Alterations in Fluids and Electrolytes During Refeeding Syndrome

Barbara Magnuson, PharmD, BCNSP

Objectives

• Define refeeding syndrome including its signs and symptoms
• Identify fluid and electrolyte disturbances in refeeding syndrome
• Review risk factors for the development of refeeding syndrome
• Describe the infusion nurse’s role in delivering care to a patient with refeeding syndrome

Refeeding Syndrome (RS)

• Occurs when enteral or parenteral nutrition is reinitiated to a starved or severely malnourished person
• May present with neurologic, pulmonary, cardiac, neuromuscular, and/or hematologic complications
• Ranges in severity, but may result in death
• Manifests as multiple metabolic alterations and significant fluid and electrolyte disturbances
  – Phosphorus (PO₄³⁻)
  – Potassium (K⁺)
  – Magnesium (Mg²⁺)
Pathophysiology of Starvation

• **First 24-72 hours:**
  - Liver uses glycogen stores for energy
  - Skeletal muscles release amino acids for gluconeogenesis
  - Glucose required for brain, renal medulla, and red blood cells

• **Greater than 72 hours:**
  - Metabolic pathways shift to free fatty acid oxidation in attempt to spare protein catabolism from skeletal muscle
  - Ketone bodies produced as a result
    - Acetyl-acetic acid
    - Beta-hydroxybutyric acid
    - Acetone


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Pathophysiology of Starvation

• **Adaptive mechanisms occur after prolonged starvation:**
  - Decreased liver gluconeogenesis
  - Decreased basal metabolic rate
  - Increased use of free fatty acids by the brain
  - Hormonal changes
    - Decreased insulin secretion
    - Increased growth hormone secretion
    - Increased cortisol secretion

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Prolonged Starvation

• Weight loss
• Decreased overall total cell mass
• Electrolyte abnormalities
  - Decreased serum phosphorus
  - Depleted potassium and magnesium stores
• Increased extracellular water
  - Secondary to decreased oncotic pressures resulting from decreased albumin
• Cardiac Effects
  - Reduced cardiac mass and output
  - Reduced total cardiac volume, end diastolic volume and left ventricular mass with severe malnutrition

Knochel J P. Arch Intern Med 1977;137:203-220
O’Connor L, et al. SEM 1977:297:901-904

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Refeeding Physiology

- Occurs when enteral or parenteral nutrition is provided after a period of starvation

- Sudden shift back to glucose as a primary fuel
  - Increased demand for phosphorylated intermediates of glycolysis (ATP)
  - Insulin levels increase to force glucose into cells for utilization

Refeeding Physiology

- Effects of increased insulin release
  - Potassium, magnesium and phosphorus rapidly enters the cells
    - Exacerbates already low electrolyte levels
  - Extracellular water compartment expands
    - Sodium and water retention may be due to anti-diuretic effect from the elevated insulin levels
  - Ventricular volume returns to normal while left ventricular mass remains reduced
    - Leads to fluid retention and congestive heart failure


Refeeding Physiology

- Thiamine is an essential co-factor for carbohydrate metabolism
  - Possible deficiency during starvation may be worsened during refeeding
- Thiamine deficiency may lead to
  - Wernicke’s encephalopathy/Korsakoff’s syndrome
  - Short-term memory loss
  - Ocular disturbance
  - Confusion
  - Ataxia
  - Coma
Phosphorus

- Normal serum value: 2.5–4.5 mg/dl
  - Major intracellular anion
  - 80% in skeleton, 20% in soft tissue and muscle
  - Average consumption: 1000–1400 mg/day
  - Absorption: jejunum
  - Elimination: 90% renal, 10% by the GI tract
- Degree of hypophosphatemia may be associated with symptoms
  - Moderate: 1.5–2.2 mg/dl
  - Severe: <1.5 mg/dl

Hypophosphatemia

- Hypophosphatemia and associated abnormalities were described in an observational series of 19 trauma patients
- 8 patients inadvertently were given PN without phosphate supplementation
- Results:
  - Patients who developed hypophosphatemia also found to have decreased levels of ATP and 2,3-DPG
  - Significant correlation between total calories administered and fall in serum phosphorus concentration
  - Significant correlation between the amount of phosphate administered and the increase in serum phosphorus concentration


Hypophosphatemia

Physiologic Manifestations:

- Neurologic
  - Paresthesias, weakness, confusion, disorientation, encephalopathy, seizures, coma, and death
- Respiratory
  - Acute failure due to impaired diaphragm contractility
- Immune function
  - Impaired leukocyte chemotaxis and phagocytosis


Hypophosphatemia

Physiologic Manifestations:

- Cardiac
  - **Contractile alterations** due to atrophy and ATP depletion
  - Hypotension
  - **Decreased stroke volume** & ventricular stroke work
  - Decreased mean arterial pressure
  - Increased pulmonary artery wedge pressure
  - Volume overload
    - From aggressive fluid replacement along with Na and water retention
  - **Arrhythmias**

Potassium

- Normal serum value: 3.5–5.0 mEq/L
  - Major intracellular ion: 98% intracellular
  - Average consumption: 4700 mg/day
  - Absorption: Small intestine
  - Elimination: Renal elimination regulated at the distal tubule
    - Complex regulation:
      - Aldosterone, Alkalosis
      - High potassium diet
      - Increased sodium delivery to the distal tubule


Potassium

- Regulates electrical cellular membrane potential, cellular metabolism, glycogen, and protein synthesis

Hypokalemia

- Degree of hypokalemia associated with symptoms
  - Mild to moderate (2.5–3.5 mEq/L)
    - Nausea & vomiting
    - Constipation
    - Weakness
  - Severe (<2.5 mEq/L)
    - Paralysis
    - Respiratory compromise
    - Rhabdomyolysis & muscle necrosis
    - EKG changes & cardiac arrhythmias

**Magnesium**

- Normal serum value: 1.8–2.5 mg/dl
- Major intracellular ion: 2nd most abundant intracellular cation
  - 99% in the bone
  - Serum levels do not properly reflect body's total Mg++ stores or intracellular stores
- Absorption: Only 30% of oral Mg++ is absorbed
  - Oral magnesium creates a laxative effect further complicating absorption in the presence of short bowel syndrome or malabsorption diseases
- Elimination: renal

**Cofactor for many biochemical reactions and enzymes**

**Participates in oxidative phosphorylation**

![Diagram](http://www.mgwater.com/ft/schroll/schrfig1.gif)

**Hypomagnesemia**

- Degree of hypomagnesemia may be associated with symptoms
  - Symptoms resemble hypokalemia and hypophosphatemia
- Mild to moderate (<1.5 mg/dl)
  - Weakness, muscle twitching/tremor, altered mental status, anorexia, nausea, vomiting, diarrhea
- Severe (<1.0 mg/dl)
  - EKG changes
  - Cardiac arrhythmias: Vtach/Torsades de Pointes
  - Tetany: intermittent muscle spasms
  - Convulsions/seizures
  - Coma/death

Hypomagnesemia-Induced Torsades de Pointes

Thiamine (Vitamin B₁)

Deficiency

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Sodium and Fluid Alterations

- Occurs in the early phase of RS
- Sodium retention
- Extracellular expansion results in fluid overload
  - Pulmonary edema/respiratory failure
  - Cardiac decompensation/failure

RS

<table>
<thead>
<tr>
<th>PO4</th>
<th>Mg</th>
<th>Vit B1</th>
</tr>
</thead>
<tbody>
<tr>
<td>K</td>
<td>Fluid</td>
<td></td>
</tr>
</tbody>
</table>

Cardiac dysfunction
- Arrhythmias
- Congestive Heart Failure
- Hypotension
- Death

Respiratory failure
- Decrease in ATP
- Decrease in 2,3 DPG
- Diaphragmatic Depression
- Mechanical Ventilation
- Death

Neurologic deficits
- Lethargy
- Confusion
- Coma
- Death

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CPS Observational Study

- Prospective study: 1944–1946
- 36 male civilian public service (CPS) volunteer participants
- Evaluated physiologic effects of 6 months of semi-starvation
- Results:
  - Decreased cardiac reserve
  - Cardiac failure
- Renewed interest → current focus on early aggressive nutrition strategies in the critically ill patient population

World War II

- Significant complications observed when victims of WWII were restored to normal food and liquid intake after prolonged periods of malnutrition/starvation
  - Lenengrad: peripheral edema, hypertension and cardiac insufficiency increased markedly
  - Japan: Peripheral edema
  - Netherlands: Neurological complications, including coma and convulsions

Parenteral Nutrition Fatalities

- 2 chronically malnourished women (both <IBW) started on TPN
  - 40% IBW and 70% IBW
  - Low potassium and phosphorus levels before TPN initiated
  - TPN dosed @ 75kcal/kg dextrose & 3.5g/kg protein
  - Electrolyte disturbances were consistent in both patients
    - Severe hypophosphatemia
    - Severe hypokalemia
    - Severe hypomagnesemia
  - Within 48 hours, both patients required mechanical ventilation
  - Cardiac abnormalities developed and both patients died

References:

Keys et al. MINNESOTA STUDY OF STARVATION, 1950.
**Parenteral and Enteral Nutrition Cohort Study**

- 148 patients with mild to severe malnutrition that received parenteral or enteral nutrition support for more than 7 days
- Incidence of RS:
  - 48% had symptoms consistent with RS
  - Hypomagnesemia, hypokalemia, hypophosphatemia
  - 55% of RS appeared at day 3 of nutrition support
- Patient Outcomes:
  - RS associated with 10 day longer hospital stay
  - 15 patients with RS died


**RS Case Report Review**

- Included 27 cases reported by 20 authors
- Ages of cases ranged from 10–90 yrs
  - Many were adolescents with anorexia nervosa
- Most pts received enteral nutrition
  - Six received TPN
- RS symptoms occurred within =first 5 days of feeding


**RS Case Report Review**

- Results
  - Of the patients with low BMI or poor PO for greater than 48 hours prior to refeeding:
    - 96% experienced hypophosphatemia
    - 51% experienced hypomagnesemia
    - 46% experienced hypokalemia
    - 46% had BOTH hypophosphatemia and hypomagnesemia
    - 42% patients had 3 or more abnormal labs

RS in the Critically Ill

- 62 med/surgical ICU pts refed with TPN or EN after least 48hrs NPO
- Patients were NOT previously malnourished
- Evaluated incidence of refeeding hypophosphatemia
  - Drop in serum phosphorus by at least 0.5 mg/dL to less than 2 mg/dL
- Results:
  - 34% pts exhibited refeeding hypophosphatemia
  - 10% pts developed a phosphorus < 1 mg/dL
  - Phosphorus nadir occurred after @ 2 days of feeding
  - Patients with hypophosphatemia had significantly prolonged lengths of mechanical ventilation and hospital stay
- A low prealbumin was the only risk factor identified that predicted the development of refeeding hypophosphatemia


RS Risk Factors

- Obvious protein & calorie malnutrition
  - Edema
  - Cachexia
- Less than 85% IBW
- Anorexia nervosa
- Chewing or swallowing difficulties
  - Patients after stroke
- Residents admitted from skilled nursing facilities
- History of excessive alcohol intake

RS Risk Factors

Chronic diseases causing under-nutrition
- Cancer
- COPD
- Cardiac cachexia
- Cirrhosis
- Poor PO intake
  - Head /Neck tumor/radiation
  - Esophageal tumors/surgery
  - GI fistulas
  - High output ileostomy drainage
RS Risk Factors

- Morbid obesity with rapid or massive weight loss
- Major surgery with previous prolonged NPO status
- ICU patients unable to be fed for 2–5 days
  - The more hypermetabolic, the sooner RS develops
  - Multiple-trauma patient, traumatic brain injury, burns
  - Maintenance IV fluids contain potassium but not phosphorus
- Children with failure to thrive, prolonged vomiting, and malabsorptive diseases

RS Management

- Prevent refeeding syndrome
  - Identify patients at risk
  - Escalate nutrition slowly
    - Administer 10% dextrose @ 40ml/hr for the first day
    - Continue 10% dextrose if hypoglycemia occurs
  - Assess electrolyte levels and aggressively correct all electrolyte disorders
  - Consult GI or Nutrition Support Service
  - Provide supportive care as needed

Prevention of RS

- Avoid overfeeding
  - The patient did become malnourished overnight and you can NOT correct it overnight!
- “START LOW & GO SLOW”
  - Only 25% of the calories on day one
  - Advance slowly over 3–5 days toward caloric goal
  - If severely malnourished, consider advancing over 5–7 days
- Assess and correct baseline electrolytes BEFORE initiating nutrition support
Prevention of RS

- Initially provide ONLY the minimal needs to prevent overfeeding.
- Carbohydrates
  - Provide minimum requirement
    - Ex: 100–150 gm/day for a 70 kg male
  - Carbohydrate administration will:
    - Suppress gluconeogenesis
    - Spare protein catabolism
    - Supply energy to CNS


Prevention of RS

- Protein
  - Requirements:
    - 1.5 gm/kg/day or 2.0 gm/kg/day if severely underweight
  - Patients with increased protein requirements:
    - Trauma, Head injury, or burns
    - Continuous renal replacement therapy
    - Hepatic dysfunction & Cirrhosis
    - Decubitus ulcers/skin breakdown
  - Patients with decreased protein requirements:
    - Renal Failure with uremia
  - Multivitamin, & Trace elements - supplemented daily
  - Thiamine - Use higher dose initially 50–100 mg/day x 3 days

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ASPEN Guidelines

- Identify patients at risk of RS
- Correct electrolyte abnormalities prior to the initiation of nutrition support.
- Initiate nutrition support at 25% of the estimated goal and advanced over 3–5 days to the goal rate.
- Monitor serum electrolytes and vital signs carefully after nutrition support is started

**Enteral Phosphorus Replacement**

- Mild/asymptomatic (1.5–2.2 mg/dl)
  - Enteral supplementation is reasonable

<table>
<thead>
<tr>
<th>Product</th>
<th>P mmol</th>
<th>Na⁺ mEq</th>
<th>K⁺ mEq</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>D/C Neutra-Phos® (capsule/pkt)</strong></td>
<td>8 (250mg)</td>
<td>7.1 (160mg)</td>
<td>7.1 (280mg)</td>
</tr>
<tr>
<td><strong>D/C Neutra-Phos K® (capsule/pkt)</strong></td>
<td>8</td>
<td>0</td>
<td>14.25</td>
</tr>
<tr>
<td>Skim milk per 8 oz (1 cup)</td>
<td>8</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>K-Phos® Neutral (tablet)</td>
<td>8</td>
<td>13</td>
<td>1.1</td>
</tr>
<tr>
<td>Fleet® Phospho Soda (soln)/ml</td>
<td>4.15</td>
<td>4.82</td>
<td>0</td>
</tr>
</tbody>
</table>

1 mmol = 93 mg phosphorus (MW = 31)

- Special Considerations:
  - Note the potassium and sodium content when selecting a product
  - Phosphorus powder packets can be given by mouth or feeding tube
  - Can cause osmotic laxative effect in critically ill patient which may worsen hypophosphatemia

- **MEDICATION ERROR ALERT:**
  - PhosLo® (calcium acetate) – is a phosphorus BINDER used to DECREASE phosphorus
    - NOT a phosphorus supplement

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**Parenteral Phosphorus Replacement**

- Severe/symptomatic (<1.5 mg/dl)
  - Parenteral replacement is most effective

<table>
<thead>
<tr>
<th>Product</th>
<th>P mmol</th>
<th>Na⁺ mEq</th>
<th>K⁺ mEq</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium Phosphate (ml)</td>
<td>3</td>
<td>0</td>
<td>4.4</td>
</tr>
<tr>
<td>Sodium Phosphate (ml)</td>
<td>3</td>
<td>4.0</td>
<td>0</td>
</tr>
</tbody>
</table>

3 mmol = 93 mg phosphorus (MW = 31)
**Parenteral Phosphorus Replacement**

- **UK HealthCare: Phosphorus Sliding Scale**

<table>
<thead>
<tr>
<th>Phosphorus</th>
<th>IV Replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild: 2.7–1.9 mg/dl</td>
<td>9 mmol over 2 hours *</td>
</tr>
<tr>
<td>Moderate: 1.8–1 mg/dl</td>
<td>18 mmol over 3 hours *</td>
</tr>
<tr>
<td>Severe: &lt;1 mg/dl</td>
<td>27 mmol over 4 hours *</td>
</tr>
</tbody>
</table>

  * Time of administration is for ICU only

- Slower infusion time required for non-ICU patient monitoring areas
- Central IV access preferred


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**Parenteral Phosphorus Replacement**

- Weight-based replacement has been evaluated:

<table>
<thead>
<tr>
<th>Phosphorus</th>
<th>Replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild: 2.5–3 mg/dl</td>
<td>0.32 mmol/kg *</td>
</tr>
<tr>
<td>Moderate: 1.6–2.2 mg/dl</td>
<td>0.64 mmol/kg *</td>
</tr>
<tr>
<td>Severe: 1.5 mg/dl</td>
<td>1 mmol/kg *</td>
</tr>
</tbody>
</table>

  * Actual BW if ≤130% of IBW, Adjusted body wt if >130% IBW
  
  \[
  \text{Adj BW} = \frac{\text{IBW} + 0.25(\text{actual body weight} - \text{IBW})}{1.30}
  \]

- Results:
  - Improved serum phosphorus levels by day 2
  - Mean serum phosphorus concentrations were within normal limits in all groups by day 3


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**Parenteral Phosphorus Replacement**

- Special Considerations:
  - Note the potassium and sodium content when selecting a product
  - Rates of administration depends on ability to monitor patient and must account for potassium concentrations
  - Check IV compatibilities prior to administration
  - May combine enteral and parenteral supplements to rapidly increase phosphorus levels
  - Caution for infusing with calcium products or same IV line
  - Both IV potassium and sodium phosphate have been on manufacturer shortage since 2012

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Phosphorus Monitoring

- Recheck phosphorus 2–4 hours after dose
- Continue to supplement
  - Until asymptomatic
  - Phosphorus is within normal limits
- Check phosphorus daily for first week of nutrition support if patient is at risk for refeeding syndrome

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Treatment: Hypokalemia

- Mild/asymptomatic (2.5–3.5 mEq/L)
  - Enteral supplementation preferred
  - Safe and well absorbed
  - Very GI/stomach irritating
    - May cause severe cramping
    - Take with food and 4–6 ounces of water or juice
  - Can cause osmotic laxative effect in critically ill patient which may worsen hypophosphatemia
- Severe/symptomatic (<2.5 mEq/L)
  - Parenteral replacement is preferred

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<table>
<thead>
<tr>
<th>Potassium</th>
<th>Replacement (PO or IV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild: 4.0–3.7 mEq/L</td>
<td>40 mEq</td>
</tr>
<tr>
<td>Moderate: 3.6–3.4 mEq/L</td>
<td>60 mEq *</td>
</tr>
<tr>
<td>Severe: &lt;3.3 mEq/L</td>
<td>80 mEq *</td>
</tr>
</tbody>
</table>

* If given as PO doses, give in 3 divided doses

- For severe hypokalemia, give both IV and PO
- Central IV access preferred
- Special considerations required for renal dysfunction:
  - Typically give 50% of recommended replacement
  - CRRT: May need higher than normal replacement and more frequent monitoring

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**Treatment: Hypokalemia**

- IV Infusion rate:
  - Peripheral line: 10 mEq/hr
  - Central line: 10-40 mEq/hr
- IV Maximum Concentration
  - Peripheral line: 40 mEq/L–80 mEq/L
  - Central line: 80 mEq/L–120 mEq/L
  - 40 mEq/L at UK HealthCare
  - 80 mEq/L at UK HealthCare
  - TPN: 80mEq/L at UK HealthCare

**MEDICATION ERROR ALERT:** In adult patients, Do not give potassium as IV push

**Potassium Monitoring**

- Careful monitoring required, especially during IV replacement
  - IV site monitoring for possible extravasation
  - ECG may be required especially if potassium is less than 2.5 mEq/L
- Recheck potassium every 1–4 hrs after dose, continue to supplement until asymptomatic or potassium is within normal limits
- Check potassium daily for first week of nutrition support if patient is at risk for RS

**Treatment: Hypomagnesemia**

- Parenteral route **preferred**
  - Magnesium sulfate salt
- Enteral magnesium oxide is available
  - Slow onset and poor absorption
  - Laxative effect that may exacerbate hypomagnesemia
- Renal elimination is FAST
  - ~50% of magnesium dose is excreted
- Rapid administration rates may simply increase urinary excretion of magnesium
**Treatment: Hypomagnesemia**

- Dilute 1 gram of Mg sulfate in at least 10 ml NS or D5W
- Non-emergent hypomagnesemia:
  - Administer 6 grams of magnesium sulfate over 6–12 hours
  - Administer higher doses of magnesium over 12–24 hours
    - Maximum administration rate: 1 gram over 1 hour
  - Total repletion may take several days
- Severe symptomatic hypomagnesemia:
  - Aggressive dosing may be required in the acute care setting
  - 1 gram over 30 min, preferred
  - Maximum: 10 minutes (non-ICU); 7 minutes (ICU)

  Iannello S, Belfiore F. Panminerva Med. 2001 Sep;43(3):177-209

**Magnesium Monitoring**

- Recheck magnesium every 12–24 hours after dose
- Magnesium has a slow equilibration time between serum, intracellular space, and tissues
- Check magnesium daily for first week of nutrition support if patient is at risk for RS

  Rio A. et al BMJ Open. 2013 Jan 11;3(1)

**Special Considerations of RS in Children**
Prevention of RS in Children

Prior to initiation of/during nutrition support:
- Evaluate hydration and nutritional state
- Obtain serum electrolytes, prealbumin and albumin levels, and weight
- Monitor pulse, ECG with or without echocardiogram
- Note that early weight gain may be secondary to fluid retention (1)
- Severely malnourished patients with celiac disease are at risk of developing potentially life-threatening refeeding syndrome (2)


Prevention of RS in Children

- Oral feeding regimen: Provide 75% of total daily needs initially
  - 0-7 years old: 80–100 kcal/kg/day
  - 7–10 years: 75 kcal/kg/day
  - 11–14 years: 60 kcal/kg/day
  - 15–18 years: 50 kcal/kg/day
- If the initial food challenge is tolerated, this may be increased over 3–5 days
- Each requirement should be tailored to an individual’s need and the above values may need to be adjusted by as much as 30%


Prevention of RS in Children

- Frequent small feeds
- Minimum of 1 kcal/ml to minimize volume overload
- If milk-based feed induces diarrhea, use hydrolysates
- Initial regimen for malnourished children
  - 0.6–1 g/kg/day (in a formula rich in essential amino acids)
  - Gradually increase to 1.2–1.5 g/kg/day

Prevention of RS in Children

Supplements:
- Replace sodium, potassium, and magnesium PRN
- Replace phosphorus:
  - IV: 1 mmol/kg/day
  - PO: 100 mmol/day for children over 5, oral supplements up to 7 years of age
  - Hypocalcaemia may occur during phosphate supplementation
- Vitamins: thiamine, folic acid, riboflavin, ascorbic acid, pyridoxine, fat-soluble vitamins A, D, E and K
- Trace elements: combination including selenium


Potassium Infusion Guidelines: UK Children's Hospital

- Medication Safety Policy: IV bolus of KCl are NOT permitted for general use except:
  - Pediatric ICU: (0.25mEq/kg–0.5mEq/kg)
    - 10 mEq/hour, not to exceed 1 mEq/kg/hr
  - Neonatal ICU: (0.25mEq/kg–0.5mEq/kg)
  - Ped Heme/Onc svc: (0.25mEq/kg) by senior resident or attending physicians ONLY
- Replace low K+ levels:
  - Increasing K+ in MIVF for a limited number of hours
  - Providing oral K+ replacement

Conclusion

- RS is not only a historical phenomenon
- RS is frequently encountered in modern clinical practice and is relatively poorly recognized or understood
- RS is associated with significant morbidity and mortality
- Pathophysiology includes disturbances of glucose, fluid balance, and electrolytes
- Phosphate, potassium, and magnesium drastically decrease during the refeeding phase and should be aggressively monitored and replaced
- Nutrition support therapies, both enteral and parenteral, should be introduced methodically and slowly over several days to minimize complications
The key to treating refeeding syndrome is preventing it!

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