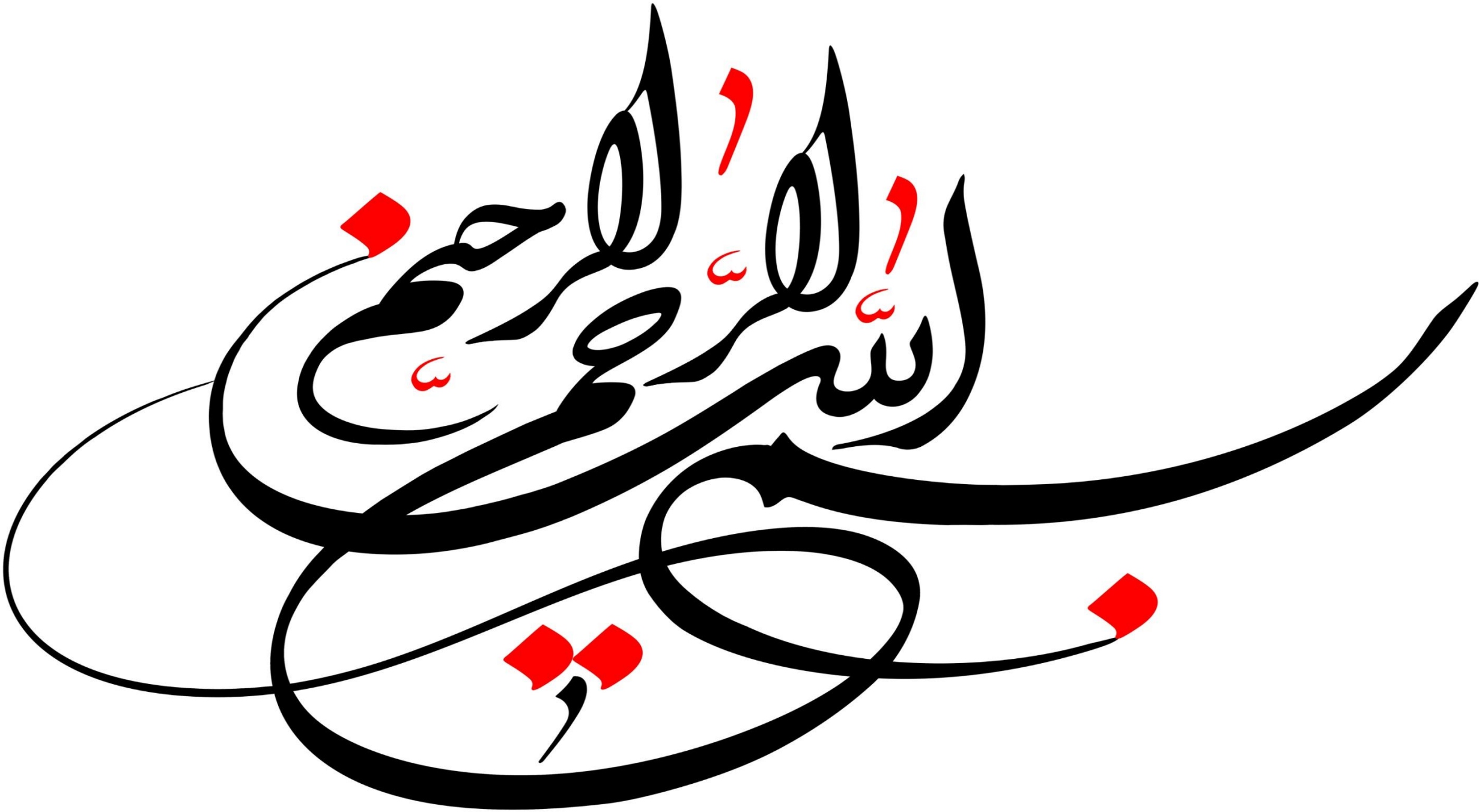

hypovolemic shock

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INTRODUCTION

- Shock is a life-threatening condition of circulatory failure, causing inadequate oxygen delivery to meet cellular metabolic needs and oxygen consumption requirements, producing cellular and tissue hypoxia.
- effects of shock are initially reversible, but rapidly become irreversible, resulting in multiorgan failure (MOF) and death.
- When a patient presents with undiferentiated shock, it is important to immediately initiate therapy and rapidly identifying the etiology so that definitive therapy can be administered to reverse shock and prevent MOF and death.

DEFINITION

- Shock is defined as a state of cellular and tissue hypoxia due to:
  - Reduced oxygen delivery
  - increased oxygen consumption
  - inadequate oxygen utilization, or a combination of these
- This most commonly occurs when there is circulatory failure manifested as hypotension (ie, reduced tissue perfusion); however a patient in shock can present hypertensive, normotensive, or hypotensive.
- Shock is initially reversible, but must be recognized and treated immediately to prevent progression to irreversible organ dysfunction.
- "***Undiferentiated shock***" refers to the situation where shock is recognized but the cause is unclear

CLASSIFICATION AND ETIOLOGY

- Four types of shock are recognized:
  1. distributive
  2. cardiogenic
  3. Hypovolemic
  4. obstructive
- However, these are not exclusive, and many patients with circulatory failure have a combination of more than one form of shock (multifactorial shock)

EPIDEMIOLOGY

1. Septic shock, a form of distributive shock, is the most common form of shock among patients admitted to the intensive care unit
2. Cardiogenic shock
3. hypovolemic shock
4. obstructive shock is rare

As an example, in a trial of 1600 patients with undiferentiated shock:

- septic shock occurred in 62 percent
- cardiogenic shock in 16 percent
- hypovolemic shock in 16 percent
- other types of distributive shock in 4 percent (eg, neurogenic shock, anaphylaxis)
- obstructive shock in 2 percent

Hypovolemic

- Hypovolemic shock is due to reduced intravascular volume (ie, reduced preload), which, in turn, reduces CO.
- Hypovolemic shock can be divided into two categories:
  - hemorrhagic
  - nonhemorrhagic

Hemorrhagic

- Reduced intravascular volume from blood loss can result in shock.
- There are multiple causes of hemorrhagic shock:
  - blunt or penetrating trauma (includes multiple fractures without vessel injury) is the most common
  - upper (eg, variceal hemorrhage, peptic ulcer)
  - lower (eg, diverticular, arteriovenous malformation) gastrointestinal bleeding
  - Less common causes

Less common causes:

- intraoperative and postoperative bleeding
- Ruptured abdominal aortic or left ventricle aneurysm
- aortic–enteric fistula
- hemorrhagic pancreatitis
- iatrogenic (eg, inadvertent biopsy of arteriovenous malformation, severed artery)
- tumors or abscess erosion into major vessels
- postpartum hemorrhage
- uterine or vaginal hemorrhage from other causes (infection, tumors, lacerations)
- spontaneous peritoneal hemorrhage from bleeding diathesis
- ruptured hematoma

Nonhemorrhagic

- Reduced intravascular volume from fluid loss other than blood
- Volume depletion from loss of sodium and water can occur from a number of anatomic sites :

  - ✓ Gastrointestinal losses (diarrhea, vomiting, external drainage)
  - ✓ Skin losses (heat stroke, burns, severe dermatologic conditions including Stevens-Johnson syndrome)
  - ✓ Renal losses (excessive drug-induced or osmotic diuresis, salt-wasting nephropathies, hypoaldosteronism)
  - ✓ Third space losses into the extravascular space or body cavities (postoperative and trauma, intestinal obstruction, crush injury, pancreatitis, cirrhosis)

Patients often present with combined forms of shock:

- Patients with shock from sepsis or pancreatitis primarily have:

1-distributive shock (due to the effects of inflammatory and anti-inflammatory cascades on vascular permeability and peripheral vasodilation)

2-hypovolemic component (due to decreased oral intake, insensible losses, vomiting, diarrhea)

3- cardiogenic component (due to inflammation-related myocardial depression).

- Patients with underlying cardiomyopathy may present with :

1- hypovolemic shock (from over-diuresis)

2- cardiogenic shock (from inadequate compensatory tachycardia and/or stroke volume).

- Patients with severe traumatic injury may have :

1- hemorrhagic shock from blood loss

2- distributive shock from SIRS or, less commonly, fat embolism

CLINICAL MANIFESTATIONS

- Hypovolemic patients may present with a variety of symptoms, physical examination findings, and laboratory abnormalities.
- Symptoms may be related to :
  - ✓ volume depletion itself, such as lassitude and postural dizziness
  - ✓ to the underlying cause of volume depletion, such as vomiting, diarrhea, or polyuria

- physical examination :
  - ✓ decreased skin turgor
  - ✓ low arterial blood pressure or postural hypotension
  - ✓ reduced jugular venous pressure

- laboratory abnormalities:
  - ✓ elevated serum creatinine and blood urea nitrogen (BUN)
  - ✓ hypernatremia or hyponatremia
  - ✓ hyperkalemia or hypokalemia
  - ✓ metabolic alkalosis or metabolic acidosis

Symptoms

Three sets of symptoms may occur in hypovolemic patients:

- Those due to volume depletion
- Those related to the cause of fluid loss
- Those due to the electrolyte and acid-base disorders that can accompany volume depletion

DIAGNOSIS

- hypovolemia is clinical diagnosis based upon characteristic manifestations and confirmed by a **low urine sodium concentration**.
- An accurate history and physical examination not only provides evidence for the presence of volume depletion but may also help determine the etiology.
- In most individuals, the history should identify the source of fluid loss.
- In older adults, the history may not identify the cause of hypovolemia.
  Such elements of the history may be absent in these patients because of confusion or cognitive issues.

- A low urine sodium concentration (or low urine chloride concentration in patients who have metabolic alkalosis) is **strongly suggestive** of reduced tissue perfusion, and it is usually present in hypovolemic patients unless there is :
  - Salt wasting state (diuretics, underlying kidney disease),
  - selective renal ischemia(acute glomerulonephritis or bilateral renal artery stenosis),
  - very low sodium diet

- presence of a low urine sodium does not necessarily mean that the patient has true volume depletion →
  - ✓ edematous patients with heart failure
  - ✓ cirrhosis with ascites
  - ✓ nephrotic syndrome also avidly conserve sodium
- These disorders are characterized by reduced effective arterial blood volume due to a primary reduction in cardiac output (heart failure)
- to splanchnic vasodilatation and sequestration of fluid in the peritoneal cavity and arterial shunts (cirrhosis)
- to a low plasma oncotic pressure (in some patients with severe or acute nephrotic syndrome)

Less specific laboratory tests

- Other laboratory tests can provide evidence for the presence of hypovolemia or reduced effective arterial blood volume, but are less specific than a low urine sodium or chloride concentration:
  - fractional excretion of sodium
  - the urine osmolality
  - specific gravity
  - urinalysis

Treatment of severe hypovolemia or hypovolemic shock in adults

- Fluid resuscitation is the mainstay of therapy in patients with severe
hypovolemia.
- severe hypovolemia may be present when loss of blood or extracellular fluids results in decreased peripheral perfusion.
- Hypovolemic shock is considered present when severe hypovolemia
results in organ dysfunction as the result of inadequate tissue perfusion.
- In patients with severe hypovolemia or hypovolemic shock, delayed fluid therapy can lead to ischemic injury and irreversible shock with multiorgan system failure

PRINCIPLES OF MANAGEMENT

Management of hypovolemia involves :

- assessing and treating the underlying cause
- identifying electrolyte and acid-base disturbances
- assessing and treating the volume deficite

all of which influence the choice of fluid and rate at which it should be administered

Identify and treat the etiology

- Clinicians should identify the etiology (or etiologies) contributing to hypovolemia so that therapies can be directed at the underlying cause of volume loss.
- Potential etiologies of hypovolemia include gastrointestinal, renal, skin, hemorrhage, and third-space losses.
- Therapies may include :
  - anti-emetics to treat vomiting
  - cessation of diuretics
  - controlling bleeding

Identify electrolyte and acid-base disturbances

- Biochemical analysis will alert the clinician to electrolyte (hypo- or hypernatremia, hypo- or hyperkalemia) and acid-base disturbances (contraction alkalosis, metabolic acidosis) which may affect choice of replacement fluid and rate of repletion.
- In some cases, an arterial blood gas may be needed if mixed acid-base disturbance is suspected

Assess the volume deficit

Clinical assessment involves:

- estimating pre- and post-deficit body weight
- assessment of clinical and laboratory parameters including:
  - blood pressure
  - jugular venous pressure
  - urine sodium concentration, urine output
  - Lactate
  - if bleeding has not occurred, hematocrit
- Ultrasonographic assessment of collapse of the inferior vena cava on inspiration may be useful in some situations

CHOICE OF REPLACEMENT FLUID

For patients with hypovolemic shock, three major classes of replacement fluids are:

- Crystalloid solutions (eg, saline solutions, bufered [ie, also known as balanced or chloride-restrictive] solutions [eg, Lactated Ringer, Plasma-Lyte, bicarbonate bufered 0.45 percent saline]).
- Colloid-containing solutions (albumin solutions, hyperoncotic starch, dextran, gelatin).
- Blood products (packed red blood cells) or blood substitutes

- The choice of replacement fluid depends in part upon the type of fluid that has been lost
- For example, blood components are indicated in patients who are bleeding.
- Isotonic crystalloids are generally preferred for the management of patients with severe volume depletion not due to bleeding

Hemorrhagic shock

Blood products :

- The mainstay of therapy for patients with intravascular volume depletion due to bleeding is the replacement of volume loss with blood products, typically packed red blood cells.
- Patients may receive fluid replacement, usually with crystalloid (eg, 0.9 percent saline), while waiting for blood products.

Nonhemorrhagic shock

- isotonic or near-isotonic crystalloids (0.9 percent saline solutions with or without dextrose, bufered crystalloids [eg, Lactated Ringer, bicarbonate bufered 0.45 percent saline])
- colloid-containing solutions (albumin solution, hyperoncotic starch, dextran, gelatin) can be used to effectively replace the extracellular fluid deficit.
- Hyperoncotic starch solutions, although effective, should be avoided since they increase the risk of acute kidney injury (AKI), need for renal replacement therapy, and mortality

- First-line: Crystalloid solutions
- Second-line: Colloid solutions

First-line: Crystalloid solutions

- In general, for patients with severe volume depletion or hypovolemic shock not due to bleeding, crystalloids are typically preferred over colloid-containing solutions.
- Among the crystalloids, normal saline (0.9 percent saline) is the most commonly used solution for initial repletion

Choosing between 0.9 percent saline and buffered crystalloid

- Normal saline (0.9 percent saline) is hyperchloremic relative to plasma
- large volume resuscitation using 0.9 percent saline may be associated with hyperchloremic metabolic acidosis
- isotonic fluids with lower chloride concentration be used instead of 0.9 percent saline for large volume resuscitation and include:
  - fluids such as Lactated Ringer solution (or Hartmann solution), 0.45 percent saline solution with 75 mmol/L of sodium bicarbonate.

- choice between bufered solutions and normal saline is individualized:
  - patient chemistries
  - estimated volume of resuscitation
  - potential adverse effect of the solution used (eg, hyponatremia [Lactated Ringer] and hyperchloremic acidosis [normal saline])
  - institutional and clinician preference.

Normal saline (crystalloid)

- For most patients, normal saline (0.9 percent saline) , which contains 154 mEq/L of sodium chloride, is an effective and inexpensive initial resuscitation fluid for the management of patients with hypovolemia and hypovolemic shock not due to bleeding
- Complications of 0.9 percent saline : hyperchloremic acidosis

  peripheral edema (extravascular distribution)
- Hyperchloremia associated with 0.9 percent saline also associated with hyperkalemia due to transcellular shifts of potassium.

Bufered crystalloid

- alternative as either the initial resuscitation fluid or as a secondary fluid to be used if large volumes of resuscitation fluids are necessary
- or if hyperchloremic acidosis is a concern.
- contain small amounts of potassium, their contribution to extracellular potassium concentration is small unless very large volumes are infused.
- have nearly the same plasma-expanding properties as isotonic crystalloid solutions

- As with 0.9 percent saline, administration of bufered crystalloids may be associated with the development of peripheral edema.
- Because the available bufered crystalloids are modestly hypotonic, they are more commonly associated with development of hyponatremia.

Second-line: Colloid solutions

- rarely used as first-line resuscitative fluids for the management of hypovolemia and hypovolemic shock not due to bleeding.
- However, some clinicians advocate the administration of colloid solutions, particularly albumin, in those with limited response to crystalloid solutions or those in whom hypoalbuminemia is thought to be contributing to shock, although data to support these indications are limited.
- Hyperoncotic starch should be avoided since its use is associated with an increased risk of kidney dysfunction and mortality

Albumin

- use of hyper-oncotic (20 to 25 percent) albumin has been used in individuals with intravascular volume depletion but total body volume overload, such as patients with cirrhosis. However, little data exists to support this strategy
- Intravenous albumin can expand the intravascular volume effectively but it is expensive and data have not shown consistent benefit with its use as an initial resuscitation fluid compared with crystalloids.

Fluids to avoid: hyperoncotic starch (colloid)

- Hyperoncotic starch solutions are not recommended for patients with hypovolemia.
- Concern has been raised about risks associated with the use of hyperoncotic starch solutions (pentastarch, hydroxyethyl starch [HES])
- increased risk of AKI, and in some studies, increased mortality

INITIAL RATE OF FLUID REPLETION

- The rate of fluid repletion should be individualized depending upon:
  - the underlying etiology and rate of fluid loss
  - estimated total body deficit
  - underlying electrolyte abnormalities
  - predicted future losses
- While there is no one ideal initial rate, many clinicians model the rate of fluid administration on rates similar to those recommended in patients with sepsis and septic shock, although data to support this strategy is lacking

MONITORING THE RESPONSE

- Clinical parameters including:
  - heart rate
  - blood pressure
  - urine output
  - skin turgor
  - mucus membrane integrity
  - mental status
- For most patients, the period of observation lasts for the duration of fluid resuscitation (eg, 6 to 48 hours, longer for ongoing fluid loss).
- While there are no recommended ideal measurable.

- Measuring laboratory parameters including chemistries and lactate level within 6 hours and urinary sodium within 24 hours of replacement
- Use of urine sodium concentration does not apply to edematous patients with heart failure or cirrhosis

►►►the urine sodium concentration is a marker of effective circulating volume depletion but not of the need for more fluid or more salt.

Thanks for your attention