Tori Vogt
Continuing Education Host
The Rosewood Institute

• Original Founders had a dream for the Rosewood Treatment Centers.....

• An Education and Training branch of The Rosewood Centers for Eating Disorders

• The dream unfolded.......
What Does TRI Do?

• Continuing Education training (Internal & External)
  – Approved Provider with: NBCC, NAADAC, CBBS and CDR

• Training professionals on the specifics of eating disorders/addictions and co-morbid complications
  1. West Coast Symposium for Addictive Disorders
  2. Cape Cod Symposium for Addictive Disorders

• Present/participate in eating disorder conferences
  – IAEDP, AED, NEDA, BEDA, BFI
TRI Initiatives and Goals:

• Invite Guest Speakers to share their specialties with our treatment staff

• Monthly Webinars and On-demand trainings

• Co-Sponsor Workshops/events and present locally and nationally with fellow field professionals

• Outreach & Education Projects for K-12 Grades & College
  – Operation RecoverED, Shannon Hershkowitz

• Internship program and Curriculum
  – Dr. Dena Cabrera, Clinical Director
Medical Complications of Anorexia Nervosa: Refeeding Syndrome

Amelia Davis, M.D.
Medical Director
Chief of Adult Psychiatry and Eating Disorders Unit
Rosewood Center for Eating Disorders
• Importance of knowing about refeeding syndrome across disciplines
• Review of DSM-5 diagnostic criteria and overview of anorexia nervosa
• Medical complications of anorexia nervosa
• Description of Refeeding syndrome
• Prevention of refeeding syndrome
• Conclusion
Why talk about refeeding syndrome?

• Important for physicians, clinicians, dietitians, nurses, nursing staff, therapists, intake staff, admissions coordinators, utilization review to:
  – Help educate patients and families on why inpatient hospitalization may be recommended for treatment
  – Help encourage patients to comply with lab draws and improve compliance with treatment
  – Recognize and document signs and symptoms of refeeding syndrome
  – Optimal management requires multidisciplinary team
A. Restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health.

B. Intense fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain, even though at a significantly low weight.

C. Disturbance in the way in which one’s body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.
Subtypes for Anorexia Nervosa (AN)¹

• **Restricting type:** During the last 3 months, the individual has not engaged in recurrent episodes of binge eating or purging behavior (i.e., self-induced vomiting or misuse of laxatives, diuretics, or enemas). This subtype describes presentations in which weight loss is accomplished primarily through dieting, fasting, and/or excessive exercise.

• **Binge-eating/purging type:** During the last 3 months, the individual has engaged in recurrent episodes of binge eating or purging behavior
DSM-5 Criteria for Anorexia Nervosa (AN)\(^1\)

Specify if:

• **In partial remission:** After full criteria for anorexia were previously met, Criterion A (low body weight) has not been met for a sustained period, but either Criterion B (intense fear of gaining weight or becoming fat or behavior that interferes with weight gain) or Criterion C (disturbances in self-perception of weight and shape) is still met.

• **In full remission:** After full criteria for anorexia were previously met, none of the criteria have been met for a sustained period of time.
Specify current severity: (based on adults, on current body mass index (BMI) or for children and adolescents, on BMI percentile)

- **Mild:** BMI ≥ 17 kg/m²
- **Moderate:** BMI 16-16.99 kg/m²
- **Severe:** BMI 15-15.99 kg/m²
- **Extreme:** BMI < 15 kg/m²
Epidemiology of AN

• Lifetime prevalence of 0.9% among adult females\textsuperscript{2,3}
• 0.3% among males\textsuperscript{2,3}
• Occurs more commonly in females; 10:1 females to male ratio\textsuperscript{4,5,6}
• Peak age of onset is between 14-18 years\textsuperscript{4,7}
• Highest mortality rate of any mental illness of 5-18%\textsuperscript{4,8}. Problems include cardiac, osteopenia, infertility, stunted growth, kidney damage, leukopenia\textsuperscript{4,9}. 
Common medical complications of AN

- **Cardiovascular**
  - Decreased cardiac muscle mass and shrinking of left ventricle, low cardiac output and hypotension
  - Bradycardia and other dysrhythmias (irregular heart rates)
  - Risk for myocardial infarction and congestive heart failure
  - Pancytopenia including leukopenia, anemia, and thrombocytopenia due to inadequate nutrition

- **Neurologic**
  - Decline in serotonin and norepinephrine
  - Hypothermia related to abnormal temperature regulation and diminished fat tissue

- **GI/hepatic**
  - Decreased gastric emptying; intermittent constipation and diarrhea
  - Elevated hepatic enzymes related to diffuse fatty liver

- **Skin**
  - Lanugo hair/dry and brittle hair and nails

- **Low levels of zinc and magnesium**
- **Hypercholesterolemia** (do not treat with statins)
- **With binge-purging type, can have electrolyte abnormalities as well**
Common medical complications of AN

• Endocrine
  – Amenorrhea - low estrogen, FSH, and LH in females. Prolactin level is generally normal
  – Euthyroid sick syndrome (do not treat with synthroid as often will resolve with weight restoration)
  – Elevated growth hormone and cortisol. Dexamethasone suppression test will be non-suppressing
  – Low testosterone in males
  – Hypoglycemia

• Skeletal
  – Osteoporosis (due to estrogen deficiency and inadequate calcium intake)

• Urinary
  – Decreased or erratic vasopressin secretion, leading to difficulty concentrating urine and leading to appearance of diabetes insipidus
  – Increased BUN and decreased GFR—related to metabolic problems and dehydration
What is Refeeding Syndrome?
What is Refeeding Syndrome?

- First described in WWII, after prisoners of war began eating again after a period of prolonged starvation that seemed to precipitate cardiac failure.\(^{10,11}\)
- The term “refeeding syndrome” was coined and brought to attention in 1981 by Weinsier and Krumdieck when they reported death of two malnourished patients who were fed “overzealously”\(^{12,13}\)
- It can be defined as potentially fatal shifts in fluids and electrolytes that occur in malnourished patients after refeeding.\(^{13}\)
- The hallmark is hypophosphatemia, however, may also feature changes in sodium, fluid balance, changes in glucose, protein and fat metabolism, hypokalemia, hypomagnesemia, and vitamin deficiency (ie B1;thiamine)\(^{13}\)
“I am healthy as all my lab results were normal”

– A 26 year old woman with anorexia nervosa, restricting type for eight years is 5’1” and weighs 65 lbs, or 62% of IBW, BMI 12.3 kg/m2. She continues to run 2 miles daily and states up until recently, she was running marathons. Her family has forced her to accept inpatient treatment. She has no symptoms except early fullness and bloating when she does eat and says all her labs from a month ago were “normal.”
• On admission, vital signs show a temperature of 95.7 F, heart rate of 54, and BP of 95/72. Exam shows a severely emaciated young woman with lanugo hair growth, dry skin, otherwise unremarkable. Her electrolytes are normal.

• Her WBC is 8.6 (3.4-10k/uL). Hematocrit is 43 (37-47%). Platelets are 169 (150-400k/uL). Liver tests, iron, and B12 levels are normal. Vitamin D level is 20 (30-74 ng/pL). She struggles with progressive GI symptoms and has a hard time taking in all her meals. Five days into treatment, blood tests show a low phosphorous of 1.7 (2.5-4.5 mg/dL) as well as mild elevation in liver function tests (AST: 59 and ALT: 104). Her sodium has also decreased from 134 on admission to 130. She also notes that her ankles and feet are painfully swollen. During the first week of treatment, she has gained 7 lbs.
Case Synopsis

• Early signs of Refeeding syndrome
  – Low phosphorus
  – Elevations in liver function tests
  – Swelling in legs
  – Rapid weight gain indicating fluid overload

• Vital sign abnormalities

• Gastroparesis
Potentially deadly syndrome that occurs when a starved person begins to take in nutrition
Changes during Prolonged Fasting (catabolism)

- During periods of prolonged fasting, there is catabolism of adipose tissue and muscle, resulting in loss of lean body mass, and fat, electrolyte, and vitamin depletion\textsuperscript{13, 14}
- While intracellular electrolytes become severely depleted, serum concentrations remain falsely normal during starvation state due partly to loss of total body water\textsuperscript{13, 14}
- Glycogen storage in the liver is depleted as well in nutritional depletion due to decreased gluconeogenesis with prolonged fasting\textsuperscript{13, 14}
Changes with Refeeding Syndrome (switch to anabolism)

• During refeeding after being starved, the body switches from a catabolic state to anabolic state\textsuperscript{13,14}

• Sudden introduction of adequate nutrition is perceived as “stress” by the body

• In response to high glucose, insulin levels are increased and there is increase in fat, glycogen and protein synthesis\textsuperscript{13,14}
Refeeding Syndrome

• Multiple processes occur:
  1. Intracellular shifts of electrolytes, primarily phosphorus, potassium and magnesium. This occurs as increase in glucose causes increase in secretion of insulin, which promotes cellular uptake of the electrolytes.¹⁵
Phosphorus serves many important functions:

a) Important for synthesis of ATP, which is basic biochemical energy storage as well as DNA and protein

b) Phosphorylation of red blood cells, which without impairs delivery of oxygen to tissues and can lead to multiple organ failure

c) Without ATP, leads to muscle breakdown, which leads to weakness (diaphragm weakness) and rhabdomyolysis
Refeeding Syndrome

2. Hypervolemia (Fluid Overload): occurs from sodium retention and effects of hyperglycemia and hyperinsulinemia (increases in glucose and insulin)\(^1^4\)

Fluid overload can appear as peripheral edema (swelling) and rapid weight gain. Fluid can enter the air spaces in the lungs, leading to pulmonary edema and can lead to congestive heart failure (heart failure).
Refeeding Syndrome

3. Vitamin deficiency: rapid depletion of vitamins during refeeding due to role in various biochemical functions. For example, thiamine is necessary for glucose metabolism and depletion can precipitate Wernicke’s encephalopathy and lactic acidosis
Hypokalaemia
Hypomagnesaemia
Hypophosphataemia
Thiamine deficiency
Salt and water retention - edema

↑ Glucose uptake
↑ Utilization of thiamine
↑ Uptake of $K^+$, $Mg^{2+}$ & $PO_4^{2-}$

↑ Protein and glycogen synthesis

Refeeding syndrome
CLINICAL CONSEQUENCES OF SEVERE HYPOPHOSPHATEMIA, HYPOMAGNESEMI A, AND HYPOKALEMIA

Hypophosphatemia

• Neurologic
  – Seizures
  – Weakness
  – Paresthesia
  – Altered higher functions
  – Acute encephalopathy

• Muscular
  – Weakness
  – Myalgia
  – Rhabdomyolysis
  – Decreased cardiac contractility
  – Cardiomyopathy

• Hematologic
  – Dysfunction of platelets and leukocytes
  – Thrombocytopenia
  – Hemolysis
  – Reduction of erythrocyte 2,3-diphosphoglycerate and adenosine triphosphate

• Respiratory
  – Impaired respiratory muscle function sometimes resulting in respiratory failure or ventilator dependency

• Bone
  – Osteomalacia

• Renal
  – Acute tubular necrosis
  – Tubular defects

Hypomagnesemia

• Neurologic
  – Tetany
  – Paresthesiae
  – Seizures
  – Ataxia
  – Tremor
  – Weakness

• Cardiac
  – Arrhythmias, e.g., torsade de pointes
  – Hypertension

• Gastrointestinal
  – Anorexia
  – Abdominal pain

• Electrolyte
  – Hypokalemia
  – Hypocalcemia
Hypokalemia

- Neurologic
  - Paralysis
  - Paresthesia
  - Rhabdomyolysis
- Respiratory depression
  - Weakness
- Cardiac
  - Arrhythmias
  - Hypotension
  - Digoxin toxicity
  - Cardiac arrest
- Gastrointestinal
  - Constipation
  - Paralytic ileus
- Renal
  - Decreased urinary concentrating ability
- Metabolic
  - Metabolic alkalosis
  - Glucose intolerance
Patients at Risk\textsuperscript{14, 19}

- Patients with anorexia nervosa
- Patients with chronic alcoholism
- Oncology patients
- Postoperative patients
- Elderly patients (comorbidities, decreased physiological reserve)
- Patients with uncontrolled diabetes mellitus (electrolyte depletion, diuresis)
- Patients with chronic malnutrition:
  - Marasmus
  - Prolonged fasting or low energy diet
  - Morbid obesity with profound weight loss
  - High stress patient unfed for >7 days
  - Malabsorptive syndrome (such as inflammatory bowel disease, chronic pancreatitis, cystic fibrosis, short bowel syndrome)
- Long term users of antacids (magnesium and aluminium salts bind phosphate)
- Long term users of diuretics (loss of electrolytes)
Patients at Risk

Criteria from the guidelines of the National Institute for Health and Clinical Excellence for identifying patients at high risk of refeeding problems (level D recommendations*):16

Either the patient has one or more of the following:
• Body mass index (kg/m²) <16
• Unintentional weight loss >15% in the past three to six months
• Little or no nutritional intake for >10 days
• Low levels of potassium, phosphate, or magnesium before feeding

Or the patient has two or more of the following:
• Body mass index <18.5
• Unintentional weight loss >10% in the past three to six months
• Little or no nutritional intake for >5 days
• History of alcohol misuse or drugs, including insulin, chemotherapy, antacids, or diuretics

Extreme cases, for which BMI is less than 14 kg/m or negligible intake for more than 15 days

National Institute for Health and Clinical Excellence.
Patient at risk

Check potassium, calcium, phosphate, magnesium

Before feeding starts, administer thiamine 200-300 mg daily orally, vitamin B high potency 1-2 tablets 3 times daily (or full dose intravenous vitamin B), and multivitamin or trace element supplement once daily

- Start feeding 0.0418 MJ/kg/day*
- Slowly increase feeding over 4-7 days

Rehydrate carefully and supplement and/or correct levels of potassium (give 2-4 mmol/kg/day), phosphate (0.3-0.6 mmol/kg/day), calcium, and magnesium (0.2 mmol/kg/day intravenously or 0.4 mmol/kg/day orally)

Monitor potassium, phosphate, calcium, and magnesium for the first 2 weeks and amend treatment as appropriate

*If patient is severely malnourished (for example, body mass index (kg/m²) <14) or if intake is negligible for 2 weeks, start feeding at maximum of 0.0209 MJ/kg/day
Hospital Protocol

- Start on low calorie diet. Per NICE guidelines, 10 kcals/kg/day or 5 kcals/kg/day for extreme cases.
- Slowly advance caloric intake—differing opinions. Based on clinical judgement and monitoring.
- Check electrolytes (potassium, magnesium, phosphorus) on presentation and either daily or three times a week and replace electrolytes if indicated.
- Monitor CBC, BMP, AST/ALT frequently (daily or weekly depending on risk and/or lab values).
- EKG on admission and cardiac monitoring in extreme cases.
- Daily vital signs, daily weights.
- Start thiamine 200mg-300mg daily, 1-2 tabs of vitamin B complex three times a day, and multivitamin.
- Monitor for signs or symptoms of refeeding syndrome.
Signs and Symptoms

**Electrolyte imbalance**
- hypokalemia
- hypophosphatemia
- hypomagnesemia

**Neurologic**
- weakness
- seizure
- paresthesia
- altered mental status
- paralysis
- tetany

**Musculoskeletal**
- weakness
- myalgia

**Hematologic**
- thrombocytopenia
- platelet dysfunction
- anemia
- infections
- bleeding

**Respiratory**
- hypoxia

**Gastrointestinal**
- anorexia
- abdominal pain
- constipation
- diarrhea

**Cardiovascular**
- dysrhythmias
- hypotension

**Renal**
- edema
- elevated blood urea nitrogen
- elevated creatinine

**Metabolic**
- metabolic alkalosis
- metabolic acidosis

**Nutritional**
- vitamin deficiency, such as deficiency of vitamin B₁ (thiamine)
- low albumin and prealbumin levels.
**Recommendation for Replacement**

<table>
<thead>
<tr>
<th>Mineral</th>
<th>Dose</th>
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</thead>
<tbody>
<tr>
<td><strong>Phosphate</strong></td>
<td></td>
</tr>
<tr>
<td>Mild hypophosphatemia (2.0-2.5 mg/dL)</td>
<td>1000 mg/d oral replacement. Dose of 250 mg po QID (K-phos tab or Neutra-phos tab)</td>
</tr>
<tr>
<td>Moderate hypophosphatemia (1.0-2.0 mg/dL)</td>
<td>1000 mg/d oral replacement. Dose of 250 mg po QID (K-phos tab or Neutra-phos tab)</td>
</tr>
<tr>
<td>Severe hypophosphatemia (&lt;1 mg/dL)</td>
<td>Send to ER. Managed with IV replacement of 0.08-0.16 mmol/kg over 2-6 hours</td>
</tr>
<tr>
<td><strong>Magnesium</strong></td>
<td></td>
</tr>
<tr>
<td>Mild hypomagnesemia (1.5-2.0 mg/dL)</td>
<td>400mg magnesium oxide PO for 1 dose</td>
</tr>
<tr>
<td>Moderate hypomagnesemia (1.2-1.4 mg/dL)</td>
<td>400mg magnesium oxide PO every 4 hours for 2 doses</td>
</tr>
<tr>
<td>Severe hypomagnesaemia (&lt;1.2 mg/dL)</td>
<td>Send to ER. 4 g MgSO4 IV then as for mild to moderate hypomagnesaemia (above)</td>
</tr>
<tr>
<td><strong>Potassium</strong></td>
<td></td>
</tr>
<tr>
<td>Mild hypokalemia (3.0-3.5 mEq/L)</td>
<td>Give KCL 20 meq po every 2 hours for 2 doses</td>
</tr>
<tr>
<td>Moderate hypokalemia (2.5-3.0 mEq/L)</td>
<td>Give KCL 20 meq po every 2 hours for 4 doses</td>
</tr>
<tr>
<td>Severe hypokalemia (&lt;2.5 mEq/L)</td>
<td>Send to ER. ECG monitoring is imperative and IV potassium</td>
</tr>
</tbody>
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Concluding Points

• Refeeding syndrome is a potentially fatal, but preventable condition, caused by rapid initiation of refeeding after a period of under nutrition
• It is characterized by hypophosphatemia, fluid and electrolyte shifts, and metabolic and clinical complications
• Patients at high risk include chronically undernourished patients who have had little or no intake for more than 10 days
• Refeeding should be started at low level of energy replacement with vitamin supplementation
• Correction of electrolyte and fluid imbalances before feeding is not necessary and should be done alongside feeding through monitoring of electrolytes at least three times a week
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Rosewood Center for Eating Disorders
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THANK YOU!
References


2015 Webinars At-A-Glance

Go to www.rosewoodinstitute.org to view recorded CE webinars

- **JAN 16**: After the Binge: Using the Mindful-based Strategies to Treat Binge Eating Disorders by Nancy Romanick, RDN, MBA, MAPC, LAC

- **FEB 27**: Treating Complex Trauma: A Sensorimotor Psychotherapy Approach by Holly Finley, LPC, CEDS

- **MAR 27**: Shaping Foundations: How Three Decades of Prevention Research has Transformed Education Outreach and Treatment of Eating Disorders by Dr. Michael Levine

- **APR 24**: Do no Harm-Ethics and Eating Disorder Treatment" / "The Implementation of Ethics and Eating Disorder Treatment by Dr. Jessica Rodriquez

- **MAY 22**: Nutrition Medicine: The Acute Care of Eating Disorders, Stabilization and Recovery by Megan Kniskern, RD, CEDRD

- **JUNE 19**: Does DSM V Really Change Anything? A New Look at Classifying Eating Disorders by Jessica Setnick, MS, RD, CEDRD

- **JULY 24**: Refeeding Syndrome: Complications of Anorexia Nervosa, by Amelia Davis, MD

- **AUGUST 21**: Mirror, Mirror? Treating the Body Image Experience by Cheryl Musick, EAGALA Advanced Certified

- **SEPTEMBER 18**: Axis II: Treating the other 50% by Dr. Dena Cabrera

- **OCTOBER 16**: Being Whole Again: Keys to Families Gaining and Holding on to Recovery by Billie Church, MC, LPC

- **NOVEMBER 13**: The Diabetic Dilemma: Treating Eating Disorders in Diabetic Adolescents and Adults with Eating Disorders by Kim Vavrosky, MS, RD

- **DECEMBER 11**: Trends, Truths and Travesties: The Real Scoop on Food Allergies and Intolerances by Miriam Anand MD, FACP, FAAAI, FACAAI and Megan Kniskern MS, RD, CEDRD
Questions & Answers

Email us at TRI@rosewoodranch.com

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