Medical Complications of Anorexia Nervosa and Bulimia

Duke University Psychiatry Grand Rounds
January 2017

Philip S. Mehler, MD, FACP, FAED, CEDS
Medical Director & CMO Eating Recovery Center, Denver
Medical Director, ACUTE at Denver Health
Glassman Professor of Medicine, University of Colorado
Disclosures

• No conflict of interest or pertinent financial disclosures to report
• Will present evidence based recommendations when available
• Any off-label use of medications or interventions will be disclosed
Learning Objectives:

• Identify medical problems related to bulimia nervosa, and how to manage them
• Identify medical problems related to anorexia nervosa, and how to manage them
• Recognize when a patient needs a higher level of care for ongoing management of medical problems
Richard Morton, 1684

“Mr. Duke’s daughter in St. Mary Axe, in the year 1684, and the Eighteenth Year of her age, in the month of July fell into a total suppression of her Monthly Courses. From which time her Appetite began to abate, and her Digestion to be bad; her Flesh also began to be flaccid and loose, and her looks pale, with other Symptoms The Spring following by the Prescription of some Emperick, she took a Vomit, till at last being brought to the last degree of a Consumption, and thereupon subject to frequent fainting Fits, she apply’d her self to me for advice.

I do not remember that I did ever in all my Practice see one, that was conversant with the Living so much wasted with the greatest degree of a Consumption, (like a Skeleton only clad with skin) yet there was no Fever, but on the contrary a coldness of the whole Body; no Cough, or difficulty of breathing. She beg’d that the whole Affair might be committed again to Nature, whereupon consuming every day more and more, she was after three Months taken with a Fainting Fit and Died.”
Anorexia Nervosa (DSM 5)

• Restriction of energy intake, leading to significantly low body weight, intense fear of weight gain, distortion of body image

• Restricting subtype
  ✓ Restrict intake

• Binge-eating/purging subtype
  ✓ Restrict intake, binge and/or purge; purging involves exercise, laxatives, diuretics

• Mild, moderate, severe, extreme
Bulimia Nervosa

- Recurrent binge-eating
  - Unusually large amount of food (by social comparison) in a short amount of time
- Feeling out of control
- Compensatory behavior
  - Vomiting
  - Laxative abuse
  - Excessive exercise
  - Fasting
- Extreme focus on shape/weight
Eating Disorders are Common

- 90% Female
- 10-20 million girls and women
- Anorexia Nervosa .5-1% of females 13-35
- Bulimia Nervosa 2-5% of females 13-35
- Industrialized countries
Why does anorexia develop?

• Gene–environment interaction
• Eating disorders are familial illnesses
  – 12 x risk for anorexia nervosa and 4 x for bulimia nervosa
• Heritability estimates
  – 50-80% genetic influence
• What is inherited?
  – Temperamental traits
  – Co-morbid anxiety, depression, perfectionistic tendencies
The Problem

- 60% of teen girls report feeling fat despite being normal weight
- 60% of teen girls in westernized countries are on a diet at any one time
- Pre-teen girls report that they are more afraid of Fat than Cancer
- 45% of girls who are smoking cigarettes are smoking as a form of weight control
Common Precipitants

• The immediate precipitating factors is almost always an internal or external experience of being out of control
  – Onset of puberty between the ages of 11-14, i.e. four years the average young women gains 40 pounds with a disproportionate fat ratio
  – Major transitions: separation, individuation, & identity
    • 90% of young women who develop an eating disorder do so between the ages of 12 and 25
Common Precipitants, continued

– Traumatic events: abuse, rejection, failure
– Family difficulty: divorce, disengagement
– Onset of co-morbid illness: anxiety, depression
– Innocent weight loss: increased exercise, performance enhancement
Fact

- Eating Disorders run in families

Genetics Loads the Gun...
Environment Pulls the Trigger
Genetic Studies

• Individuals with a mother or sister who had suffered from Anorexia Nervosa are:
  – 12 times more likely to develop Anorexia Nervosa
  – 4 times more likely to develop Bulimia Nervosa
Prognosis of anorexia nervosa

- Mean duration of illness to recovery: 7 years
- 50% completely recover
- 30% partially recover
- 20% remain severely ill
- Crude mortality rate > 5%
- Meds don’t work

Hoek 2006, Keel 2010, Arcelus 2011
Recovery Awaits
J. Clinical Psych, 2016

• Mass General longitudinal study
• Assessed at 16, 20 and 25 years of follow up
• 62% of A.N. and 68% B.N. patients recovered
• Half of those with A.N. at 10 years who had not recovered, progressed to recovery at 22 years.
• Recidivism is “normal” in EDs
Risk factors for death

- Older age at first presentation
- Lower weight (BMI) at first presentation
- Duration of illness
- Alcohol and diuretic abuse
- Comorbid mood disorders
- Psychiatric hospitalization
- History of suicide attempts, self harm

Anorexia nervosa: mortality

• Mortality rate 10 years after hospitalization: 10x greater than age/gender matched population

• ½ of deaths occurred in first three years after hospitalization

• Risk predicted by chronicity and seriousness of illness

• Most frequent causes of death: suicide and medical complications of anorexia
Medical Complications

- In anorexia a direct result of starvation and weight loss
- Anorexia nervosa has the highest mortality rate of any mental illness
- In bulimia directly correlated with mode and frequency of purging
- Litany of medical problems associated with these eating disorders
- Most are reversible and treatable
- Some are associated with permanent harm
Medical Complications of Anorexia Nervosa

**Cardiovascular**
- Bradycardia and hypotension
- Mitral valve prolapse
- Sudden death - arrhythmia
- Refeeding syndrome
- Echo changes
- Pericardial effusions

**Endocrine and Metabolic**
- Amenorrhea
- Unintended pregnancy & miscarriages
- Osteoporosis
- Thyroid Abnormalities
- Hypercortisolemia
- Hypoglycemia
- Neurogenic diabetes insipidus
- Hypophosphatemia

**Dermatologic**
- Dry skin
- Alopecia
- Lanugo hair
- Starvation-associated pruritis
- Acrocyanosis

**Hematologic**
- Pancytopenia
- Decreased sedimentation rate

**Neurologic**
- Cerebral atrophy

**Ophthalmic**
- Lagopthalmos

**Auditory**
- Patulous eustachian tube dysfunction

**Gastrointestinal**
- Constipation
- Refeeding pancreatitis
- Acute gastric dilatation
- Delayed gastric emptying
- Hepatitis
- Dysphagia
- SMA syndrome

**Pulmonary**
- Aspiration pneumonia
- Respiratory failure
- Spontaneous pneumothorax
- Emphysema
Medical Complications of Bulimia Nervosa

**Gastrointestinal**
- Dental erosion and caries
- Parotid gland swelling
- Esophageal rupture
- Gastroesophageal reflux (GERD)
- Constipation due to laxative abuse
- Rectal prolapse
- Mallory-Weiss tear

**Pulmonary-Mediastinal**
- Aspiration pneumonitis
- Pneumomediastinum

**Ophthalmic**
- Scleral hemorrhage

**ENT**
- Epistaxis
- Pharyngitis

**Cardiac**
- Arrhythmias
- Diet pill toxicity
- Palpitations
- Emitele cardiomyopathy

**Endocrine**
- Irregular menses
- Mineralocorticoid excess
- Diabulimia

**Metabolic**
- Hypokalemia
- Dehydration
- Nephropathy
- Metabolic alkalosis
- Pseudo Bartter's syndrome

**Dermatologic**
- Russel’s sign
- Edema
Criteria for Hospitalization (Anorexia)

• Weight <70% of expected IBW (highest grade)
• Continued weight loss despite intensive outpatient therapy
• Unstable vital signs: pulse < 40, temp <35, SBP <85 mmHg
• Arrhythmias
• Suicidality
• Albumin levels **Not** predictive-normal
• Prealbumin levels are useful and low
• Other levels of care (Residential, PHP, IOP) if less severe
When to Hospitalize Medically? (Bulimia)

For Bulimia Nervosa

– Potassium < 2.8 mmol/L
– Bicarbonate > 37 mmol/L
– Excessive edema
– History of edema with cessation of purging behaviors
– Severe constipation
HEENT (Bulimia)

- Perimyolysis (anterior-lingual surfaces)
- Caries
- Subconjunctival hemorrhages
- Epistaxis
- Sialadenosis
Sialadenosis

NOTE: Begins 3-4 days after cessation of vomiting
Lagophthalmos - Anorexia

• Failure of eyelids to close fully due to sunken eyes
• Causes dry, irritated eyes
• Can lead to corneal scarring
• Use tears, ointment and eye taping nightly
• Resolves rapidly with hydration, modest weight restoration
Gastrointestinal (Bulimia)

- Esophagitis – GERD
- Barrett’s
- Mallory-Weiss tears
- Boerhaave’s syndrome
- Dysphagia/strictures
- Cathartic colon – severe and recurrent constipation from stimulant laxatives
Gastrointestinal (Anorexia)
Factors Impeding Refeeding

• Delayed gastric emptying – gastroparesis
• Acute gastric dilatation
• Reflex hypofunctioning of colon
• Diarrhea – Diamine oxidase
• SMA - syndrome
Superior Mesenteric Artery (SMA) Syndrome

- Third portion of duodenum becomes compressed between the aorta and the vertebral column, due to weight loss and loss of the fat pad that normally surrounds the blood vessels
- Present with weight loss, abdominal pain, distention, nausea and vomiting
- Functionally: a small bowel mechanical obstruction that can only be fixed by weight restoration
- Diagnosed by CT or UGI
Superior Mesenteric Artery (SMA)
Liver Dysfunction

Liver function tests (LFTs) are often elevated in severe AN

- **Starvation mediated:**
  - Autophagy on biopsy (apoptosis) correlates with nadir BMI and hypoglycemia, recovers with refeeding. More common.

- **Refeeding mediated:**
  - Steatohepatitis, recovers with slowed refeeding

How to tell which is causing elevated LFTs?
(very different approach)
Time Trends in the prevalence of Celiac Disease
JAMA Int Medicine 176:1716-1719, 2016

Key notes
• NHANES data (2008-2014)
• 22,278 persons interviewed
• Prevalence of celiac disease remained stable
• Prevalence of gluten-free diets tripled!!
Celiac Disease

Practice Implications

• Digestive symptoms and functional GI disorders are frequently reported by patients with Anorexia Nervosa
• Dyspepsia, gassiness, distention, diarrhea and pain complaints are common in this population
• Intestinal absorption and villi integrity may be impaired in Anorexia Nervosa and cause diarrhea early on in refeeding
• But, gluten sensitivity concerns are exaggerated, and dietary modifications need judicious deliberation.
MESSAGE

Caveat emptor
Let the buyer beware!!
Cardiac (Anorexia)

- Heart muscle atrophy with loss of Ventricular mass
- Attenuated BP response to exercise & reduced exercise capacity
- Low blood pressure (70/40)
- Mitral valve prolapse
- Bradycardia (resting pulse 40-60)
- Pericardial effusions
- Fibrosis by MRI
Sinus Bradycardia and Anorexia

- Sinus Bradycardia is the most common arrhythmia in anorexics
- Increased cardiac vagal activity is responsible for the marked bradycardia
- Junctional bradycardia – Exercise extinguishes
- A normal heart rate (>70) is **NOT** normal and should prompt evaluation for possible cause of increased sympathetic tone
- Hypotension is the rule (SBP<90). Thus, don’t treat

*BMC Cardiovascular Disorders 4:10, 2004*
Cardiac (Anorexia)

- Increased risk of sudden death
- Clean coronaries on post-mortem
- ??QT prolongation $\rightarrow$ Torsade De Pointes, $\rightarrow$ sudden death (the old)
- Increased QT interval dispersion on EKG, is associated with propensity to ventricular arrhythmias (the new)
- Abnormally low heart rate variability (the new)
QT Dispersion and Metabolic Rate

QTdisp = 150.710 - (0.0886*KCAL)

R = 0.673  \quad R^2 = 0.453

P = 0.004
Refeeding Syndrome

- Classic study by Keys in 1940’s (“Minnesota Experiment”)
- Seen with unintentional refeeding experiments involving victims of WWII. Once released they were given candy by soldiers and precipitously died. Those given chocolate milk survived??
- These severe complications of refeeding rediscovered with introduction of TPN
- Anorexia nervosa serves as a sobering model for the possible calamity inherent in refeeding severely malnourished patients and manifests with peripheral and pulmonary edema, CHF, hypophosphatemia and death
Refeeding Hypophosphatemia

Increased Demand for Phosphorous to provide phosphorylated intermediates of Glycolysis and high energy compounds like ATP

Normal Serum Phosphorous

Insulin Secretion

Glycolysis Intracellular Influx of Phosphorous

Tissue Hypoxia Myocardial Myopathy

Hypophosphatemia

2,3-DPG ATP Glycolysis

Renal Excretion of Phosphorous & Muscle Catabolism

CARBOHYDRATES
Recommendations to Avoid the Refeeding Syndrome

1. Recognize the “patient at risk”
2. Carefully test for and correct electrolyte abnormalities before initiating any nutrition support whether PO, NG or TPN
3. Judiciously restore circulatory volume, closely monitor vitals and exam, Never administer rapid IV fluids
4. Increase caloric delivery slowly, but faster than before
5. Carefully monitor the electrolytes especially over the 1st week, including: Phosphorous, Potassium and Magnesium
7. Start 1400-1800 kcal/day and increase by 300-400 after 3-4 days
Consequences of Hypophosphatemia
(Normal 2.6 - 4.0)

- RBC dysfunction (hemolysis)
- Rhabdomyolysis (↑ CPK and ↑ AST)
- CNS dysfunction (Seizures)
- Myocardial failure
- Respiratory paralysis (diaphragm doesn’t move)
Timing of Hypophosphatemia

- DKA ~ 12 hours
- ETOH Re-Feeding ~ 24-48 hours
- Hyperalimentation ~ 24-48+ hours
- Anorexia Nervosa ~ 24-72+ hours

(Generally at risk for it for first few weeks of consistent refeeding)


Cardiac (Bulimia)

- Ipecac induced cardiomyopathy
  - Due to alkaloid emetine in Ipecac
  - Each bottle contains 30 mg of emetine
  - Cumulative toxicity after 1250 mg to heart & skeletal muscle with heart failure and muscle weakness

- Arrhythmias due to acid-base & electrolyte disorders
  a) Low potassium – hypokalemia (<3.4)
  b) Metabolic alkalosis – high bicarbonate level (>28)
  c) Metabolic acidosis – low bicarbonate level (<19)
Pulmonary – Anorexia Nervosa

• Spontaneous pneumothorax, slow healing
• Early emphysema
• Aspiration – cough/pneumonia
• Weakened respiratory muscles
• Hypoxia – hypercarbia (↑ CO₂ and ↓ O₂)
Hyponatremia (Low Sodium) (Anorexia)

• Due to inability to clear free water because of low renal solute load versus normal-weight individual

• Hyponatremia occurs at much lower volumes of water intake and can result in severely low levels of serum sodium

• SSRI’s may exacerbate

• Not SIADH
A Clinicians Guide to the Bulimic’s Medicine Cabinet

60% Self-induced vomiting/Ipecac

30% Laxatives (stimulant type)

5% Diet Pills

5% Diuretics
Symptoms from Purging

- Vomiting
  - Epistaxis
  - Visual “floaters”
  - Oral pain
  - Hygienic issues
  - Dental erosions
Symptoms from Purging

- Laxatives
  - Abdominal pain
  - Constipation
  - Diarrhea
  - Rectal bleeding
  - Volume depletion and dizziness
  - Paresthesias
  - Irreversible cathartic colon syndrome
Electrolyte Abnormalities

- Hypokalemia (all)
- Metabolic alkalosis (vomit-diuretic)
- Non-Gap, metabolic acidosis (diarrhea)
- Pseudo Bartter’s Syndrome (all modes of purging)

- Water intoxication hyponatremia (sodium <130 mmole/dl)
- Hypophosphatemia (phosphorous <2.4)
Metabolic Alkalosis

- Most common acid/base disturbance seen with bulimics
- Both vomiting and diuretics create a contraction alkalosis secondary to loss of NaCl resulting in intravascular volume depletion
- Laxatives in the acute setting can cause a non-gap metabolic acidosis. With chronic use a mild metabolic alkalosis with severe hypokalemia is more likely
- Bicarbonate >38 is very suggestive of self-induced vomiting
Hypokalemia (Low Potassium)

- Seen in all types of purging disorders
- Finding of **significant hypokalemia** in an otherwise healthy appearing young woman is **highly specific** for bulimia nervosa (Teach!)
- Predisposes bulimics to palpitations and cardiac arrhythmias
Why is it Medically Difficult to Cease Purging??

- Psychological!! **BUT,**
- A predictable constellation of troubling symptoms which ensue with abrupt cessation of purging, regardless of specific type of purging
- Memories of these medical events are disincentives to cease purging and create angst when it is suggested and contribute to bulimia’s increased death risk of 1.93-
Pseudo-Bartter's Syndrome

- Normotensive hypokalemic hypochloremic metabolic alkalosis
- Secondary hyperaldosteronism from the adrenal glands
- Tendency to severe edema formation with abrupt cessation of any mode of purging, especially if receive rapid IVF
Turning Off Aldosterone - Preemptive

- Restoring intravascular volume turns off aldosterone…but this takes **WEEKS**
- Rapid infusion of IVF will result in significant edema formation and weight gain
- Don’t bolus IV saline in these patients!! (ED docs)
- Replace potassium and magnesium
- Transient use of spironolactone (25-200 mg/day) will help turn off aldosterone, promote potassium retention and prevent edema formation (7-14 days)
How Does Pseudo-Bartter Impact ED Management?

- Treatment requires correction of the stimulus for hyperaldosteronism
  - Correct volume contraction *(Slowly!!)*
  - Correct hypokalemia  *Annals Emerg Med, 2013*

- Patient dissatisfier!
National Survey of Eating Disorder Training

Key Notes

• 887 training programs in Internal Medicine, Family Practice, Pediatrics, Adolescent Medicine and Child & Adolescent Psych

• Surveyed to ascertain amount of Eating Disorder training in the 3 and 4 year residencies

• 514 offered no formal teaching

• 323 offered very limited teaching

• 42 offered formal scheduled training

• Child Psych & Peds offered most
Idiopathic Cyclical Edema

- Subject of discussion since early 1950’s
- Confined to females
- Negative work-up for cardiac, hepatic, lymphatic, nutritional, or renal disorder
- Diuretics prescribed after being labeled “hormonal” as if a specific diagnosis is made
- Idiopathic ≠ iatrogenic
Idiopathic Cyclical Edema

- Many reports demonstrating that diuretic abuse may actually be the primary factor in initiating some cases
- Reflex edema following their usage due to stimulation of Renin-Angiotensin-Aldosterone system
- Treatment is to taper diuretics, restore volume, restrict salt and be patient
Pathophysiology of Early Refeeding Edema in Anorexia Nervosa (restricting subtype)

- Not due to albumin oncotic pressure
- Not Pseudo-Bartter syndrome
- Hyperinsulinemia →↑ distal tubular sodium reabsorption-retention and antinatriuresis
Dermatological

- Xerosis
- Diffuse hypertrichosis
- Acrocyanosis
- Brittle nails
- Starvation–associated pruritis
- Cartonemia

- Russell’s sign (bulimia)
Heme – Anorexia Nervosa

- Trilinear hypoplasia
- Serous fat atrophy – gelatinous marrow
- Diminished fever response
  \(\text{(Don’t see real high temps)}\)
- Risk of infection??
- High B-12 levels
Neurological (Anorexia Nervosa)

- Cerebral atrophy with enlarged ventricles
- Impaired cognitive function
- Permanent cognitive deficits??
- Impaired thalamic function
- Skeletal muscle weakness
• By the end of the second decade, more than 90% of peak bone mass has been achieved.
• By the end of their second decade, most anorexic patients have suffered for years from severe malnutrition and amenorrhea and fail to lay down crucial bone mass.
• 50% of adolescents have osteopenia or osteoporosis, 90% adults in anorexia nervosa.
Osteoporosis in Males with Anorexia Nervosa

Don’t Overlook!

Endocrine

- Hypoestrogenic and low T
- Amenorrhea – hypothalamic (GnRH)
- Intact fertility, preterm infant & miscarriage risk increased 2-fold (A.N.)
- Euthyroid sick syndrome (nml TSH). Don’t treat!
- Increased growth hormone
- Hypoglycemia
- High cortisol levels
- Diabetes mellitus (B.N.) “Diabulimia”
Medical Treatments (Anorexia)

**GI**
- Low dose metoclopramide and Erythro-Azithro
- Patience – colonic transit normalizes in a few weeks with adequate fluid and nutritional intake. Avoid “too much fiber”

**Endo**
- Menses return at >90% of IBW
- No long term infertility
- Recombinant Leptin induced menstrual cycle
- Euthyroid sick syndrome is self limiting
Osteoporosis: Bisphosphonates & Rx

- Definitely indicated in men with osteoporosis if testosterone is normal
- Document informed consent with women of childbearing age and definitive osteoporosis
- A “bridge” until full weight restoration is achieved
- Check DEXA every 2 years and not more frequently
- Order DEXA in all patients with anorexia nervosa after just one year of diagnosis
- Teriapartide (PTH) – likely
- Transdermal estrogen, not BCP’s
- Prolia - ??
- Not exercise
**Constipation (Laxative Abuse)**

- Cease stimulant laxatives – no logic to taper!
- Replete potassium to > 4.5 mg/L
- Reality – Rome III Criteria
- Abdominal x-rays
- The right laxative to obligate intraluminal water retention (polyethylene glycol).
  - Osmotic-type
- Lubiprostone or linzess – role??
- Reassurance, patience, and admonition against ongoing stimulant-type laxatives
Medical Treatments (Anorexia)

**Cardiac**
- Bradycardia & HRV return to normal
- MVP recedes
- Left ventricle wall thickens back to normal

**Neuro**
- Brain volume recovers mostly
- Neurocognitive function may recover

**Heme**
- Bone marrow reconstitutes all cell lines

**GI**
- Gastroparesis resolves as does SMA. Role for reglan ± erythro BID or Azithro
Medical Treatments (Bulimia)

**Oral**
- Gentle brushing & use of fluoride mouthwash
- Application of heat, tart candies, oral pilocarpine

**UGI**
- PPI (Omeprazole)
- Metoclopramide – **low dose**
- Barrett’s esophagus ??
Acute sialadenosis

- Goes away slowly with NSAIDs (e.g. ibuprofen), tart candies, and warm compresses
Metabolic rate or total energy expenditure (TEE) has four components:

1. Sleeping energy expenditure (SEE)
2. Resting energy expenditure (REE)
3. Dietary-induced thermogenesis
4. Activity-induced thermogenesis
REE

- Key determinant of dietary prescription
- Can be calculated using Harris-Benedict equation
- Can be measured using indirect calorimetry
- Some of REE determined genetically
- Accounts for 60% of TEE
• Absolute REE is low in underweight A.N. patients
• REE increases substantially (20% increase in REE/5kg) with refeeding
• Accurate prediction of the calorie level to promote weight restoration is needed especially in later steps of weight gain (>4,000 Kcal/d)
• Leptin does not contribute either to the reduced REE in prefed A.N. patients or to the increase in REE during refeeding
• Majority of predictive formulas overestimate REE in the early refeeding stages of anorexia nervosa
Weight Restoration in Anorexia

• Becoming more aggressive – but use caution
• Goal is to achieve >90% - 100% of IBW
• Rate of weight gain: 1-1.5 lbs/week (outpt) and 2-3 lbs/week (inpt)
• Start 1400-1600 Kcal and increase 300-400 Kcal every 3-4 days
• No role for supplemental antibiotics as part of management of severe malnutrition in anorexia *(NEJM)*
Plateau Effect

- During the course of refeeding, the number of Kcal needed to gain weight increases and REE increases (>4000 Kcal/day)
- Clinical observations consistently suggest that anorectics gain weight with great difficulty
- Calculations for caloric needs necessary to assure weight gain and are continuously upwardly adjusted
- Mechanism unknown: ↑anxiety, ↑T₃
Enteral vs TPN vs Oral

- Little attention accorded to innovation in nutritional rehabilitation for the treatment of anorexia (no RCT’s)
- Staged oral refeeding remains the standard, but may need additional help
- PEG – NG feedings, or via PEJ
- Supplemental nocturnal tube refeeding successfully used
- Has been challenged clinically and ethically
- TPN not as dangerous?? (NEJM- 2014)
- Diet composition without basis
Extreme Anorexia Nervosa

- NHANES-4
- DSM- A.N.
- ACUTE @ DH (BMI 6.2-14.7)
Case: HD

- 20 year old from New Jersey with restrictive AN, admitted for syncope at her university hospital
- Complications during her 6 months of hospitalization at outside hospitals:
  - PICC line placement with air embolism
  - Severe refeeding syndrome
  - Mismanagement of intravenous glucose
  - Severe edema
  - Falls
  - Lack of psychiatric support
  - Chronic nasogastric tube use due to surgeon discomfort with placing a PEG
  - Despite continuous hospitalization, ongoing weight loss from August to December
But medical questions remain:

1. Who gets gastroparesis and how long does it persist?
2. Who gets SMA and when does it resolve?
3. What is the optimal mode and rate of refeeding?
4. What is the role of supplements in the refeeding process?
5. When do orthostatic changes in pulse and blood pressure revert to normal in A.N.? Slower in younger? In men?
6. What is optimal treatment for osteoporosis?
7. Who gets pseudo-bartters edema?
8. How long do aldosterone levels remain high?
9. What is optimal oral hygiene practice in B.N.?
10. What is the medical issue that most frustrates your patients or you?
Take Home Points: Bulimia

- Medical problems are caused by the mode and frequency of purging
- Severe fluid and electrolyte shifts can be life-threatening
- Patients with a history of severe edema, difficult diuretic detoxification, or severe hypokalemia should be managed in a hospital setting with experienced providers
Take Home Points: Anorexia

- Medical problems are caused by starvation and weight loss
- Most medical problems reverse with judicious nutritional rehabilitation and weight restoration. Osteoporosis is an exception
- **Refeeding can be deadly** and should not be undertaken with severely underweight patients in the outpatient setting
- Patients weighing **less than 70% of IBW** should optimally refeed in a hospital setting with experienced providers
- Many patients with severe AN languish in hospitals without sufficient expertise and cannot move on to much-needed inpatient psychiatric care