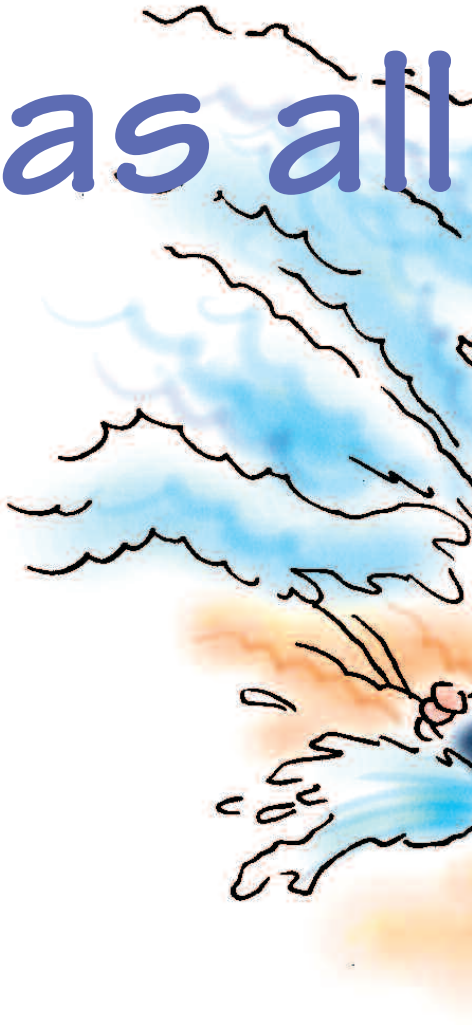
Third-spacing: Where has all



Suppose your patient has edema—indicating that there's enough fluid in his body—but his vital signs and urine output suggest that he's hypovolemic. What's going on? He's experiencing third-spacing, a shifting of fluid into interstitial spaces. Find out what needs to be done to get that fluid back where it belongs.

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The author has disclosed that she has no significant relationships with or financial interest in any commercial companies that pertain to this educational activity.

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YOU'RE TAKING REPORT on John Miller, who had a colectomy 2 days ago because of a ruptured diverticulus. You learn that his heart rate has increased over the past 24 hours, yet his blood pressure has been gradually falling and he's had marginal urine output (30 mL/hr). Mr. Miller weighs 4 kg more than before surgery, and he has generalized edema.

The health care team has decided not to increase Mr. Miller's maintenance intravenous (I.V.) infusion of lactated Ringer's solution. The nurse from the previous shift says not to worry: His fluid will "mobilize" and he'll "make urine" soon.

Not sure what she means by that, you head off to check out Mr. Miller's condition for yourself.

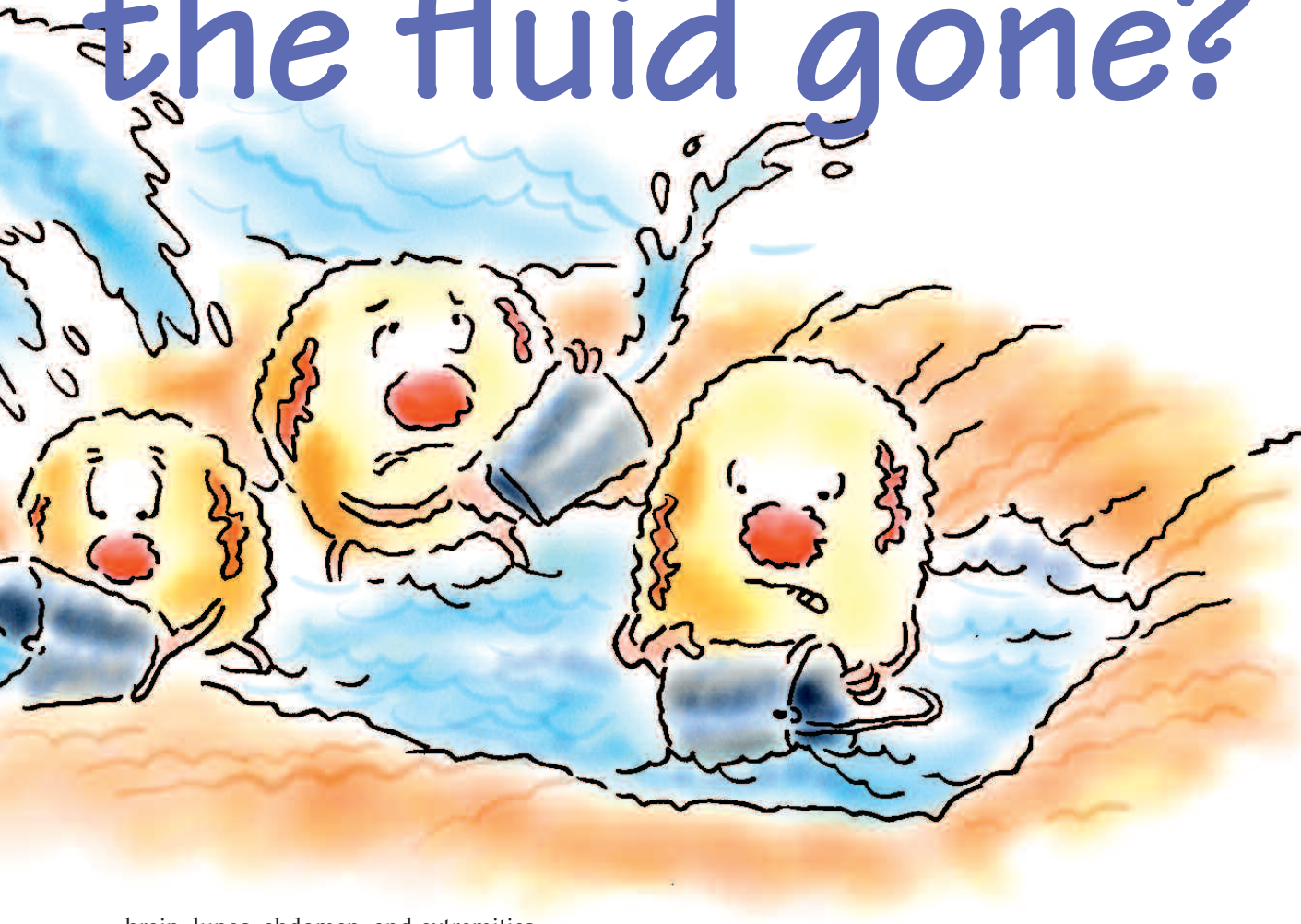
During your assessment, you find that Mr. Miller has 2+ edema, warm skin, and palpable peripheral pulses. His heart rate is 108 beats/min, his blood pressure is 110/64 mm Hg, and his urine output remains marginal at 30 mL/hr. Mr. Miller's abdomen is firm and distended, with hypoactive bowel sounds. He says his pain is well controlled with his patient-controlled analgesia infusion.

Mr. Miller's edema indicates that he has enough fluid in his body. But his vital signs and urine output seem to tell a different tale—hypovolemia. How can you reconcile these differences?

Who's on third?

Mr. Miller is experiencing third-spacing, which happens when fluid is trapped in the interstitial spaces. It can occur in the

the fluid gone?



brain, lungs, abdomen, and extremities. Let's look at the physiology of third-spacing and what you need to know to care for Mr. Miller.

You may remember from your pathophysiology class that fluid moves from intravascular (inside the blood vessel) to extravascular (outside the blood vessel) spaces and from intracellular (inside the cell) to extracellular (outside the cell) spaces (see *Fluids 101*). Fluid is constantly on the move to maintain balance (see *On the move*).

Intravascular-to-extravascular movement of fluid occurs through diffusion, which is controlled by hydrostatic and capillary plasma oncotic pressures.

Hydrostatic pressure is the pressure that fluid places on the wall of a blood vessel. If the vessel wall is strong, as with an artery,

On the move

Fluids are constantly on the move, seeking to keep the body in equilibrium. Here's how they do it.

- *Diffusion:* This is passive movement of molecules across a membrane from an area of higher concentration to an area of lower concentration.
- *Osmosis:* Water moves through a selectively permeable membrane from an area of lower concentration of ions to an area of higher concentration of ions.
- *Active transport:* This is movement of molecules against a concentration as they move from an area of lower concentration to an area of higher concentration. The movement requires energy.

fluid will be held in. But if the wall is weak or semipermeable, as with a capillary, hydrostatic pressure will force fluid out of the vessel. Hydrostatic pressure pushes fluid from the arterial side of the capillary into the interstitial space.

Intravascular or intracapillary *oncotic pressure* is determined by plasma proteins in the bloodstream. The proteins keep fluid in the blood vessel, with albumin protein molecules doing 70% of the work.

When oncotic pressure goes bad

Loss of albumin or protein leads to decreased oncotic pressure. Fluid can now leak from the intravascular space into the interstitial space and stay there, causing edema. Because this fluid is lost from the circulating blood volume, cardiac output decreases. You can see albumin loss in patients with liver failure, liver dysfunction, or malnutrition. The low albumin levels lead to loss of fluid into the peritoneum, causing ascites.

At some point, a patient with decreased oncotic pressure might need *hypertonic* or colloid fluids, such as albumin, to "pull" the fluid in the interstitial space back into the intravascular space and increase the circulating volume. Giving *hypotonic* fluids causes fluid to shift from the intravascular space into the interstitial space, increasing interstitial fluid and edema. I.V. fluids that are *isotonic* should move in and out of the vascular space equally, so that's what Mr. Miller is receiving (see *How fluids affect cells* and *Quick guide to I.V. solutions*).

Fluids 101

Fluids bring nutrition and oxygen to cells and take away cell waste for metabolism or excretion. This important role may be why 60% of an adult's weight is fluid. This fluid is divided into two compartments: intracellular and extracellular.

Intracellular fluid, located inside the cells, makes up 40% of the body's total fluid. The remaining 60% of the body's total fluid is extracellular fluid, which is, logically, located outside the cell. Extracellular fluid is further divided into interstitial (between cells, in tissue) and intravascular (inside the blood vessels) fluid.

The body's fluid should be in balance, with the volume entering the body equal to what's leaving. Fluid loss can occur through urine, sweat, stool, and incidental losses from respiratory effort.

And another thing...

Even when the oncotic pressure and albumin levels are normal, fluid can still leak out of the vessels if there's damage to the capillary membranes. That's what happened to Mr. Miller. During surgery, his bowel was handled, poked, and prodded as it was repaired, damaging tissues and destroying the endothelial cells lining the capillaries. Not good news for Mr. Miller: Endothelial cells help maintain vascular integrity, which facilitates osmosis, diffusion, and active transport of fluids and electrolytes.

Once Mr. Miller's endothelial cells were destroyed, permeability of his capillary membranes increased, allowing fluid to move from the intravascular space to the interstitial space, but not back again. Oncotic pressure fell and fluid was trapped, causing edema. (Remember Mr. Miller's 2+ generalized edema?) The excessive fluid in the abdomen (ascites) causes increased pressure and firmness. The fluid in the interstitial space and tissue compliance determine the level of pressure.

Periods of hypotension, which cause hypoperfusion, along with hypoxia and ischemia, can also destroy endothelial cells and trap fluid in the interstitial space. This can occur in the brain as a result of a cardiac arrest. In the lungs, fluid accumulation can lead to cardiogenic or noncardiogenic pulmonary edema. As you saw with Mr. Miller, excessive fluid in the extremities leads to peripheral edema. And if the fluid

Quick guide to I.V. solutions

A solution is isotonic if its osmolarity falls within (or near) the normal range for serum (240 to 340 mOsm/L). A hypotonic solution has a lower osmolarity; a hypertonic solution, a higher osmolarity. This chart lists common examples of the three types of I.V. solutions and provides key considerations for administering them.

Solution	Examples	Nursing considerations
Isotonic	• Lactated Ringer's (275 mOsm/L) • Ringer's injection (275 mOsm/L) • 0.9% sodium chloride (308 mOsm/L) • 5% dextrose in water (D_5W; 260 mOsm/L) • 5% albumin (308 mOsm/L) • Hetastarch (310 mOsm/L) • Normosol (295 mOsm/L) (295 mOsm/L)	• Because isotonic solutions expand the intravascular compartment, closely monitor the patient for signs of fluid overload, especially if he has hypertension or heart failure. • Because the liver converts lactate to bicarbonate, don't give lactated Ringer's solution if the patient's pH exceeds 7.5. • Avoid giving D_5W to a patient at risk for increased intracranial pressure (ICP) because it acts like a hypotonic solution. (Although usually considered isotonic, D_5W is actually isotonic only in the container. After administration, dextrose is quickly metabolized, leaving only water—a hypotonic fluid.)
Hypotonic	• 0.45% sodium chloride (154 mOsm/L) • 0.33% sodium chloride (103 mOsm/L) • 2.5% dextrose in water (126 mOsm/L)	• Administer cautiously. Hypotonic solutions cause a fluid shift from blood vessels into cells. This shift could cause cardiovascular collapse from intravascular fluid depletion and increased ICP from fluid shift into brain cells. • Don't give hypotonic solutions to patients at risk for increased ICP from stroke, head trauma, or neurosurgery. • Don't give hypotonic solutions to patient at risk for third-space fluid shifts (abnormal shifts into the interstitial space)—for example, patients with burns, trauma, or low serum protein levels from malnutrition or liver disease.
Hypertonic	• 5% dextrose in 0.45% sodium chloride (406 mOsm/L) • 5% dextrose in 0.9% sodium chloride (560 mOsm/L) • 5% dextrose in lactated Ringer's (575 mOsm/L) • 3% sodium chloride (1,025 mOsm/L) • 25% albumin (1,500 mOsm/L) • 7.5% sodium chloride (2,400 mOsm/L)	• Because hypertonic solutions greatly expand the intravascular space, administer them by I.V. pump and closely monitor the patient for circulatory overload. • Hypertonic solutions pull fluid from the interstitial space, so don't give them to a patient with a condition that can cause cellular dehydration, such as diabetic ketoacidosis. • Don't give hypertonic solutions to a patient with impaired heart or kidney function—his system can't handle the extra fluid.

accumulates in the abdomen, the patient can develop edema of the bowel, which may result in intra-abdominal hypertension or abdominal compartment syndrome if not reversed over the first 24 to 48 hours after surgery.

What's going on inside the cells?

Interstitial fluid trapping causes compression of the microvasculature in the distal circulation. As the cells swell and compress the capillaries around them, blood flow is further impaired, leading to hypoperfusion and ischemia. Anaerobic metabolism kicks in (except in the brain cells) to sustain the cells until perfusion is restored. But when anaerobic metabolism eventually fails, the sodium/potassium pump inside the cell starts to fail too. Sodium and potassium switch places: Sodium moves into the intracellular space while potassium moves into the extracellular space.

An increased level of intracellular sodium causes water to be pulled into the cell. The cell wall membrane stretches and releases cytokines and other mediators. Once released, mediators become active and create local inflammation, which further damages the cells.

Mediators also get into the systemic circulation, where the blood gives them a ride to other parts of the body. This free ride can lead to systemic inflammatory response syndrome (SIRS). Organ failure may also occur, which leads to further inflammation and dysfunction, release of mediators, and progression to multiple organ dysfunction syndrome (MODS), which increases the incidence of mortality (see *Mediators of SIRS and MODS*). Let's hope Mr. Miller doesn't go down this road!

Back to Mr. Miller

Now that you understand more about the fluid changes that occur with third-spacing, Mr. Miller's vital signs aren't surprising. The shift of fluid into the interstitial tissues decreased his intravascular circulating volume. The baroreceptors in the aorta and carotid arches sensed the lower volume and told the sympathetic nervous system (SNS) to get busy. The SNS did its job by causing release of epinephrine and norepinephrine, which lead to vasoconstriction of the peripheral vessels and an increasing heart rate. Let's take a closer look at this process.

Vasoconstriction shunts blood from the periphery to the major organs, which can compromise circulation to the extremities. Compromised circulation may lead to hypoxia, ischemia, and SIRS.

You'll want to keep a sharp eye on Mr. Miller's perfusion: Check peripheral pulses, skin temperature and sensation, and capillary return on the hands and feet.

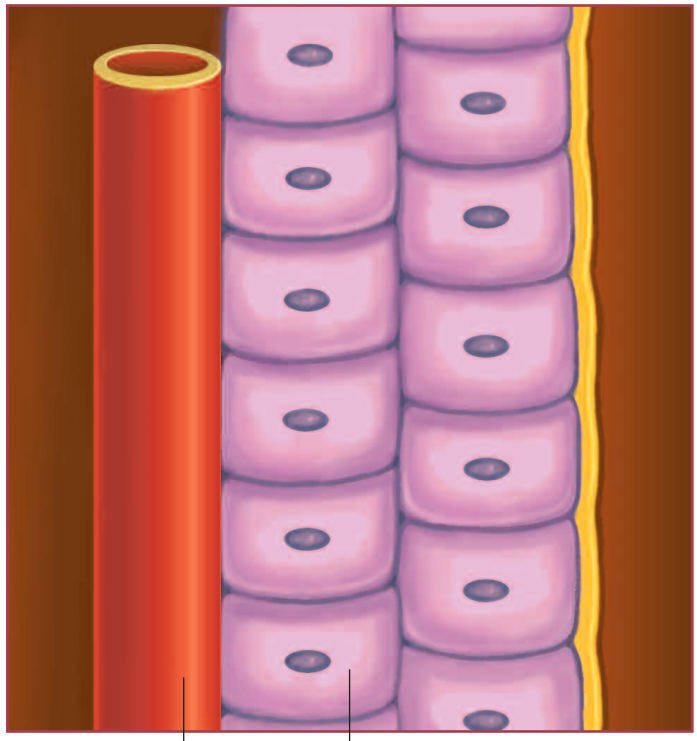
An *increased heart rate* kicks up cardiac output so that the body's oxygen requirements can be met. But the heart can only do so much. If myocardial oxygen demand keeps rising, the heart won't be able to supply enough oxygen; myocardial ischemia or infarction may result. Be ready to respond quickly if Mr. Miller shows signs or symptoms of myocardial ischemia, such as chest pain.

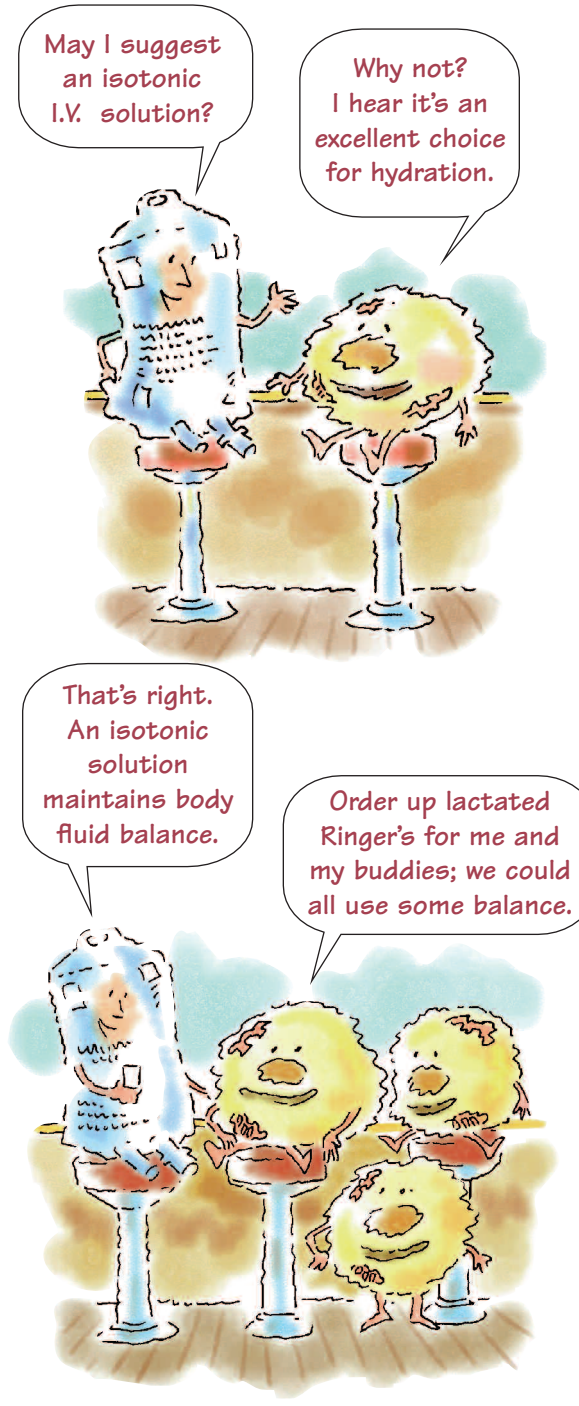
Mr. Miller's *kidneys* are doing their part to help out. When they sensed the decrease in glomerular filtration rate, which would happen with his marginal urine output, they launched the renin-angiotensin-aldosterone system. This system causes peripheral vasoconstriction from the effects of angiotensin II and fluid retention from the release of aldosterone. Antidiuretic hormone is also released in response to low circulatory volume, and it tells the kidneys to absorb more sodium from the tubules; this will also increase the absorption of water. The goal is to increase circulating volume, thereby boosting cardiac output and blood pressure. Without this compensatory mechanism, Mr. Miller's blood pressure would be lower than it is right now.

How fluids affect cells: Isotonic solutions

An isotonic solution has the same solute concentration (or osmolarity) as serum and other body fluids. Infusing the solution doesn't alter the concentration of serum; therefore, osmosis doesn't occur. (For osmosis to occur, there must be a difference in solute concentration between serum and the interstitial fluid.)

The isotonic solution stays where it's infused, inside the blood vessel, and doesn't affect the size of cells.

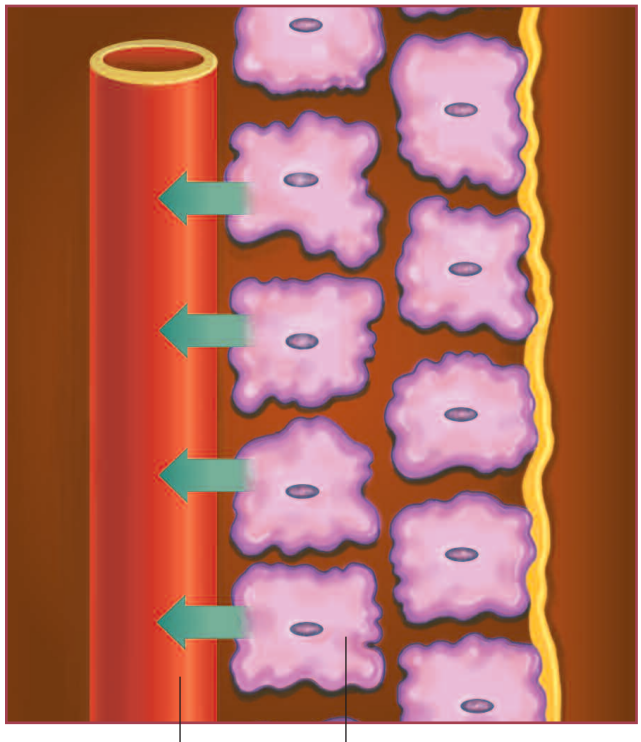


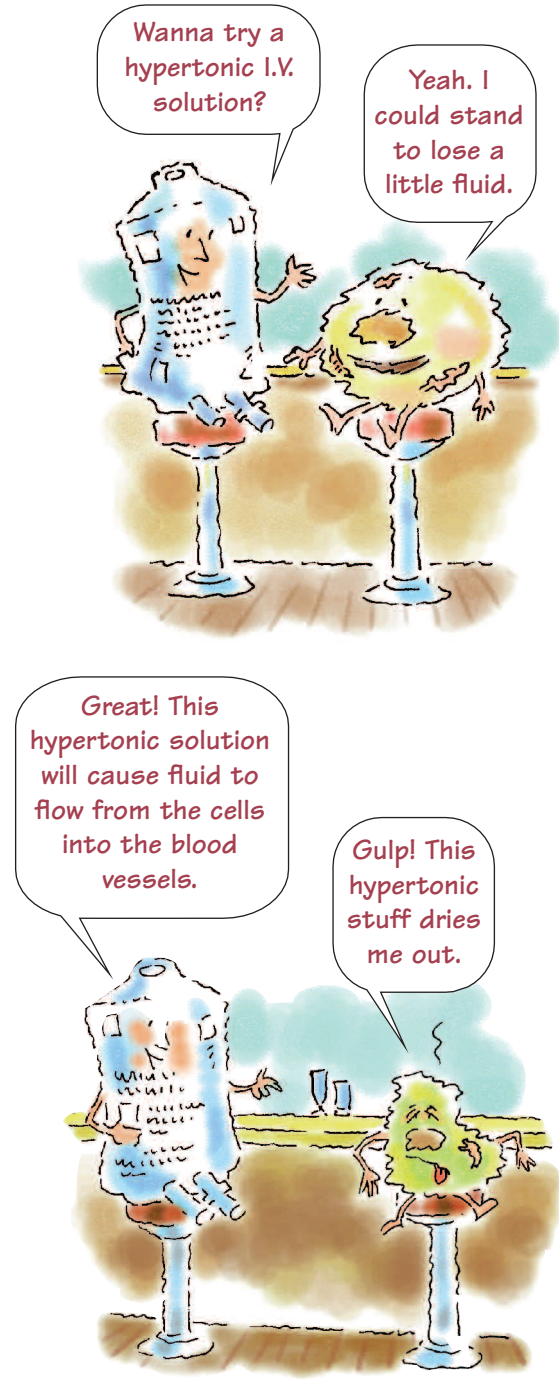


How fluids affect cells: Hypertonic solutions

A hypertonic I.V. solution has a solute concentration higher than the solute concentration of serum. Infusing a hypertonic solution increases the solute concentration of serum. Because the solute concentration of serum is now different from the interstitial fluid, osmosis occurs. Fluid is pulled from the cells and the interstitial compartment into the blood vessels.

Many patients receive hypertonic fluids postoperatively. The shift of fluid into the blood vessels reduces the risk of edema, stabilizes blood pressure, and regulates urine output.

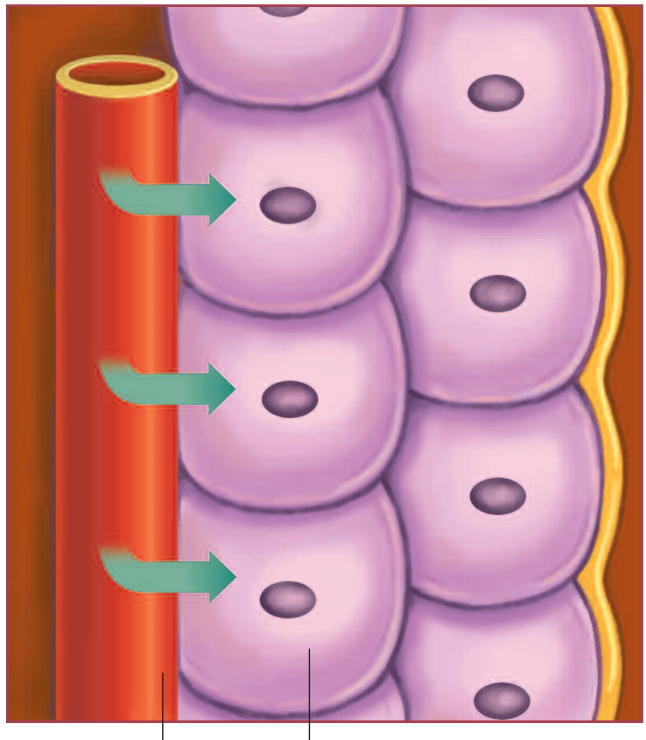


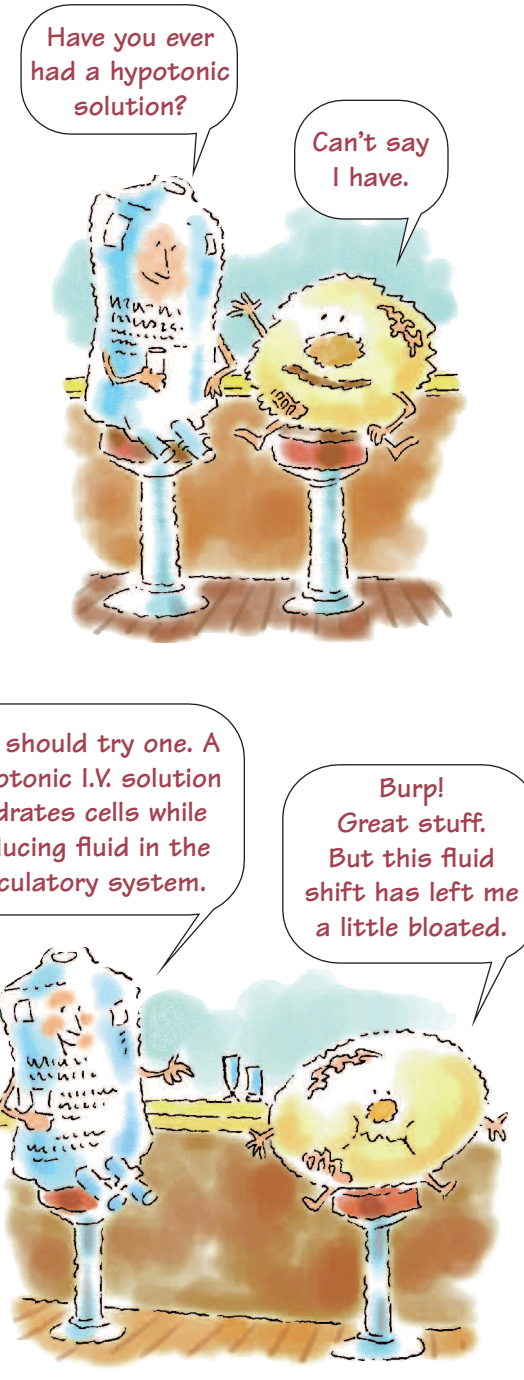


How fluids affect cells: Hypotonic solutions

A hypotonic I.V. solution is the opposite of a hypertonic solution. It has a lower solute concentration than serum. Infusion of a hypotonic solution causes the solute concentration of serum to decrease. Because the solute concentration of serum is now different from the interstitial fluid, osmosis occurs.

This time, the fluid shift is in the opposite direction than that of a hypertonic fluid. Fluid shifts out of the blood vessels and into the cells and interstitial spaces, where the solute concentration is higher.



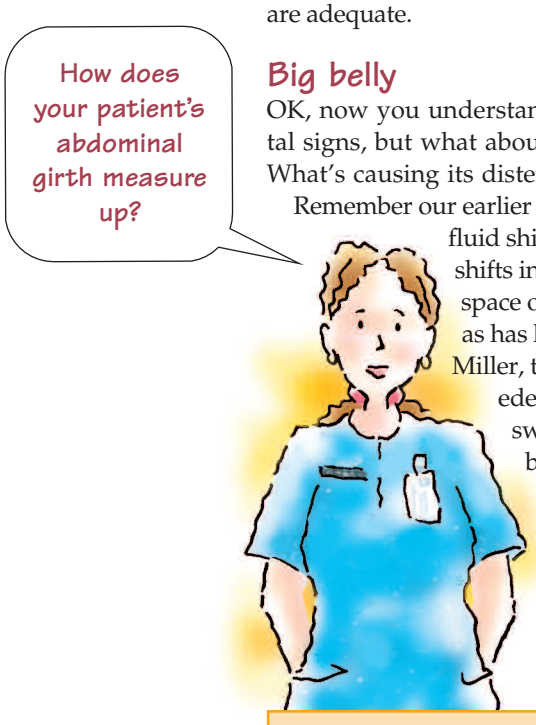


One more thing: Know what Mr. Miller's heart rate and blood pressure normally are so you can determine if his current readings are adequate.

Big belly

OK, now you understand Mr. Miller's vital signs, but what about his abdomen? What's causing its distension?

Remember our earlier discussion about fluid shifts? When fluid shifts into the interstitial space of the bowel tissue, as has happened with Mr. Miller, the tissue becomes edematous. The swelling causes the bowel to expand in the peritoneum. The abdominal skin stretches to accommodate the edema, similar to the way a balloon expands when you blow air into it.



We monitor this effect by measuring abdominal girth, and now we can also measure bladder pressure to determine the amount of pressure the edematous bowel is generating.

To measure abdominal girth, wrap a tape measure around the patient's abdomen at the umbilicus and record the measurement. Remeasure abdominal girth every 4 to 8 hours, making sure to place the tape measure on the same spot. Report any increased measurements to the health care team.

If the patient has a urinary catheter in place, you can measure abdominal pressure by obtaining bladder pressure measurements (if you have the capability of doing these measurements). After clamping the urinary catheter, aseptically insert an 18-gauge Angiocath in the catheter's sampling port or connect an IAP bladder pressure monitor. Connect the system to a

Mediators of SIRS and MODS

Mediator	Effects
Tumor necrosis factor	• Vasodilation • Activation of other proinflammatory mediators
Histamine	• Intense vasodilation • Increased capillary membrane permeability
Bradykinins	• Vasodilation • Activation of the coagulation cascade
Platelet-activating factor	• Platelet aggregation • Activation of the coagulation cascade
Myocardial depressant factor	• Depressed myocardial function
Arachidonic acid cascade, an inflammation-related cascade, including:	
• Leukotrienes	• Increased tissue permeability
• Thromboxanes	• Vasoconstriction of arterioles
• Prostaglandins	• Vasodilation

pressure transducer and level the transducer to the iliac crest. Instill 50 mL of sterile 0.9% sodium chloride into the bladder, turn the stopcock or valve, and obtain the pressure measurement at end expiration. If the intra-abdominal pressure is more than 12 mm Hg, suspect that the patient has intra-abdominal hypertension (see *Making the grade*).

Elevated pressure in the abdomen indicates increased bowel edema. As the bowel edema progresses (as a result of cellular ischemia and hypoperfusion) and there's more pressure in the abdomen, the blood return to the right side of the heart is impaired; so is blood flow out of the left ventricle. The increased abdominal pressure can impair lung expansion as well, leading to respiratory distress. It can also exert pressure on the renal circulation, leading to renal dysfunction.

Mr. Miller's edema happened quickly because of his surgery, so his skin can't keep pace with the swelling. That's why his abdomen is firm. Rising pressure in the abdomen from third-spacing compresses the major blood vessels running through it, which causes the following problems:

- in the *vena cava*, reduced preload (venous return to the heart), leading to decreased cardiac output, which results in decreased blood pressure
- in the *aorta* and the *iliac and femoral arteries*, increased afterload (pressure in the peripheral circulation), further reducing cardiac output and blood pressure
- in the *renal vessels*, impaired kidney function
- in the *spleen's vasculature*, impaired blood flow to the bowel, liver, and spleen.

If the fluid shift isn't corrected and pressure keeps rising, Mr. Miller will develop intra-abdominal hypertension or abdominal compartment syndrome. This situation results in a downward spiral of bowel ischemia and tissue death leading to necrosis. So you'll want to watch him closely for signs of intra-abdominal hypertension and abdominal compartment syndrome: increasing abdominal girth or bladder pressure and increasing pain not controlled with the previous medication dosing. Early suspicion of intra-abdominal hypertension or abdominal compartment syndrome will allow time for interventions to prevent or minimize tissue damage.

Making the grade

A patient's intra-abdominal pressure (IAP) determines if he has intra-abdominal hypertension. In a consensus statement, the World Society of Abdominal Compartment Syndrome defines intra-abdominal hypertension as "sustained or repeated pathologic elevation of IAP $\geq$ 12 mm Hg." There are four grades of intra-abdominal hypertension, according to this group:

- ***Grade I:*** IAP of 12 to 15 mm Hg
- ***Grade II:*** IAP of 16 to 20 mm Hg
- ***Grade III:*** IAP of 21 to 25 mm Hg
- ***Grade IV:*** IAP of > 25 mm Hg.

The higher the number, the more severe the condition and the greater the risk of complications.

What's next?

Mr. Miller's vital signs are slightly abnormal but stable, so he seems to be tolerating the fluid shifting. Over the next several hours (up to 48 hours), the fluid shift will be resolved or he will continue to develop bowel edema and, eventually, ischemia.

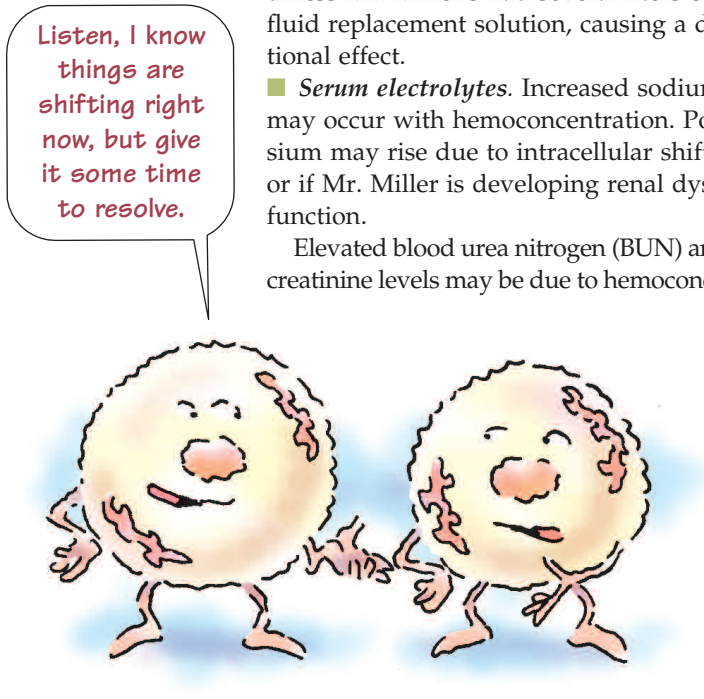
During this time, closely monitor Mr. Miller's vital signs, urine output, and peripheral perfusion. Report any changes to the health care provider. Here are other areas to keep tabs on:

- ***Mental status.*** Is Mr. Miller responsive and able to communicate and answer questions appropriately? If not, his blood pressure isn't high enough for adequate perfusion.
- ***Ventilation/perfusion status.*** Can he maintain adequate ventilation to support his oxygen needs? Does he have crackles? Is his oxygen saturation greater than 97% on room air, or does he need supplemen-

tal oxygen therapy?

- ***Hematocrit and hemoglobin.*** Rising hematocrit and hemoglobin levels indicate hemoconcentration of serum due to fluid shifting to the interstitial space; decreases in these values may indicate bleeding—unless Mr. Miller's had several liters of fluid replacement solution, causing a dilutional effect.
- ***Serum electrolytes.*** Increased sodium may occur with hemoconcentration. Potassium may rise due to intracellular shifting or if Mr. Miller is developing renal dysfunction.

Elevated blood urea nitrogen (BUN) and creatinine levels may be due to hemoconcentration, but a rising creatinine with a normal BUN level may signal intrarenal dysfunction.



Lactate is produced as a byproduct of anaerobic metabolism. In patients who've had bowel surgery, an elevated lactate level may indicate bowel ischemia—unless the patient has liver dysfunction or failure and received several liters of lactated Ringer's solution for fluid resuscitation. The lactate in lactated Ringer's solution is converted to bicarbonate in a healthy liver. But in a dysfunctional or diseased liver, lactate isn't converted; it remains in the blood.

- ***Abdominal pressure.*** Measure abdominal girth or bladder pressure at least every 4 to 8 hours while Mr. Miller's vital signs are abnormal and his urine output is low.
- ***Fluid resuscitation.*** You'll continue to give Mr. Miller a maintenance I.V. infusion of isotonic fluid, as well as intermittent boluses of a colloid, such as albumin. Albumin will pull fluid from the interstitial space into the intravascular space. If the kidneys can't get rid of the extra fluid on their own, a small dose of a loop diuretic like furosemide (Lasix) can help. Remember, colloid fluids are plasma proteins, so their higher molecular structures allow you to give less volume to support Mr. Miller's blood pressure.

If his hemoglobin is low, infusing blood products, such as packed red blood cells, as needed will help increase oxygen-carrying capacity, as well as increase intravascular oncotic pressure and pull fluid from the interstitial space.

did you know?

Why does fluid follow a protein like albumin? Protein is a large molecule with a negative charge. It attracts the most abundant extracellular fluid ion—sodium—which has a positive charge. You probably remember that water follows sodium. So, sodium follows protein, and water follows sodium.

What if...?

If the health care provider suspects bowel ischemia or necrosis, he may order a kidney-ureter-bladder (KUB) X-ray and computed tomography (CT) scan.

A KUB image will show the extent of bowel edema and any "free" air, which would indicate bowel perforation. A CT scan detects worsening bowel edema, lack of adequate perfusion, or hematomas formed from bleeding.

A patient whose vital signs are deteriorating and who has decreasing urine output and increasing abdominal girth or bladder pressure readings will likely return to the operating room; he may have a perforated bowel that needs to be repaired. If the bowel

isn't perforated, the surgeon still may have to open up the abdomen to allow the edema to subside; it'll be closed later.

A trip to the operating room is definitely in order if the patient's KUB film shows free air; he needs to have that perforated bowel repaired *STAT!* While they're in there, the surgical team will also explore the abdomen for any further damage to the bowel related to perforation or edema.

It's up to you

Your clinical assessments and index of suspicion as to what could be going on in Mr. Miller's abdomen will help put him on the path of continued recovery. And, yes, now you know just what the nurse on the other shift meant by "mobilize fluid." ■

Learn more about it

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Third-spacing: Where has all the fluid gone?

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Third-spacing: Where has all the fluid gone?

GENERAL PURPOSE: To provide the registered professional nurse with an overview of the pathophysiology, signs and symptoms, and nursing care of a patient who is experiencing third-spacing of fluids. **LEARNING OBJECTIVES:** After reading this article and taking this test, you should be able to: 1. Describe the pathophysiology and complications of third-spacing. 2. Identify nursing assessments, monitoring, and interventions for patients with third-spacing.

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1. Third-spacing refers to fluid trapped in the
a. intravascular spaces.
b. interstitial spaces.
c. intracellular spaces.

2. Third-spacing typically occurs in the extremities, abdomen, and
a. lungs.
b. liver.
c. kidneys.

3. In third-spacing, fluid in the body moves from the
a. intravascular spaces to the extravascular spaces.
b. extravascular spaces to the intracellular spaces.
c. extracellular spaces to the extravascular spaces.

4. Hydrostatic pressure primarily affects the
a. lymph system.
b. veins.
c. capillaries.

5. Loss of albumin or protein can cause
a. increased cardiac output.
b. decreased oncotic pressure.
c. decreased hydrostatic pressure.

6. Ascites is caused by
a. a low albumin level and fluid accumulation in the peritoneum.
b. decreased plasma proteins in the peritoneum.
c. excess fluid in the intracellular spaces of the liver.

7. Hypertonic I.V. fluids are used as therapy because they
a. pull fluid from intravascular space into interstitial space.
b. replace lost proteins.
c. increase circulating volume.

8. Normally, the body's fluid is distributed as
a. 75% intracellular and 25% extracellular.
b. 60% extracellular and 40% intracellular.
c. 20% intracellular and 80% extracellular.

9. Albumin is effective in treating third-spacing because it
a. decreases oncotic pressure.
b. attracts sodium and water.
c. increases hydrostatic pressure.

10. How does third-spacing relate to systemic inflammatory response syndrome (SIRS)?
a. Swollen cells of interstitial edema release mediators.
b. Histamine release causes capillary vasoconstriction.
c. Bradykinin release inhibits the coagulation cascade.

11. Decreased intravascular circulating volume leads to
a. vasoconstriction and increased heart rate.
b. vasodilation and decreased heart rate.
c. vasodilation and increased blood pressure.

12. Compression of the vena cava from abdominal third-spacing can cause
a. increased blood pressure.
b. decreased cardiac output.
c. increased preload.

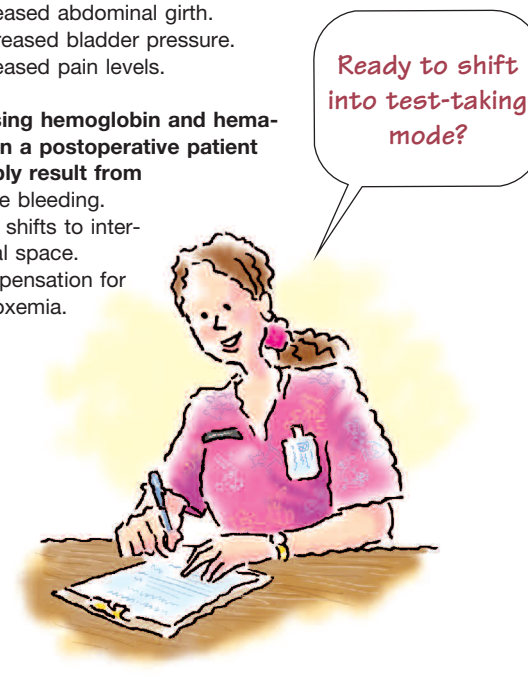
13. Which lab finding may indicate postoperative bowel ischemia?
a. increased sodium
b. increased blood urea nitrogen
c. increased lactate

14. Five percent dextrose in ½ normal saline is classified as
a. isotonic.
b. hypotonic.
c. hypertonic.

15. Patients receiving hypertonic I.V. solutions should be monitored for which adverse effect?
a. circulatory overload
b. increased peripheral edema
c. decreased intravascular volume

16. Signs of abdominal compartment syndrome include each of the following *except*
a. increased abdominal girth.
b. decreased bladder pressure.
c. increased pain levels.

17. Rising hemoglobin and hematocrit in a postoperative patient probably result from
a. acute bleeding.
b. fluid shifts to interstitial space.
c. compensation for hypoxemia.



Go to the next page for the CE Enrollment Form.

CE

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A. Registration Information:

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Address _____

City _____ State _____ ZIP _____

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Job title _____ Specialty _____

Type of facility _____ Are you certified? ❑ Yes ❑ No

Certified by _____

State of license (1) _____ License # _____

State of license (2) _____ License # _____

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❑ Please fax my certificate to me.

Acute pancreatitis: Inflammation gone wild (page 18)

B. Test Answers: Darken one circle for your answer to each question.

	a	b	c
1.	❍	❍	❍
2.	❍	❍	❍
3.	❍	❍	❍
4.	❍	❍	❍
5.	❍	❍	❍
6.	❍	❍	❍
7.	❍	❍	❍
8.	❍	❍	❍
9.	❍	❍	❍
10.	❍	❍	❍
11.	❍	❍	❍
12.	❍	❍	❍
13.	❍	❍	❍
14.	❍	❍	❍
15.	❍	❍	❍
16.	❍	❍	❍
17.	❍	❍	❍

Registration deadline: October 31, 2008
Contact hours: 2.5
Fee: $22.95
Test code: NMIE1306

C. Course Evaluation*

1. Did this CE activity's learning objectives relate to its general purpose? ❑ Yes ❑ No
2. Was the journal home study format an effective way to present the material? ❑ Yes ❑ No
3. Was the content relevant to your nursing practice? ❑ Yes ❑ No
4. How long did it take you to complete this CE activity?___ hours___minutes
5. Suggestion for future topics

_____

Bad blood: Tips for preventing CR-BSIs (page 30)

B. Test Answers: Darken one circle for your answer to each question.

	a	b	c
1.	❍	❍	❍
2.	❍	❍	❍
3.	❍	❍	❍
4.	❍	❍	❍
5.	❍	❍	❍
6.	❍	❍	❍
7.	❍	❍	❍
8.	❍	❍	❍
9.	❍	❍	❍
10.	❍	❍	❍
11.	❍	❍	❍
12.	❍	❍	❍
13.	❍	❍	❍
14.	❍	❍	❍
15.	❍	❍	❍
16.	❍	❍	❍
17.	❍	❍	❍

Registration deadline: October 31, 2008
Contact hours: 2.5
Fee: $22.95
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C. Course Evaluation*

1. Did this CE activity's learning objectives relate to its general purpose? ❑ Yes ❑ No
2. Was the journal home study format an effective way to present the material? ❑ Yes ❑ No
3. Was the content relevant to your nursing practice? ❑ Yes ❑ No
4. How long did it take you to complete this CE activity?___ hours___minutes
5. Suggestion for future topics

_____

Third-spacing: Where has all the fluid gone? (page 42)

B. Test Answers: Darken one circle for your answer to each question.

	a	b	c
1.	❍	❍	❍
2.	❍	❍	❍
3.	❍	❍	❍
4.	❍	❍	❍
5.	❍	❍	❍
6.	❍	❍	❍
7.	❍	❍	❍
8.	❍	❍	❍
9.	❍	❍	❍
10.	❍	❍	❍
11.	❍	❍	❍
12.	❍	❍	❍
13.	❍	❍	❍
14.	❍	❍	❍
15.	❍	❍	❍
16.	❍	❍	❍
17.	❍	❍	❍

Registration deadline: October 31, 2008
Contact hours: 2.5
Fee: $22.95
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C. Course Evaluation*

1. Did this CE activity's learning objectives relate to its general purpose? ❑ Yes ❑ No
2. Was the journal home study format an effective way to present the material? ❑ Yes ❑ No
3. Was the content relevant to your nursing practice? ❑ Yes ❑ No
4. How long did it take you to complete this CE activity?___ hours___minutes
5. Suggestion for future topics

_____

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