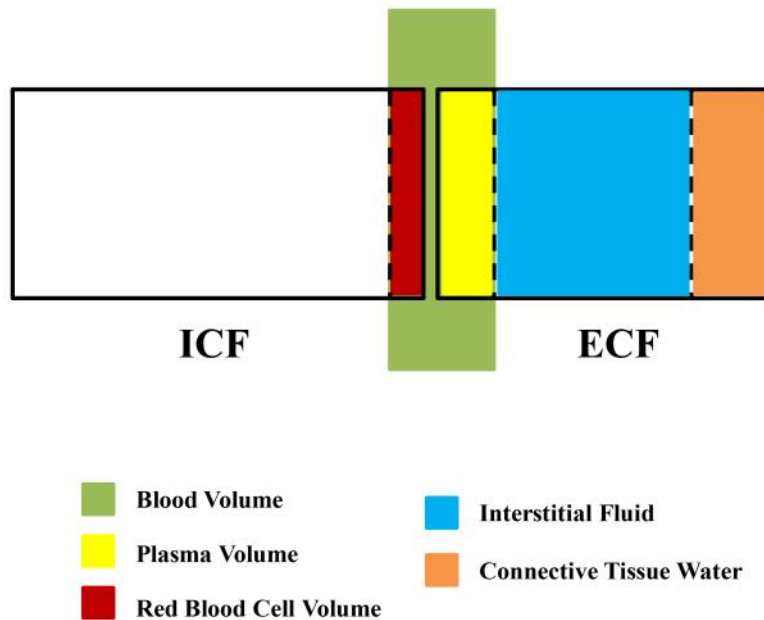


FLUIDS AND ELECTROLYTES

Epidemiology/Pathophysiology

In an otherwise healthy individual, daily volume requirement estimations may be derived from calculations of weight (30-40ml/kg/day), body surface area (1.5L/meter²/day), and metabolic rate (100ml/100kcal). However, these methods may be inaccurate in particular patients (ex: a sedentary obese patient, a frail elderly patient, a patient with cancer). Further, surgical conditions such as trauma, acute peritonitis or abscess can result in acute volume loss, inadequate intake and altered metabolic requirements that must be considered when designing a treatment strategy.

A patient's homeostasis is related to total body water and its distribution between intra and extracellular spaces. Electrolytes are distributed throughout the system and held in various concentrations dependent upon gradients created by active and permissive transport across cell membranes. Up to 60% of the patient's weight is comprised of water that is distributed between intracellular and extracellular fluid compartments and is dependent upon solute concentrations, osmolarity and the semi-permeable cellular membranes.



Source: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4096820/figure/F1/>

Volume status is a term that directly relates to the extracellular space. This is comprised of intravascular ('blood': cell + plasma) and interstitial volume. Common causes of intracellular electrolyte and volume loss are directly related to extracellular causes.

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Table 2: Causes of Intravascular Volume Loss
<i>Hemorrhagic hypovolemia</i>
Chest: Aortic disruption, pulmonary parenchyma trauma, pulmonary/intercostals vascular injury, hemoptysis
Abdomen/pelvis/retroperitoneum: GI hemorrhage (esophageal varices, ulcers, vascular anomalies), solid-organ injuries (liver, spleen, kidney), vascular (trauma, aneurysmal rupture)
Gynecologic disorders: ruptured ectopic pregnancy, peripartum hemorrhage, abnormal uterine bleeding, ovarian cyst rupture, etc
Orthopedic: Large bone fractures, pelvic fracture, multiple fractures
Extremity and skin surfaces
Major vascular injuries
Large soft tissue injuries
<i>Nonhemorrhagic hypovolemia</i>
GI disorders: vomiting, diarrhea, ascites
Burns
Environmental exposure or neglect
Renal salt wasting

Source: <http://img.medscapestatic.com/pi/meds/ckb/33/26533.jpg>

As the access to the system is either through the GI tract (oral intake) or via intravenous administration, treating the extracellular space is key to accessing and managing the intracellular space. Although the term resuscitation is commonly used to describe an immediate intervention to restore circulating intravascular volume and cardiac output, the ultimate goal is the restoration of cellular perfusion and correction of electrolyte and metabolic disarray.

In the presence of disease, estimations of fluid requirements become less reliable and fluid/electrolyte administration must relate closely to clinical context (ex: hemorrhage, emesis, diarrhea, oliguria with acute renal failure, third spacing of fluid in CHF or cirrhosis). An understanding of the patient’s underlying medical conditions (ex: congestive heart failure, renal insufficiency, diabetes mellitus) and how they might contribute to the pathophysiological state ensures that the clinician applies proper goal-directed fluid and electrolyte therapy, maximize cardiovascular sufficiency, and restoration of cellular respiration.

Resuscitation versus Maintenance Volume Administration

Volume resuscitation implies restoration of cardiac output through the use of intravenous solutions with an osmolarity that will allow it to remain within the circulating volume and not be lost quickly into the intracellular or interstitial space. Maintenance fluids take into consideration the daily needs of the entire system for normal homeostatic function in order to maintain a euvolemic state.

Determination of the need for intravenous volume administration is considered with each clinical context and objective data. For example, a patient with a large scalp laceration, concurrent tachycardia, hypotension, tachypnea and cool extremities with diminished pulses is exhibiting signs and symptoms of acute volume depletion from hemorrhage. Providing maintenance volume will not restore adequate vascular volume quickly enough to establish normal cardiac output and avoid progressive hypoxia and metabolic acidosis. Once adequate vascular volume expansion and treatment of the anemia/coagulopathy has taken place, restoration of

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maintenance fluid needs can be determined and provided in order to maintain stabilization, until the patient is capable of adequate oral intake. Proper monitoring such as trends of the patient's vital signs and urine output can assist with determining whether or not the initial maintenance fluid calculations are adequate or additional resuscitative vascular volume expansion may be required.

Endogenous Factors that Affect Renal Control of Sodium and Water Excretion

Fluid requirements begin with the understanding of losses incurred from normal homeostasis (obligatory water loss). In an average adult, this comes from insensible sources (exhalation, sweat), feces and urine. External sources of volume come from oral fluids, food and IV infusion. Internal sources include metabolic water production from oxidation of food (Krebs cycle).

Urine output is required to remove metabolic byproducts and ingested excess solute. This volume is dependent in part upon the daily solute excretory load and the kidney's ability to concentrate urine. If urine volume is less than this amount, solutes will accumulate and renal failure will evolve. Alternatively, water ingestion beyond that required for homeostasis will be excreted. Thus water and electrolyte balance requires adequate renal filtration, urinary concentration and excretion capability.

In the presence of adequate intake, volume regulation is predominantly exerted by renal function - water and electrolyte compositions are maintained by ingestion of more salt and water than is needed, and by the renal capacity to excrete the excess.

Volume status is regulated through the monitoring of systemic solute per unit volume, or osmolarity. Sodium, the most prominent electrolyte 'solute' in extracellular fluid, is used to monitor extracellular osmolarity. A disproportionate loss of water relative to sodium results in a concentrating osmolar effect. The system will need to conserve/retain water relative to sodium. Alternatively, if losses of extracellular volume are proportionate, for example, whole blood loss during surgery, the system will need to conserve both sodium and water in order to maintain normal osmolarity.

Water balance in the presence of sodium concentration, or hyperosmolarity, is controlled by hypothalamic antidiuretic hormone (ADH) and thirst response. Hyperosmolality and volume depletion are sensed in part by baroreceptors in the carotids, aorta and heart and contribute ADH secretion. Thirst response contributes by altering water intake. ADH stimulates water reabsorption from the nephron's distal collecting duct.

Dilution of the extracellular space is prevented in part by regulating sodium concentration. The adrenal hormone Aldosterone stimulates distal renal tubular cells to absorb sodium; in exchange potassium and hydrogen are excreted. Aldosterone effects do not concentrate urine directly, because it exchanges one ion for another. However, the system is foremost attempting to conserve and maintain volume by monitoring its extracellular osmolarity; adjustments in water and sodium reabsorption are performed towards this goal with the balance shifting depending upon the volume's concentration.

For example, in dehydration with hyperosmolarity, the balance between ADH stimulation and water reabsorption is greater than the production and effect of aldosterone. The result will be a return to normal osmolarity by returning water back to the system and allowing sodium to be

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excreted (lower urine volume, higher concentration). In conditions of volume overload and hypo-osmolar state, aldosterone is created to increase sodium conservation, ADH is suppressed (urine volume increased and becomes dilute) and osmolarity is returned to baseline.

Cardiac output and blood pressure play a role in volume regulation. As above all else, the body must preserve the central nervous system and cardiac perfusion at all cost, the sympathetic nervous system will commence shunting blood away from the remaining organ systems and the periphery to preserve flow to these central organs. The result will be a reduction in kidney perfusion and with it reduced glomerular filtration.

Glomerular blood flow and filtration is sensed by the arterial juxtaglomerular apparatus resulting in either suppression (adequate or high GFR) or stimulation (low GFR) of these cells to produce renin.

With hypovolemia (ex: blood loss, volume depletion), reduced GFR will be sensed by the kidney's juxtaglomerular cells in the afferent and efferent arterioles resulting in renin production and stimulation of renin-angiotensin cascade.

In this case, the body is trying to conserve volume. Hence not only has vasopressin been stimulated though central arterial pressure loss and osmolar shifts but aldosterone is also called upon to assist with sodium retention to maintain sodium concentrations and iso-osmolarity. The result will be a low urine volume, but its osmolarity may not be as high as with hyperosmolar dehydration (ex: dehydration from sweating or fever, volume depletion from emesis, diarrhea) because both ADH and aldosterone are working in balance here rather than ADH greater than aldosterone.

In conditions where excessive amounts of volume and electrolytes are lost (ex: enterocutaneous fistula) renal function may be effected by secondary hyperaldosteronism provoked by sodium loss through the fistula. Urinary sodium can be monitored. A concentration below 20 mM suggests inadequate sodium replacement, and this is certain when the level falls below 10 mM. Thus it is important to monitor fistula output and replace the losses with a solution containing adequate amounts of sodium.

With hypervolemia and sodium overload, glomerular flow is adequate, suppressing renin-angiotensin cascade and aldosterone production, allowing excretion of sodium. When a positive free water balance lowers the serum sodium concentration (ex: <135 mMol/L), cell volume receptors in the hypothalamus inhibit the secretion of ADH. Free water is excreted and circulating sodium is returned to normal levels. Atrial natriuretic factor (ANF) hormone is released by cardiac myocytes in response to elevated blood volume. Its effect counters that of renin-angiotensin-aldosterone and promotes natriuresis by promoting glomerular filtration and reducing distal tubular sodium reabsorption.

An important distinction must be made between dehydration and volume depletion, as the terms are mistakenly used interchangeably. Dehydration refers to a loss of total body water that can produce overall hypertonicity. Alternatively, volume depletion occurs when there is a loss of extracellular fluid volume. While both conditions can occur simultaneously, the management, including the rate and type of fluids used may differ. Resuscitation with restoration of the extracellular space's intravascular volume is a priority, though once this has been re-established, focus must shift to address intracellular needs.

SIGNS AND SYMPTOMS

Symptoms, Signs and Physical Findings of Volume Depletion and Dehydration

As with other information, the objective data must be taken in the context of the patient's presentation and relevant medical history. Patients may complain of thirst, nausea, emesis or stool output at rates greater than volume input. Lethargy, confusion and obtundation may occur. Vital signs are an important diagnostic clue, especially if the patient's values when (s)he was previously well are available for comparison. Similar to laboratory values, trends over time are more helpful than singular, random numbers. Progressive tachycardia, clinical orthostasis, postural hypotension, narrowed pulse pressure are helpful clues. Tachypnea may occur from volume loss and resultant metabolic acidosis. Additional physical examination findings that suggest intracellular in addition to extracellular/vascular volume loss may include dry mucous membranes, reduced skin/tongue turgor, and prolonged capillary refill (most effective in infants/children). Muscle weakness can occur from potassium, magnesium and calcium electrolyte disarray. Patients may have reduced urine output ($< 0.5\text{cc/kg/hr}$), commonly with a change in urine color and concentration. Oliguria may be an initial finding in acute kidney injury (AKI) whose etiology is commonly related to pre-renal etiologies including volume depletion.

Relevant Diagnostic Studies

Metabolic Panel

Basic: Serum Sodium, Potassium, Chloride, Bicarbonate and Glucose; Blood Urea Nitrogen and Creatinine

Complete: includes Magnesium, Calcium, Total Protein, Albumin, Globulin, Total Bilirubin, Alkaline Phosphatase, AST, ALT, Glomerular Filtration Rate (GFR)

Serum Lactate

Complete Blood Count: Hematocrit

Urinalysis: Specific Gravity, Ketones, Protein, Sodium, Osmotic Concentration (Osmolarity)

PEARLS

Chloride will be low with gastric fluid loss (emesis, NG Tube aspiration, pyloric stenosis) leading to hypochloremic hypokalemic metabolic alkalosis with paradoxical aciduria.

Poor tissue perfusion in volume depletion and dehydration results in lactic acidosis.

Bicarbonate is reduced in metabolic acidosis (ex: lactic, diabetic ketoacidosis [DKA])

Bicarbonate may also be lost in diarrheal stool output (non anion gap acidosis).

Glucose may be high from DKA or hyperosmolar hyperglycemic nonketotic coma (HONK)

BUN and creatinine levels may be elevated (BUN/Cr ratio $> 20:1$ suggests volume depletion pre-renal hypoperfusion)

Hemoconcentration in severe dehydration may lead to Hematocrit and albumin elevation, whereas rapid acute volume loss from hemorrhage may not change initial hematocrit or albumin levels.

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Serum Osmolarity (nl 275–295 mosm/kg) is elevated in dehydration, hyperglycemia (DKA, HONK), Diabetes Insipidus

Serum Osmolarity is decreased in SIADH, Hyponatremia (CHF, Cirrhosis)

Oliguria may suggest significant volume depletion and/or dehydration

Urine Specific Gravity may be elevated in volume depletion and dehydration (an exception: Diabetes Insipidus)

Ketonuria may be present in dehydration

Urine Sodium may be low (< 20meq/L) in volume depletion (aldosteroma)

Urine chloride may be low in metabolic alkalosis (emesis)

Consider electrolyte analysis of fluid from drains to assist with replacement fluid needs (ex: pancreatic or biliary drain, gastric fluid)

Urine Osmolarity may be elevated (> 400mosm/kg) in volume depletion and dehydration

Serum Electrolytes: Normal Ranges for use with Management Needs Assessment

<u>Electrolyte</u>	<u>Ref Range & Units</u>
SODIUM	135 - 145 mmol/L
POTASSIUM	3.4 - 5.0 mmol/L
CHLORIDE	98 - 108 mmol/L
CO ₂	23 - 32 mmol/L
BUN	8 - 25 mg/dL
CREATININE	0.60 - 1.50 mg/dL
GLUCOSE	70 - 110 mg/dL
CALCIUM	8.5 - 10.5 mg/dL
EGFR	>60 mL/min/1.73m ²
ANION GAP	3 - 17 mmol/L
WHOLE BLOOD GLUCOSE	70 - 110 mg/dL
Calculated Osmolarity	200-300 mOsm/L
$2 [\text{Na}(\text{mEq/L})] + 2 [\text{K}(\text{mEq/L})] + [\text{Glucose}(\text{mEq/dL})]/18 + [\text{BUN}(\text{mEq/dL})]/2.8$	

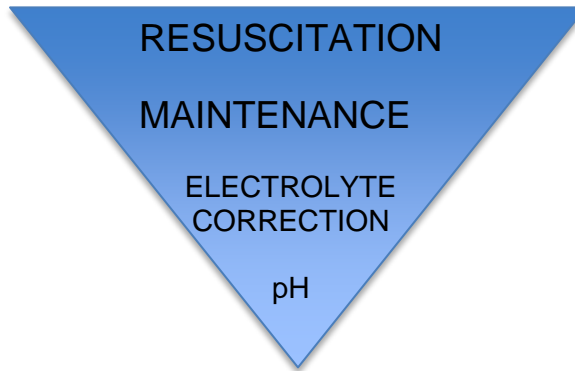
It is important to review the treating institution's normal values. Analysis of a patient's basic metabolic panel should be done in the context of the patient's presentation. Previous known values are equally important to assist with establishing trends. Anticipating potential evolving metabolic derangements is helpful in designing treatment and management plans to reduce or avoid their occurrence (ex: nasogastric tube drainage, ileostomy output). If resuscitation is underway, the provider will be expected to adjust focus on maintenance of volume and metabolic/electrolyte correction as needed with restoration of cardiovascular sufficiency.

Management

Regardless of the etiology of volume depletion, the mainstay of treatment strategy is a goal-directed resuscitation to restore intravascular volume. This will contribute to improved cardiovascular function and tissue perfusion. As the extracellular space is restored, focus can begin to shift towards maintaining the current extracellular volume state and restoring intracellular volume. Electrolyte disarray may be anticipated from the clinical context and

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verified using diagnostic tests. Correction can occur during and after the maintenance phase is established. With adequate tissue perfusion, most pH abnormalities should correct without the need for exogenous buffer. The inverted pyramid below illustrates the focus of acute management in descending order of priority:



Measuring Fluid Balance

There are several tools available to monitor patients' intravascular resuscitation. The clinical context is always very helpful in anticipating potential volume and metabolic derangements and in creating a management strategy. Monitoring for changes in vital signs, including the development of fever, is used to determine resuscitation as well as adjusting for needs. Trend analysis of laboratory values such as serum and urine electrolytes and osmolarity, BUN, creatinine and GFR can be used to tailor specific needs. Requesting staff assistance with 'Strict Inputs & Outputs' on patients by accurately measuring each source of input (ex: IV fluid and medication volumes, oral intake calculations) and output sources (urine, stool/ostomy [emesis/diarrhea volume], and drains [ex: NGT, JP, chest tube]). These will assist with identification of input adjustment needs relative to calculated output measured. These can be trended and re-calculated with each shift.

Sources of Fluid Loss or Gain may include:

Loss

- Hemorrhage
- Emesis
- Stool/ostomy output
- Drains
- Diuresis

Gain

- Excessive IV fluids
- Excessive sodium intake
- Reduced Inotropy (ex: CHF)
- Renal Parenchymal Disease
- Cirrhosis

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Physical Findings suggestive of Volume Depletion/Dehydration or Fluid Overload:

Finding	Volume Depletion/Dehydration	Overload
Body Weight	Loss	Gain
Pulse Pressure	Wide (early) Narrow (Late)	Wide
Integument	Dry, Low Turgor	Pitting Edema
Mucous Membranes	Dry	Moist

Compare and Contrast Various Resuscitation Strategies for Patients who are Volume Depleted or in Hemorrhagic Shock

Initial management of Volume Depletion includes restoration of intravascular circulating volume. 20cc/kg boluses of isotonic crystalloid (ex: 0.9%NS, LR) can be instituted. Lactated Ringer’s solution is the fluid of choice for large volume resuscitation, i.e. trauma patients. Goals of treatment include improvement of vital signs and establishment of urine output.

Acute hemorrhage: Given a clinical context (ex: blunt, penetrating trauma, acute GI bleeding, leaking aortic aneurysm), in-hospital resuscitation discussion including the use of crystalloid versus Type O Rh -, type-specific and cross-matched packed red blood cells followed by infusion ratio of packed RBCs, Fresh Frozen Plasma and Platelets in transfusion resuscitation and massive transfusion protocols.

Once resuscitation is successful, focus can shift towards maintenance of extravascular volume, resuscitation of intracellular volume, and correction of metabolic disarray.

Electrolyte Composition of the Following Solutions

- Normal Saline (0.9%NaCl)
- Ringer’s Lactate (Balanced Solution, Hartmann’s Solution)
- 1/2 Normal Saline (0.45%NaCl)
- 5% dextrose in water (D5W)

Solution	pH	Na ⁺	Cl ⁻	K ⁺	Ca ⁺⁺	Lactate	Glucose	Osmolality
.9% Normal Saline	5.0	154	154	0	0	0	0	308
Lactated Ringers	6.5	130	109	4	3	28	0	275
5% Dextrose in Water (D ₅ W)	4.0	0	0	0	0	0	50 g/L	252
.45% Normal Saline with Dextrose (D ₅ 1/2 NS)	4.5	77	77	0	0	0	50 g/L	406
0.45% Normal Saline (1/2 NS)	5.0	77	77	0	0	0	0	155

Source: <http://www.medscape.org/viewarticle/503138>

Hypertonic solution has an osmolarity greater than 340mOsm/kg; Isotonic solutions have an osmolarity between 240-340mOsm/kg; Hypotonic solutions have an osmolarity less than 250mOsm/kg. Hypertonic and isotonic solutions are considered for resuscitation. Isotonic and hypotonic solutions may be chosen for maintenance and electrolyte correction.

POSTOPERATIVE CARE

Specific Problems

Patients must be monitored closely with frequent vital sign checks for evolving tachycardia and pulse pressure shifts from volume output exceeding input. Continued measurements of intake versus output are required. Maintenance fluid therapy may require supplemental resuscitation when output exceeds input. Resuscitation may be provided in the form of bolus isotonic solutions (ex: 10-20cc/kg) followed by maintenance infusion. Losses from drains (ex: NG tube,

biliary drains) are replaced ml for ml with solutions whose electrolyte composition closely mirrors that of the fluid lost. For example, nasogastric aspirate may be replaced using NaCl; peripancreatic fluid loss may be replaced ml for ml using Lactated Ringers solution. This administration is added to, not in replacement for the maintenance infusion.

In the following situations, serum Na, K, HCO₃, Cl will remain stable (0), rise considerably (++) , rise moderately (+), fall moderately (-), or fall considerably (--):

- Excessive Gastric Losses**
- High Volume Pancreatic Fistula**
- Small Intestinal Fistula**
- Biliary Fistula**
- Diarrhea**

Excessive Gastric Losses (Eventual Chloride-Responsive Metabolic Alkalosis)

Na	K	HCO ₃	Cl
Fall ++	Fall +	Rise	Fall ++

High Volume Pancreatic Fistula (Hyponatremic Hypokalemic Metabolic Acidosis)

Na	K	HCO ₃	Cl
Fall ++	Fall +	Fall ++	Stable

Small Intestine Fistula (Hyponatremic Hypokalemic Metabolic Acidosis)

Na	K	HCO ₃	Cl
Fall ++	Fall +	Fall ++	Fall +

Biliary Fistula

Na	K	HCO ₃	Cl
Fall +	Fall +	Stable	Fall +

Diarrhea (Non-Anion Gap Hyperchloremic Metabolic Acidosis)

Na	K	HCO ₃	Cl
Rise	Fall ++	Fall ++	Rise

Sources of average daily fluid production/loss:

- Salivary glands 1.5L
- Gastric glands 1.5 L
- Pancreas 1 L
- Bile Production 1 L

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Duodenal (ex: Brunner's glands) 200 mL
Jejunum + Ileum 1.5-2 L

Depending on the daily oral intake, 8 to 10 L of fluid passes through the jejunum each day, (more in the presence of inflammation, infection or obstruction).

98% of this fluid is normally absorbed most occurring proximal to the ileocecal valve (colon absorbs up to 2L); usually 100 to 200 mL of fluid is excreted in the stool.

Composition of the enteral/organ secretions at various levels of the gastrointestinal tract includes the following:

Gastric: H, Cl, Na, K,
Duodenal: H, Cl, Na, K
Bile: Na, Cl
Pancreas: Na, K, HCO₃
Small Intestine: Na K Mg HCO₃

In general, there is a potential for acute fluid loss and electrolyte imbalance with pancreatic or proximal intestinal fluid loss from conditions such as pancreatic or small intestinal fistulae. Water and electrolytes (especially sodium, magnesium and bicarbonate) inevitably accompany this fluid, as do essential nutrients.

In general, gastric losses from emesis and NG tube aspiration will contain higher concentrations of H, Na, K and Cl, resulting in a volume depletion and chloride-responsive metabolic alkalosis. Losses from peripancreatic, and intestinal pathologies below the pylorus such as EC fistulae and diarrhea will contain more K and bicarbonate anion resulting in a volume depletion and metabolic acidosis. Choice of IV resuscitation and maintenance fluids may be chosen in anticipation of such risk as well as to reflect these conditions that are verified by diagnostic serum electrolyte tests.

Normal 'Fed' versus 'Fasting' state and electrolyte variations

Electrolyte secretions differ significantly between the fasting and fed states. Feeding increases the H ion concentration from about 50 mM to up to 100 mM, increases Cl from 90 to 120 mM, but decreases sodium from 40 to about 25 mM. Na concentration of about 140 mM is independent of the activity state of the pancreas, but the HCO₃ concentration increases from 40 to up to 145 mM following stimulation.

Fistulous discharge at the level of the upper jejunum is therefore associated with significant losses of sodium, chloride, and bicarbonate ions.

Fluid volumes can be replaced intravenously or orally. In general each liter of fluid lost from a stoma or fistula contains 100 mmol of sodium. Treatment must include isotonic solutions containing Na. This is replaced cc for cc collected from fistulae and stomas. In general, most patients will not be able to manage more than 1.5 to 2.0 L in 24 hours period. If greater volumes are required, IV support is needed.

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In the following situations, indicate whether serum and urine Na, K, HCO₃, Cl and osmolality will remain stable (0), rise considerably (++) , rise moderately (+), fall moderately (-), or fall considerably (--):

- Acute Tubular Necrosis**
- Dehydration**
- Inappropriate ADH secretion (SIADH)**
- Diabetes Insipidus**
- Congestive Heart Failure**

ATN (ex: Nephrotoxic)

Na	U Na	K	HCO ₃	Cl	Osm
Fall	Rise	Rise or Fall	Fall	Fall	Rise

Dehydration (Determine Etiology ex: Emesis, Diarrhea, Sweating + Poor PO Intake)

Na	U Na	K	HCO ₃	Cl	Osm
Rise or Fall	Fall	Rise or Fall	Rise or Fall	Rise or Fall	Rise

SIADH

Na	U Na	K	HCO ₃	Cl	Osm
Fall	Rise	Stable	Stable	Stable	Fall (< U.Osm)

DI

Na	U Na	K	HCO ₃	Cl	Osm
Rise	Fall	Rise	Stable	Rise	Rise

CHF (reduced GFR, elevated renin-angiotensin-aldosterone, vasopressin [ADH])

Na	U Na	K	HCO ₃	Cl	Osm
Stable/Fall	Fall	Fall	Rise	Fall	Fall

Describe the possible causes, signs and symptoms, appropriate laboratory studies needed, and treatment of the following conditions:

- Hypernatremia**
- Hyponatremia**
- Hyperkalemia**
- Hypokalemia**
- Hypomagnesemia**
- Hyperchloremia**
- Hypochloremia**

HYPERNATREMIA

Causes:

Electrolytes are ingested and retained without corresponding amounts of water or when water is lost at a rate greater than the electrolytes.

Normally excess salt intake results in thirst response (increasing water intake) and ADH release

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(reducing water loss).

Hypotonic fluid losses (ex: vomitus, infectious or cathartic induced diarrhea, glycosuria, osmotic or loop diuretic use, Diabetes Insipidus)

Hypothalamic atrophy or disease can reduce thirst response, (elderly), and aberrant ADH release (Diabetes Insipidus).

Normal range serum osmolality: 275-295 mosmol/L.

If both hypothalamic and renal function are intact, a rise in the serum sodium concentration (ex: 150meq/L) should result in plasma osmolality > 295mosmol/kg. This should normally cause ADH secretion to maximally concentration urine (> 600mosmol/kg). Exogenous ADH should not produce a further rise in the urine osmolality.

Diabetes Insipidus:

Urine osmolality is less than the plasma osmolality (< 300 mosmol/kg), consider central or nephrogenic diabetes insipidus. Exogenous ADH should result in reduced urine output and increased urine osmolarity.

Lab Tests: Basic metabolic panel, including glucose, creatinine; serum and urine osmolarity, urine Na

Intermediate urine osmolality (300 - 600 mosmol/kg), consider an osmotic diuresis or diabetes insipidus. ADH effect is maximal in hypernatremia from osmotic diuresis. Thus exogenous ADH will have no effect.

High urine osmolarity (> 600 mosmol/kg) is most likely due to extrarenal water losses in a dehydrated patient. Urine osmolality > 600 mosmol/kg, indicates secretion of and response to ADH. The treatment includes resuscitation of intravascular volume followed by isotonic or hypotonic solutions.

A volume depleted patient with high plasma osmolality and a low urine osmolality, consider diabetes insipidus.

Significant volume depletion (ex: emesis, diarrhea) will likely result in urine sodium < 25meq/L (aldosterone, sodium reabsorption).

Urine sodium > 100meq/L with normal renal function suggests excessive salt ingestion relative to free water (ex: 3%NaCl).

Calculations: What is the Free Water Deficit that must be infused to treat Hypernatremia?

$$\text{Water deficit} = \text{current TBW} \times \frac{\text{Serum Na}}{140} - 1$$

TBW (total body water) = approx 50% lean body weight

Ex: 75yo female 60kg with a serum Na 160

Water deficit = $0.50 \times 60 \left(\left[\frac{160}{140} \right] - 1 \right) = 4.3$ liters

NB: does not include estimated losses from sweat, isosmotic loss in diarrhea or osmotic diuresis

Acute or Chronic Hypernatremia:

Acute: deficit replaced in 24 hours; hourly infusion rate should exceed the water deficit divided by 24: Hourly infusion rate (mL/hour) > Water deficit in mL ÷ 24 hours; for patient above, $4300\text{ml} \div 24 = 180\text{ml/hr}$

or

D5W 3-6 ml/kg/hr; measure serum Na every 1-2 hours until 145meq.

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D5W at 1ml/kg/hr until serum Na is 140meq

Chronic: a fraction of the water deficit is replaced in 24 hours (i.e., enough water to lower the serum sodium by 10meq/L)

Desired water replacement in the first day in mL = 3 mL/kg body weight x 10
180ml X 10 = 1800ml/24hr

or

1.35ml/hr X pt.'s wt. (kg) – goal is approx. 10eq/l/24hours; measure serum Na every 6 hours

HYPONATREMIA

Elevated ADH: Volume Depletion, CHF, Cirrhosis, SIADH, Thiazide Diuretics, Adrenal Insufficiency

Low ADH: Chronic kidney disease, water intoxication (primary/psychogenic polydipsia)

Elevated Osmolarity: Hyperglycemia, Alcohol Ingestion

Normal Osmolarity: Elevated triglycerides, lipoproteins (jaundice), myeloma, mannitol

Lab Tests: Basic metabolic panel, including glucose, creatinine; consider CBC, LFTs, calcium

Timing: Acute (<24hrs ex: marathoners hydrating with water) vs Chronic (> 48hrs)

Symptom Severity:

Mild/Moderate: fatigue nausea confusion ataxia (serum Na 121-129)

Severe: obtundation, seizure, coma (serum Na < 120)

Treatment Goal: Increase the serum sodium by 4 to 6 meq/L over 4-24 hours but not to exceed 8 meq/L in 24-hour period (danger of too rapid a correction is central pontine myelinolysis).

Treat Underlying Disease

Volume Depletion: isotonic, hypertonic saline (suppressing nl. ADH release, promoting excess free water excretion)

Adrenal Insufficiency: glucocorticoids (directly suppressing ADH release)

Secondary SIADH: reduce/discontinue use of Desmopressin (DDAVP), SSRIs [fluoxetine, sertraline]

Solutions

Hypertonic (moderate/severe symptoms, serum Na < 121) isotonic (mild/moderate symptoms, serum Na > 121)

Hypertonic Saline (3%NaCl): 1 mL/kg body weight of 3 percent saline = 1 meq/L increase in serum Na (note: rough estimate; serum values must be checked regularly. Overcorrection can occur when

a) hypovolemia is corrected, removing ADH release and causing excess water excretion resulting in faster/higher serum Na levels.

b) SIADH – causes excretion of serum Na (normal aldosterone and atrial natriuretic peptide function) with water retention, resulting in slower/lower serum Na levels

Isotonic saline (0.9%NaCl): Raises serum Na by 1meq/L for each liter infused. Consider in volume depletion states (emesis, diarrhea, diuretic use). This will reduce ADH release, and increase free water loss.

Volume restriction is preferred for edematous hyponatremic states (ex: CHF); isotonic saline may increase total body water with only minimum improvement in serum Na.

HYPERKALEMIA

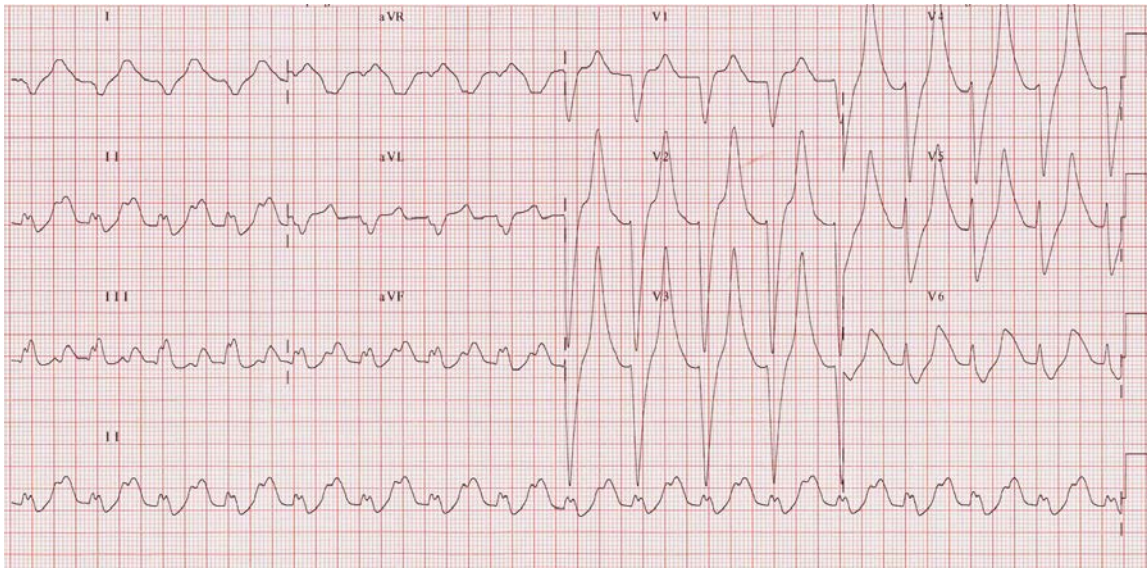
Causes:

Renal dysfunction causing urinary K excretion (parenchymal disease; inhibition of renin-angiotensin-aldosterone [underlying disorder, drug-induced]).

Redistributive hyperkalemia most commonly occurs in uncontrolled hyperglycemia (e.g., diabetic ketoacidosis or hyperosmolar hyperglycemic state). Impaired insulin production or response, serum hyperosmolality shifts intracellular potassium into the ECF. Volume repletion and insulin will restore condition, though patients may still have whole body potassium deficit (ex: renin-aldosterone).

Lab Tests: Basic metabolic panel, including glucose, creatinine

Identification of EKG Abnormalities Consistent with Hyperkalemia (EPA-3)



Scenario: Patient with end-stage renal disease (ESRD) and missed dialysis; Crush injury; Hypertension treatment with ARB/ACE inhibitors, diuretic use such as spironolactone or amiloride; Transplant patient on tacrolimus or cyclosporine.

ECG: (Hyperkalemia)

(Source: <http://cdn.lifeinthefastlane.com/wp-content/uploads/2011/02/ECG-Potassium-9.2.jpg>)

ECG findings:

- AV block
- Prolonged PR interval
- Peaked T waves
- Widened QRS
- Fusion of P and T wave into QRS

Hyperkalemia Treatment

Calcium chloride (central access) 500-1000mg (5-10cc of a 10% solution) infused over 2-3minutes (4.6meq elemental Ca in 10ml)

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Calcium gluconate (peripheral access) 1000mg (10ml of a 10% solution) infused over 2-3 minutes (13.6 meq elemental Ca in 10ml)
(rapid onset of action)

Insulin (10 units regular) bolus injection with either D50 (50ml) or in 500ml D10W over 60 minutes
(onset action in 30-60 minutes)

Glucose may be held if serum level ≥ 250

Albuterol 10-20mg in 5ml saline nebulization (4X greater than usual bronchodilation dose); increases skeletal muscle Na-K-ATPase pump

(onset action variable)

Cation exchange resins (e.g., sodium polystyrene sulfonate, 15-30g) are indicated:

Potentially life-threatening hyperkalemia

Dialysis is not readily available

Other therapies to remove potassium (e.g., diuretics, rapid restoration of kidney function) have failed or are not possible

(onset action in hours)

Contraindications:

Postoperative patients

Patients with an ileus, small or large bowel obstruction or receiving opiates

Repeated doses of insulin and glucose is preferred in such patients until dialysis is available

Dialysis hyperkalemia is severe and expected to increase rapidly

HYPOKALEMIA

Causes:

GI losses (diarrhea, vomiting)

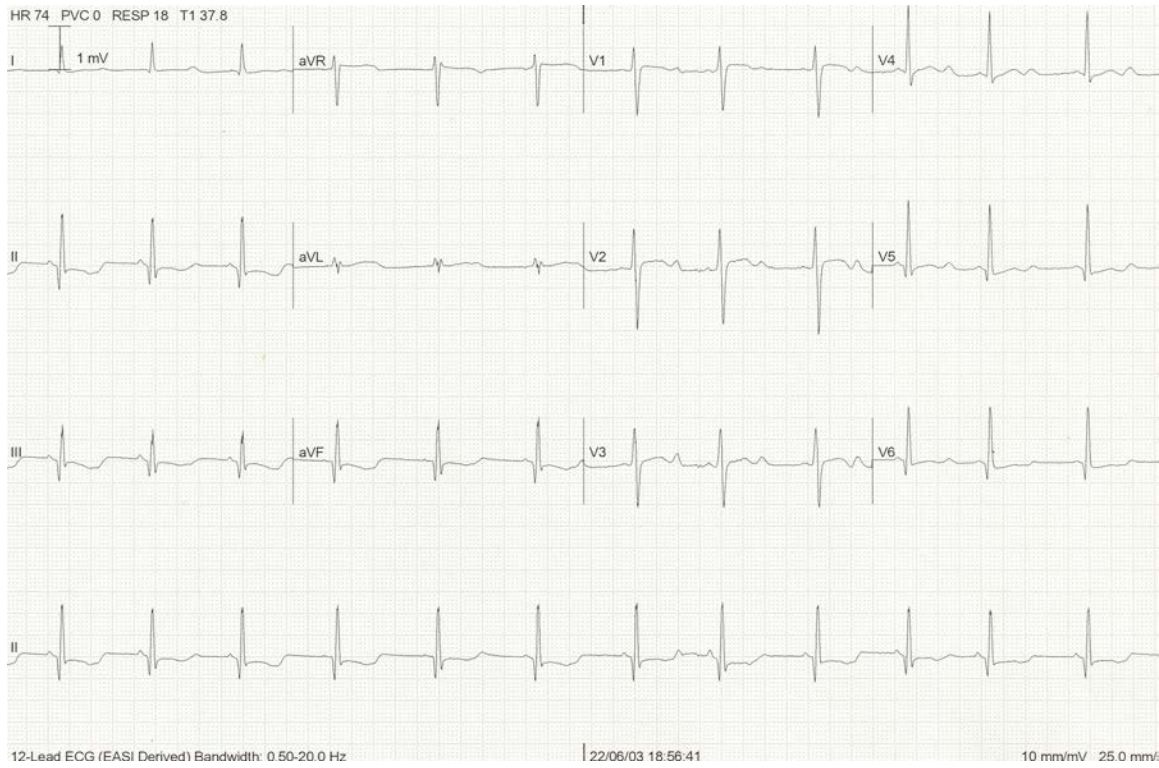
Renal (diuretics, renal artery stenosis)

Tests: Basic metabolic panel, including glucose, creatinine, Mg, urine K, ECG (U wave)

Urine K < 20meq/L in volume depletion from diarrhea, insulin use,

Urine K > 40meq/L with diuretic use, vomiting, mineralocorticoid excess, Bartter and Gitelman syndromes (autosomal recessive renal disorders causing hypokalemic metabolic alkalosis)

Identification of EKG Abnormalities Consistent with Hypokalemia (EPA-3)



Scenario: Patient with enteritis, diarrhea and vomiting; diabetic ketoacidosis (as the pH improves with therapy, the serum K may fall precipitously); diuretic use (thiazides, furosemide); primary hyperaldosteronism

ECG (Hypokalemia)

(Source: <http://cdn.lifeinthefastlane.com/wpcontent/uploads/2011/02/hypokalaemia2.jpg>)

- ECG findings:
- Tachyarrhythmias
- T wave inversion
- ST depression
- U waves
- Prolonged QU interval

Hypokalemia Treatment

For every 1 mEq/L decrease in serum potassium, the potassium deficit is approximately 200-400 mEq.

Note that potassium values are difficult to correct when magnesium level is also low; both may need to be corrected.

Oral replacement when possible; most efficient, especially when larger doses are required more quickly.

Consider providing supplements with serum potassium levels < 3.8 mEq/L.

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1-3 meq/kg/day in 2 - 4 divided doses to a maximum of 120 mEq over 6 hours; titrate to desired level – wait at least 1 hour after administration before checking serum K values

Note that KDur™ should be avoided when values need to be corrected quickly.

IV Treatment:

Peripheral IV

Maximum concentration through a peripheral line is 10 mEq/100 ml.

Minibag therapy: maximum of 20 mEq.

maximum infusion rate is 10 mEq per hour.

Central IV

20 mEq/100 ml is standard; maximum concentration of 20 mEq/50 ml for fluid restricted patients

Minibag therapy: maximum of 20 mEq.

Maximum infusion rate via central line is 20 mEq/hr with cardiac monitoring

Recommended to wait at least 1 hour after administration before checking serum K values

Oral + IV administration: maximum IV + PO of 120 mEq over 6 hour period with serum checks.

HYPOMAGNESEMIA

Causes:

Starvation

Alcohol dependence

Pancreatitis

Vomiting and nasogastric suction

Diarrhea

Ostomy and fistula output

Diuretics - Loop diuretics, osmotic diuretics, and long-term use of thiazides

Diagnostic Tests: Serum Mg, Total Protein, Serum K, Ca and Phos levels

Extracellular magnesium is protein bound; poor protein values may be a contributor.

Majority of Mg is reabsorbed in the kidney's ascending limb, and a lesser extent the PCT, thus diuretics may contribute to low Mg.

Hypomagnesemia may lead to hypokalemia; thus treating magnesium levels are important while supplementing potassium.

Hypomagnesemia may lead to Hypocalcemia; Ca levels should be checked and addressed when evaluating magnesium values.

Treatment

Immediate treatment

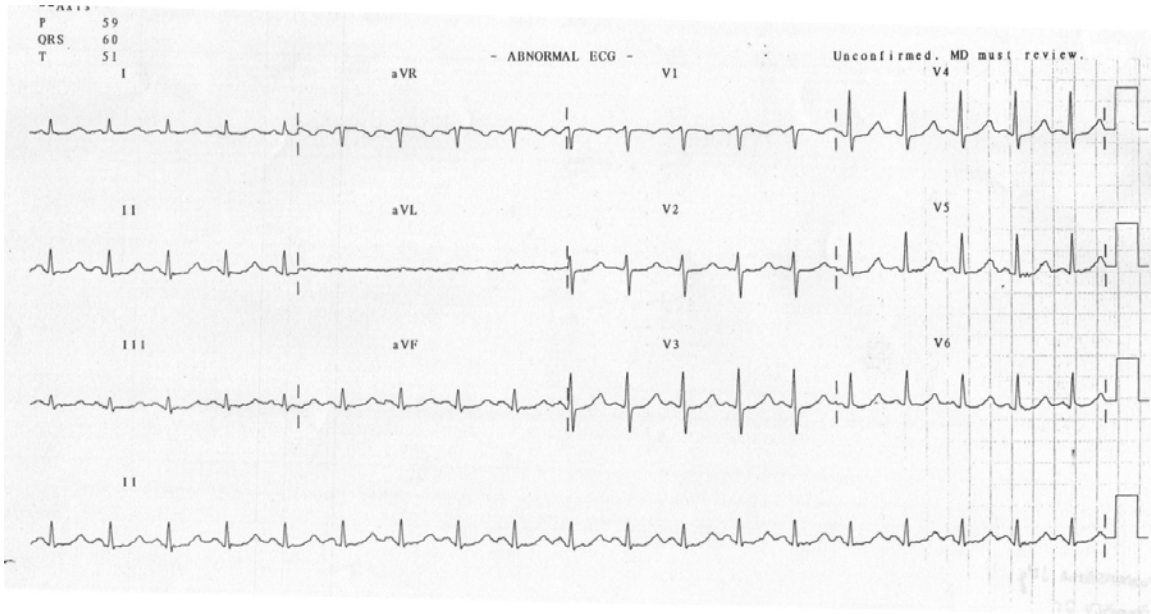
Indications: Ventricular arrhythmia, tetanic muscular spasms

IV: MgSO₄ 1-2 g slow IV (diluted in 50-100 mL D5W) over 5-60 minutes, then 0.5-1 g/hr IV

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Note: serum magnesium concentration regulates renal magnesium reabsorption. An abrupt elevation in magnesium concentration may reduce magnesium retention resulting in paradoxical urinary excretion. Thus serum levels should be checked frequently to ensure stabilization.

Identification of EKG Abnormalities Consistent with Hypokalemia (EPA-3)



Scenario: Patient with enteritis and diarrhea; chronic pancreatitis (saponification of Mg and Ca); Proton pump inhibitor use in peptic ulcer disease; renal losses from diuretic use (thiazides); chronic alcohol abuse

ECG (Hypomagnesemia)

(Source: http://cdn.lifeinthefastlane.com/wp-content/uploads/2011/03/ECG90402-Hypomagnesaemia_prolonged-QT.jpg)

Note: Hypomagnesemia is commonly seen with hypokalemia; both values should be checked; higher risk of ventricular tachyarrhythmias

ECG findings:

Ectopy

Prolonged QT interval

Tachyarrhythmias (ex: torsades de pointes)

HYPERCHLOREMIA

Causes:

Dehydration

Diarrhea (loss of HCO₃)

Renal insufficiency (RTA, nephrotic syndrome)

Diuretics (HCTZ, Acetazolamide-induced hyperchloremic acidosis)

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Lab Tests: Basic metabolic panel, including glucose, creatinine; consider LFTs (albumin)
Serum anion gap = Measured cations - Measured anions

Serum anion gap = $\text{Na} - (\text{Cl} + \text{HCO}_3)$

Note: 'Normal values vary from lab to lab analysis (check your hospital's range); most normal values for the serum anion gap range from 3 to 10 meq/L (averaging 6 meq/L)

Note: Some institutions also incorporate use serum K value in the calculation: Serum anion gap = $(\text{Na} + \text{K}) - (\text{Cl} + \text{HCO}_3)$

If used, the normal anion gap range will increase by approximately 4 meq/L

Note: serum albumin is the anion responsible serum anion gap; poor liver function or nephrosis with low albumin will reduce the gap.

Hyperchloremic Metabolic Acidosis (non-anion gap acidosis: unmeasured anions lower, Cl- elevated)

Cause:

Loss of HCO_3 (ex: diarrhea; RTA; ileal loop)

Reduced acid loss from kidney (Normal function: sulfuric + phosphoric acid from protein metabolism:

- a) Glomerular NaSulfate + NaPhosphate
- b) Distal tubular HSulfuric + HPhosphoric [Na saved]
- c) Reduced GFR: increased anions (ex: sulfate, phosphate)
- d) Tubular dysfunction: reduced H excretion

RTA (Type 1, 'Distal') reduced H excretion; sulfates and phosphates are already filtered so no unmeasured anion retention thus normal anion gap

Reduced Aldosterone

Treatment:

Hydration solutions containing bicarbonate, acetate, citrate, or phosphate salts in exchange for chloride. Bicarbonate replacement of 1-2 meq/kg/day with a goal serum HCO_3 value 22-24 meq/L. Serum K is also commonly low in distal RTA. Addition of K citrate as needed.

RTA (Type 2, 'Proximal') reduced proximal bicarbonate reabsorption resulting in reduced serum bicarbonate concentration. Increases in filtered bicarbonate load above the reduced reabsorptive capacity, resulting in a metabolic acidosis. Example includes Fanconi Syndrome; reduced proximal tubular function results in phosphaturia, glycosuria, proteinuria.

Treatment:

Bicarbonate supplementation (10 to 15 meq/kg/day); close monitoring of serum K and addition on potassium citrate as needed.

HYPOCHLOREMIA

Causes:

Vomiting,

Diarrhea,

Gastrointestinal suction,

Diuretics,

Syndrome of inappropriate antidiuretic hormone secretion, (SIADH),

Water intoxication,

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Excessive sweating,
Adrenal insufficiency,
Hyperaldosteronism,
Drugs (ex: laxatives, corticosteroids, bicarbonate)

Lab Tests: Basic metabolic panel, including glucose and creatinine; hypochloremia can occur with significant volume depletion and dehydration with resultant concurrent hypokalemia, hyponatremia, and metabolic alkalosis

Treatment

Resuscitation with chloride rich isotonic fluid, ex: 0.9% normal saline to restore intravascular volume. Following this, maintenance fluid management with judicious correction of concurrent hypokalemia. With restoration of chloride levels however, serum bicarbonate values will return to normal and serum potassium concentration will rise. Frequent checks of the basic metabolic panel may be required.

SPECIAL CONSIDERATIONS

Pediatric Resuscitation

Case example: A 7 year-old child 3 days sp laparoscopic appendectomy arrives after 8 hours of progressive abdominal pain distention and recurrent nonbloody emesis thought related to an adynamic ileus. His vital signs include a heart rate of 140, blood pressure 90/40 respirations 20 O2 sat 100%RA; his abdomen is distended tympanic and mildly tender diffusely without peritonitis or focal tenderness. Your nurse asks if you would like to commence fluid treatment. What is the most appropriate next step?

Aggressive fluid resuscitation using isotonic solution 10-20 mg/kg bolus, pre-emptive assumption of evolved metabolic alkalosis, chloride responsive condition best managed using a saline solution (ex: 0.9%NaCL); after stabilization of vital sings calculation of maintenance infusion (ex: 4-2-1 rule). The risk of concurrent K losses requiring a serum K check as well as consideration of K replacement and its various administration options. If an NG Tube is placed, additional losses must be calculated and added separately to the total input needs (ex: replacement of NGT losses cc for cc every 8-hours, in addition to the 4-2-1 maintenance calculation).

Burns – Adult and Pediatric - Parkland Formula for both Children and Adults

Case example: A 5 year-old child weighing 22kg suffers partial and full thickness burns to the entire left leg, left arm and back in a house fire within the last hour. EMS have placed an IV but have not begun IV resuscitation. The nurse asks you how to proceed.

The Rule of Nines (the child scale age limit is 3 so the Adult formula should be used; but that the formula is somewhat inaccurate and discussions may be useful regarding Lund-Browder Classification), calculate the %TBSA and calculate the resuscitation needs for his given weight and timing (50% within the first 8 hours). Students must also recognize that the formula does not take into consideration the child's maintenance needs (adding volume based upon the 4-2-1 method to calculate the maintenance needs over 24 hours; also utility of colloids may be discussed for TBSA >30%).

MASSIVE TRANSFUSION

Case example: A 22 year-old female arrives after being struck by an automobile. She was found unconscious and unresponsive at the scene with a heart rate of 130 and a BP 82/40 mm Hg. She is intubated and immobilized for transport with 2 peripheral IVs placed and 1 liter of LR running. On arrival; her vital signs are unchanged, her abdomen is distended with an unstable pelvis and a GCS of 7. FAST is positive. The nurse asks you what you would like to do next.

Discussion of the indications for massive transfusion protocol (ex: Assessment of Blood Consumption/ABC score, persistent hemodynamic instability in context, active bleeding requiring operative or IR embolization, ongoing blood transfusion), what the blood bank will require, what types of products, infusion rate and product ratios [ex: 2:1 – 1:1]) should be given.

Questions

1. A 55 year-old 70kg patient with a history of cholelithiasis presents with 2 days of progressive epigastric abdominal pain, fever, anorexia, episodes of nonbloody, nonbilious emesis. The last bowel movement was 24 hours prior to arrival without hematochezia or melena. The patient has the following vital signs: T 101.2, HR 106 BP 96/56 RR 22 100%. The mucous membranes are dry. Lungs are clear, the heart tachycardic but without murmurs or gallops. There is significant and diffuse epigastric tenderness to gentle palpation with an equivocal Murphy's tenderness. Labs reveal an elevated WBC count and leftward shift, elevated amylase, lipase and LFTs. Right upper quadrant ultrasound reveals gallstones with sonographic Murphy's tenderness and a dilated common bile duct of 9mm. As the clinician, you suspect sepsis and request to commence treatment.

What is the MOST appropriate choice of intravenous fluid therapy?

- A. D5 0.45%NaCl at 110cc/hr
B. D5 0.45%NaCl + 40meqKCl/L at 110cc/hr
C. 2 units crossmatched packed red blood cells
D. 0.9% NaCl, 2.0 liters over 30minutes/1 hour
E. Lactated Ringers solution 1 liter over 1 hour
2. Paramedics deliver a 27 year-old patient whom they found on the pavement after accidentally falling from the second-story window of his apartment. The patient had an initial heart rate of 134, blood pressure 86/52 and shallow respiratory rate of 20. Two 14-gauge IVs were placed and 2 liters of lactated ringers solution were being infused during transport. On arrival, the patient remains unresponsive with clear equal breath sounds, heart rate of 138, blood pressure of 88/56 with absent radial pulses, multiple contusions and abrasions along the right lower lateral chest and abdominal wall and right hip with ecchymosis in the right groin and perineum. FAST exam is positive and the right iliac wing feels unstable.

What is the MOST appropriate next step in the resuscitation to stabilize the patient's blood pressure?

- A. Complete the 2 liters of LR and infuse an additional 2 liters; then reassess.
B. Allow hypotension to persist
C. Activation of Massive Transfusion Protocol with a ratio of RBC:Plasma of 2:1
D. Arrange for transfusion of crossmatched blood and FFP, RBC:Plasma ratio of 2:1
E. Complete the 2 liters of LR and infuse 2 units of type O Rh-negative donor blood.
3. A 27 year-old patient suffers from a gunshot wound to the abdomen involving the liver. His presenting vital signs after a 30-minute transport from the field included heart rates in the 150s and blood pressures of 60s/40s. The patient is managed aggressively and stabilized 3 hours after the event.

What is the MOST LIKELY effect of this condition on the patient's renal function?

- A. Vasopressor use will spare renal perfusion.

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- B. Maintained GFR and creatinine clearance should be expected.
 - C. Hemodilution preserves renal function.
 - D. As with other tissues, the kidneys can tolerate up to 6 hours of ischemia.
 - E. Acute renal insufficiency is likely with elevated BUN values despite urine output > 1.0cc/kg/hr.
4. A 65 year-old patient with a history of jejunal-cutaneous fistula requires an admission. The fistula output over the last 24 hours is 1500cc of watery, non-bloody material.

Which of the following solutions would BEST REPLACE the patient's losses?

	Na(meq)	K	Ca	Cl	HCO ₃	
A.	39	0	0	39	0	(0.25%NaCl)
B.	154	0	0	154	0	(0.9%NaCl)
C.	130	4	3	109	28	(LR)
D.	77	0	0	77	0	(0.45%NaCl)
E.	513	0	0	513	0	(3%NaCl)

5. You are asked by your senior surgical resident to calculate the maintenance fluid needs for the next 8 hours of an ICU patient on the team. The patient is POD#2 from abdominal surgery. He weighs 70kg, remains intubated, and has an orogastric tube, a Foley catheter, and two Jackson-Pratt drains in the abdominal cavity. The patient's RN has recorded the output from each of the drains above over the last 8-hour shift:

Urine: 600cc
OGT: 450cc
JP #1: 200cc
JP#2: 50cc

Which value below is the best estimate of MAINTENANCE FLUID NEEDS for this patient over the next 8 hours?

- A. 1300cc
- B. 700cc
- C. 2000cc
- D. 600cc

Answers

1. **D.** The patient has clinical evidence of inadequate perfusion. The cause of the condition is acute progressive cholangitis from a common biliary duct obstruction. This has progressed from an acute systemic inflammatory response to sepsis. Progressive tissue hypoxia from poor perfusion can lead to progressive base deficit and lactic acidosis. Fluid resuscitation should be initiated as early as possible in patients with severe sepsis. Early recognition of sepsis and septic shock and appropriate fluid resuscitation can improve the patient's outcome. Early goal-directed therapy (EGDT) decreases the in-hospital mortality of patients with sepsis. The Surviving Sepsis Campaign guidelines recommends that septic patients with suspected hypovolemia receive 20-30cc/kg of isotonic crystalloids as soon as possible. Greater volumes may be required. Answer A is neither adequate fluid concentration nor infusion rate. KCl (answer B) should not be administered to patients until adequate volume resuscitation and resumption of urine output has occurred. The patient has no clinical evidence of acute hemorrhage making transfusion inappropriate (answer C). The volume of Lactated Ringers solution is inadequate to resuscitate this 70kg septic patient (answer E).

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Sartelli et al. Complicated intra-abdominal infections in Europe: a comprehensive review of the CIAO study; *World Journal of Emergency Surgery* 2012;7:36
DOI: 10.1186/1749-7922-7-36

2. **C.** The patient has persistent tachycardia and hypotension, absent distal pulses with FAST exam evidence of intraperitoneal hemorrhage as well as examination findings of pelvis fracture with concurrent risk of retroperitoneal hemorrhage. The patient has an ABC (Assessment of Blood Consumption) score of 3, suggesting the potential need for massive transfusion of 10 units or more of PRBCs and associated products in order to stabilize ongoing losses. The ABC score has a sensitivity and specificity ranging from 75% to 90% and 67% to 88% with a score of 3 or more. Criteria to consider the need for massive transfusion of red blood cells and plasma include and ABC score >2, persistent hemodynamic instability and active bleeding (positive FAST, pelvis fracture). Continued resuscitation with crystalloid is inappropriate as it not only dilutes both O₂ carrying capacity and clotting factors, but can also lead to hypothermia and metabolic acidosis. The patient has evidence of multiple ongoing bleeding sources from blunt trauma and

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allowing continued and progressive hypotension carries the risk of progressive tissue hypoxia, worsening base deficit and lactic acidosis. Permissive hypotension is not a treatment. It is never an alternative for definitive hemorrhage control (surgery, embolization), and it currently only applies to trauma patients with active bleeding in the prehospital arena or emergency department while awaiting resuscitation with blood products and emergent surgical intervention.

There is no indication that 2 units of donor blood will be an adequate resuscitation. Appropriate management is to arrange for the possibility of ongoing blood product resuscitation with a RBC:Plasma ratio of 2:1, and capacity to add platelets.

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3. **E.** Kidneys may tolerate 1-2 hours of ischemia before acute injury evolves. With an acute reversible ischemia, decreased creatinine clearance should be expected transiently. Suspect high-output renal insufficiency from reversible ischemia in the presence of elevating BUN values despite adequate urine output. Suspect severe renal insufficiency in the presence of elevated BUN, reduced creatinine clearance and oliguria. Hemodilution from aggressive crystalloid infusion may lead to worsened tissue hypoxia and ischemic-induced renal insufficiency.

4. **C.** High output fistula are defined as those draining > 500cc/day. Small bowel secretions, bile, and pancreatic secretions serve to neutralize gastric acid secretion. Bile contains sodium and chloride, pancreatic secretions contain sodium and bicarbonate. Jejuna lesions are associated with significant losses of Na, Cl, HCO₃. Of the solution choices listed above, Lactated Ringers solution provides the best balance of electrolytes and bicarbonate to assist with replacing these proximal intestinal losses.

5. **D.** There is a distinction between maintenance volume needs and the total volume required from all losses. The patient remains NPO and requires intravenous fluids in order to maintain euvoolemia and homeostasis. This initial determination is based upon the patient's weight (ex: using the 4-2-1 Rule) and would be estimated to be 110cc/hour or approximately 560cc over the next 8 hours. The calculation does not consider, however, additional losses from the drains listed above. Therefore, the patient will also require an additional volume equal to the total lost from the drains. For patients with normal cardiac function, this is commonly replaced in addition to the maintenance fluid calculation over the subsequent 8 hours. Therefore the TOTAL fluid requirement for this patient over the next 8 hours is 1300cc.

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