The South Island Eating Disorders Service

The A-B-C of Eating Disorders

Dr Roger Morgan
Psychiatrist
Princess Margaret Hospital
Christchurch
What are we dealing with?

Eating disorders are the 3rd most common disease of young women

High mortality

High physical morbidity

High psychiatric co-morbidity

Eating disorders are a group of serious conditions in which you're so preoccupied with food and weight that you can often focus on little else.  

Mayo Clinic

The assessment of eating disordered patients may reveal that they look sick, but they are often much worse.
What are we dealing with?

We often deal with:-

- Severe starvation/malnutrition
- Multiple vitamin mineral and protein deficiencies
- Loss of fat
- Loss of glycogen
- Bulimia associated with multiple metabolic and cardiac problems

These clinical findings are not necessarily reflected in laboratory tests.
Classification (DSM IV)

- Anorexia Nervosa
- Bulimia Nervosa
- Eating Disorder NOS (EDNOS)
- Binge Eating Disorder (in DSM V)
Classification

• Anorexia Nervosa

  Significant loss of body weight

  Intense fear of gaining weight

  Body image disturbance

  Amenorrhea (excluded in DSM V)

  Restricting, Binge-eating/Purging subtypes

  Cognitive inflexibility as prevalent thinking style
Classification

- Bulimia Nervosa
  
  Recurrent episodes of binge eating
  
  Recurrent compensatory behaviour
  
  Distinct sense of loss of control
  
  Frequency 2/week for 3 months
  
  Self-evaluation influenced by shape/weight
  
  AN excluded
  
  Purging/non-purging subtypes
Classification

- Eating Disorder NOS (FEDNEC: Feeding or Eating Disorder Not elsewhere classified)

  Meets criteria for AN and/or BN **BUT**

  Females have menses (excluded in DSM V)

  May have significant weight loss but weight is in the normal range

  Binging and purging occur less frequently than in BN
Other Types of Clinical Eating Disturbance

- Binge Eating Disorder
  
  Very common, 3.5% females, 2% males.
  
  Does not exercise control over consumption of food.
  
  Feels loss of control over eating during binge.
  
  Eats when depressed or bored.
  
  Eats large amounts of food even when not really hungry.
  
  Feels disgusted, depressed, or guilty after binge eating.
  
  Experiences rapid weight gain/sudden onset of obesity.
Other Types of Clinical Eating Disturbance

- Generally seen in childhood:
  - Pervasive refusal syndrome
  - Food avoidance emotional disorder
  - Functional dysphagia /food phobia
  - Selective eating/extreme faddiness
  - Restrictive eating/poor appetite
Demographics

Anorexia Nervosa

- Lifetime prevalence for women 0.5%
- Point prevalence 15-19 years 0.5%
- Incidence 20/100,000 females/yr
- 10% of presentations are males
- Younger onset
- 20% mortality
Demographics

Bulimia Nervosa

- Lifetime for women 1-3%
- Point prevalence 1% young women
- Incidence 30/100,000 females/yr
- Rapid increase in diagnosis since described in 1979
- Thought to be a culturally bound syndrome (inaccurate)
- Now stable or declining rates but local experience shows an increase in male presentations
Demographics

Eating Disorder NOS

Point prevalence young women 3-5%

More than half of clinical samples

Virtually unstudied
**Prognostic Factors**

**Good**

- young age/first episode/short duration
- relatively preserved body weight
- intact family
- established other roles
- absence of co-morbidity
Prognostic factors

Bad

purging anorexia

chronicity > 6 years

alcohol and drug abuse/dependence

psychiatric co-morbidity, especially personality disorder

unrelenting lack of insight
Morbidity

It is timely to remember that patients with Anorexia Nervosa have a 10 fold risk of death compared with healthy controls.

A 50 times risk of death with concurrent type 1 diabetes or alcohol dependence

A 20% mortality at 20 years

Causes of death

Complications of Anorexia Nervosa – malnutrition, methods of weight control 54%

Suicide 27%

Other/unknown 19%
Morbidity

Specific causes of death

Rarely documented
Dehydration
Electrolyte disturbance
Metabolic complications (renal, hepatic)
Infections (bronchopneumonia, sepsis)
Cardiac complications
Rupture of the GI tract
Pathogenesis

Eating disorders are undoubtedly complex and multi-factorial disorders.

With AN, theories abound implicating social and cultural attitudes, genetic endowment, trauma, and family and interpersonal relationships.

Research has shown that there is a familial aggregation of eating disorders phenotypes:-

- Anorexia 48 – 74%
- Bulimia 54 – 83%
- BED 57%
Pathogenesis

ED symptoms are heritable (46 – 72%) :-

- Dietary restraint
- Binge eating
- Self-induced vomiting

Attitudinal symptoms of eating and weight, weight concerns, weight pre-occupation, and body dissatisfaction have heritabilities of between 32 -72%.

It is clear that an individual’s genotype plays a part in behaviours seen in eating disorders and that eating disorders are brain disorders.
A summary of findings from neuroscience research on eating disorders and the brain

Structural changes

One of the major tenets of a neuroscience approach to eating disorders is that starving the body will also starve the brain.

CT findings in AN and starvation show :-

- dilated ventricles
- widened sulci
- widening of the inter-hemispheric fissure

All indicating an overall reduction in brain volume.
A summary of findings from neuroscience research on eating disorders and the brain

Structural changes:

There is also a reduction in:

- neuronal cell numbers
- the number of synaptic connections

This may account for:

- Increased cognitive inflexibility
- Poor attentional functioning

“Sticky brain” readily describes what is seen clinically
A summary of findings from neuroscience research on eating disorders and the brain

Structural changes:

Cerebral atrophy does not seem to have severe consequences for the neuropsychological or psychopathological status of patients with eating disorders.

On the negative side, CT scans on recovered AN patients showed a persistence of grey matter volume deficits.

This suggests that there is an irreversible component to structural brain changes.
Risk Factors for AN and BN

Female (only 10% cases are male)

Adolescent/young adult

Western culture

Dieting

Family history of

eating disorder

depression

obesity (AN and BN)
Individual Characteristics

Low self-esteem (AN / BN)
Perfectionism (AN)
High achievement (AN)
Over-compliance (AN)
Excessive exercise (AN / BN)
OCD/OCPD traits (AN)
Anxiety (AN / BN)
Diagnosing Eating Disorders

Cues to Anorexia nervosa

- Unexplained weight loss
- Failure to gain weight in proportion to height
- Secondary amenorrhea
- Bradycardia
- Hypotension
Diagnosing Eating Disorders

Cues to Anorexia nervosa

- Hypothermia
- Peripheral cyanosis
- Lanugo hair, brittle hair, hair loss
- Hypercarotenemia
- Preoccupation with additional weight loss despite thinness
Diagnosing Eating Disorders

Cues to Binge-Purge behaviour

- Swollen or tender parotid glands
- Dental enamel erosion / many new caries
- Calloused scarred area on back of hand
- Yo-yo weight pattern
- Hypokalemia
Diagnosing Eating Disorders

The SCOFF questionnaire.

Do you make yourself Sick because you feel uncomfortably full?

Do you worry you have lost Control over how much you eat?

Have you recently lost more than One stone in a 3 month period?

Do you believe yourself to be Fat when others say you are too thin?

Would you say that Food dominates your life?

One point for every "yes"; a score of >=2 indicates a likely case of anorexia nervosa or bulimia BMJ. 1999 Dec 4;319(7223):1467-8.
Physical complications and management

Metabolic abnormalities

Metabolic alkalosis
Hypokalemia
Hypochloremia
Hyponatremia
Hypomagnesimia

Monitor -> HCO₃, K+, Na+, Cl-, Mg++
Physical complications and management

Hypokalemia (muscle weakness, fatigue, constipation)

Regular monitoring of K+ if purging > 3 times per day. More so if combined with laxative abuse.

If K+ < 3.0 mmol/l -> daily monitoring initially with ECG

Treatment -> Oral K+, Slow K 1-2 tabs tds with fluids

If K+ < 2.5 mmol/l or there has been a rapid drop in levels, or progression of ECG changes -> ED dept / hospitalise.
Physical complications and management

Constipation and laxative abuse

Constipation is due to low calorie intake, chronic laxative abuse, hypokalemia

Constipation is aggravated by chronic use of stimulant laxatives (Bisacodyl, Senna, etc)

Treatment -> Education

Switch from stimulant laxatives to non-stimulant varieties (Metamucil, fibre, fluids)

Then no laxatives
Physical complications and management

Cardiovascular complications

Small heart, hypotrophic muscle

Fatigue, decreased exercise tolerance

Hypotension

Bradycardia

Cardiac arrhythmia (increased QTc, electrolytes)

Hypothermia
Physical complications and management

Cardiovascular complications

Cardiac risk increased by ->

Severe and or rapid weight loss

High purge frequency

Drastic re-feeding and rehydration

Excessive exercise
Physical complications and management

Cardiovascular complications

Treatment ->

Normalise electrolyte imbalances

Weight restoration

Refer severe cases to Cardiologist

Avoid drugs causing QTc prolongation

tricyclic antidepressants
antipsychotics
cisapride

Restrict activities
Physical complications and management

Osteoporosis

Due to low oestrogen, high hydrocortisone

When early onset of amenorrhea, peak bone mass won’t be achieved

Diagnosis -> DEXA bone scan

Treatment -> Weight restoration

   Calcium supplement 1g daily
   Cholecalciferol 1.25mg month
   ? Oestrogen / Progesterone replacement
Physical complications

Medical indications to consider hospitalisation

Severe malnutrition (BMI < 13) (Mass in Kg / Height in M squared)

Rapid weight loss (> 4kg in 6 weeks)

Severe dehydration

K+ below 2.5 – 3.0 mmol/l

Prolonged QTc interval (>450 msec)

Dysrhythmias
Physical complications

Medical indications to consider hospitalisation

- Hypothermia  (< 35.5 C)
- Bradycardia  (< 40 b/m)
- Pulse differential  ( > 30 b/m)
- Rapidly diminishing exercise tolerance
- Frequent exercise induced chest pain
- Oligouria  (< 400 ml/day)
- Low phosphate during initial re-feeding
Treatment

Bulimia Nervosa/ BED:

- Good evidence to support the use of CBT (16-20 sessions)
- IPT could be used as an alternative to CBT
- SSRI’s

Anorexia Nervosa:

- Limited evidence base
  - Some evidence for olanzapine
  - Range of drugs may be used in the treatment of co-morbid conditions.
- Limited evidence for therapy **Except FBT in those under 18**

Adults *without* chronic conditions

- Consider CBT and IPT, accompanied by regular monitoring of patient’s physical state.
- Aim: reduce risk; encourage weight gain, reduce eating disorder cognitions and to facilitate psychological and physical recovery
In Summary:

Patients with eating disorders present complex challenges.

Treatment involves careful monitoring of physical parameters.

Psychological support is needed.

**Weight gain must always be a priority.**