WA Eating Disorders Outreach & Consultation Service (WAEDOCS)

Handbook for the medical management of eating disorders in the inpatient setting
Additional information for medical staff

Adapted from the Royal Brisbane Women’s Hospital Eating Disorder’s Unit Inpatient Guide
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Introduction

Eating Disorder (ED) patients often present a unique constellation of signs and symptoms spanning across both the psychological and medical realm. This handbook is meant to serve as a guide for managing the more common complications seen in ED patients but should never replace sound clinical judgement and if in doubt you should always discuss with your Registrar and/or Consultant, with additional sub-specialist consultation with a physician experienced in the management of people with complex eating disorders if required.

It also provides a guide on how to discuss these medical complications with ED patients – who often tend to minimise their symptoms, or fail to understand their severity. Increasing their understanding of these risks can be a pivotal factor in helping to treat these patients – and is a place where you can play a very important role towards their recovery!

The most common patients you will be treating in the inpatient setting will be patients who have been admitted for medical compromise due to their eating disorder, which most commonly will be Anorexia Nervosa (AN). Generally those at highest medical risk are those who have associated purging behaviours (vomiting/laxative abuse) or co-morbid drug and alcohol misuse. Other patients may be admitted with normal BMI but are at risk due to rapid weight loss or to help them break purging behaviours – often seen in Bulimia Nervosa (BN). Conversely, others may be admitted due to their suicide risk – another equally fatal complication of eating disorders. (See appendix for admission criteria).

Eating Disorder patients carry a high mortality risk, and have the highest mortality rates of all psychiatric patient groups. Deterioration can occur rapidly, including those who have previously appeared stable. Early recognition and treatment of complications will help prevent serious outcomes.

Some things to bear in mind when treating ED patients:

- For numerous reasons, ED patients may not tell you their symptoms. A medical history is therefore not always reliable. If you are concerned, investigate anyway, despite being told “I’m fine”. Unlike most patients – these patients may not seek out your help for symptoms, and may actually try and put you off treating them! Do not be swayed – go with your clinical judgement.

- Do not treat these patients as you usually may for a patient of the same age i.e., “young girl so is unlikely to have cardiac chest pain”. This is not a young resilient heart or body you are dealing with. Actually similar to treating a heart failure patient.

- The majority of what you will be managing will be acute complications secondary to starvation and purging, as well as the risk of refeeding syndrome. However, it is also worth thinking about the longer term complications such as osteoporosis; bone fractures; colonic atony from laxatives; or Barret’s oesophagus from vomiting, and what measures may be put in place to monitor this or reduce the risk of their onset.
Refeeding Syndrome

- Refeeding syndrome is a potentially fatal medical complication arising from the aggressive refeeding of an individual who has been malnourished for a prolonged period.
- The process by which it occurs is complex but put simply it is thought to be due to a switch from fasting gluconeogenesis to carbohydrate-induced insulin release.
- This triggers a rapid shift of potassium, phosphate and magnesium into cells to metabolise carbohydrates.
- Already low body stores of these electrolytes from starvation leads to a rapid onset of hypophosphataemia, hypomagnesia and hypokalaemia.
- Additionally triggering of insulin release in addition to depleted glycogen stores can lead to post-prandial hypoglycaemia, including in the early hours of the morning.
- Given this, a low carbohydrate meal plan is the safest when refeeding, with close monitoring and replacement of electrolytes and blood glucose.
- Refeeding syndrome can have wide ranging effects from neurological, cardiopulmonary, haematological, cardiac and even death. The onset can be rapid (within days) and the risk can continue up to two weeks after refeeding has commenced depending on progress.

Management:

- Daily monitoring of electrolytes particularly Ca^{2+}/Mg^{2+}/PO_4^{3-}, until outside of risk period
- Prompt replacement of electrolytes if low. Refer to local guidelines (or for example Therapeutic Guidelines) for PO vs IVI replacement depending on severity of deficiency. Preferably by PO replacement though,
- Regular thiamine orally 300mg OD daily
- Commence supplementation 1 Vitamin and mineral supplement OD and 1 B complex OD
- Commence prophylactic 500mg Phosphate Sandoz BD QID 1 – 2 hours postprandial and 0200 BSLs with replacement if low - even after they’ve eaten!

Hypoglycaemic episodes often occur in early re-feeding of severely malnourished patients and those at risk of refeeding syndrome. Low BGLs (<4.0mmol/l) should be managed according to hospital protocol.

It is important to note also that as excess simple carbohydrate can precipitate rebound hypoglycaemia in these patients secondary to inadequate glycogen stores, a slow acting carbohydrate (e.g., one of: Tetrapak of Sustagen / Ensure Plus / Fortisip / glass milk and three crackers), **should be given in addition to a fast acting carbohydrate at the same time**, for those patients not receiving continuous nasogastric feeding.
Cardiovascular

- Cardiovascular complications are one of the major contributors to the increased mortality rate in eating disorder patients.
- Studies have found multiple structural changes in the heart of AN patients, most commonly, reduced LV mass and LV chamber with resultant reduction in cardiac output.
- Histological examination has also suggested changes such as widespread interstitial fibrosis.
- These changes predispose patients to clinically significant symptoms such as bradycardia, hypotension and arrhythmias. Combined with electrolyte disturbances this can easily become fatal.
- Therefore, consider that even in younger patients – this is a weakened, deconditioned heart that can easily become overwhelmed from factors such as minimal exertion and IV fluids.
- As such, low BP and HR are not necessarily due to hypovolaemia. In these patients they are more likely to be adaptive responses to conserve energy. But they are also indicators of reduced cardiac reserve and risk of imminent cardiac deterioration. Therefore only should be treated with fluids if there are clear clinical indicators of dehydration, and then should be done very carefully to reduce the risk of fluid overload.
- Note also that absence of cardiac symptoms does not imply less clinical significance. Patients will often not recognise or not report their symptoms. (e.g., Due minimisation or fear of consequences)
- Although it is critical to recognise and respond to these complications acutely, they are reversible with nutritional rehabilitation and weight restoration.

Hypotension

- Transfer to a medical unit if SBP < 80
- Usually due to a combination of cardiac deconditioning (with reduced contractility and HR), conservation of energy and dehydration with consequent risk of syncope, falls and reduced organ perfusion.

Management:

- May need to remain on bed rest
- Mainstay of treatment is with nutrition - will normalise with adequate refeeding
- If clinically dehydrated fluids preferably PO
- If IVF required do so slowly and carefully over a short period and monitor for fluid overload.
- If severely low – will need to consider monitored beds/Contacting ICU

ECG changes

- ECG changes are common in ED patients.
- Most commonly seen is sinus bradycardia, and also prolonged QT interval.
- Sinus Bradycardia is seen in up to 95% of patients and is thought to be predominantly due to increased vagal tone.
- If <40bmp should be transferred medically.
QT prolongation has been suggested in some studies to indicate increased risk for fatal arrhythmias. It is still uncertain if this just related to hypokalaemia and increased vagal tone rather than being intrinsic to AN.

Other findings seen may be secondary to electrolyte disturbances, (e.g., U waves in hypokalaemia), other arrhythmias or ST-T changes.

Any arrhythmias should be treated very seriously. Bear in mind (especially in patients that have a longstanding illness), the structural changes in a malnourished heart, haemodynamic compromise, and electrolyte disturbance may predispose to serious and fatal consequences of conduction abnormalities.

Management

- Check and replace any electrolyte deficiencies immediately
- Consider safest environment for the patient
- Discuss with the team and suspend any leave temporarily
- Discuss with the medical or cardiology registrar
- Check for medications that may be increasing the QT interval

Orthostatic Changes

- Orthostatic changes are common in AN and are reversible with refeeding.
- Postural tachycardia can be a sign of an undernourished heart and indicator of risk of arrhythmias
- HR has been found to be a more sensitive indicator of vital sign in/stability than blood pressure and a postural tachycardia can often be seen with no change in the BP.
- Defined as:
  
  Postural Hypotension = SBP >20mmHg or DBP >10mmHg drop on standing  
  Postural Tachycardia = HR >20bpm increase on standing

- Postural hypotension can lead to syncope and injury from falls.
- Resolution of postural changes is a good indicator of adequate nutritional rehabilitation and medical stability.

Management

- Mainstay of treatment is with nutrition – will normalise with adequate refeeding.
- Medical transfer should be discussed if the findings are in excess of the above ranges and keep on bed rest in the meantime.

Other less common findings:

Tachycardia: >110bpm can also be a sign of cardiac decompensation due to the heart getting inadequate nutrition and an indicator of risk

Pericardial Effusion: silent pericardial effusion can be commonly seen in AN. It usually resolves with restoration of weight. However there are some rare case reports of cardiac
tamponade needing urgent pericardiocentesis. In most cases it will not need any intervention. Discuss urgently with the medical registrar if there are any concerns of it causing cardiac compromise.

**Mitral Valve prolapse:** can occur in the setting of LV atrophy. May predispose to palpitations and chest pain. Usually no intervention required - treated expectantly with follow up ECHO to check for resolution with weight restoration.

**Electrolyte Disturbances**

- Electrolyte disturbances can occur for a number of reasons in eating disorders.
- The most common causes are from behaviours such as purging (vomiting, laxative abuse), refeeding syndrome, or from malnutrition itself.
- Other disturbances may arise in the setting of alcohol abuse (a high risk situation in eating disorders) renal failure, or medication related.
- The cause may not always be apparent from the history, as certain behaviours may not have been disclosed, such as purging. It is important to try and establish the cause so that the team can decide how to best manage it. (eg supervised bathroom visits if there is concern about purging), and address it with them.
- Disturbances are almost always clinically significant in being an indicator of either refeeding syndrome, increasing risk of fatal complications such as arrhythmia’s, or indicating severity of eating disordered behaviours such as purging.

**A guide to interpreting electrolyte findings in patients with eating disorders**

<table>
<thead>
<tr>
<th>Purging mode</th>
<th>Sodium</th>
<th>Potassium</th>
<th>Chloride</th>
<th>Bicarbonate</th>
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<tbody>
<tr>
<td>Vomiting</td>
<td>↓ Or</td>
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<td>Laxatives (short term)</td>
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<td>Laxatives (long term)</td>
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<tr>
<td>Diuretics</td>
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**Sodium:** may or may not be affected by purging (as above). Hyponatraemia may be seen in “fluid loading” done prior to weighing to increase BMI temporarily, and is associated with risk of seizures and cerebral oedema. In rare cases of fluid overload, fluid restriction may be needed. Usually management is expectant. A urinary chemistry with sodium can help establish cause. Also consider other causes e.g., SIADH. Check urine specific gravity prior to weighing to discourage fluid loading.

**Potassium:** hypokalaemia is seen in around 1/3 of all hospitalised ED patients. Usually due to purging (potassium exchanged for hydrogen ions in the kidney to compensate for that lost
through vomiting, to buffer consequent alkalosis). When chronically low – symptoms are often absent.

**Phosphate**: first to decrease in the setting of refeeding syndrome, usually first 1-3 days. Also seen in diuretic abuse, renal failure and rapid correction of hypokalaemia. Most patients will have been started on supplements prophylactically for refeeding syndrome. If levels are high – just monitor and continue on supplements while in the acute setting.

**Magnesium**: mostly seen in purging and refeeding, also can be due to diarrhoea, or diuretic and alcohol misuse. Does not decrease in refeeding until days after phosphate does, around 5-6 days so monitoring needs to continue. Can also lead to hypokalaemia and ECG changes.

**Calcium**: hypocalcaemia may be secondary to malnutrition, as well as alkalosis. May be associated with ECG changes.

**Bicarbonate and Chloride**: often deranged in purging. Vomiting can cause critically high bicarbonate and low chloride. The most important thing is to recognise what this is, and discuss with the team. Also monitor potassium which is likely to precipitously drop in the hours after.

### Management

- Establish the cause where possible
- Do ECG in case of potassium/magnesium/calcium
- Deficiencies can usually be replaced PO
- If IVI needed - do so slowly
- RBWH has IVI guidelines on when to replace IV vs PO
- Otherwise can check on eTherapeutic Guidelines
- If there are associated complications such as ECG changes - usually requires IVI and rechecking
- In severe cases may need to transfer medically for medical monitoring/telemetry. Discuss with the medical registrar
- If potassium is not improving despite replacement remember to check magnesium or consider Pseudo-Bartter’s Syndrome.
- Discuss at next ward round re behavioural interventions to reduce purging behaviours.

### Pseudo-Bartter’s Syndrome

- Dehydration (reduced intake/ purging) can lead to up-regulation of the renin-angiotensin-aldosterone system.
- Aldosterone secretion causes uptake of sodium and bicarbonate in the kidneys with subsequent water retention to protect against the ongoing potential for dehydration.
- In chronic situations - this results in a metabolic alkalosis and hypokalaemia. Taken together – this is known as pseudo-Bartter’s syndrome (a secondary hyperaldosteronism)
- Aldosterone continues to be up-regulated even after purging ceases – the subsequent fluid retention that occurs in this phenomenon may lead to development of oedema, particularly if rapid fluids have been given to correct dehydration
- Thus – slow and careful correction of dehydration is key
Haematological

- Starvation causes a hypoplasia of the bone marrow that is reversible with nutrition.
- Bone marrow biopsies from starved patients with ED’s have revealed there is gelatinous deposition in the marrow and hypoplasia affecting all three cell lines. As BMI falls severity of these changes increases.
- Most commonly seen is neutropenia and leucopenia.
- Anaemia is also commonly seen, and less commonly thrombocytopenia.
- An association between peripheral blood parameters and nutritional status has recently been demonstrated – as such the FBC is used on the ward as a marker for improving nutritional status.
- Malnutrition is associated with immune suppression, and potential for presentation with unusual or overwhelming infections.
- Long term bone marrow suppression is also linked with poorer bone health and osteoporosis.

Management

- Usually management is expectant as it is reversible with nutrition and there are few serious complications
- Check iron, B12/Folate if not already done
- Discuss with a medical registrar if neutrophils are consistently <0.5
- Does not require transfusion unless platelets or Hb is critically low, or there is active bleeding.
- Transfusion only in consultation with a medical registrar

Gastroenterological

There are many changes in the gastrointestinal physiology in patients with an eating disorder. Some of these are indicative of a chronic nature of this disorder.

Constipation

- Commonly occurs in drastic weight loss as the reduced caloric intake leads to reflex hypo-functioning of the colon, or to slowed colonic transit.
- Frequently follows cessation of laxatives.
- The most widely abused laxatives are from the stimulant class (eg. Senna based).
- In prolonged laxative use, return to normal bowel function can take an extended time, and on rare occasions can have serious outcomes (see laxative abuse).

Management

- Reassure that bowel movements will improve as nutrition improves, but can take some time
- If symptoms persist an upright abdominal x-ray may exclude abnormal colonic distension.
- Stimulant laxatives should always be avoided even in absence of misuse history.
- If needed osmotic laxatives such as movicol are safe to use
Laxative Abuse

- After self-induced vomiting laxative abuse is the second most common type of purging.
- Its medical complications usually manifest in two ways:
  - direct effects on the GI system
  - hypovolaemia and electrolyte disturbances.
- Beyond the more common prolonged constipation that follows cessation of laxatives, habituation of the colon to chronic laxative abuse may lead to an atonic colon and refractory constipation. In severe cases this may become irreversible resulting in requirement of a ileo/colo/stomy.
- Excessive laxative use with diarrhoea will ultimately lead to dehydration and hypovolaemia.
  Electrolytes lost include Cl, K+, HCO₃ and Calcium.
- Chronic diarrhoea leads to a hypochloraemic, hypokalaemic metabolic alkalosis, secondary to hypovolaemia induced hyperaldosteronism.
- Acute diarrhoea leads to a hyperchloraemic metabolic acidosis without an increased anion gap.
- Severe oedema can also occur with abrupt cessation of laxatives in abuse settings.

Management:

- Likely to need regular osmotic based laxatives e.g. movicol up to 3 or 4 times daily
- Ensure adequate hydration (PO) to oblige colonic absorption of water and stool softening.
- Monitor electrolytes and watch for fluid retention.
- If not resolving after reasonable period of regular osmotic laxatives do an abdominal examination, consider abdominal x-ray and discuss with the gastroenterology registrar.

Gastro-oesophageal Reflux Disease and Excessive vomiting

- Common in people who frequently purge via self-induced vomiting, microtrauma to the oesophageal epithelial cells occurs due to the repeated exposure to stomach acid.
- Consequences can include oesphagitis, ulcers, bleeding and Barrett’s oesophagus.
- One of the most serious although very rare consequences to repeated vomiting is Boerhaave’s Syndrome (oesophageal rupture) which is a surgical emergency. It manifests as chest pain, SOB and unique complaint of painful yawning, with tachycardia and tachypnoea.

Management:

- Manage as usual GERD with PPIs, antacids.
- Refer for endoscopy:
  - if there is a prolonged history non-urgently
  - more urgently if there significant symptoms
  - urgently if there is concern about bleeding.
- Suspected Boerhaaves should be treated as a surgical emergency.

Management:

- Explain how the bowel has lost its own intrinsic ability to contract due to excessive laxative use. Explain the risks (as above) of prolonged use and thus why stimulant laxatives need to be stopped. Discuss that it may take some time for the bowel to regain its usual function again, and we can use more gentle laxatives to help in the meantime that don’t over stimulate the bowel.

- Explain that repeated exposure to stomach acid from vomiting is traumatic to the oesophagus. While medications may lessen this, there is still discomfort, a risk of ulcers, bleeding and long term can increase the risk of cancer.
Other Findings:

Hepatitis/Deranged LFTs
Mildly elevated transaminases (around 2-3 x normal) is often seen in around 50% of AN patients due to weight loss and fasting. It also occurs in around 1/3 of hospitalised AN patients treated in hospital if carbohydrate intake is too high causing a steatosis. Discuss this with the ward dietitian – carbohydrate calories may be temporarily decreased.

If LFTs are significantly raised prior to refeeding it may indicate organ failure. This is usually not seen until BMI is <12. It should improve with refeeding. An ultrasound will show an enlarged liver in refeeding (ie fatty liver) whereas it will be small in a starvation induced hepatitis.

If the clinical picture is not suggestive of this – investigate for other causes of hepatitis. Note: Don’t forget that alcohol abuse can often be co-morbid in ED and is linked with higher medical risks and complications.

Pancreatitis
Rare but may occur during refeeding or binge eating. Abdominal examination and check amylase and lipase. Bear in mind amylase may be raised in purging - levels should be at least 3 fold above normal to indicate acute pancreatitis.

Renal Impairment / Acute Renal Failure
Reduced oral intake, purging and laxative abuse can all induce hypovolaemia and dehydration, with resultant risk of pre-renal failure and potentially acute kidney injury, which can be irreversible. Advice from a renal physician or specialist physician experienced in the management of physical complications of eating disorders is recommended.

During refeeding and prolonged periods of nasogastric and / or high calorie nutritional restoration, specialist dietetic advice is recommended to prevent excessive protein load to the kidneys.

Endocrine/hormonal changes
Many endocrinological changes are seen in eating disorders, especially anorexia nervosa, some of which are benign and others which can cause chronic irreversible problems such as osteoporosis and infertility.

Amenorrhea and Pregnancy
- Although no longer part of the diagnostic criteria for AN, amenorrhoea occurs in the majority of AN patients and up to 40% of patients with BN.
- Results from low levels of GnRH, LH, FSH and oestrogen.
- Essentially it is a “hypothalamic amenorrhoea syndrome” in which there is inhibition of the hypothalamic secretion of GnRH leading to inadequate LH and FSH to stimulate menstruation.
- It can occur before significant weight loss in some, and not until severe weight loss in others, but overall is most strongly correlated with loss of body weight.
- Amenorrhoea has affects long term on both fertility and bone density.
There are studies to suggest that people with a history of AN have a higher risk of infertility or difficulties conceiving even after recovery.

- Important to note that cessation of menstruation in these patients does not always stop ovulation and some may still fall pregnant.
- If patients do fall pregnant, they are at higher risk of pregnancy and neonatal complications including miscarriage.

Management: with nutrition/refeeding.
- At this stage, there is no evidence for clinical benefit with oestrogen replacement in these patients.
- Oral contraceptives used for withdrawal bleeds may only serve to provide false reassurance of improving health which may hinder motivation and weight gain, therefore they should only be used when contraception is needed.

**Thyroid hormones**
Abnormalities in AN patients usually resemble that of the euthyroid sick syndrome with decreased T3 and T4 but TSH usually remains normal. This is reversible with refeeding and weight gain.

Management
- Predominantly expectant
  - Re-check 3-6 months’ time depending on weight gain
- Treating unnecessarily with thyroid hormone replacement may not only cause harm in reducing weight gain efforts, but also reduce bone density.
- If there is concern about co-morbid thyroid disease then seek endocrine opinion.
- Thyroid tests will often have been performed on routine bloods before admission to rule out thyroid causes of weight loss or psychiatric symptoms.

**Growth Hormone (GH):** raised – probably to mobilise fat tissue to combat hypoglycaemia. The clinical significance of this is not yet clear.

**Cortisol:** is also increased. These patients will often have blunted dexamethasone suppression tests. The clinical significance of this is unknown, but may contribute to reduced bone mineral density in AN patients.

**Musculoskeletal**

- One of the most serious medical complications occurring in AN is loss of bone density with up to 85% of women with AN having either osteopenia or osteoporosis.
- Those who develop anorexia nervosa as a child or adolescent are particularly at risk given that accrual of bone formation occurs only through to late 20’s and thus they may never reach normal peak bone mass.
- This leads to high risk of bone fractures, delayed or non-union of fractures, potentially early invalidism, increased hospitalisations and increased suffering in these patients.
- The aetiology is likely due to several mechanisms that both increased bone resorption and/or decreased bone formation.
- The most significant risk factor is thought to be oestrogen deficiency reflected by amenorrhea, producing a picture similar to that seen in post-menopausal women.
Testosterone is also thought to play a role with male AN patients also having significantly reduced BMDs – that in some studies have been worse than that seen in females with AN.

Other factors thought to play a role include elevated GH and IGF-1, reduced body mass, hyperactivity and excess malnutrition.

Diagnosis is generally done by DEXA scan of BMD with:
- Osteopenia defined as T score between -1 and -2.5
- Osteoporosis T score of 2.5 standard deviations or more below the mean bone mass density of a healthy adult.

Management:

- Routine bloods may indicate other causes for bone loss (eg liver failure) and should be further investigated if found.
- Check for medications that may worsen bone density (eg long term glucocorticoids)
- DEXA should be done if 6 months of being underweight with or without amenorrhea
- It is important to note the lowest score at each anatomical site measured (eg lumbar spine or pelvis) to gain an understanding of overall bone health, rather than the global score given on the report.
- DEXA should be repeated every 2 years of ongoing difficulty with eating disorder
- As of yet, studies looking at the role of pharmacotherapy for treatment or prevention of osteoporosis in ED patients have been either lacking, unsuccessful or inconclusive and thus at this stage, medications are not used.
- Treatment is therefore aimed at refeeding and restoration of normal BMI and menses.

Neurological

Seizures

- Around 5% of ED patients are affected by seizures.
- Disturbances in glucose, calcium and sodium metabolism are all potential likely causes of seizures in this patient group.
- May present as myoclonic or generalised tonic-clonic seizures

Management

- Should be treated as medical emergency
- Call a MET call and commence ‘ABC’ approach
- Can start basic investigations to find cause such as BSL and baseline bloods
- Will need transfer back to the medical wards
- Other causes should also be investigated after stabilisation e.g., CT brain, EEG
Other Findings:

Cerebral Changes: Recent studies suggest that AN is associated with a degree of brain atrophy that may not be reversible. The changes are variable but likely significant, with severe cases appearing similar on MRI brain to that of a patient with Alzheimer’s dementia. Findings include decreased cortical substance and enlarged ventricles.

Q amazingly is often not diminished, however, as weight decreases, concentration becomes impaired and therefore the ability to sustain reasoning or make decisions deteriorates. This can be significant when considering the ED patients capacity to consent to treatments.

Neuromuscular: main symptoms include general muscle weakness, headaches and peripheral neuropathy. In severe cases, pulmonary function may be disturbed due to impairment of diaphragmatic contractility. Check B12/folate when case of suspected peripheral neuropathy.

Cognitive Changes: starvation can lead to predictable changes in cognition as seen in Ancel Keys starvation study. This includes obsessive ruminations about food, rigid thinking, anxiety, depression and intense fear of weight gain. This tends to reverse with nutrition with patients demonstrating improved flexibility of thinking and insight.

Dental/oral cavity

- With repeated vomiting – several abnormalities can be seen in the oral cavity.
- Repeated acid exposure damages the enamel of the teeth leading to its loss or decay. This is irreversible, and can lead to cracking or breaking of the teeth
- Other complications include gingivitis, periodontal disease and xerostomia (dry mouth).
- Erosions may be apparent as early as 6 months after onset of regular self-induced vomiting.

Management:

- Specific education should be given to patients about their dental care:
  - Advise to not brush immediately after vomiting (acid softens the enamel for 30mins after purging and brushing can cause further abrasion). Instead instruct to rinse mouth with water or a mixture of water and baking soda to neutralise the acid.
  - Outside of this time, a high fluoride toothpaste or mouthwash should be used to reduce decay.
  - General mouth rinses provide no benefit.
  - Floss and brush teeth twice daily
  - Stay hydrated to maintain good saliva levels
- A non-urgent dental appointment should be made in patients thought to be at risk
- An OPG (Orthopantomogram) will need to be performed prior to dental appointment

Other Findings:

Sialadenosis: hypertrophy of the salivary glands occurs in chronic vomiting. The bilateral parotids are the most commonly involved, but submandibular swelling may also be seen creating a “chipmunk-like” facies around 3-4 days after cessation of vomiting. Can be treated with low-dose anti-inflammatory’s (eg ibuprofen) short term as PRN if causing discomfort or pain.
Cutaneous changes

- Skin changes can be seen as a result of declining weight and nutrition, as well as secondary to compensatory behaviours.
- With decreasing weight comes dry skin sometimes with fissures; lanugo (fine downy hair seen on the side of the face and down the spine), and loss of subcutaneous tissue predisposes to easy bruising and decubitus ulcers.
- Repeated self-induced vomiting by repetitively inserting the fingers into the mouth can also cause abrasions to the hand called “Russell’s sign”

Management:

- In most cases – only treatment will be with nutrition
- Barrier creams for dry and cracking skin
- May need to consider pressure mattress in severe malnutrition if ulcers are present.
- If skin changes appear due to other causes, seek dermatology opinion.

Management on Admission

Investigations: Ensure they have had baseline bloods as well as TFTs checked, b12/folate and Iron studies also done. An ECG should also have been done and reviewed.

Obtain recent or pertinent investigations from the GP (eg previous ECG for baseline, last BMD scans, investigations done for other causes of weight loss) or other results depending on the medical history.

Medications: if not already done so – ensure as a minimum they have been stated on:
Thiamine 300mg for 3 days then 100mg daily
  - unless at high risk of Wernicke’s encephalopathy – then consider initial 3 days be given as IMI
  - thiamine should always be given before any glucose preparations
Multivitamin daily

Monitoring

After admission to the unit, unless otherwise stated by the team, all eating disorder patients are commenced on daily monitoring with:

- Vitals: initially QID including postural BP and HR
- BSLs: QID and at 0200hours
- Daily bloods: FBC, UECs, Ca/Mg/PO4, LFTs
- ECG

Your job will be checking these daily – and responding to any abnormalities

Progress

Every week, a ward round will be held with the entire “Eating Disorder Team”.


It is here that the majority of decisions are made such as changes to meal plans, frequency of physical monitoring required (e.g., twice weekly instead of weekly bloods), leave from the ward, whether bathroom visits need to be supervised etc.

It is important that these decisions are made as a team to ensure consistency and reduce the chance for ‘splitting’ of team members. If you are being asked by patients to reduce how frequently they are having bloods done for example – it is best to answer that “it will be discussed at the next ward round”.

Your role will be to inform the team at the ward round about the patient’s medical status in regards to the monitoring being done, and how it has progressed since the last ward round (eg “neutrophils are trending up”, “BP is stable”), any new medical concerns that may have arisen, and the results of any recent investigations or referrals made.

As a team, a decision will then be made on what ongoing monitoring is required depending on the patient’s physical status, weight gains, co-operation with meals and any compensatory behaviours that may be occurring.
### Appendix 1. Indicators for consideration for psychiatric and medical admission for adults

**RANZCP Clinical Practice Guidelines for the Treatment of Eating Disorders (2014)**

<table>
<thead>
<tr>
<th>Psychiatric or Medical Admission * Indicated (level of acuity can usually be managed in either setting)</th>
<th>Acute Medical Admission * Is Required (level of acuity usually requires a medical ward)</th>
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<tr>
<td><strong>Rapid weight loss</strong></td>
<td>Rapid weight loss (i.e. 1kg/week over several weeks) or grossly inadequate nutritional intake (&lt;100kcal daily) or continued weight loss despite community treatment</td>
</tr>
<tr>
<td><strong>Re-feeding Risk</strong></td>
<td>High (if markers below are present)</td>
</tr>
<tr>
<td><strong>Systolic BP</strong></td>
<td>&lt;90 mmHg</td>
</tr>
<tr>
<td><strong>Postural BP</strong></td>
<td>&gt;10 mmHg drop with standing</td>
</tr>
<tr>
<td><strong>Heart rate</strong></td>
<td>&gt;10 mmHg drop with standing</td>
</tr>
<tr>
<td><strong>Temperature</strong></td>
<td>&lt;35.5°C or cold/blue extremities</td>
</tr>
<tr>
<td><strong>12-lead ECG</strong></td>
<td>Any arrhythmia including QTc prolongation, nonspecific ST or T-wave changes including inversion or biphasic waves</td>
</tr>
<tr>
<td><strong>Blood sugar</strong></td>
<td>Below normal range*</td>
</tr>
<tr>
<td><strong>Sodium</strong></td>
<td>&lt;130 mmol/L*</td>
</tr>
<tr>
<td><strong>Potassium</strong></td>
<td>Below normal range*</td>
</tr>
<tr>
<td><strong>Magnesium</strong></td>
<td>Below normal range*</td>
</tr>
<tr>
<td><strong>Phosphate</strong></td>
<td>Below normal range*</td>
</tr>
<tr>
<td><strong>Albumin</strong></td>
<td>Below normal range</td>
</tr>
<tr>
<td><strong>Liver enzymes</strong></td>
<td>Mildly elevated</td>
</tr>
<tr>
<td><strong>Neutrophils</strong></td>
<td>&lt;1.5 × 10⁹/L</td>
</tr>
<tr>
<td><strong>eGFR</strong></td>
<td>&lt;60ml/min/1.73m² or rapidly dropping (25% drop within a week)</td>
</tr>
<tr>
<td><strong>Weight</strong></td>
<td>Body Mass Index (BMI) &lt;16kg/m²*** BMI &lt;14kg/m² (&gt; 85% ideal body wt 16-18yrs)</td>
</tr>
<tr>
<td><strong>Risk assessment</strong></td>
<td>Suicidal ideation; Active self-harm; Moderate to high agitation and distress</td>
</tr>
<tr>
<td><strong>Severe ED symptoms</strong></td>
<td>Bulimia Nervosa with hypokalaemiaand/or without control of vomiting; Vomiting &gt;4 times daily</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td>Not responding to outpatient treatment</td>
</tr>
</tbody>
</table>

Starvation Syndrome can occur at any weight therefore weight should not be the deciding factor for admission location or use of the Mental Health Act.

* Patients who are not as unwell as indicated above may still require admission to a psychiatric or other inpatient facility.
* Medical admission refers to admission to a medical ward, short stay medical assessment unit or similar.

*Any biochemical abnormality which has not responded to adequate replacement within the first 24 hours of admission should be reviewed by a medical registrar urgently

** This additional information is taken from NSW Health/CEDD Guideline for Inpatient Management of Eating Disorders in General Medical and Psychiatric Setting in NSW (2014).
References and Resources:


It is also recommended you familiarize yourself with the eating disorder patient handout including leave guidelines.