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 	 Serum hypernatremia (> 145 mEq/L) Urine hypo - osmolality (< 500 mOsm/kg) Low urine specific gravity (1.001 - 1.005) ADH levels low-
	Elevated ADH levels	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)

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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

How to respond to shock

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This article describes three common types of shock—hypovolemic, septic, and cardiogenic—and how to recognize and respond appropriately. Shock has many causes, but the underlying disease process is the same, and prompt, aggressive intervention is the key to improved patient outcomes. [DIMENS CRIT CARE NURS 2001:20(2):22-27]

uickly identifying the type of shock and ensuring correct, aggressive treatment are key to patient survival. This article describes three types of shock—hypovolemic, septic, and cardiogenic—and how to recognize and respond appropriately to them.

Although shock has many causes, the disease process is the same: Reduced or poorly distributed blood volume leads to inadequate tissue perfusion. The body attempts to compensate (see Figure 1), but if the patient does not receive treatment to improve oxygenation and blood flow, the compensatory mechanisms fail. Continued hypoperfusion leads to an inflammatory response and the release of mediators, eventually resulting in organ dysfunction and death. Because hypovolemic shock is the most common type, we will start there.

HYPOVOLEMIC SHOCK

When the body loses circulating blood volume, venous return to the heart (preload) decreases; reduced vascular pressure triggers the sympathetic nervous system. Cardiac output increases and central and peripheral blood vessels constrict in an attempt to raise blood pressure (BP). Because of cardiac and cerebral autoregulation measures, this compensatory vasoconstriction has little effect on cardiac and cerebral vessels. Suspect hypovolemic shock due to fluid loss or fluid shifts in a patient who has experienced soft-tissue trauma, burn injuries, vomiting, diarrhea, or bleeding (including gastrointestinal blood loss). Do not give vasopressors without adequate fluid replacement; alone, vasopressors can cause cardiac decompensation and hemodynamic deterioration, especially in patients with ischemic heart disease.

Consider the case of John Sloane, 22, who was involved in a head-on motor vehicle collision. He was wearing his seat belt, and his driver's-side air bag deployed. When the paramedics bring him into the emergency department (ED), he is alert, slightly confused, and pale. He complains of pain in the right upper abdominal quadrant.

The nurse starts cardiac monitoring and pulse oximetry and begins the assessment. Mr. Sloane's vital signs are: heart rate, 136; BP, 88/60; respira-



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	Class I	Class II	Class III	Class IV
Blood loss	15%	15%-30%	30%-40%	>40%
(percent of	(750 ml)	(750-1,500 ml)	(1,500-2,000 mi)	(>2,000 ml)
total volume)				
BP	Normal	Normal	Decreased	Decreased
Heart rate	< 100	>100	>120	>140
Level of	Slightly anxious	Mildly anxious	Anxious and	Confused and
consciousness			confused	lethargic
Respirations (per min)	14-20	20-30	30-40	>40
Urinary output (ranges, in mi/hr)	>30	20-30	5-16	<5

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 Spo₂ 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 intraabdominal 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.

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 intraaortic 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

		TABLE 2 Assessing shock	
Common sessessment findings (early)	Hypovolemic • Pale, clammy skin • Thready peripheral pulses, nar- rowing pulse pressure, collapse of veins • Tachycardia, tachypnea • Anxiety, restlessness • Decreased urine output • Dilated pupils • Hypoactive bowel sounds	 Septic Warm, flushed skin Bounding pulses, widened pulse pressure Tachycardia, tachypnea (respiratory alkalosis) Decreased level of consciousness Hypotension Fever or hypothermia Agitation 	Cardiogenic • 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 Tachypnsa, then bradypnea Absent bowel sounds Decorticate or decarebrate positioning
Diagnostic criteria	 Lab values: hemoglobin changes depending on the cause and dura- tion of fluid loss and fluid replace- ment; elevated serum lactate; in- creased urine specific gravity; de- creased 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; car- diac output (CO) variable but even- tually below normal; systemic vas- cular resistance (SVR) increased Other diagnostic tests: X-rays, gastroscopy, gastric contents tested for occult blood 	 Lab values: leukocytosis, throm- bocytopenia, elevated serum lac- tate, increased urine specific gravity and osmolality, and decreased urine sodium Hemodynamic values: increased CO and low SVR in early shock; de- creased 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 Hernodynamic values: decreased systolic BP with narrow pulse pres- sure; cardiac index less than 2.1 liters/min/m²; pulmonary artery wedge pressure greater than 20 mm Hg; SVR greater than 1,800 dynes/sec/cm⁵ Other diagnostic tests: ECG; chest X-ray for pulmonary edema; echocardiography; ventriculogra- phy 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. 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 thirdspacing 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.

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Shock Study Guide and Reference

I. Definitions

A. Shock: Inadequate tissue perfusion to vital organs that typically manifests itself as respiratory failure, renal failure, altered mental status, DIC Decreased tissue perfusion leads to decreased delivery of vital oxygen/nutrients and energy to cells. This leads to eventual cell necrosis and acidosis and if left untreated can lead to cell death and organ damage

B. Compensatory mechanisms of shock include:

Massive vasoconstriction: tachycardia, shunting of blood from less vital organs to vital organs (cool, clammy skin; decreased capillary refill, decreased urine output, hypoactive bowel sounds; depending on the organs affected)

II. Types of shock:

A. Cardiogenic/Obstructive

- 1. Impaired tissue perfusion resulting from severe cardiac dysfunction and or obstruction to blood flow
- **2.** Causes: Papillary muscle rupture, acute valvular dysfunction, trauma, PE, aortic dissection
- **3.** Signs and symptoms include: Distended neck veins, large differences in extremity pressures, pulmonary edema, murmurs, distant heart sounds, (tamponade), elevated CVP
- **4.** Diagnostics: EKG, Echo, CXR, TEE, Cardiac catheterization, labs (ABG, CBC, Electrolytes, Cardiac enzymes troponin, CPK)
- 5. Pharmacology: + Inotropes, vasopressors, anticoagulants, diuretics

B. Neurogenic

- 1. Impaired tissue perfusion caused by sympathetic nervous system dysfunction
- 2. Rarest form of shock
- 3. Causes: Trauma, spinal anesthesia, spinal shock, spinal cord injury
- Signs and symptoms: Bradycardia, absence of thermoregulation (sweating, temperature control), paralysis profound hypotension not relieved with conventional treatment, decreased CVP Symptoms depend on the level of injury
- 5. Diagnostics: Radiologic imaging (MRI/CT)
- 6. Pharmacology: Volume, vasopressors, steroids, atropine

C. Septic

- 1. Impaired tissue perfusion caused by massive infection
- 2. Causes: Anything that can cause infection
- 3. Signs and symptoms: Tachycardia, hypotension, increased WBC, hyper/hypothermia, decreased CVP, confirmed infection
- 4. Diagnostics: Radiology (CXR), labs (WBC, lactate)
- 5. Pharmacology: Volume, antibiotics, vasopressors

D. Hypovolemic

- 1. Impaired tissue perfusion resulting from decreased intravascular volume
- Causes: Fluid shifts (burns), hemorrhage, dehydration (vomiting, diarrhea, DKA, DI, heat stroke), extravascular fluid loss/3rd spacing, ascites, ruptured spleen, pancreatitis, hemothorax
- 3. Signs and symptoms: Collapsed neck veins, poor capillary refill, dry/pale/grey skin, tenting, decreased CVP
- 4. Diagnostics: CXR, CBC, BUN/Creatinine
- 5. Pharmacology: Volume, treat the cause

E. Anaphylactic

- 1. Impaired tissue perfusion related to an allergic reaction
- 2. Causes: Allergens contrast dye, drug/food allergies, insects/animal/snake bites
- 3. Signs and symptoms: Stridor/wheezing, hives, abdominal symptoms, decreased CVP
- 4. Diagnostics: WBC, CXR
- 5. Pharmacology: SC Epi, steroids, racemic epi nebulizer, IV Benadryl

III. Assessment and care

- A. Primary survey: Is the patient breathing, do they have a pulse, are the awake?
- B. Secondary survey: Is the patient breathing effectively/can they maintain an airway, what are the vital signs, what is their mental status, do they have IV access, what is going on?
- C. Positioning: Reverse Trendelenburg for hypotension, or upright/tripod for ease of breathing
- D. Maintain adequate nutrition (TPN needed if NPO for a prolonged period)
- E. Infection prevention/infection control
- F. Invasive monitoring (Arterial line, CVP monitoring, central line)
- G. Treat the underlying cause
- H. Nursing care would consist of:
 - 1. Frequent vitals and physical assessment
 - 2. Pulmonary toileting/pulmonary prevention bundle (C&DB, incentive spirometer, early ambulation)
 - 3. Early and frequent ambulation (improves respiratory status, helps maintain perfusion, helps decrease risk of complications, improves patient morale)
 - 4. Administration of medications, fluids, blood products, electrolytes
 - 5. Frequent and accurate monitoring of I/O

IV. Goals of care

- A. Return patient to their baseline
- B. Increase systolic pressure to maintain adequate perfusion (Is the HR/BP in WNL for the patient?)
- C. Maintain adequate oxygenation (Improve pulmonary congestion)
- D. Maintain adequate fluid and electrolyte balances
- E. Prevent complications and delays in care/treatment
- V. References:

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