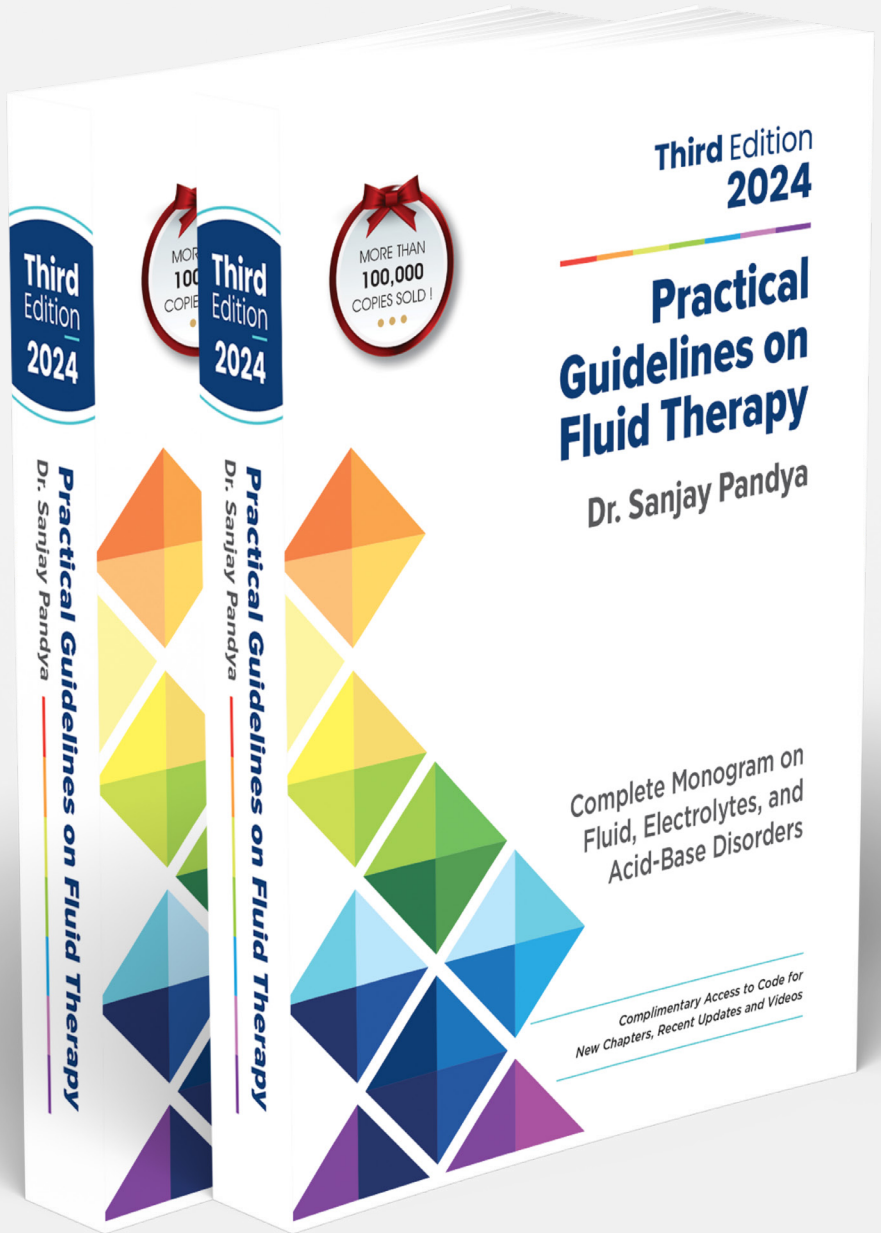
fluid therapy.org

Chapter 15:

Fluid Assessment and Monitoring



To get a copy of the book, visit: **www.fluidtherapy.org**

15 Fluid Assessment and Monitoring

Both inadequate or excessive fluid replacement is detrimental [1, 2], and optimum fluid replacement is beneficial and, at times, lifesaving. So, meticulous evaluation and, on its basis, planning of proper administration of intravenous (IV) fluid is critical.

The accurate assessment of body fluid volume status requires proper history, clinical examination, hemodynamic monitoring, and laboratory investigations (Table 15.1). There is no single parameter that alone can precisely assess the hydration status [3]. Therefore, after initial resuscitation, frequent reassessment and monitoring are mandatory for the appropriate subsequent fluid administration.

HISTORY

Detailed history provides valuable information about body fluid volume status and associated illnesses. Important history to be elicited are:

1. Fluid intake (volume and type of oral, nasogastric, or intravenous fluids).
2. Abnormal loss of fluid (diarrhea, vomiting, drains, high fever, and insensible losses).
3. Urine volume.
4. Symptoms of hypovolemia (increased thirst, oliguria, fatigue, weakness, or dizziness on standing).
5. Symptoms of hypervolemia (swelling, weight gain, shortness of breath with aggravation on exertion or lying down flat).
6. Coexisting illness and comorbidities (diabetes, hypertension, ischemic heart diseases, congestive heart failure, kidney failure, cirrhosis of the liver, hypoproteinemia, and malnutrition).
7. Medication (antihypertensive, diuretics, laxatives).

Table 15.1 Assessment of volume status

- History
- Clinical examination and monitoring
- Hemodynamic monitoring
- Laboratory investigations

CLINICAL EXAMINATION

It is essential to assess the volume status of the patient by proper physical examination. Based on the clinical examination, the predictability of the severity of volume depletion is poor. However, in critical patients with severe

volume depletion and shock, prompt clinical assessment and proper medical history are sufficient to initiate the right treatment [4]. It is essential to know the features of hypovolemia and hypervolemia to determine the volume status.

Signs of hypovolemia: Signs of volume depletion on clinical evaluation do not always correlate with the severity of hypovolemia. However, the features, based on the degree of extracellular fluid (ECF) volume depletion, are summarized below.

Mild volume depletion (<5% reduction of ECF)

- Diminished skin turgor
- Concentrated urine
- Loss of weight

Moderate volume depletion (5 to 10% reduction of ECF), as above plus

- Oliguria (<400 ml/day)
- Orthostatic tachycardia and hypotension (fall of ≥20 mm Hg systolic blood pressure or 10 mm Hg diastolic blood pressure)

Severe volume depletion (>10% reduction of ECF), as above plus

- Hypotension
- Low pulse volume, tachycardia, tachypnea
- Cold extremities, dry tongue, sunken eyeballs
- Prolonged capillary refill time (>2 seconds), and reduced skin turgor (doughy feel)
- Abnormal mental status and confusion

Difference between dehydration and hypovolemia: Terms dehydration and hypovolemia are often confusing and used interchangeably. But dehydration and hypovolemia are fundamentally different clinical disorders with different pathogenesis, clinical presentations, biochemical features, and management [5, 6]. The term dehydration refers to pure water loss producing hypertonicity and intracellular volume contraction. While hypovolemia refers to a combined loss of salt and water, causing extracellular fluid volume deficit and contraction of the blood volume. Characteristic features of dehydration are normal blood pressure, no orthostatic hypotension, and hypernatremia. Hypovolemia is characterized by orthostatic hypotension, tachycardia, decreased skin turgor, and normal or low serum sodium.

Signs of hypervolemia: Common signs of volume overload on clinical evaluation are:

- Peripheral edema, sacral edema in bedridden, and recent weight gain.
- Distended jugular vein.
- The third spacing of intravascular fluid causing ascites and pleural fluid.
- Tachycardia, tachypnea, and orthopnea.
- On auscultation, basal crepitation in the chest and third heart sounds.

MONITORING OF FLUID BALANCE

Important steps to monitor fluid balance are:

1. Intake and output chart: Maintain a proper chart of fluid administered (enterally or parenterally) and fluid losses (urine output, abnormal losses such as diarrhea, vomiting, or losses from gastrointestinal drainage tubes, and insensible losses) to assess the fluid balance.

2. Urine flow rate: Maintaining hourly urine output is a standard practice in all hemodynamically unstable patients. Urine output reflects tissue perfusion and is an important indicator of hydration (in the absence of glycosuria, osmotic diuresis, or diuretic therapy). Fluid therapy aims to achieve a urine output of approximately 0.5 mL/kg/h or more.
3. Daily weight: The day-to-day weight chart is an accurate indicator for detecting the changes in the patient's volume status. Weight gain suggests fluid excess, while weight loss suggests fluid deficit. The inability to obtain weight is a limitation in sick patients.

HEMODYNAMIC MONITORING

Hemodynamic monitoring is a cornerstone in the management of hemodynamically unstable patients. The goal of hemodynamic monitoring is to ensure optimal tissue perfusion and to optimize the oxygenation of the tissues. Less than 50% of hemodynamically unstable critical patients are 'fluid responders', and in the rest 50% of patients, fluid administration may be harmful [7, 8], and therefore proper evaluation before administration of fluid is advisable.

Various hemodynamic monitoring techniques and parameters ranging from simple bedside examination to advanced complex methods are available (Table 15.2).

The selection of modality varies depending on the severity of the underlying disease, resources and local expertise available at each institution, predictability of the modality, and cost-effectiveness.

Table 15.2 Techniques and parameters used for hemodynamic monitoring

A. Basic and non-invasive hemodynamic monitoring techniques Non-invasive blood pressure measurement, pulse oximetry, continuous electrocardiography, ultra-sonogram, echocardiography, and X-ray chest
B. Static hemodynamic monitoring techniques Inferior vena cava assessment, central venous pressure monitoring, arterial cannulation, and pulmonary artery catheter monitoring
C. Dynamic hemodynamic monitoring **1. Provocative techniques to detect fluid responsiveness** The fluid challenge, passive leg raising, and end-expiratory occlusion test **2. Dynamic parameters to predict fluid responsiveness** Pulse pressure variation, stroke volume variation, cardiac output, and plethysmographic variability index **3. Methods and monitors used for the assessment** **a. Noninvasive cardiac output monitoring** Transthoracic echocardiography, bioimpedance or bioreactance, radial applanation tonometry, volume clamp method, ultrasound cardiac output monitoring (USCOM), and plethysmographic variability index **b. Minimally invasive cardiac output monitoring** Transesophageal echocardiography, transpulmonary thermodilution, lithium dilution, arterial pulse contour analysis, and partial CO_2 rebreathing **c. Invasive cardiac output monitoring** Pulmonary thermodilution (Intermittent bolus or continuous)

There is no single hemodynamic technique or parameter which is enough to provide sufficient information in all patients [9]. Less invasive systems are safer and therefore preferred for the initial evaluation. But invasive methods provide more predictable information and therefore are preferred in critical, unstable patients or patients with shock who do not respond to initial therapy.

Non-critical patients who are hemodynamically stable require non-invasive monitoring techniques such as continuous ECG monitoring, frequent non-invasive blood pressure measurement, and peripheral pulse oximetry to assess their oxygen saturation.

The limitations of this primary monitoring modalities is a less precise or inaccurate data, but it avoids risks of harmful invasive approaches.

On the other hand, hemodynamically unstable critical patients need more precise or highly accurate data, so they require advanced hemodynamic monitoring approaches such as arterial pulse contour analysis, transesophageal echocardiography, and transpulmonary thermodilution for continuous hemodynamic monitoring [3, 10].

Noninvasive echocardiography and ultrasonogram are currently rapidly growing and preferred modalities for the initial hemodynamic assessment of shock and perioperative evaluation instead of more invasive technologies.

Dynamic parameters such as pulse pressure variation (PPV), stroke volume variation (SVV), and cardiac output (CO) are shown to be more accurate for the assessment of the volume status. Therefore, they are preferred in predicting fluid responsiveness over static invasive measures (e.g., central venous pressure and pulmonary artery catheters) [3].

However, monitoring these dynamic parameters is not routinely recommended for patients in shock who are responding well to initial treatment. Complex, high-risk patients who do not respond to initial fluid administration need measurements of dynamic parameters to evaluate the response to fluids or inotropes [3].

LABORATORY INVESTIGATIONS

Various laboratory investigations performed considering the clinical context for the monitoring of fluid therapy are:

- Routine investigations: CBC, BUN/creatinine, transaminases, and serum electrolytes.
- Additional investigations: Serum lactate level, arterial blood gas (ABG) analysis, cardiac biomarkers, coagulation profile, and urinary electrolytes.

Serum lactate

Serum lactate estimation is the most useful and valuable laboratory parameter for monitoring critically ill patients, and the high lactate level is strongly associated with the severity of sepsis [11, 12].

Elevated lactate is multifactorial: Increased serum lactate level reflects tissue hypoxia is a misconception [13]. Increased serum lactate level is a non-specific finding which can occur due to multiple causes such as impaired tissue oxygenation, stimulation of beta-2 adrenergic receptors due to increased aerobic glycolysis, medications (adrenaline, beta-2 agonists), liver failure, thiamine deficiency, or other causes [14]. So always interpret the value of serum lactate carefully in the clinical context.

Clinical application of lactate measurement

- A tool to diagnose severe sepsis or septic shock: The measurement

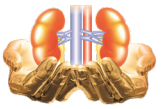
of lactate is useful to establish the diagnosis of severe sepsis [15] and is included in the definition of recent major sepsis guidelines such as National Quality Forum Sepsis Update [16], and third international consensus definitions for sepsis and septic shock [17, 18].

- Prognostic marker on admission: Increased lactate level in the pre-hospital measurement or the emergency department is associated with increased mortality even in patients with initial normal vital signs [18–22]. So early elevated lactate is useful to detect occult shock, uncover subtle organ hypoperfusion, and help to detect patients who are at higher risk for deterioration and require aggressive management.
- As a component of resuscitative algorithms: Serum lactate is included as a component of resuscitative algorithms in different guidelines (e.g., Surviving Sepsis Campaign Bundle and Surviving Sepsis Campaign International Guidelines) [23, 24].
- Lactate monitoring for a shock: In addition to clinical assessment, trends in the blood lactate levels are useful to guide resuscitation [25]. Serial serum lactate measurements during resuscitation predict mortality among septic shock patients [26, 27]. So, serum lactate level is generally remeasured every 6 hours until it becomes normal [27, 28]. Lactate-guided therapy significantly reduces hospital mortality [29–31] and has a greater mortality benefit than even early goal-directed therapy [32].
- Lactate normalization predicts a favorable prognosis: Normalization of lactate within 6 hours of initial resuscitation is the strong predictor of survival [33–36]. In a recent study, even delayed normalization of lactate (i.e., within 24 hours), independently predicts decreased mortality [37].
- Lactate clearance as a prognostic marker: Early lactate clearance reduces in-hospital mortality and strongly predicts the survivor [33, 38–41]. On the contrary, persistent hyperlactatemia increases morbidity and mortality and is a strong adverse prognostic factor [38, 42, 43].

REFERENCES

1. Mandal M. Ideal resuscitation fluid in hypovolemia: The quest is on and miles to go! Int J Crit Illn Inj Sci. 2016;6(2):54–55.
2. Claure-Del GR, Mehta RL. Fluid overload in the ICU: evaluation and management. BMC Nephrol 2016;17(1):109.
3. Cecconi M, De Backer D, Antonelli M, et al. Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. Intensive Care Med. 2014;40(12):1795–1815.
4. Marx G, Schindler AW, Mosch C, et al. Intravascular volume therapy in adults: Guidelines from the association of the scientific medical societies in Germany. Eur J Anaesthesiol 2016:33(7):488–522.
5. Bhave G, Neilson EG. Volume depletion versus dehydration: how understanding the difference can guide therapy. AJKD 2011;58(2):302–309.
6. Asim M, Alkadi MM, Asim H, et al. Dehydration and volume depletion: How to handle the misconceptions. World J Nephrol. 2019;8(1):23–32.
7. Marik PE, Monnet X, Teboul JL. Hemodynamic parameters to guide fluid therapy. Ann Intensive Care. 2011;1(1):1.
8. Bentzer P, Griesdale DE, Boyd J, et al. Will this hemodynamically unstable patient respond to a bolus of intravenous fluids? JAMA. 2016;316(12):1298–309.
9. Vincent JL, Rhodes A, Perel A, et al. Clinical review: Update on hemodynamic monitoring - a consensus of 16. Crit Care. 2011;15(4):229.
10. Aseni P, Orsenigo S, Storti E. et al. Current concepts of perioperative monitoring in high-risk surgical patients: a review. Patient Saf Surg 2019;13:32.
11. Gutiérrez HB, Concepción YA, Pérez JS1, et al. Prognostic value of serum lactate levels in critically ill patients in an intensive care unit. J Crit Care Med (Targu Mures). 2020;6(1):59–64.
12. Gattinoni L, Vasques F, Camporota L, et al. Understanding lactatemia in human sepsis: potential impact for early management. Am J Respir Crit Care Med 2019;200(5):582–589.
13. Levy B. Lactate and shock state: the metabolic view. Curr Opin Crit Care 2006;12(4):315–321.

14. Levy B, Desebbe O, Montemont C, et al. Increased aerobic glycolysis through beta2 stimulation is a common mechanism involved in lactate formation during shock states. Shock. 2008;30(4):417–21.
15. Fuller BM, Dellinger RP. Lactate as a hemodynamic marker in the crtically ill. Curr Opin Crit Care 2012;18(3):267–72.
16. National Quality Forum (NQF). #0500 Severe Sepsis and Septic Shock: Management Bundle, Last Updated: Jan 05, 2015 https://emcrit.org/wp-content/uploads/2015/06/0500.pdf Accessed May 23, 2020.
17. Shankar-Hari M, Phillips GS, Levy ML, et al. Developing a new definition and assessing new clinical criteria for septic shock: For the third international consensus definitions for sepsis and septic shock (Sepsis-3). JAMA 2016;315(8):775–87.
18. Singer M, Deutschman CS, Seymour CW, et al. The third international consensus definitions for sepsis and septic shock (Sepsis-3). JAMA 2016;315(8):801–10.
19. Jansen TC, van Bommel J, Mulder PG, et al. The prognostic value of blood lactate levels relative to that of vital signs in the pre-hospital setting: a pilot study. Crit Care. 2008;12(6):R160.
20. Mikkelsen ME, Miltiades AN, Gaieski DF, et al. Serum lactate is associated with mortality in severe sepsis independent of organ failure and shock. Crit Care Med. May 2009;37(5):1670–1677.
21. Soliman H, Vincent JL. Prognostic value of admission serum lactate concentrations in intensive care unit patients. Acta Clinica Belgica. 2010;65(3):176–81.
22. Casserly B, Phillips GS, Schorr C, et al. Lactate measurements in sepsis-induced tissue hypoperfusion: results from the Surviving Sepsis Campaign database. Crit Care Med. 2015;43(3):567–573.
23. Levy MM, Evans LE, Rhodes A. The surviving sepsis campaign bundle: 2018 update. Intensive Care Med 2018;44(6):925–928.
24. Rhodes A, Evans LE, Alhazzani W, et al. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock: 2016. Intensive Care Med. 2017;43(3):304–77.
25. Weiss SL, Peters MJ, Alhazzani W, et al. Surviving sepsis campaign international guidelines for the management of septic shock and sepsis-associated organ dysfunction in children. Pediatr Crit Care Med. 2020;21(2):e52–e106.
26. Ryoo SM, Lee J, Lee YS, et al. Lactate level versus lactate clearance for predicting mortality in patients with septic shock defined by Sepsis-3. Crit Care Med 2018;46(6):e489–e495.
27. Ryoo SM, Ahn R, Lee J, et al. Timing of repeated lactate measurement in patients with septic shock at the emergency department. Am J Med Sci 2018;356(2):97–102.
28. Martin ND, Codner P, Greene W, et al. Trauma Surg Acute Care Open 2020;5(1):e000411.
29. Jansen TC, van Bommel J, Schoonderbeek FJ, et al. Early lactate-guided therapy in intensive care unit patients: a multicenter, open-label, randomized controlled trial. Am J Respir Crit Care Med. 2010;182(6):752–761.
30. Jones AE, Shapiro NI, Trzeciak S, et al. Lactate clearance vs central venous oxygen saturation as goals of early sepsis therapy: a randomized clinical trial. JAMA. 2010;303(8):739–746.
31. Lyu X, Xu Q, Cai G, et al. Efficacies of fluid resuscitation as guided by lactate clearance rate and central venous oxygen saturation in patients with septic shock. Zhonghua Yi Xue Za Zhi. 2015;95(7):496–500.
32. Ding XF, Yang ZY, Xu ZT, et al. Early goal-directed and lactate-guided therapy in adult patients with severe sepsis and septic shock: a meta-analysis of randomized controlled trials. J Transl Med. 2018;16(1):331.
33. Nguyen HB, Rivers EP, Knoblich BP, et al. Early lactate clearance is associated with improved outcome in severe sepsis and septic shock. Crit Care Med 2004;32(8):1637–42.
34. Puskarich MA, Trzeciak S, Shapiro NI, et al. Whole blood lactate kinetics in patients undergoing quantitative resuscitation for severe sepsis and septic shock. Chest. 2013;143(6):1548–1553.
35. Jones AE. Lactate clearance for assessing response to resuscitation in severe sepsis. Acad Emerg Med. 2013;20(8):844–847.
36. Chertoff J, Chisum M, Garcia B, et al. Lactate kinetics in sepsis and septic shock: a review of the literature and rationale for further research. J Intensive Care 2015;3:39.
37. Ryoo SM, Ahn R, Shin TG, et al. Lactate normalization within 6 hours of bundle therapy and 24 hours of delayed achievement were associated with 28-day mortality in septic shock patients. PLoS ONE 2019;14(6):e0217857.
38. Arnold RC, Shapiro NI, Jones AE, et al. Multicenter study of early lactate clearance as a determinant of survival in patients with presumed sepsis. Shock. 2009;32(1):35–9.
39. Zhang Z, Xu X. Lactate clearance is a useful biomarker for the prediction of all-cause mortality in critically ill patients: a systematic review and meta-analysis. Crit Care Med 2014;42(9):2118–25.
40. Gu WJ, Zhang Z, Bakker J. Early lactate clearance-guided therapy in patients with sepsis: a meta-analysis with trial sequential analysis of randomized controlled trials. Intensive Care Med 2015;41(10):1862–3.
41. Simpson SQ, Gaines M, Hussein Y, et al. Early goal-directed therapy for severe sepsis and septic shock: A living systematic review. J Crit Care 2016;36:43–48.
42. Zhang Z, Chen K, Ni H, et al. Predictive value of lactate in unselected critically ill patients: an analysis using fractional polynomials. J Thorac Dis. 2014;6(7):995–1003.
43. Dezman ZDW, Comer AC, Smith GS, et al. Failure to clear elevated lactate predicts 24-hour mortality in trauma patients. J Trauma Acute Care Surg. 2015;79(4):580–585.

KidneyEducation 

Join the Mission to Fight Kidney Diseases

Explore the world's largest multilingual website created by a global team of over 100 nephrologists.

www.KidneyEducation.com

» Read online or download the 200-page book "Save Your Kidneys" in 40 languages—completely free.

» This comprehensive resource offers valuable information on preventing and managing common kidney problems, tailored for kidney patients and their families.

» It's an authentic guide, prepared by nephrologists and free from any external funding.