand the plasma volume is dependent on its volume of distribution and the metabolic fate of the solute, so that while colloids are mainly distributed in the intravascular compartment, once the dextrose is metabolized, dextrose containing solutions are distributed through the total body water and hence have a limited and transient blood volume expanding capacity (Table 3 and Fig. 2).

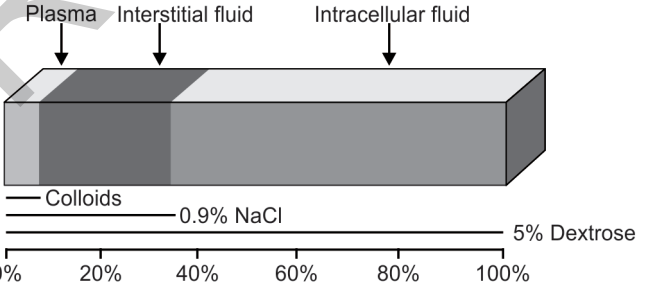


Fig. 2: Distribution of infused fluids in the body water compartments.

One myth regarding crystalloid selection deserves further assessment—calcium-containing solutions are not absolutely contraindicated in the setting of transfusion. Most studies demonstrating clotting were based on citrate-phosphate-dextrose-adenine preserved packed red blood cells; newer preservatives, such as AS-3, contain less plasma and more citrate, and are much less likely to be overwhelmed by the small amount of calcium present in isotonic crystalloids.[3]

The cornerstone of treating patients with shock remains as it has for decades—IV fluids. Surprisingly, dosing IV fluid during resuscitation of shock remains largely empirical.

JAYPEE BROTHERS

Too little fluid may result in tissue hypoperfusion and worsen organ dysfunction; however, over-prescription of fluid also appears to impede oxygen delivery and compromise patient outcome. Recent data suggest that early aggressive resuscitation of critically ill patients may limit and/or reverse tissue hypoxia, progression to organ failure, and improve outcome.[5] In a landmark study, Rivers et al. demonstrated that a protocol of early goal-directed therapy reduces organ failure and improves survival in patients with severe sepsis and septic shock.[6] Similarly, a protocol to optimize preload and cardiac output in patients undergoing major surgery reduced postoperative complications and length of stay.[7] Uncorrected hypovolemia, leading to inappropriate infusions of vasopressor agents may increase organ hypoperfusion and ischemia.[8]

However, overzealous fluid resuscitation has been associated with increased complications; increased length of intensive care unit (ICU) and hospital stay, and increased mortality. The first step in the hemodynamic management of critically ill patients is to determine the adequacy of tissue/organ perfusion. Although the signs of shock may be obvious, those of subclinical hypoperfusion may be more subtle (Box 1).

In those patients with indices of inadequate tissue perfusion, fluid resuscitation is generally regarded as the first step in resuscitation. Clinical studies have, however, demonstrated that only approximately 50% of hemodynamically unstable critically ill patients are volume-responsive. The resuscitation of the critically ill patient requires an accurate assessment of the patient's intravascular volume status (cardiac preload) and the likelihood that the patient will respond (increase stroke volume) to a fluid challenge (volume responsiveness).

Box 1: Clinical indices of the adequacy of tissue/organ perfusion.

- Mean arterial pressure—cerebral and abdominal perfusion pressures
- Urine output
- Mentation
- Capillary refill
- Skin perfusion/mottling
- Cold extremities (and cold knees)
- Blood lactate
- Arterial pH, BE and HCO_3
- Mixed venous oxygen saturation ($SmvO_2$) or central venous oxygen saturation ($ScvO_2$)
- Mixed venous pCO_2
- Tissue pCO_2
- Skeletal muscle tissue oxygenation (StO_2)

Fluid challenge has to be given to hypotensive patient presenting either in the emergency, or in clinical scenario in the ICU. How much fluid and how rapidly it is to be administered, and which fluid is to administered is very crucial for a good outcome. Clinical examination may yield important clue to the requirement of fluids, signs of dehydration like diminished skin turgor, thirst, dry mouth, hypernatremia, elevated hematocrit are suggestive of fluid deficit. Circulatory signs of hypovolemia like tachycardia, hypotension, elevated lactate along with signs of decreased renal perfusion like concentrated urine, increased BUN all suggest fluid deficit. A 500 mL of 0.9% saline or balance solutions (plasmalyte) is to be administered over 15–20 minutes; Partial resuscitation using albumin may be considered in hypoalbuminemic patients and in patients with chronic liver disease, heart failure, where large volume crystalloid resuscitation may be very challenging. In the light of present evidence, the use of HES is not recommended in patients with severe sepsis or septic shock.

Fluid challenge may always not be successful. It is effective only in 50% of the cases as various meta-analyses have shown. So the various indices used to predict fluid responsiveness are static and dynamic indices.

Static indices:

- Central venous pressure (CVP)
- Pulmonary artery occlusion pressure (PAOP)

Dynamic indices:

- Passive leg raising (PLR)
- Pulse pressure variation (PPV), or stroke volume variation (SVV)
- *Ultrasonography*: Inferior vena cava (IVC) collapsibility and 2D echocardiogram of the heart chamber.

Central venous pressure has been traditionally used as a guide to fluid therapy. If the CVP is less, then CVP guided fluid challenge can be given as described in Table 4:

Table 4: Fluid challenge: CVP, cm H_2O ("5–2" rule).

Observe CVP for 10 min	<8 cm H_2O	200 mL × 10 min
	<14 cm H_2O	100 mL × 10 min
	>14 cm H_2O	50 mL × 10 min
During infusion 0–9 min	>5 cm	STOP
Following infusion	>2 cm and <5 cm	Wait 10 min
	>2 cm	Wait STOP
	<2 cm	Continue infusion

(CVP: Central venous pressure).

JAYPEE BROTHERS

Table 5: Critical components of the fluid challenge—two possible types of response are presented.

	Example 1			*Example 2*		
	Baseline	+10 min	+20 min	Baseline	+10 min	+20 min
1. *Type of fluid*: Normal saline						
2. *Rate of infusion*: 500 mL/30 min						
3. *Clinical end points*: MAP of 65 mm Hg	MAP 50	MAP 60	MAP 65	MAP 50	MAP 60	MAP 60
4. *Pressure safety limit*: CVP of 15 mm Hg	CVP 6	CVP 8 Continue	CVP 12 Stop	CVP 9	CVP 14 Continue	CVP 15 Stop
		Successful fluid challenge			Unsuccessful fluid challenge	

(CVP: Central venous pressure; MAP: Mean arterial pressure).

the "5–2" rule, which was popularized in the 1970s, is still widely used today for guiding fluid therapy. According to this rule, the change in CVP after a fluid challenge is used to guide subsequent fluid management decisions (Table 5).

The CVP is a good approximation of right atrial pressure, which is a major determinant of right ventricular (RV) filling. It has been assumed that the CVP is a good indicator of RV preload. Furthermore, because RV stroke volume (SV) determines left ventricular (LV) filling, the CVP is assumed to be an indirect measure of LV preload. However, due to the changes in venous tone, intrathoracic pressures, LV and RV compliance, and geometry that occur in critically ill patients, there is a poor relationship between the CVP and RV end-diastolic volume. Furthermore, the RV end-diastolic volume may not reflect the patients' position on the Frank-Starling curve and therefore his/her preload reserve.

More than 100 studies have been published to date that have demonstrated no relationship between the CVP (or change in CVP) and fluid responsiveness in various clinical settings.[9,10] Based on this information, we believe that the CVP should no longer be routinely used for guiding fluid management in the ICU, operating room or emergency room.

Until recently, it has been unclear as to which hemodynamically unstable patients are volume-responsive and likely to benefit from fluid resuscitation (fluid boluses). However, during the past decade, a number of dynamic tests of volume responsiveness have been reported. These tests dynamically monitor the change in SV after a maneuver that increases or decreases venous return (preload). These tests allow the clinician to determine the individual patient's position on his/her Frank-Starling curve, and thus determine whether the patient is likely to be fluid-responsive. These techniques use the change in SV during mechanical ventilation or after a PLR maneuver to assess fluid responsiveness.

Spontaneous breathing: During spontaneous breathing systolic blood pressure fluctuates, normally by 5–10 mm Hg also known as "respiratory swing". Pulsus paradoxus is a phenomenon in which the difference in systolic blood pressure between inspiration and expiration is more than 10 mm Hg. Reverse pulsus paradoxus occurs during controlled mechanical ventilation when arterial pressure rises during inspiration and falls during expiration due to changes in intrathoracic pressure secondary to positive pressure ventilation.

Passive Leg Raising

Lifting the legs passively from the horizontal position induces a gravitational transfer of blood from the lower limbs toward the intrathoracic and the volume of blood transferred to the heart during PLR is sufficient to increase the left cardiac preload and thus challenge the Frank-Starling curve. Apart from being easy to use, this method has the advantage of reversing its effects once the legs are tilted down. Therefore, PLR may be considered a reversible "autotransfusion".

The best way to perform a PLR maneuver to predict volume responsiveness is to elevate the lower limbs to 45° (automatic bed elevation or wedge pillow) while at the same time placing the patient in the supine from a 45° semirecumbent position. This induces a larger increase in cardiac preload because it induces the shift of venous blood not only from both the legs but also from the abdominal compartment. The maximal hemodynamic effects of PLR occur within the first minute of leg elevation, it is important to assess these effects by a direct measure of cardiac output or SV (Fig. 3).

JAYPEE BROTHERS

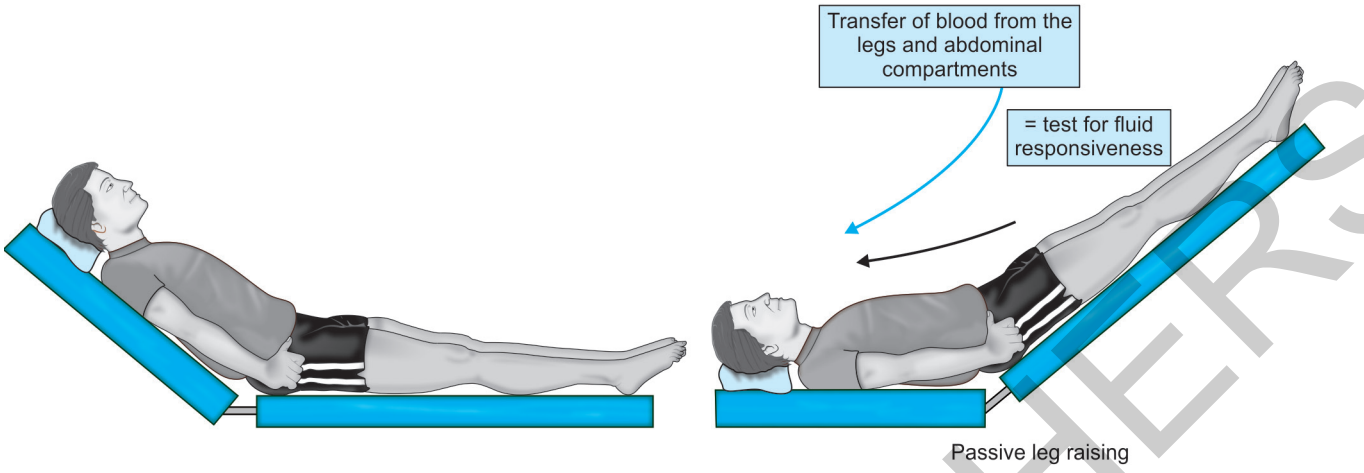


Fig. 3: Passive leg raising. The passive leg raising test consists in measuring the hemodynamic effects of a leg elevation up to 45°. A simple way to perform the postural maneuver is to transfer the patient from the semirecumbent posture to the passive leg raising position by using the automatic motion of the bed.

Stroke Volume Variation or Pulse Pressure Variation

An impressive number of studies have demonstrated that the PPV derived from analysis of the arterial waveform, the SVV derived from pulse contour analysis, and the variation of the amplitude of the pulse oximetry plethysmographic waveform to be highly predictive of fluid responsiveness. A variation of greater than 12–13% has been reported to be highly predictive of volume responsiveness.[11]

Stroke volume variation is a naturally occurring phenomenon in which the arterial pulse pressure falls during inspiration and rises during expiration due to changes in intrathoracic pressure secondary to negative pressure ventilation (spontaneously breathing). Variations over 10 mm Hg have been referred to as pulsus paradoxus. The normal range of variation in spontaneously breathing patients has been reported between 5 mm Hg and 10 mm Hg. Reverse pulsus paradoxus is the same phenomenon with controlled mechanical ventilation, however, in reverse. Arterial pressure rises during inspiration and falls during expiration due to changes in intrathoracic pressure secondary to positive pressure ventilation. In addition to reverse pulsus paradoxus, it has also been referred to as paradoxical pulsus, respiratory paradox, systolic pressure variation and PPV. Traditionally SVV is calculated by taking the $SV_{max} - SV_{min}/SV_{mean}$ over a respiratory cycle or other period of time. SVV and its comparable measurement, PPV, are not indicators of actual preload but of relative preload responsiveness (Fig. 4).

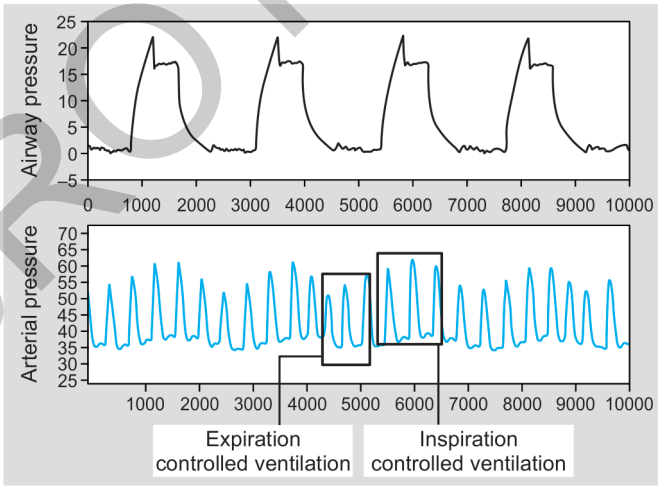


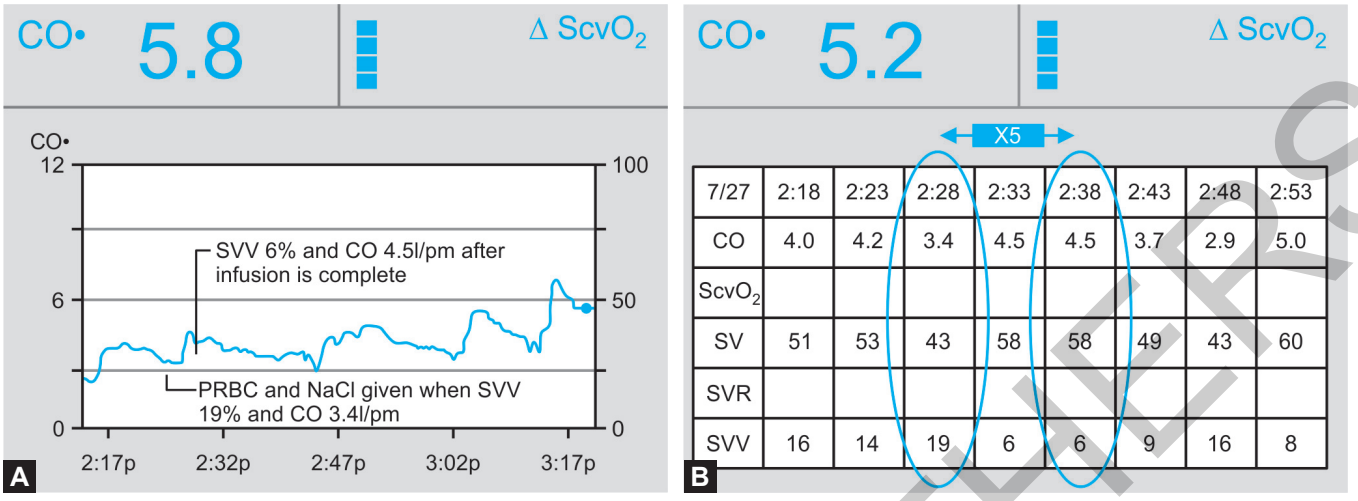
Fig. 4: Pulse pressure variation. The arterial pulse pressure falls during inspiration and rises during expiration.

Normal SVV values are less than 10–15% on controlled mechanical ventilation. Figures 5A and B demonstrate using SVV as a guide for volume resuscitation with a goal SVV of less than 13%. SVV increased to 19% with a SV of 43 mL/beat, blood and saline were given to obtain an SVV of 6% and an SV of 58 mL/beat.

The SVV has got its limitations:

- *Mechanical ventilation*: SVV can be used only on patients who are 100% mechanically (control mode) ventilated with tidal volumes of more than 8cc/kg and fixed respiratory rates.
- *Spontaneous ventilation*: SVV cannot be used on with patients who are spontaneously breathing due to the irregular nature of rate and tidal volumes.

JAYPEE BROTHERS



Figs 5A and B: Stroke volume variation.

- *Arrhythmias*: Arrhythmias can dramatically affect SVV values. Thus, the utility of SVV as a guide for volume resuscitation is greatest in absence of arrhythmias.
- *Positive end-expiratory pressure*: Increasing levels of positive end-expiratory pressure may cause an increase in SVV, the effects of which may be corrected by additional volume resuscitation if warranted.
- *Vascular tone*: The effects of vasodilatation therapy may increase SVV and should be considered before treatment with additional volume.

Ultrasound modalities to evaluate preload sensitivity include transthoracic echocardiography, transesophageal echocardiography (TEE), esophageal Doppler and ultrasound of the IVC.

Inferior Vena Cava Diameter Variation

Respiratory change in IVC diameter is an accurate predictor of fluid responsiveness in septic patients.[12] The IVC is a highly compliant vessel with no valves and can be easily distended. The IVC can be identified using either a standard cardiac or abdominal two-dimensional (2D) transducer by a subcostal approach (Fig. 6).[13] Measurements should be made at the hepatic segment of the IVC just cephalad to the origin of the hepatic vein. The IVC is identified with 2D imaging. The respiratory variation in the diameter of the IVC is measured from a frozen M-mode image using the electronic calipers of the machine coincident with ventilator cycling (Fig. 7). Respiratory variation of IVC size in a passive patient on mechanical ventilation in regular heart rhythm has been described as a method of determining volume responsiveness that is easily mastered by the critical care physician.

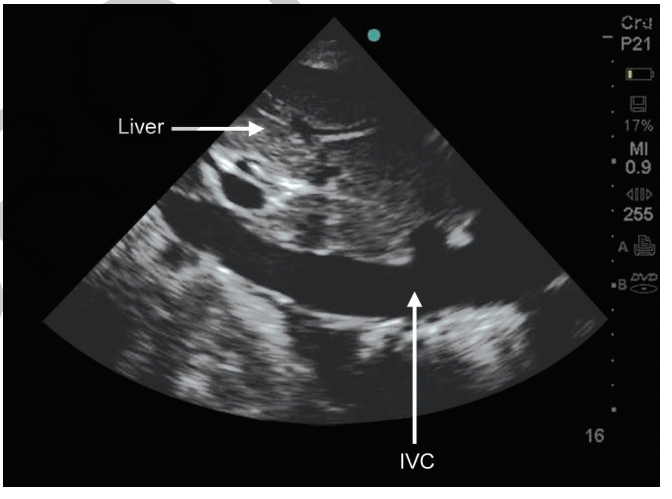


Fig. 6: Longitudinal image of the IVC using a subcoastal view.

A limitation of IVC variation use in determining fluid responsiveness is that the patient must be passive in their interaction with the ventilator. Intra-abdominal hypertension may invalidate this method.

Transthoracic Echocardiography or Transesophageal Echocardiography

One of the major applications of bedside echocardiography is the determination of preload sensitivity.

Ventricular Size

In the patient with shock, the finding of a small hyperdynamic LV with effacement of the end-systolic cavity ("kissing LV walls") strongly suggests major preload sensitivity and is a simple visual clue for immediate volume

JAYPEE BROTHERS

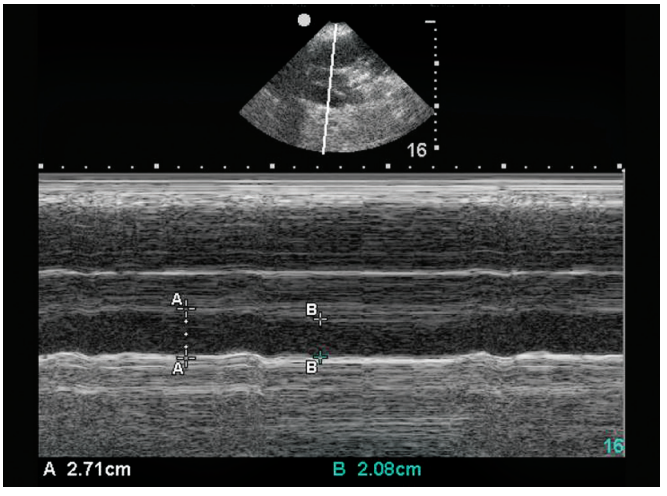


Fig. 7: Measurement of respiratory variation in the diameter of the IVC from a frozen M-mode image. The diameter of the IVC is calculated as [(A – B)/B].

resuscitation of a hypotensive patient. This pattern can be easily identified during goal directed cardiac ultrasound from a parasternal long, short or subcostal view. Cannesson et al.[14] measured changes in LV diastolic area with TEE in the short axis view finding that a 16% variation of LV diastolic area between inspiration and expiration was predictive of preload sensitivity.

CONCLUSION

Fluid balance is an integrated function of various systems working in tandem to maintain an internal milieu, sepsis and septic shock can lead to a gross imbalance in the fluid state of the body. Role of colloids is limited and their use may cause more harm to the patient than benefit, so continued use of colloids is not justified. Fluid challenge and early goal directed therapy is warranted to prevent further damage by septic shock. Fluid challenge responsiveness should be assessed by using various static and dynamic parameters. Dynamic parameters are gaining more and more popularity due to the increased sensitivity in predicting the responsiveness to fluid challenge. IVC collapsibility and echocardiography are tools which can be easily mastered and can give very accurate measurement of preload and response to fluid challenge, so increasing use of new technology is warranted more in emergency room and ICUs.

REFERENCES

1. Finfer S, Bellomo R, Boyce N, et al. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. N Engl J Med. 2004;350(22):2247-56.
2. Schierhout G, Roberts I. Fluid resuscitation with colloids or crystalloid solutions in critically ill patients; a systematic review of randomised trials BMJ. 1998;316(7136): 961-4.
3. Albert K, van Vlymen J, James P, et al. Ringer's lactate is compatible with the rapid infusion of AS-3 preserved packed red blood cells. Can J Anaesth. 2009;56(5):352-6.
4. Lamke LO, Liljedahl SO. Plasma volume changes after infusion of various plasma expanders. Resuscitation. 1976;5(2): 93-102.
5. Levy MM, Macias WL, Russell JA, et al. Failure to improve during the first day of therapy is predictive of 28-day mortality in severe sepsis. Chest. 2004;124(Suppl):120 S.
6. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med. 2001;345(19):1368-77.
7. Lopes MR, Oliveira MA, Pereira VO, et al. Goal-directed fluid management based on pulse pressure variation monitoring during high-risk surgery: a pilot randomized controlled trial. Crit Care. 2007;11(5):R100.
8. Murakawa K, Kobayashi A. Effects of vasopressors on renal tissue gas tensions during hemorrhagic shock in dogs. Crit Care Med. 1988;16(8):789-92.
9. Michard F, Taboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. Chest. 2002; 121(6):2000-8.
10. Marik PE, Baram M, Vahid B. Does the central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. Chest. 2008;134 (1):172-8.
11. Marik PE, Cavallazzi R, Vasu T, et al. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients. A systematic review of the literature. Crit Care Med. 2009;37(9): 2642-7.
12. Barbier C, Loubières Y, Schmit C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. Intensive Care Med. 2004;30(9):1740-6.
13. Feissel M, Michard F, Faller JP, et al. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. Intensive Care Med. 2004;30(9):1834-7.
14. Cannesson M, Slieker J, Desebbe O, et al. Prediction of fluid responsiveness using respiratory variations in left ventricular stroke area by transesophageal echocardiographic automated border detection in mechanically ventilated patients. Crit Care. 2006;10(6):R171.

JAYPEE BROTHERS

for detailed information on medicine books, visit our website **www.jaypeebrothers.com,** for detailed information on medicine books, visit our website **www.jaypeeb**

CRITICAL CARE

Salient Features

- Critical Care is a lucid, illustrated, specialty book, which emphasizes on practical aspects in critical care.
- Eminent Indian critical care physicians from top hospitals and institutions have contributed to the book. It gives a glimpse of the critical care as practiced in India.
- Includes topics that are relevant to India like poisoning, tropical infections, pregnancy-related critical illness and the end-of-life care issues.
- Also covers the practical aspects of mechanical ventilation.
- This book would not only provide essential information to the postgraduate trainees and Superspeciality fellows (IDCCM, IFCCM, FNB, DM) but will also benefit the practicing physicians, who are exposed to the ICU in their practice.



Narendra Rungta MD FISCCM FCCM FICCM is an illustrious alumni of SMS Medical College, Jaipur, Rajasthan, and entered into Critical Care at a time when there were no formal training programs in India. He was fascinated by ISCCM programs and conferences and trained at various institutions in India and abroad for short periods of time. Being awarded with FCCS Instructorship in 2001, stimulated him to train doctors all over India in the basics of critical care. Criticare 2003 at Jaipur proved to be another milestone as organizing secretary. He is a recognized teacher in critical care and has served the ISCCM in various capacities. He has been the President of the Indian Society of Critical Care Medicine and was instrumental in developing guidelines on Tropical fever and ICU planning and designing in India, as Chairman of the Guidelines Committee. He was also elected the council member of APACCM and then became founder President of Association of SAARC Critical Care Societies. These positions gave him wide opportunity to travel abroad as a participant and speaker in International conferences and programs connected to Critical Care. Being selected to speak at World Congress at Seoul was special to him.



Rajesh Pande MD PDCC (SGPGIMS) FICCM FCCM (USA) is an alumnus of GSVM Medical College, Kanpur, and Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, Uttar Pradesh. He is a well-known Indian expert in Critical Care, who is currently working as Director, Critical Care and Emergency Medicine at BLK Superspeciality Hospital, New Delhi. He has earlier been associated with many prestigious institutions like AIIMS, SGPGIMS, Sir Ganga Ram Hospital and Fortis Hospital. He is a recognized teacher to various critical care courses like Fellowship of National Board (FNB), Indian Fellowship (IFCCM) and Diploma in Critical Care Medicine (IDCCM), as well as the Indian Diploma in Critical Care Nursing. He is a fellow of the Indian College of Critical Care Medicine and the American College of Critical Care Medicine. His area of interest includes infection control and ventilation in the critically-ill patients.



Manish Munjal MD FICCM is an alumnus of SMS Medical College, Jaipur and is currently the Vice President at Jeevan Rekha Hospital in Jaipur, Rajasthan. He is a member of Editorial Board of Indian Journal of Critical Care Medicine, an indexed journal published monthly by the Indian Society of Critical Care Medicine and Associate Editor, Critical Care Communication, the Newsletter of ISCCM. He is a Fellow of Indian College of Critical Care Medicine (FICCM) and has served as the Secretary of National Executive of the Indian Society of Critical Care Medicine. In the last 12 years, he has successfully organized 125 Fundamentals of Critical Care Support (FCCS), 12 Pediatric Fundamentals of Critical Care Support (PFCCS) and 5 Beginner's All-purpose Symbolic Instruction Code (BASIC) Provider & Instructor Courses in India. He has been honored by many societies for his contribution in intensive care. He is a life member of ISCCM, SCCM (USA), ISA, IACTA, ITACCS, IMA and MPS.



Sudhir Khunteta MD is an alumnus of SMS Medical College, Jaipur and JLN Medical College, Ajmer, Rajasthan. He is presently working as Chief Internist and Critical Care Specialist with Shubh Hospital, Jaipur, Rajasthan. He has organized many critical care courses like BLS, FCCS for the nurses and healthcare providers. He was involved in the organization of the International Critical Care Congress in 2003 and the 18th Asia-Pacific Congress of Critical Care in 2014. He is also the author of the book Critical Care 2003. He has served in the national executive of the Indian Society of Critical Care Medicine in various capacities and has contributed to the development of its website.



Available at all medical bookstores
or buy online at www.jaypeebrothers.com

JAYPEE BROTHERS
Medical Publishers (P) Ltd.
www.jaypeebrothers.com

Join us on facebook.com/JaypeeMedicalPublishers

Shelving Recommendations
CRITICAL CARE
INTERNAL MEDICINE

ISBN 978-93-5152-213-3