

How to
recognize and
respond to

refeeding syndrome

**If your patient is malnourished,
you need to assess her carefully before she
restarts nutrition by any route to avoid
potentially lethal consequences.**

By Mary Ann Yantis, RN, PhD,
and Robyn Velandar, RD, LD, CNSD

YOU'RE CARING FOR Betty Dawson, 73, who's newly admitted with dehydration and malnutrition. She's 5 feet 6 inches and weighs 121 pounds (55 kg). Her admission basic metabolic panel is normal except for a slight increase in serum sodium (149 mEq/L; normal range, 136 to 145 mEq/L) and a decrease in potassium (3.3 mEq/L; normal range, 3.5 to 5.2 mEq/L) and albumin (3.2 grams/dL; normal range, 3.5 to 4.8 grams/dL). Her baseline magnesium level is 2.2 mg/dL (normal range, 1.8 to 2.6 mg/dL), and her baseline phosphorus level is 3 mg/dL (normal range, 2.5 to 4.5 mg/dL). She's initially treated with dextrose 5% in 0.45% sodium chloride solution for hydration and I.V. potassium to correct her hypokalemia. Ms. Dawson is diagnosed with inflammatory bowel disease and malnutrition.

On hospital day two, she's started on parenteral nutrition (PN) at a rate that delivers 25 to 35 kcal/kg or 1,375 to 1,925 kcal/day, and her condition is stable. But when you assess her early in the morning on hospital day three, you note lethargy, shortness of breath, and muscle weakness. All of her vital signs are normal except her pulse, which has become irregular. Would you realize that these nonspecific signs might indicate a potentially life-threatening disorder?

Severely malnourished patients can experience significant fluid shifts and electrolyte imbalances after aggressive nutritional support is initiated. This potentially lethal

disorder, known as *refeeding syndrome* (RFS), is usually associated with PN, but it can also occur with enteral nutrition (EN), oral intake, or dextrose-containing I.V. fluids.¹

Although information about RFS is available in nutrition journals, it isn't widely discussed in general nursing literature. We'll fill that gap with this article by discussing how to identify this dangerous complication and what to do about it.

Disorder discovered

This syndrome was first observed and described after World War II when victims of starvation were noted to experience cardiac or neurologic dysfunction or both after being reintroduced to food. Today, we rarely see patients who are as severely malnourished as these peo-

ple were, but PCM (protein-calorie malnutrition) is common among hospitalized patients: Almost one-third are malnourished and nearly two-thirds are at risk for malnutrition.²

As you think of the changes in refeeding syndrome, you probably envision a thin, emaciated patient, but even an overweight or obese patient can be malnourished.

Electrolyte disturbances (primarily decreased levels of phosphorus, magnesium, or potassium) occur immediately upon the rapid initiation of refeeding—commonly within 12 to 72 hours—and can continue for the next 2 to 7 days. Cardiac complications can develop within

the first week, often within the first 24 to 48 hours, with neurologic signs and symptoms developing somewhat later.³

The following patients are at particular risk for RFS:

Hospitalized patients. Refeeding syndrome occurs primarily in hospitals. One study found that in 51 patients who received vigorous nutritional support, 80% of the patients developed depletions in potassium, magnesium, or phosphorus. Ninety-three percent of the patients in this study who were considered "at nutrition risk" and 74% of those "not at nutrition risk" had electrolyte imbalances.⁴ These results show the importance of closely monitoring electrolytes in *all* patients who are receiving EN or PN support.

Children. Although RFS is reported most often in adults, it can also occur in children. In a review of the literature, we found reports of 27 children who developed this syndrome after initiation of oral or enteral feeding; 9 of them died as a result of related complications.⁵

Patients with chronic malnutrition. Refeeding syndrome can develop in patients who are chronically malnourished for any reason (see *Who's at risk for malnutrition?*). At particular risk are those who:

- have lost at least 10% of their weight within the preceding 4 weeks
- haven't eaten for more than 7 days, such as a patient experiencing nausea and vomiting or anorexia from chemotherapy
- have chronic alcoholism
- have had rapid and massive weight loss after previously being obese.⁶

Understanding starvation

To understand what happens during RFS, first review the pathophysiology of malnutrition. Normally, glucose is the body's preferred fuel, coming from the intake of carbohydrates. As the malnourished body loses access to carbohydrates, it shifts to catabolism of fat and protein. With this shift, the body's production of insulin drops in response to a reduced availability of glucose. This adaptive change to protein breakdown during prolonged malnutrition also leads to a gradual loss of cellular and muscle mass, often resulting in atrophy of vital organs and other internal structures, including the heart, lungs, liver, and intestines.

Serious complications may occur as respiratory and cardiac function declines due to muscular wasting and fluid and electrolyte imbalances. Metabolic rate, cardiac output, hemoglobin level, and renal concentration capacity also decrease.⁷ The body is now surviving by very slowly consuming itself.

As you think of these changes, you probably envision a thin, emaciated patient, but even an overweight or obese patient can be malnourished.

What goes wrong in RFS

When a malnourished patient is given aggressive nutritional support, such as PN, a number of events ensue. These are primarily driven by the change in insulin secretion as a result of the shift from protein metabolism to carbohydrate metabolism. The increase in glucose levels, which

results from the composition of the nutritional support formula, increases insulin release by the pancreas. This in turn promotes cellular uptake of glucose along with electrolytes, primarily phosphorus, magnesium, and potassium. The result can be a life-threatening depletion of these vital electrolytes.^{1,8} (See *How electrolyte levels change in RFS*.)

First, let's look at some normal functions of phosphate. It's needed to produce adenosine triphosphate (ATP), which provides energy for almost all cellular functions. Phosphate is an essential part of RNA and DNA, and it's needed in red blood cells for 2,3-diphosphoglycerate production for easier release of oxygen to the tissues. Patients with signs and symptoms of hypophosphatemia or phosphate levels below 2 mg/dL require oral or intravenous (I.V.) phosphate replacement.⁹

Refeeding-induced severe hypophosphatemia (serum concentrations less than 1 mg/dL) can result in respiratory failure from a decrease in available ATP, which is needed to maintain the diaphragm's normal contractility. In addition, hypophosphatemia can also cause red and white blood cell dysfunction, muscle weakness, and seizures. Other factors that can contribute to hypophosphatemia include vitamin D deficiency and excessive intake of antacids, which block phosphate absorption.¹⁰

Hypokalemia (serum levels below 3.5 mEq/L) and hypomagnesemia (serum levels below 1.8 mg/dL) are also frequently associated with RFS. Mild decreases of potassium and magnesium may cause nausea, vom-

Who's at risk for malnutrition?

Assess and closely monitor patients with these conditions:

- chronic alcoholism
- anorexia nervosa
- cancer, especially when on chemotherapy
- massive weight loss after being morbidly obese
- chronic gastrointestinal disease, such as Crohn's disease
- prolonged I.V. hydration with nonnutritional fluids
- stroke
- protein calorie malnutrition
- prolonged fasting, defined as no oral intake for more than 7 days
- loss of 10% of body weight or more within the preceding 4 weeks
- decreased intake for any reason after surgery.

Sources: McCray¹; Crook.³

iting, constipation, diarrhea, muscle twitching, or weakness. A more severe depletion of the serum concentrations of potassium and magnesium can cause dysrhythmias, cardiac dysfunction, skeletal muscle weakness, seizures, and metabolic acidosis.¹⁰

Collaboration is key

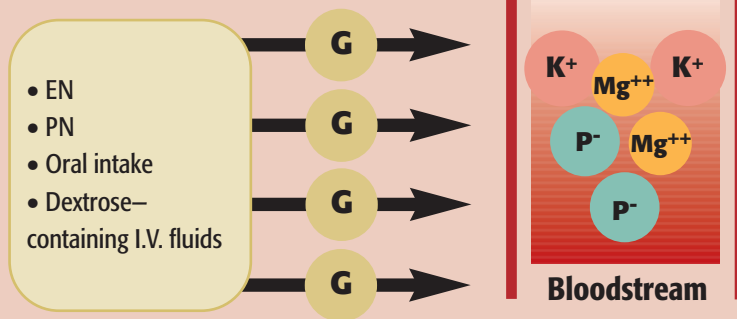
To prevent RFS or identify early signs and symptoms, you'll need to communicate and coordinate often with health care providers, dietitians, pharmacists, and other nurses. (See *Watch for these signs and symptoms of RFS*.) Your role centers on identifying patients who are at risk and recognizing signs and symptoms early on. You may be the first person to take a patient's history and perform a physical assessment, so you may be the first to learn about her preadmission weight loss or problems that prevent adequate nutritional intake, such as nausea, vomiting, and dysphagia. Once you identify risk factors for RFS, alert her health care provider and a registered dietitian.

How electrolyte levels change in RFS

Here's what happens once a malnourished patient begins aggressive nutritional support:

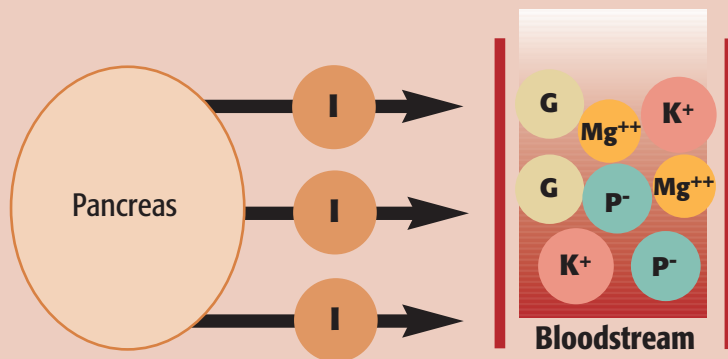
Step 1

Glucose (G) enters bloodstream by enteral, parenteral, oral, or I.V. route.



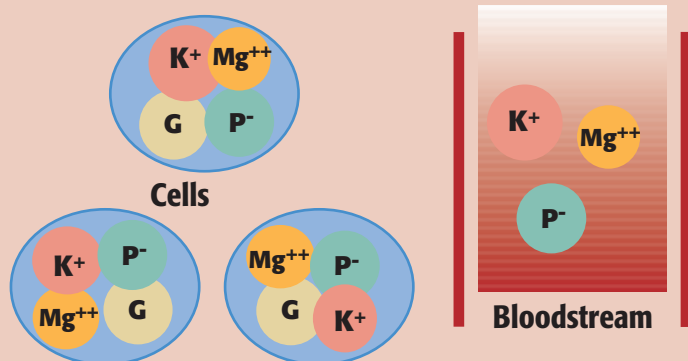
Step 2

Pancreas secretes insulin (I) in response to increase in serum glucose.



Step 3

Insulin promotes cellular uptake of glucose, potassium (K^+), magnesium (Mg^{++}), and phosphorus (P^-). Low serum electrolyte levels for potassium, phosphorous, and magnesium result.



Enter the dietitian

The dietitian can identify the level of malnutrition (mild, moderate, or severe) based on the patient's rate of weight loss and her current body weight, and by evaluating the patient's visceral protein status based on levels

of serum albumin, prealbumin, and transferrin or her total lymphocyte count. A dietitian will help determine the appropriate method of nutrition support (parenteral, enteral, or oral) and request additional lab tests.

Tailoring nutrition support to the

patient's condition requires the calculation of her basic energy requirement, or basal energy expenditure—the amount of energy required to maintain the body's normal metabolic activity at rest. The dietitian is well educated in evaluating a patient's

caloric need and can provide guidance on how and when to increase the nutritional load of the nutrition support formula.

Your role in patient care

If your patient is at risk for RFS, follow these guidelines to prevent or minimize problems.

- *Monitor serum electrolyte levels and make sure they're normal before beginning nutritional support.* Watch particularly for abnormal levels of potassium, phosphate, and magnesium. If they aren't normal, they should be corrected promptly. These levels also need to be checked every 6 hours, 12 hours, or daily for at least 3 days after nutrition is initiated.¹

- *Carefully assess blood pressure, pulse rate, and intake and output.* Your patient may be dehydrated and hypovolemic before she begins therapy. Carefully restore volume and sodium if necessary. If your patient is severely malnourished, her fluid intake may need to be restricted. She should also avoid excessive sodium intake. She may have an altered cardiac rate or rhythm at the start of therapy, and changes in pulse rates can indicate a change in her clinical status. Tell her health care provider about any such clinical changes.

- *Closely monitor the refeeding rate.* When resuming nutrition, start slowly (especially when PN is used), at a rate that delivers 15 to 20 kcal/kg/day (about 1,000 kcal/day for adults) for the first 1 to 3 days, before gradually advancing to the desired daily levels over 5 to 7 days. The health care provider will advance nutritional support when the serum electrolytes are close to the normal range or are actively being replaced.

- *Provide patient education.* If a patient is showing signs of RFS after oral food intake, you should help slow down the reintroduction of carbohydrates by encouraging her to eat

Watch for these signs and symptoms of RFS

Electrolyte imbalance <ul style="list-style-type: none">• hypokalemia• hypophosphatemia• hypomagnesemia	Gastrointestinal <ul style="list-style-type: none">• anorexia• abdominal pain• constipation• diarrhea
Neurologic <ul style="list-style-type: none">• weakness• seizure• paresthesia• altered mental status• paralysis• tetany	Cardiovascular <ul style="list-style-type: none">• dysrhythmias• hypotension
Musculoskeletal <ul style="list-style-type: none">• weakness• myalgia	Renal <ul style="list-style-type: none">• edema• elevated blood urea nitrogen• elevated creatinine
Hematologic <ul style="list-style-type: none">• thrombocytopenia• platelet dysfunction• anemia• infections• bleeding	Metabolic <ul style="list-style-type: none">• metabolic alkalosis• metabolic acidosis
Respiratory <ul style="list-style-type: none">• hypoxia	Nutritional <ul style="list-style-type: none">• vitamin deficiency, such as deficiency of vitamin B₁ (thiamine)• low albumin and prealbumin levels

Source: Crook.³

small meals. Tell her to choose supplements or snacks that are low in sugar (for example, sugar-free juices or sugar-free puddings or gelatins) and fluids that don't contain carbohydrates. Eating food that's high in potassium can help reverse potassium repletion, but at a much slower rate than when potassium is given I.V. High-potassium foods include melons, milk, bananas, apricots, oranges, and potatoes. Giving carbohydrate-rich snacks along with a high-protein source (for example, crackers with cheese or peanut butter) can help slow the rate of digestion and may slow the rate of insulin secretion.

- *Monitor phosphorus, magnesium, and potassium levels daily for the first week or until levels become stable.* Life-threatening changes in these electrolytes commonly occur in the first 1 to 3 days of therapy. Notify the primary care provider if abnormalities develop in any of these val-

ues. You should continue closely observing your patient during correction of any of these electrolyte abnormalities. For example, while phosphorus is being administered, monitor your patient for possible adverse reactions such as tetany, hypotension, hyperkalemia, and hypernatremia. Be aware of medications that may influence the development of hypophosphatemia, hypokalemia, or hypomagnesemia. Consult with the pharmacist if necessary to identify additional medications that may contribute to electrolyte imbalances.

- *Assess vitamin levels.* Malnourished patients, especially those with alcoholism, are likely to have deficiencies of vitamins, including thiamine. Thiamine deficiency can result in confusion, ataxia, and coma. As a general rule, any malnourished patient at risk for RFS should receive thiamine supplementation.

- *Meticulously document fluid intake and output and weight.* During refeeding, a malnourished patient can rapidly develop fluid overload, so be sure to monitor her for abnormal lung sounds, which can be the first sign of fluid overload. Also monitor her intake and output and weigh her daily. Assess for edema of her lower extremities, which may be an early sign of fluid overload. Weight gain of more than 0.5 pound (0.23 kg) a day or 3.3 pounds (1.5 kg) a week probably represents fluid retention. If excessive weight gain occurs, notify the health care provider.

- *Monitor blood glucose levels frequently.* Your patient can rapidly become hyperglycemic when she's receiving glucose replacement. Generally, during the initial refeeding stage, glucose levels should be kept at or below 200 mg/dL. Closely monitor your patient for additional sources of glucose she's receiving; for example, dextrose in I.V. fluids

or drugs that can affect serum glucose such as corticosteroids and beta-blockers.

- *Monitor for neurologic signs and symptoms.* Your patient with RFS may develop muscle weakness, tremors, paresthesias, and seizures. Institute seizure precautions. In addition, she may have cognitive changes, including irritability and confusion. These cognitive changes make her safety a priority. Institute fall risk procedures.

Remain vigilant

Although not all patients who receive nutritional support develop RFS, you must be aware of this condition and identify and closely monitor those patients at risk. Your vigilance can help protect patients from the potentially devastating consequences of RFS. ♦

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How to recognize and respond to refeeding syndrome

GENERAL PURPOSE To provide nurses with an overview of RFS. **LEARNING OBJECTIVES** After reading the preceding article and taking this test, you should be able to: **1.** Describe the pathophysiologic process involved in RFS. **2.** Recognize the signs and symptoms of RFS. **3.** Discuss implications for nursing interventions in RFS.

1. RFS is *most often* associated with

- a. parenteral nutrition.
- b. dextrose-containing I.V. fluids.
- c. enteral nutrition.
- d. oral intake.

2. Electrolyte disturbances with rapid initiation of refeeding usually include

- a. increased phosphorus.
- b. decreased potassium.
- c. increased magnesium.
- d. decreased calcium.

3. After rapid initiation of refeeding, cardiac complications can develop as early as

- a. 0 to 4 hours.
- b. 6 to 12 hours.
- c. 24 to 48 hours.
- d. 3 to 5 days.

4. Which statement about RFS is *not* correct?

- a. Most signs and symptoms are due to electrolyte imbalances.
- b. It was first recognized in starvation victims of World War II.
- c. RFS occurs primarily in hospitalized patients.
- d. RFS occurs only in people with dramatic recent weight loss.

5. At particular risk for malnutrition is a patient who

- a. has lost at least 5% of his weight within the preceding 4 weeks.
- b. hasn't eaten for 5 days.
- c. has chronic alcoholism.
- d. has experienced moderate weight loss after previous obesity.

6. The body's preferred fuel is

- a. carbohydrates.
- b. glucose.
- c. fat.
- d. protein.

7. During malnutrition, all of the following occur during fat and protein catabolism *except*

- a. cardiac hypertrophy.
- b. decreased insulin production.
- c. gradual muscle mass wasting.
- d. cellular atrophy.

8. Which statement is correct about malnourished patients?

- a. The body survives by rapidly consuming itself.
- b. Children aren't at risk for complications of refeeding.
- c. Patients are always thin and emaciated.
- d. Patients can be overweight or obese.

9. The driving force behind the electrolyte shift during aggressive nutritional support is the

- a. increase in insulin secretion.
- b. shift from carbohydrate to protein metabolism.
- c. rapid fall in serum glucose.
- d. correction of dehydration.

10. Respiratory failure during refeeding is generally caused by severe

- a. hypokalemia.
- b. hypomagnesemia.
- c. hypophosphatemia.
- d. hyperinsulinemia.

11. Which blood test is *not* used to evaluate the patient's visceral protein status?

- a. albumin
- b. hemoglobin
- c. transferrin
- d. total lymphocyte count

12. Which statement is correct about fluid balance while refeeding a severely malnourished patient?

- a. Fluid intake may need to be restricted.
- b. A high sodium intake will help to achieve fluid balance.
- c. Weight gain of 2.2 to 3.3 lbs/day is desirable.
- d. Dehydrated patients need rapid restoration of fluid.

13. The recommended rate for initial refeeding of an adult is about

- a. 5 to 10 kcal/kg/day.
- b. 15 to 20 kcal/kg/day.
- c. 25 to 30 kcal/kg/day.
- d. 35 to 40 kcal/kg/day.

14. During phosphorus administration, monitor the patient for

- a. tetany.
- b. hypertension.
- c. hypokalemia.
- d. hyponatremia.

15. As a general rule, malnourished patients at risk for RFS should receive

- a. beta-blockers.
- b. calcium channel blockers.
- c. thiamine supplements.
- d. diuretics.

16. Which statement is correct about the neurologic complications of RFS?

- a. They're generally seen before any cardiac complications.
- b. Myalgia and rhabdomyolysis are common neurologic complications.
- c. Seizure precautions aren't needed.
- d. Patients should be placed on fall risk precautions.

17. Hematologic signs and symptoms of RFS include all of the following *except*

- a. thrombocytopenia.
- b. anemia.
- c. platelet dysfunction.
- d. thrombocytosis.

18. How does insulin secretion affect electrolytes?

- a. It causes increased serum potassium.
- b. It causes decreased serum magnesium.
- c. It blocks cellular uptake of phosphorus.
- d. It blocks cellular uptake of potassium.



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