Diabetes Insipidus and SIADH Reference Sheet

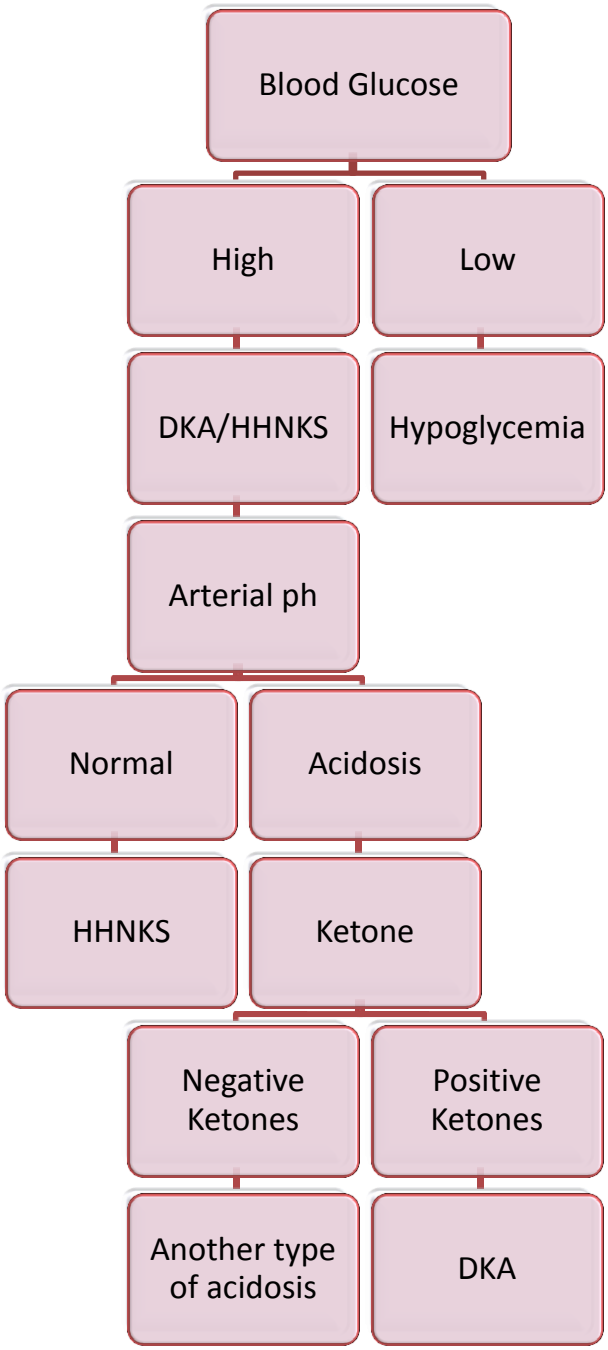
Normal Lab Values	SIADH	DI
• Serum Sodium 137 - 145 mEq/L • Urine Na (40-220 MEq/L/day • Urine osmolality (50-1200 mOsm/kg) • Urine specific gravity (1.002 - 1.028) • ADH level (0 - 4.7 pg/ml)	• Serum hyponatremia (< 137) • Urine hypernatremia (> 40 mEq/L/day) • Urine hyperosmolality (>100 mOsm/kg) • Elevated urine specific gravity • Elevated ADH levels	• Serum hypernatremia (> 145 mEq/L) • Urine hypo - osmolality (< 500 mOsm/kg) • Low urine specific gravity (1.001 - 1.005) • ADH levels low-normal

Clinical Manifestations Associated with Hyponatremia

Serum sodium levels	Associated symptoms
130 – 140 mEq/L	Impaired taste, anorexia, dyspnea with exertion, fatigue, dulled sensorium
120 – 130 mEq/L	Severe GI symptoms including vomiting and abdominal cramps
< 115 mEq/L	Confusion, lethargy, muscle twitching, convulsions

Decision Tree

Diabetic Crisis



Medications that can cause drug-induced hypoglycemia include:

- Bactrim (an antibiotic)
- Beta-blockers
- Haloperidol
- Insulin
- MAO inhibitors
- Metformin when used with sulfonylureas
- Pentamidine
- Quinidine
- Quinine
- SGLT2 inhibitors (such as dapagliflozin and empagliflozin)
- Sulfonylureas
- Thiazolidinediones (such as Actos and Avandia)

Diabetes Insipidus (DI) and Syndrome of Inappropriate Antidiuretic Hormone (SIADH) Secretion are both disorders of water metabolism. The posterior pituitary gland secretes anti-diuretic hormone (ADH). ADH is responsible for regulation of water balance and serum osmolality.

SIADH

If there is too much ADH secreted from the posterior pituitary gland your body will hold on to water. This can lead to water intoxication of not recognized and treated promptly.

Dilute blood (low sodium and low osmolality)

Concentrated urine (high urine sodium and high osmolality/specific gravity)

DI

If there is not enough ADH secreted from the posterior pituitary gland, or, your renal tubules are resistant to it, your body will be unable to conserve water. This leads to an excessive excretion of urine.

Concentrated blood (high sodium, high osmolality)

Dilute urine (low urine sodium, low osmolality/specific gravity)

Goals of care

Maintain adequate tissue perfusion

Ensure patient and family understanding of long term therapies and treatments needed/diet needs

Prevent complications (CHF/fluid overload; electrolyte abnormalities; Seizures)

Nursing Assessments

Hydration status (IV sites, orthostatic vitals, tissue perfusion)

Cardiac (rhythm abnormalities)

Neuro (mental status changes, seizure activity, neuro checks every 2-4 hours)

GU/GI (last BM, appearance of urine)

Nursing Care and Interventions

Strict I&O (every 2 hours)

Daily weights

24 hour urine collection

Dietary restrictions

Monitor, report labs/vitals/rhythm changes

Fluid therapy/restrictions

Falls prevention

Seizure precautions

Disorders of Water Metabolism Handout

Copyright ©2001. All Rights Reserved

TABLE 2
Classes of hypovolemic shock

	Class I	Class II	Class III	Class IV
Blood loss (percent of total volume)	15% (750 ml)	15%-30% (750-1,500 ml)	30%-40% (1,500-2,000 ml)	>40% (>2,000 ml)
BP	Normal	Normal	Decreased	Decreased
Heart rate	< 100	>100	>120	>140
Level of consciousness	Slightly anxious	Mildly anxious	Anxious and confused	Confused and lethargic
Respirations (per min)	14-20	20-30	30-40	>40
Urinary output (ranges, in ml/hr)	>30	20-30	5-15	<5

Note: A normal blood donation is 10% of total blood volume, or 500 ml.

tions, 24; Spo_2, 92% on room air; and temperature, 93.2° F (34° C). The cardiac monitor shows sinus tachycardia.

Place warmed blankets on Mr. Sloane and administer oxygen at 15 liters/minute via non-rebreather mask. His Spo_2 increases to 99%. Keep in mind that peripheral vasoconstriction or an automatic BP cuff on the same arm as the pulse oximetry sensor can cause inaccurate pulse oximeter readings.

Perform a brief neurologic assessment. Mr. Sloane scores 14 out of 15 on the Glasgow Coma Scale (GCS), which measures level of consciousness. His abdomen shows no evidence of ecchymosis, and bowel sounds are present, but he has rebound tenderness in the right upper abdominal quadrant.

Insert a large-bore intravenous (I.V.) device and rapidly infuse 1 liter of warmed 0.9% sodium chloride solution. The fluid should be warmed to prevent hypothermia, which can lead to metabolic acidosis and coagulopathy. Obtain samples for baseline lab studies (arterial blood gas analysis, chemistry panel, complete blood cell [CBC] count, type and crossmatch, prothrombin time [PT], and activated partial thromboplastin time).

Generally, use a 3:1 ratio for fluid resuscitation—300 ml of crystalloid for every 100 ml of fluid loss. (See Table 1 for information on estimating fluid loss.) Mr. Sloane's BP does not respond to the initial fluid bolus, so hang another liter. His abdomen is becoming firm and distended, his BP has dropped to 84/50, and his heart rate has increased to 138.

Prepare to infuse two units of packed red blood cells, as ordered. Use O-negative (the universal donor) until the type and crossmatch are complete.

As shock progresses, level of consciousness deteriorates due to decreased cerebral perfusion. Mr. Sloane is becoming more restless and agitated, and his GCS score falls to 12. He is now in a Class III shock state.

While fluid resuscitation is under way, insert a nasogastric tube and an indwelling urinary catheter, assess for other injuries, and continuously monitor vital signs, urine output, Spo_2 values, cardiac rhythm and rate, and temperature. In the meantime, the physician orders a diagnostic peritoneal lavage (DPL) to assess for intra-abdominal bleeding. (If Mr. Sloane were stable, a noninvasive diagnostic abdominal ultrasound could be done.) The DPL is grossly positive for blood, so prepare the patient for surgery.

Surgery reveals a liver laceration. Because of Mr. Sloane's hemodynamic instability, his liver laceration is packed to control bleeding instead of being repaired, and he is returned to the intensive care unit (ICU).

While Mr. Sloane is in the operating room, his temperature drops to 92.3° F (33.5° C) and coagulopathy starts to develop, as evidenced by a prolonged PT and decreased platelet count. Hypothermia impairs clotting functions and platelet aggregation, and thrombocytopenia can develop. After 6 hours of continuous warm fluid resuscitation, blood product replacement, and external rewarming in the ICU, Mr. Sloane's vital signs stabilize. His temperature rises to 96.8° F (36° C), and his PT and platelet count normalize. He returns to the operating room for removal of the liver packing and surgical repair of the laceration. After a week in the ICU, he is transferred to the trauma step-down unit.

Copyright ©2001. All Rights Reserved

SEPTIC SHOCK

Septic shock is a distributive form of shock resulting from maldistribution of blood volume and decreased oxygen uptake at the cellular level. When gram-negative bacteria in the bloodstream are destroyed by phagocytic cells, endotoxins are released from the bacterial cell wall. The presence of endotoxins causes the release of immune mediators, including cytokines. Cytokines cause vasodilation, increase capillary permeability leading to fluid shifts, decrease oxygen extraction from tissues, and decrease platelet aggregation, leading to activation of the clotting cascade. As the immune response spirals out of control, septic shock ensues. Without treatment, multiple organ dysfunction occurs.

Early signs and symptoms of septic shock may be subtle and nonspecific, but typically include tachycardia, hypotension, and a hyperdynamic cardiovascular profile (elevated cardiac output, reduced afterload, decreased systemic vascular resistance [SVR], and low preload). Other possible signs and symptoms include general malaise and evidence of perfusion deficits, such as oliguria and changes in mentation.

Anna Denardo, an active and previously healthy 65-year-old woman, was brought to the ED with a 2-day history of fever, chills, and malaise. Her skin is flushed and dry. She is awake but needs to be questioned repeatedly to complete the assessment. Her BP is 70/40; heart rate, 150; respiratory rate, 36; and temperature, 104° F (40° C) orally.

While another ED nurse attends to Ms. Denardo, ask her husband about her current illness and medical history. He says that 4 days ago, Ms. Denardo's primary health care provider started her on an antibiotic for a urinary tract infection (UTI). He cannot remember the name of the antibiotic, but says she stopped taking it after only two doses because it upset her stomach.

Based on Ms. Denardo's history and her signs and symptoms, it appears that the untreated UTI developed into sepsis and progressed to septic shock, which has a 40% to 60% mortality rate. Fast action is needed to prevent organ dysfunction, coagulopathy, and death.

Treatment for septic shock focuses on supportive measures, such as improving oxygenation and combating the underlying infection by administering broad-spectrum antibiotic therapy.

Establish I.V. access and begin infusing fluids rapidly. Ms. Denardo's Spo_2 is 88%, so administer 100% oxygen via non-rebreather mask. The cardiac monitor shows sinus tachycardia at a rate of 136. Insert an indwelling urinary catheter and obtain a urine culture; a small amount of dark amber urine drains. A chest X-ray is normal.

Obtain samples for a CBC count, chemistry, coagulation profile, and blood cultures. Then administer I.V. antibiotics as prescribed. Combined antibiotics, such as an aminoglycoside with a third-generation cephalosporin, usually are ordered, and therapy is modified as the infectious organisms are identified. Use aminoglycosides carefully in the elderly because of the increased risk of renal insufficiency. Unfortunately, antibiotics may exacerbate symptoms as damaged bacteria release additional endotoxin; these symptoms may be indistinguishable from shock symptoms.

Ms. Denardo is admitted to the medical ICU for aggressive fluid resuscitation and monitoring. Because a patient in septic shock is hypovolemic, she may need large volumes of fluids—5 to 15 liters for an adult. A pulmonary artery (PA) catheter may be placed for hemodynamic monitoring. If she has a PA catheter, maintain her pulmonary capillary wedge pressure at 12 mm Hg and monitor her continuously to avoid overload.

If fluids alone do not improve her cardiovascular status, she will need drug therapy. Dopamine or norepinephrine promote vasoconstriction and increase BP. However, nursing interventions will focus on fluid resuscitation and treating the causative organism.

Continue to administer oxygen to maintain her Pao_2 above 60 mm Hg. Ms. Denardo may need intubation if oxygenation is inadequate or if respiratory fatigue causes carbon dioxide retention.

Ms. Denardo's condition stabilizes with fluid resuscitation and antibiotic therapy. She is transferred to the medical unit on day two of her admission and is discharged home on day five.

CARDIOGENIC SHOCK

The most lethal form of shock, cardiogenic shock carries an 80% to 100% mortality rate. It develops when the heart cannot pump enough blood to meet the body's oxygenation needs. This reduced cardiac output is manifested by low BP, jugular vein distension and pulmonary edema (signs of right- and left-sided heart failure), and decreased urine output.

Myocardial infarction (MI) is the most common cause of cardiogenic shock. Treatment focuses on reducing the heart's workload with drugs or mechanical devices such as an intra-aortic balloon pump (IABP). The patient may need mechanical ventilation to decrease the work of breathing and support oxygenation.

Xiang Tan, 65, is in your unit

Copyright ©2001. All Rights Reserved

TABLE 2
Assessing shock

	Hypovolemic	Septic	Cardiogenic
Common assessment findings (early)	• Pale, clammy skin • Thready peripheral pulses, narrowing pulse pressure, collapse of veins • Tachycardia, tachypnea • Anxiety, restlessness • Decreased urine output • Dilated pupils • Hypoactive bowel sounds	• Warm, flushed skin • Bounding pulses, widened pulse pressure • Tachycardia, tachypnea (respiratory alkalosis) • Decreased level of consciousness • Hypotension • Fever or hypothermia • Agitation	• Cool, pale, clammy skin • Tachycardia, tachypnea • Anxiety, restlessness • Hypoactive bowel sounds
Common assessment findings (late)	• Cold, mottled, cyanotic skin • Tachycardia • Tachypnea, then bradypnea • Absent bowel sounds • Decorticate or decerebrate positioning	• Cold, mottled skin and weak pulses • Tachycardia and hypotension • Tachypnea and hypoxemia • Obtunded state or coma • Decreased CO • Anuria	• Cold, mottled, cyanotic skin • Tachycardia • Tachypnea, then bradypnea • Absent bowel sounds • Decorticate or decerebrate positioning
Diagnostic criteria	• Lab values: hemoglobin changes depending on the cause and duration of fluid loss and fluid replacement; elevated serum lactate; increased urine specific gravity; decreased hematocrit • Hemodynamic values: central venous pressure below the normal range of 2-8 mm Hg; pulmonary artery wedge pressure below the normal range of 4-12 mm Hg; cardiac output (CO) variable but eventually below normal; systemic vascular resistance (SVR) increased • Other diagnostic tests: X-rays, gastroscopy, gastric contents tested for occult blood	• Lab values: leukocytosis, thrombocytopenia, elevated serum lactate, increased urine specific gravity and osmolality, and decreased urine sodium • Hemodynamic values: increased CO and low SVR in early shock; decreased CO and increased SVR in late shock. • Other diagnostic tests: positive blood cultures	• Lab values: cardiac enzymes to evaluate for myocardial infarction; hyperglycemia; increased blood urea nitrogen level • Hemodynamic values: decreased systolic BP with narrow pulse pressure; cardiac index less than 2.1 liters/min/m^2; pulmonary artery wedge pressure greater than 20 mm Hg; SVR greater than 1,800 dynes/sec/cm^{-5} • Other diagnostic tests: ECG; chest X-ray for pulmonary edema; echocardiography; ventriculography to check for reduced ejection fraction

with an inferior-wall MI. During cardiac catheterization, his BP drops to 90/40 and he develops more chest pain. The catheterization reveals a 99% blockage of Mr. Tan's distal right coronary artery and narrowing in several other vessels. Because of the lesion's location and the physician's inability to pass a guide wire through it, no interventional procedure can be done at this time. The cardiothoracic team is called to perform emergency bypass surgery.

Mr. Tan's heart rate is now 110 and his BP has dropped to 70/40. The cardiologist inserts an IABP to decrease afterload and increase myocardial perfusion. Mr. Tan is diaphoretic and has weak pulses. Decreased renal perfusion leads to oliguria, and decreased cerebral perfusion causes mental status changes, including confusion, disorientation, and agitation. Some patients also develop metabolic acidosis and cardiac arrhythmias.

A PA catheter and arterial line are inserted, and Mr. Tan is transferred to the coronary care unit for further monitoring and treatment until the cardiothoracic team arrives.

Copyright ©2001. All Rights Reserved

Monitor him closely for changes in status. Check his level of consciousness, heart rate, BP, Spo_2, breath sounds, and urine output frequently. Treat pain and anxiety with opioids and sedatives, but cautiously and with consideration of his BP and oxygenation status.

Mr. Tan's central venous pressure is 12 mm Hg (normal, 2 to 6 mm Hg); pulmonary capillary wedge pressure, 22 mm Hg (normal, 8 to 12 mm Hg); and SVR, 1,900 dynes/second/cm^{-5} (normal, 800 to 1,200 dynes/second/cm^{-5}). You will use the hemodynamic values to guide fluid administration. Patients in cardiogenic shock can easily develop fluid overload and pulmonary edema, which is characterized by frothy, increased secretions; decreased oxygen saturation; and crackles. If fluid overload occurs, give diuretics with caution to prevent hypotension.

Three types of medications can be used to improve cardiac output in a patient in cardiogenic shock:

- Beta-blockers such as metoprolol decrease heart rate and increase cardiac filling time.
- Vasodilators such as nitroglycerin and nitroprusside decrease preload, afterload, and SVR. Use these medications cautiously if the patient's systolic BP is less than 90 mm Hg.
- Positive inotropes such as dobutamine and amrinone increase cardiac output by increasing the force of left ventricular contraction.

Mr. Tan is started on dobutamine at 5 mcg/kg/minute and dopamine at 5 mcg/kg/minute. He is also receiving I.V. heparin and morphine. The cardiac monitor shows sinus tachycardia with frequent premature ventricular contractions. Assess his cardiac rhythm frequently; patients in cardiogenic shock are prone to arrhythmias and may need temporary pacing in addition to medications to increase the heart rate or treat arrhythmias.

Within a short time, the cardiothoracic team arrives and takes Mr. Tan to surgery. He undergoes a quadruple bypass and is discharged home 5 days later. A home health care nurse will visit daily.

Follow trends in the patient's vital signs to identify complications quickly and intervene. Maintaining a normal BP can help prevent organ damage. Patients first develop an increase in sympathetic tone as a compensatory mechanism but, eventually, peripheral vessel tone relaxes and can lead to hypotension.

AND REMEMBER...

Remember to address these other areas when caring for a patient in shock:

- *Nutrition.* Patients in shock have high metabolic needs, so start nutritional support early. A dietitian should evaluate the patient's caloric intake and make sure that he is receiving adequate nutrition by whatever route is appropriate.
- *Skin care.* Fluid overload and third-spacing of fluid put the patient at risk for skin breakdown. Monitor his skin regularly and use pressure-relieving mattresses and frequent turning to prevent pressure ulcers.
- *Patient and family teaching.* Make sure the patient and family understand the diagnosis and are updated daily on the patient's progress.

Caring for a patient in shock requires constant vigilance, but with teamwork, nurses can give patients the best possible chance for recovery. ■

SELECTED BIBLIOGRAPHY

De Jong M. Clinical snapshot: Cardiogenic shock. *AJN* 1997;97(6):40-1.

Dracup K. Heart failure secondary to left ventricular systolic dysfunction: Therapeutic advances and treatment recommendations. *The Nurse Practitioner* 1996;21(9):56-68.

Hazinski, M., et al, eds. 2000 Handbook of Emergency Cardiovascular Care for Healthcare Providers. Dallas, Tex.: American Heart Association, 2000.

Emergency Nurses Association. Shock, in *Trauma Nursing Core Course Instructor Manual*, 5th edition. Chicago, Ill., Emergency Nurses Association, 2000.

Tangredi M. Clinical snapshot: Septic shock. *AJN* 1993;98(3):46-7.

Copyright ©2001. All Rights Reserved