North East Region Eating Disorders Taskforce

Pathways to care & Care planning and coordination

Austin Health

ST VINCENT'S HEALTH AUSTRALIA

CEED THE VICTORIAN CENTRE OF EXCELLENCE IN EATING DISORDERS

eating disorders victoria

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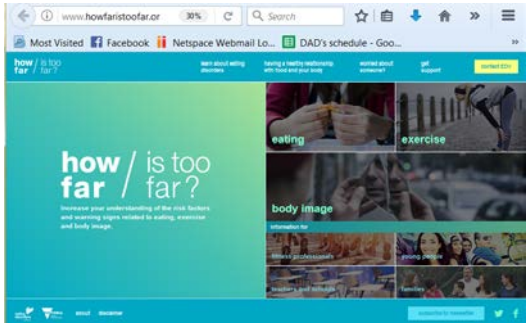
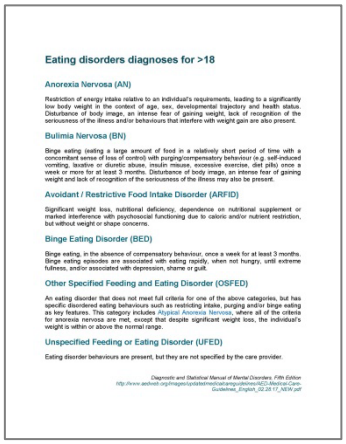


Definition of terms document



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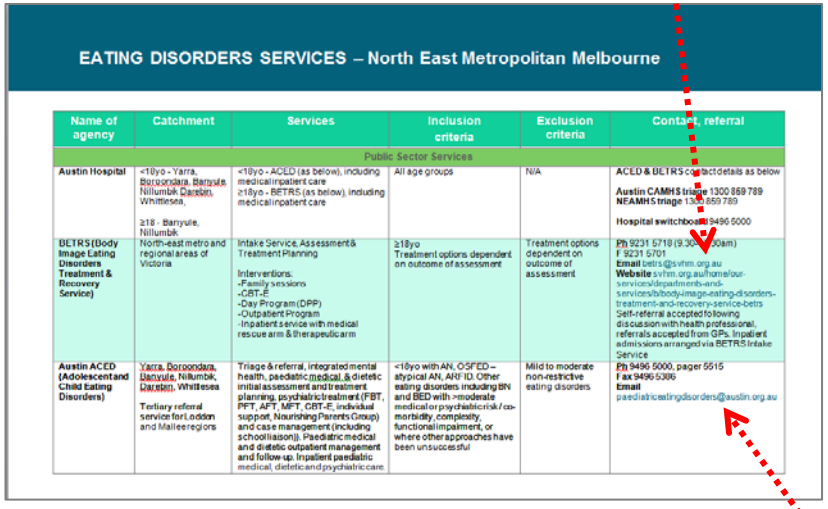

Email links

CLINICAL CARE AND REFERRAL PATHWAY

Children and adolescents under 18 years with Anorexia Nervosa

(& OSFED - Atypical Anorexia Nervosa, ARFID, complex eating disorders) ➔

North East Metropolitan Melbourne – Austin CAMHS Catchment

Regional Specialist Service: AUSTIN ACED (<18 yrs)
(Austin Adolescent & Child Eating Disorders - community & inpatient care)

LGA Catchment: Yarra, Boroondara, Banyule, Nillumbik, Darebin, Whittlesea
Service Level: AN treatment requires access to level 3 & 4 tier services

ACED (Austin Adolescent & Child Eating Disorders)

ACED nurse via switch 9496 5000, pager 5515, M-F 9.00-17.00
paediatriceatingdisorders@austin.org.au
F 9496 5386

Guidance for clinical evaluation ➔
Criteria for hospitalisation ➔
Referral information to service ➔

EMERGENCY
Medical Care

Patient requires immediate action to ensure access to medical resuscitation and stabilisation

Austin Hospital

Emergency Department
via 9496 5000

IP: tl-ŜŘĂl-úŊĂŎ ² l-ŊŘ н ² Ŝăú
ØĂl- K2ăLJĂúl-f ăøĂúŎK фпфс рллл
ó! /95 ƴdzŊăŜ LJl-ƎŜŊ ррмр ĂƴK2dzŊăΣ
2ƴŎl-ff LJl-ŜŘĂl-úŊĂŎĂl-ƴl-ŦúŜŊ K2dzŊăú

EMERGENCY
Mental Health Care

Patient requires immediate action to ensure access to mental health crisis management

Austin Hospital

tl-ŜŘĂl-úŊĂŎ Emergency Department
via 9496 5000

Austin CAMHS Triage
1300 859 789

IP: Statewide Child Inpt Unit (≤12yo)
Adolescent Inpatient Unit (13-17yo)

GP CLINICAL RESOURCES

Care and Recovery Plan
Risk/Safety Plan

Growth centile charts ➔
ViCTOR age specific observation charts ➔
RANZCP Guidelines ➔
NEDC GP Resources ➔
RACGP Resources ➔
GP eLearning Module by EDV & RACGP ➔
AED A Guide to Medical Care ➔
CEED Case Consultations ➔

HELPLINES

Butterfly Foundation
ED Hope Support Line (for patients & carers)
1800 334 673
8am-9pm Mon–Fri
EDV
ED Helpline (for patients, carers & professionals)
1300 550 236
9.30am-5pm Mon-Fri

CARER & FAMILY RESOURCES

Carer/Family Support Plan
Feed Your Instinct ➔
Butterfly Foundation ➔
EDV, How far is too far? ➔
NEDC ➔
FEAST ➔

MULTI-DISCIPLINARY ASSESSMENT

Austin ACED Multidisciplinary Initial Assessment Clinic

Private clinicians

MENTAL HEALTH MANAGEMENT

Specialist treatment, psychosocial support, case management (including school liaison)

Austin ACED, CAMHS
via 1300 859 789

Private mental health services

Other community services

MEDICAL & DIETETIC MANAGEMENT

Specialist treatment and monitoring.

Austin ACED
via ACED nurse via switch 9496 5000, pager 5515, M-F 9.00-17.00

GPs, private paediatricians
Private dietitians

CARE COORDINATION

Care Coordination (with link)
Patient / family & other carers / school
Mental health mx and psychosocial support
Nutritional rehabilitation
Medical management and monitoring

CLINICAL CARE AND REFERRAL PATHWAY

Adults over 18 years with Anorexia Nervosa

(& OSFED - Atypical Anorexia Nervosa, ARFID, complex eating disorders) ➔

North East Metropolitan Melbourne – North East (NEAMHS) Catchment

Regional Specialist Service: BETRS (≥18 yrs)
(Body Image Eating Disorders Treatment and Recovery Service - community, day program & inpatient care)

LGA Catchment: Banyule, Nillumbik
Service Level: AN treatment requires access to level 3 & 4 tier services

BETRS Clinical Intake service
9231 5718 M-F 9.30-11.30
betrs@svha.org.au
F 9231 5701

Guidance for clinical evaluation ➔
Criteria for hospitalisation
Referral information to service ➔

EMERGENCY
Medical Care

Patient requires immediate action to ensure access to medical resuscitation and stabilisation

Austin Hospital
Emergency Department
via 9496 5000
IP: General Medical Ward
BETRS Inpatient Unit via 1300 859 789
or 9231 5718

EMERGENCY
Mental Health Care

Patient requires immediate action to ensure access to mental health crisis management

Austin Hospital
Emergency Department
via 9496 5000
NEAMHS Triage
1300 859 789
IP: Acute Psychiatry Unit via 1300 859 789
BETRS inpatient Unit via 1300 859 789
or 9231 5718

MULTI-DISCIPLINARY ASSESSMENT

BETRS Assessment and Treatment Planning Service
Private clinicians

MENTAL HEALTH MANAGEMENT

Specialist treatment, psychosocial support, case management.

BETRS
NEAMHS psychiatry triage
1300 859 789
Private mental health services
Community health services

MEDICAL & DIETETIC MANAGEMENT

Specialist treatment and monitoring.

GPs
Physicians
BETRS dietitian, private dietitians
Community health services

CARE COORDINATION

Care Coordination (with link)
Patient / family & other carers
Mental health mx and psychosocial support
Nutritional rehabilitation
Medical management and monitoring

GP CLINICAL RESOURCES

Care and Recovery Plan
Risk/Safety Plan
Advanced Care Plan

BMI Centile Charts ➔
RANZCP Guidelines ➔
NEDC GP Resources ➔
RACGP Resources ➔
GP eLearning Module by EDV & RACGP ➔
AED A Guide to Medical Care ➔
CEED Case Consultations ➔

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EDV
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9.30am-5pm Mon-Fri

CARER & FAMILY RESOURCES

Carer/Family Support Plan
Feed Your Instinct ➔
Reach Out and Recover ➔
Butterfly Foundation ➔
EDV, How far is too far? ➔
NEDC ➔
FEAST ➔

CLINICAL CARE AND REFERRAL PATHWAY

Adults over 18 years with Anorexia Nervosa

(& OSFED - Atypical Anorexia Nervosa, ARFID, complex eating disorders) ➔

North East Metropolitan Melbourne – Inner Urban East (IUEAMHS) Catchment

Regional Specialist Service: BETRS (≥18 yrs)
(Body Image Eating Disorders Treatment and Recovery Service - community, day program & inpatient care)

LGA Catchment: Yarra, Boroondara
Service Level: AN treatment requires access to level 3 & 4 tier services

BETRS Clinical Intake service
9231 5718 M-F 9.30-11.30
betrs@svha.org.au
F 9231 5701

Guidance for clinical evaluation ➔
Criteria for hospitalisation ➔
Referral information to service ➔

EMERGENCY
Medical Care

Patient requires immediate action to ensure access to medical resuscitation and stabilisation

St Vincent's Hospital
Emergency Department
via 9231 2211
IP: General Medical Ward
BETRS Inpatient Unit via 1300 859 789
or 9231 5718

EMERGENCY
Mental Health Care

Patient requires immediate action to ensure access to mental health crisis management

St Vincent's Hospital
Emergency Department
via 9231 2211
IUEAMHS Triage
1300 558 862
IP: Acute Psychiatry Unit via 1300 859 789
BETRS inpatient Unit via 1300 859 789
or 9231 5718

GP CLINICAL RESOURCES

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MULTI-DISCIPLINARY ASSESSMENT

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Private clinicians

MENTAL HEALTH MANAGEMENT

Specialist treatment, psychosocial support, case management.

BETRS
IUEAMHS psychiatry triage
9231 2211
Private mental health services
Community health services

MEDICAL & DIETETIC MANAGEMENT

Specialist treatment and monitoring.

GPs
Physicians
BETRS dietitian, private dietitians
Community health services

CARE COORDINATION

Care Coordination (with link)
Patient / family & other carers
Mental health mx and psychosocial support
Nutritional rehabilitation
Medical management and monitoring

CLINICAL CARE AND REFERRAL PATHWAY

Adults over 18 years with Anorexia Nervosa

(& OSFED - Atypical Anorexia Nervosa, ARFID, complex eating disorders) ➔

North East Metropolitan Melbourne – Northern (NAMHS) Catchment

Regional Specialist Service: BETRS (≥18 yrs)
(Body Image Eating Disorders Treatment and Recovery Service - community, day program & inpatient care)

LGA Catchment: Darebin, Whittlesea
Service Level: AN treatment requires access to level 3 & 4 tier services

BETRS Clinical Intake service
9231 5718 M-F 9.30-11.30
betrs@svha.org.au
F 9231 5701

Guidance for clinical evaluation ➔
Criteria for hospitalisation ➔
Referral information to service ➔

EMERGENCY
Medical Care

Patient requires immediate action to ensure access to medical resuscitation and stabilisation

Northern Hospital
Emergency Department
via 8405 8000
IP: General Medical Ward
BETRS Inpatient Unit via 1300 859 789
or 9231 5718

EMERGENCY
Mental Health Care

Patient requires immediate action to ensure access to mental health crisis management

Northern Hospital
Emergency Department
via 8405 8000
NAMHS Triage
1300 874 243
IP: Acute Psychiatry Unit via 1300 859 789
BETRS inpatient Unit via 1300 859 789
or 9231 5718

GP CLINICAL RESOURCES

Care and Recovery Plan
Risk/Safety Plan
Advanced Care Plan

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RANZCP Guidelines ➔
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FEAST ➔

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BETRS
NAMHS (1300 874 243)
Private mental health services
Community health services

MEDICAL & DIETETIC MANAGEMENT

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GPs
Physicians
BETRS dietitian, private dietitians
Community health services

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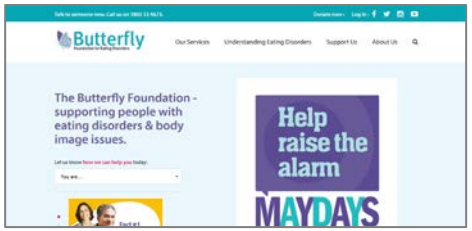
Mental health mx and psychosocial support
Nutritional rehabilitation
Medical management and monitoring

WEBSITE LINKS

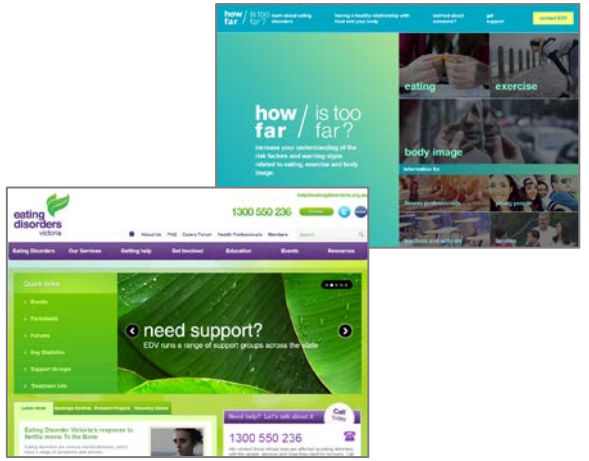
Feed your instinct
feedyourinstinct.com.au/

ROAR – Reach Out And Recover
reachoutandrecover.com.au/

Butterfly Foundation
thebutterflyfoundation.org.au/



EDV: eatingdisorders.org.au
How far is too far?
howfaristoofar.org.au/



NEDC
nedc.com.au/

FEAST
www.feast-ed.org/

MAP OF THE SYSTEM OF CARE FOR PATIENTS WITH EATING DISORDERS – North East Metropolitan Melbourne

Map of the system of care for patients with eating disorders - North East Metropolitan Melbourne (LGAs of Yarra, Boroondara, Banyule, Nillumbik, Darebin, Whittlesea)						
	Mild EDs	**Moderate EDs** e.g. BN/OSFED/UFED/BED with mild to mod medical risk, nil co-morbidity	**Severe EDs** e.g. AN, ARFID, BN/OSFED/UFED/BED with > moderate medical or psychiatric risk, co-morbidity, ED where other approaches have been unsuccessful			
				Community Treatment	**Day Program**	**Inpatient Care**
<18yo	• GP, consider paediatrician • Mental health interventions – including school welfare, Headspace • Community health / private dietitian • Involve family • Other – e.g. helplines, support groups, body image programs, guided self-help, books, online resources	• GP & paediatrician • Mental health Interventions - including school welfare, Headspace, private specialist clinicians • Private specialist dietitian • Involve family • Other – e.g. helplines, support groups, body image programs, guided self-help, books, online resources	**Mental Health**	ACED & Austin CAMHS Specialised private services	Monash Health Wellness and Recovery Centre Butterfly Day Program (12-24yo)	Austin child psychiatric inpatient unit (<13yo) Austin adolescent psychiatric inpatient unit (13-18yo)
			Dietetic **Medical**	ACED dietitian Private dietitian GP and paediatrician (ACED or private)		ACED dietitian Austin Hospital paediatric and adolescent med ward
18-24	• GP • Mental health interventions - including Headspace	• GP, consider physician • Mental health interventions - including Headspace, private	**Mental Health**	BETRS NEAMHS, NAMHS, IUEAMHS Specialised private services	BETRS Day Program Private services	BETRS inpatient eating disorders unit Private services

Map of the system of care for patients with eating disorders - North East Metropolitan Melbourne (LGAs of Yarra, Boroondara, Banyule, Nillumbik, Darebin, Whittlesea)						
	Mild EDs	**Moderate EDs** e.g. BN/OSFED/UFED/BED with mild to mod medical risk, nil co-morbidity	**Severe EDs** e.g. AN, ARFID, BN/OSFED/UFED/BED with > moderate medical or psychiatric risk, co-morbidity, ED where other approaches have been unsuccessful			
				Community Treatment	**Day Program**	**Inpatient Care**
18-24	• Community Health / private dietitian • Involve family • Other – e.g. helplines, support groups, body image programs, guided self-help, books, online resources	specialist clinicians • Private specialist eating disorders dietitian • Involve family • Other – e.g. helplines, support groups, body image programs, guided self-help, books, online resources	**Dietetic** **Medical**	BETRS dietitian Private dietitian GP, consider physician	BETRS dietitian GP	Inpatient dietitian BETRS inpatient eating disorders unit General medical ward (Austin, St Vincent's, TNH) Private services
25+	• GP • Mental health interventions • Community health / private dietitian • Involve family • Other – e.g. helplines, support groups, body image programs, guided self-help, books, online resources	• GP, consider physician • Mental health interventions – including private specialist clinicians • Private specialist eating disorders dietitian • Involve family • Other – e.g. helplines, support groups, body image programs, guided self-help, books, online resources	**Mental Health**	BETRS NEAMHS, NAMHS, IUEAMHS Specialised private services	BETRS Day Program Private services	BETRS inpatient eating disorders unit Private services
			Dietetic **Medical**	BETRS dietitian Private dietitian GP, consider physician	BETRS dietitian GP Inpatient dietitian	BETRS inpatient eating disorders unit General medical ward (Austin, St Vincent's, TNH) Private services

EATING DISORDERS SERVICES – North East Metropolitan Melbourne

Name of agency	Catchment	Services	Inclusion criteria	Exclusion criteria	Contact, referral
Public Sector Services					
Austin Hospital	<18yo - Yarra, Boroondara, Banyule, Nillumbik Darebin, Whittlesea, ≥18 - Banyule, Nillumbik	<18yo - ACED (as below), including medical inpatient care ≥18yo - BETRS (as below), including medical inpatient care	All age groups	N/A	**ACED & BETRS** contact details as below **Austin CAMHS triage** 1300 859 789 **NEAMHS triage** 1300 859 789 **Hospital switchboard** 9496 5000
BETRS (Body Image Eating Disorders Treatment & Recovery Service)	North-east metro and regional areas of Victoria	Intake Service, Assessment & Treatment Planning Interventions: • Family sessions • CBT-E • Day Program (DPP) • Outpatient Program • Inpatient service with medical rescue arm & therapeutic arm	≥18yo Treatment options dependent on outcome of assessment	Treatment options dependent on outcome of assessment	**Ph** 9231 5718 (9.30-11.30am) **F** 9231 5701 **Email** betrs@svhm.org.au **Website** www.betrs.org.au Self-referral accepted following discussion with health professional, referrals accepted from GPs. Inpatient admissions arranged via BETRS Intake Service
Austin ACED (Adolescent and Child Eating Disorders)	Yarra, Boroondara, Banyule, Nillumbik, Darebin, Whittlesea Tertiary referral service for Loddon and Mallee regions	Triage & referral, integrated mental health, paediatric medical & dietetic initial assessment and treatment planning, psychiatric treatment (FBT, PFT, AFT, MFT, CBT-E, individual support, Nourishing Parents Group) and case management (including school liaison). Paediatric medical and dietetic outpatient management and follow-up. Inpatient paediatric medical, dietetic and psychiatric care.	<18yo with AN, OSFED – atypical AN, ARFID. Other eating disorders including BN and BED with >moderate medical or psychiatric risk / co-morbidity, complexity, functional impairment, or where other approaches have been unsuccessful	Mild to moderate non-restrictive eating disorders	**Ph** 9496 5000, pager 5515 **Fax** 9496 5386 **Email** paediatriceatingdisorders@austin.org.au

Name of agency	Catchment	Services	Inclusion criteria	Exclusion criteria	Contact, referral
Austin CAMHS (Austin Child & Adolescent Mental Health Service)	Yarra, Boroondara, Banyule, Nillumbik, Darebin, Whittlesea	Triage, liaison and collaborative assessment, treatment planning and management with ACED and other specialist eating disorders services & GPs as indicated	<18yo	≥18yo	**Triage ph** 1300 859 789
NEAMHS (North East Area Mental Health Service)	Banyule, Nillumbik	Triage, assessment, referral and planning, treatment, liaison and collaborative management with GP and specialist eating disorders services as indicated, case management, consultation liaison psychiatry service, acute inpatient psychiatric care	≥18yo	<18yo	**Triage ph** 1300 859 789
St Vincent's Hospital & IUEAMHS (Inner Urban East Area Mental Health Service)	Yarra, Boroondara	Triage, assessment, referral and planning, treatment, liaison and collaborative management with GP and specialist eating disorders services as indicated, case management, consultation liaison psychiatry service, acute inpatient psychiatric care Medical inpatient care	≥18yo	<18yo	**Triage ph** 1300 558 862 **Hospital switchboard** 9231 2211
Northern Hospital & NAMHS (Northern Area Mental Health Service)	Darebin, Whittlesea	Triage, assessment, referral and planning, treatment, liaison and collaborative management with GP and specialist eating disorders services as indicated, case management, consultation liaison psychiatry service, acute inpatient psychiatric care Medical inpatient care	≥18yo	<18yo	**Triage ph** 1300 874 243 **Hospital switchboard** 8405 8000
Headspace Hawthorn	No catchment	Mental Health Services – psychologists, drug and alcohol services, Relationships Australia Victoria counsellor	Ages 12-25 years Mild to moderate presentations Early intervention	Outside of age range Medical instability Severe or crisis presentations	**Ph** 9006 6500 **Fax** 9815 0818 **Email** enquiries@headspacehawthorn.org.au Lvl 1, 360 Burwood Road, Hawthorn 3122

Name of agency	Catchment	Services	Inclusion criteria	Exclusion criteria	Contact, referral
Headspace Greensborough	No catchment	Mental Health Services: psychologists, counsellors, social workers, mental health nurses, drug and alcohol services, vocational training (education/employment), online counselling (eheadspace), youth programs	Ages 12-25 years Mild to moderate presentations Early intervention	Outside of age range Medical instability Severe or crisis presentations	**Ph** 9433 7200 **Fax** 9435 8621 **Email** headspacegreensborough@mindaustralia.org.au 78 Main Street, Greensborough 3088
Headspace Collingwood	No catchment	Mental Health Services: Better Access and ATAPS, intake and assessment team, psychologists, psychotherapists, counsellors, occupational therapists, social workers, Aboriginal and Torres Strait support worker, Youth Brief Intervention Service team (Austin), MIND worker, YSAS drug and alcohol services, employment support, group programs, accredited youth primary health clinic (GPs, practice nurse, practice manager), online counselling (eheadspace)	Ages 12-25 years Mild to moderate presentations Early intervention	Outside of age range Medical instability Severe or crisis presentations Medical instability	**Ph** 9417 0150 **Fax** 9416 3279 **Email** reception@collingwoodheadspace.org.au 16 Lulie Street, Abbotsford 3067
MIND	Yarra, Boroondara, Banyule, Nillumbik, Darebin, Whittlesea	PARCS (Prevention and Recovery Care Services), individual support, group programs, PIR (Partners in Recovery), Recovery College, Equality Clinic, in home respite	≥12yo pts with eating disorders of all levels of severity	<12yo	**Ph** 1300 286 463 **Website** www.mindaustralia.org.au
NEAMI Abbotsford & Brunswick	Yarra (& Melbourne, Moonee Valley and Moreland)	Community-based mental health and recovery support, support groups	16-64yrs Diagnosed mental illness that impacts on daily living	N/A	**Ph** NEAMI central intake 1300 379 462 Abbotsford 8679 9140 Brunswick 8383 2050 **Website** www.neaminational.org.au Cost: free
NEAMI Partners in Recovery (PIR)	Banyule, Nillumbik, Darebin	Service coordination for mental health support	Diagnosed mental illness, needs that require support from multiple services	Already receiving support with coordination	**Ph** 1300 747 247 (Mind Australia) PIR team 8459 8214 **Website** Neaminational.org.au Cost: free

Name of agency	Catchment	Services	Inclusion criteria	Exclusion criteria	Contact, referral
Northern Prevention and Recovery Care Service (PARCS)	Darebin, Whittlesea	Short term mental health support (up to 28 days) in a sub-acute residential setting	Currently receiving support from NAMHS or MHCSS	Residents are not to be drug or alcohol affected whilst on site	**Ph** 9470 3100 **Website** Neaminational.org.au Cost: free
The Bouverie Centre	N/A	Family therapy	Serious mental illness	Crisis presentation, current court proceedings, family / domestic violence	**Ph** 9385 5100 **Fax** 9381 0336 **Email** bouverie.centre@latrobe.edu.au
Mindful Moderate Eating Group, Hawthorn	N/A	Group program and individual counselling	≥18yo, women Patients with mild to severe eating disorders including BED	<18yo, men Patients with AN, problematic drug & alcohol abuse, major physical illness, severe suicidal ideation	**Ph** 9214 5528 **Email** psychprojects@swin.edu.au Swinburne Psychology Clinic Lvl 4, The George, 34 Wakefield Street, Hawthorn 3122
CHEW (Clinic for Healthy Eating and Weight), Australian Catholic University	N/A	CBT-E, OBE (overcoming binge eating)	≥18yo, physically stable with BMI ≥16.5, linked in with GP for medical monitoring	Significant co-morbidity including drug and alcohol abuse. Significant purging, hospitalisation within the last 2 years with inadequate weight restoration	**Ph** 9953 3006 **Email** melbournepsychologyclinic@acu.edu.au The Daniel Mannix Building, Australian Catholic University, Level 5, 17 Young Street, Fitzroy 3065
Boroondara Youth Services, Camberwell (360)	LGA	Information, advocacy, referral, advice, counselling, material aid, customized education sessions and outreach services for young people and parenting courses. 360 is also a 'drop in' space for young people to meet, hang out, utilize our computer lab (with WiFi access X-Box and Wii) and our soundproof band room	10-25 years with mild eating disorders	Outside of age range, moderate, severe & crisis presentations	**Ph** 9835 7824 360 Burwood Road, Hawthorn 3122
Camcare Camberwell	LGA	Counselling and wrap around services for families and carers.	All ages and severity, in partnership with other eating disorders services	N/A	**Ph** 9831 1900 **Fax** 9831 1999 51 St Johns Ave, Camberwell 3124

Name of agency	Catchment	Services	Inclusion criteria	Exclusion criteria	Contact, referral
Camcare Ashburton	LGA	Counselling and wrap around services for families and carers.	All ages and severity, in partnership with other eating disorders services	N/A	**Ph** 9809 9100 **Fax** 9809 9199 4Y Street, Ashburton 3147
Access Health & Community, Hawthorn, Ashburton, Richmond	LGA	Dietetics	Mild eating disorders of all ages.	Moderate, severe & crisis presentations	**Ph** 9885 6822 **Fax** 9818 6154
North Richmond Community Health	LGA	Dietetics and counselling	Mild eating disorders of all ages	Moderate, severe & crisis presentations	**Ph** 9418 9800 **Fax** 9428 2269 huongt@nrch.com.au
CoHealth – multiple centres inner north	LGA	Dietetics and counselling	Mild eating disorders of all ages	Moderate, severe & crisis presentations	**Ph** 8378 3500 (intake), 9948 5528 (Collingwood centre), 9948 5531 (Fitzroy centre) **Fax** 9374 2866
Banyule Community Health – West Heidelberg, Greensborough	LGA	Dietetics	Mild eating disorders of all ages	Moderate, severe & crisis presentations	**Ph** 9450 2000 21 Alamein Road, West Heidelberg 3081
healthAbility, Eltham	No catchment	Dietetics Mondays and Thursdays Cousellors, psychologist	Dietetics - disordered eating, binge eating disorder or bulimia nervosa that is medically stable (GP referral required) Mental health – mild to moderate eating disorders	Anorexia Nervosa, unstable bulimia nervosa, binge eating disorder or disordered eating	**Ph** 9430 9100 917 Main Road, Eltham 3095

Name of agency	Catchment	Services	Inclusion criteria	Exclusion criteria	Contact, referral
Darebin Community Health	Whittlesea, Darebin, Banyule, Nillumbik, Yarra (and Moreland & Hume)	Allied health including dietetics and counseling, medical services including GPs, mental health nursing	Mild eating disorders / disordered eating, such as chronic restrained eating, compulsive eating or habitual dieting Clients of all ages are welcome. Our priority population groups are: • Aboriginal and Torres Strait Islanders • Newly Arrived Refugees and Asylum Seekers • Children under 12 (with support to their parents or carers) • Adults aged 65 or over • People who are socioeconomically disadvantaged • People with chronic or complex conditions • People who live in unsafe or insecure environments • People living with a disability	Moderate to severe eating disorders; anorexia nervosa, binge eating disorder, bulimia nervosa, other specified feeding or eating disorder (OSFED), unspecified feeding or eating disorder (UFED)	**Ph** 8470 1111 **Fax** 8470 1107 **Email** info@dch.org.au **Web** www.dch.org.au Referrals can be made by clients, family/carers, health professionals, GPs. DCH has 4 sites that are located in Darebin: • 125 Blake Street, East Reservoir • 42 Separation Street, Northcote • PANCH: 300 Bell Street, Preston • East Reservoir Community Hub: 1/44 Whitelaw St, Reservoir Services are free for most priority population groups (including children 0-17 years with health care card/low-income, counseling for low- and medium income, and all DCH medical consultations). Other fees are mostly low cost and are based on household income.
Plenty Valley Community Health	LGA	Dietetics	Mild eating disorders in children <12years	Moderate, severe & crisis presentations ≥ 12 years	**Ph** 9716 9444 40-42 Walnut Street, Whittlesea 3757
School Welfare	N/A	Initial identification and referral, ongoing support and management	Attending school	Not attending school	N/A
GPs	N/A	Early identification, intervention and referral, ongoing management	N/A	N/A	N/A

Name of agency	Catchment	Services	Inclusion criteria	Exclusion criteria	Contact, referral
EDV (Eating Disorders Victoria)	N/A	Information, support, resources, community-based psychological treatment, referral, support groups	All ages, all ED presentations For psychology services must be clinically stable & willing to link in with medical & mental health support	N/A	**Ph** 1300 550 236 **Email** help@eatingdisorders.org.au Most services free or low-cost Medicare rebates available for therapy (with GP referral)
NEDC (National Eating Disorders Collaboration)	N/A	Online resources	N/A	N/A	**Website** www.nedc.com.au
The Butterfly Foundation	N/A	Butterfly National Helpline for sufferers and carers	N/A	N/A	**Ph** 1800 33 4673 **Email** support@thebutterflyfoundation.org.au
CEED (The Victorian Centre of Excellence in Eating Disorders)	N/A	Clinical consultation to public mental health and other eating disorders services, professional development, service development support, online resources	N/A	N/A	**Ph** 8387 2673 **Fax** 8387 2667 **Email** ceed@mh.org.au **Website** www.ceed.org.au
Private Sector Services					
Paediatricians	List of practitioners available via Austin ACED, BETRS & EDV				
Mental Health Clinicians and Services					
Dietitians					

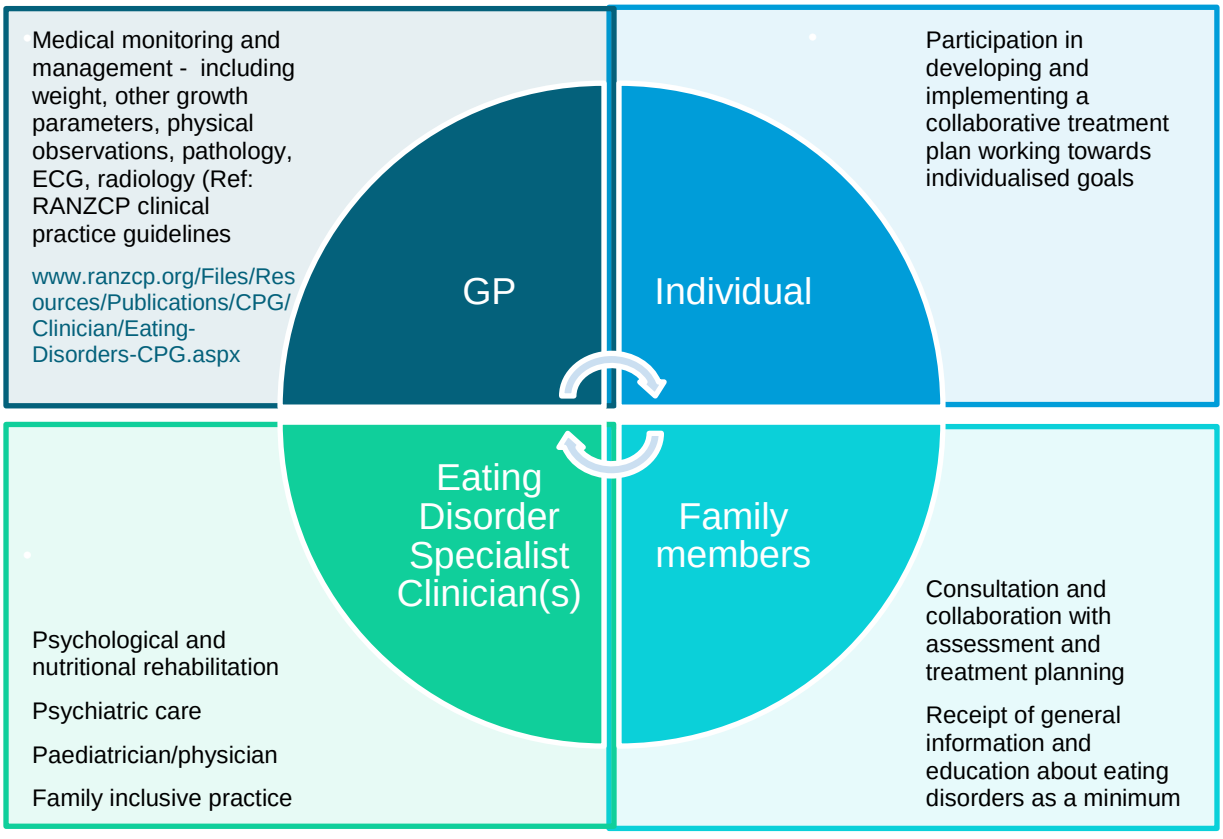
Who needs care co-ordination?

- People with a diagnosed eating disorder which is moderate to severe (e.g. BMI ≤ 17 in adults, <85% healthy BMI in adolescents, significant signs of medical risk)
- People who have more than one person in their team of care
- People with complex needs (E.g. multiple diagnoses, concern about acute psychiatric risk issues, limited psycho-social support etc.)
- People who have consented to the sharing of communication

What does good care co-ordination provide?

- The right care at the right time - a stepped care approach which is responsive to acuity
- A clear treatment plan outlining care team roles, aims and communication pathways
- Support for both the individual and the system around them - including families, carers and community supports
- A vision of recovery and the best possible outcome

Who should be involved and what is their role?

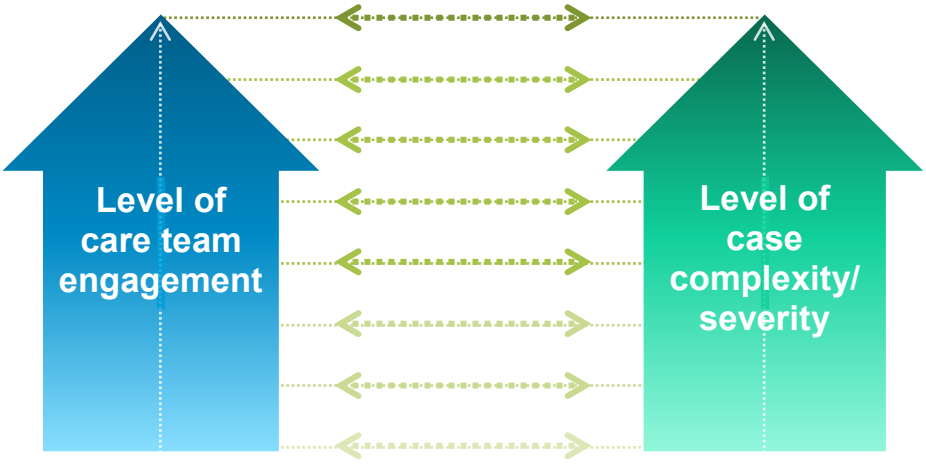


How does the care team communicate?

- A clear plan is developed with the team which includes the goals of treatment and the roles of each person involved. A copy is provided to the consumer, professionals and family, if consent is obtained.
- A written plan is developed for the management of both physical and psychiatric risk. This should include clear parameters for the escalation of care and appropriate pathways for this.
- A system is set up to enable regular communication on a weekly - monthly basis (dependent on acuity/ complexity). This may include email, face to face meetings or tele-conferencing.
- A professional within the care team is identified as having a leadership role and responsibility for organising regular reviews of the plan in place.

How long should care co-ordination last for?

- Length of care co-ordination is determined by the review of the team, including the views of the individual, the family and the system around them.
- Indicators for reduced input include progression towards agreed treatment goals, reduction of medical and psychiatric clinical risk and the level of engagement with appropriate supports.



[1]

[1] Reference: National Eating Disorders Collaboration (2012)
An Integrated Response to Complexity National Eating Disorders Framework - pages 51-56

ATTACHMENTS

Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the treatment of eating disorders

Phillipa Hay[1,2,3], **David Chinn**[1,4], **David Forbes**[1,5], **Sloane Madden**[1,6], **Richard Newton**[1,7], **Lois Sugenor**[1,8] **Stephen Touyz**[1,9] **and Warren Ward**[1,10]

Abstract

Objectives: This clinical practice guideline for treatment of DSM-5 feeding and eating disorders was conducted as part of the Royal Australian and New Zealand College of Psychiatrists (RANZCP) Clinical Practice Guidelines (CPG) Project 2013–2014.

Methods: The CPG was developed in accordance with best practice according to the National Health and Medical Research Council of Australia. Literature of evidence for treatments of anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), other specified and unspecified eating disorders and avoidant restrictive food intake disorder (ARFID) was sourced from the previous RANZCP CPG reviews (dated to 2009) and updated with a systematic review (dated 2008–2013). A multidisciplinary working group wrote the draft CPG, which then underwent expert, community and stakeholder consultation, during which process additional evidence was identified.

Results: In AN the CPG recommends treatment as an outpatient or day patient in most instances (i.e. in the least restrictive environment), with hospital admission for those at risk of medical and/or psychological compromise. A multi-axial and collaborative approach is recommended, including consideration of nutritional, medical and psychological aspects, the use of family based therapies in younger people and specialist therapist-led manualised based psychological therapies in all age groups and that include longer-term follow-up. A harm minimisation approach is recommended in chronic AN. In BN and BED the CPG

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recommends an individual psychological therapy for which the best evidence is for therapist-led cognitive behavioural therapy (CBT). There is also a role for CBT adapted for internet delivery, or CBT in a non-specialist guided self-help form. Medications that may be helpful either as an adjunctive or alternative treatment option include an antidepressant, topiramate, or orlistat (the last for people with comorbid obesity). No specific treatment is recommended for ARFID as there are no trials to guide practice.

Conclusions: Specific evidence based psychological and pharmacological treatments are recommended for most eating disorders but more trials are needed for specific therapies in AN, and research is urgently needed for all aspects of ARFID assessment and management.

Expert reviewers

Associate Professor Susan Byrne, Dr Angelica Claudino, Dr Anthea Fursland, Associate Professor Jennifer Gaudiani, Dr Susan Hart, Ms Gabriella Heruc, Associate Professor Michael Kohn, Dr Rick Kausman, Dr Sarah Maguire, Ms Peta Marks, Professor Janet Treasure and Mr Andrew Wallis.

Keywords

Clinical Practice Guideline, eating disorders, evidence-based review

Introduction

This guideline for the clinical management of eating disorders is a project of the Royal Australian and New Zealand College of Psychiatrists (RANZCP). The guideline represents the work of a core working group of health care academics and professionals and wide consultation with key stakeholders and the community.

The guideline is intended to provide current evidence based guidance on the assessment and treatment of people with eating disorders by psychiatrists and other health professionals in the Australian and New Zealand context and includes identifying further research needs. It is written with reference to other international guidelines such as those of the American Psychiatric Association and the United Kingdom National Institute for Health and Care Excellence guidelines and is intended to address both broad but also specific issues, such as those relevant to Māori and Pacific, and Aboriginal and Torres Strait Islander peoples.

Overview

Eating disorders are characterised by disturbances of eating behaviours and a core psychopathology centred on food, eating and body image concerns. Early reports of an anorexia nervosa-like illness date to the 1600s (Silverman, 1983) and anorexia nervosa as a diagnostic entity was described first in 19th century medical reports (Gull, 1874; Lasegue, 1873). In contrast, bulimia nervosa and binge eating disorder were not described until the 20th century. The American Psychiatric Association DSM-5 diagnostic criteria for anorexia nervosa (APA, 2013) include self-imposed or maintained weight loss such that the person is underweight (for age and height) and associated overvaluation of shape and weight (see

Table 1). Two subtypes of anorexia nervosa are specified: restrictive type (with or without compulsive exercise); and binge eating/purging type, with binge eating (uncontrolled overeating) and purging (vomiting, laxative or diuretic misuse). Severity is specified according to BMI (kg/m^2) status.

Bulimia nervosa and binge eating disorder are both defined in the DSM-5 by having regular and sustained binge eating episodes. People with bulimia nervosa also compensate for binge eating with regular extreme weight control behaviours (such as purging). As they do not engage in such compensation regularly, people with binge eating disorder are likely to be overweight or obese. People with bulimia nervosa also have a self-view that is unduly influenced by weight and shape overvaluation. Other specified and unspecified feeding and eating disorders (OSFED and UFED) in the DSM-5 include atypical anorexia nervosa (where BMI may be within the normal range) and sub-threshold forms of bulimia nervosa and binge eating disorder on the basis of insufficient frequency and/or duration of disordered eating behaviours.[1] A new disorder added to DSM-5 is avoidant/restrictive food intake disorder (ARFID) which, like binge eating disorder, and in contrast to anorexia nervosa and bulimia nervosa, is not characterised by body image disturbance. This departure from weight/shape overvaluation as a key feature of all eating disorders is the subject of discussion in the field and likely to also be found in the ICD-11 revision (Al-Adawi et al., 2013). Readers interested in this debate are referred to Russell (2013) and Hay (2013a).

Since the classic writings of Hilde Bruch (Bruch, 1978) the 'face' of anorexia nervosa and eating disorders has changed dramatically. Eating disorders are not the 'preserve' of females, the wealthy or 'westerners'. In the general population, lifetime prevalence of anorexia nervosa is around 1% in women and < 0.5% in men, bulimia nervosa around 2% in women and 0.5% in men, and binge eating disorder around 3.5% in women and 2.0% in men (Favaro et al., 2003; Hudson et al., 2007; Keski-Rahkonen et al., 2007; Lewinsohn et al., 2000; Oakley Browne et al., 2006; Preti et al., 2009; Raevuori et al., 2009; Striegel-Moore et al., 2003; Wade et al., 2006). Point (three-month) prevalence in Australia is estimated at around 1% for bulimia nervosa, 2% for binge eating disorder (using the DSM-5 criteria of weekly frequency of binge eating and extreme weight control behaviours) and 3% for other eating disorders (specified or unspecified according to the new DSM-5 criteria) (Hay et al., 2008). The gender ratio in bulimia nervosa is similar to that of anorexia nervosa but binge eating disorder has a more even gender distribution (Hudson et al., 2007). Accurate point prevalence has not been estimable for anorexia nervosa in Australia but 12-month prevalence in the New Zealand survey was <1% (Wells et al., 2006).

Eating disorders are associated with notable quality of life impairment and impact on home, work, personal, and social life (Jenkins et al., 2011; Mitchison et al., 2012; Mond et al., 2012) and economic cost (Butterfly Foundation, 2012). Eating disorders also frequently co-occur with other mental health disorders, particularly anxiety disorders and depression (Hudson et al., 2007). The peak age of onset of anorexia nervosa is in early to mid-adolescence but may occur at any age, including in childhood, where the gender balance is more even (Madden et al., 2009). The reasons for the greater number of boys presenting in childhood years are unclear. In bulimia nervosa and binge eating disorder onset is more commonly in later adolescence and young adulthood (Stice et al., 2013) and binge eating disorder is more likely a mid-life disorder with a much more even gender frequency. It is important to be aware that all eating disorders can, and do, arise at any age, and in both females and males. In addition, eating disorders often go undiagnosed and untreated. Thus it is common for

adults to present for treatment many years after onset, even into late middle-age (Bulik et al., 2012; Hart et al., 2011b). However, most people make a sustained recovery with treatment. This includes people with anorexia nervosa, where up to 40% of adults (and a higher percent of adolescents) will make a good five-year recovery, a further 40% a partial recovery and those with persistent illness may yet benefit from supportive therapies. For those with bulimia nervosa at least 50% fully recover and the outcomes with treatment are also good if not better for binge eating disorder (Steinhausen, 2002; Steinhausen and Weber, 2009; Steinhausen et al., 2003).

Aetiology

Socio-cultural, biological and psychological factors all contribute to the aetiology of eating disorders (Mitchison and Hay, 2014; Smink et al., 2012; Stice, 2002). The strongest socio-demographic risk factor for having an eating disorder continues to be being of female gender and being from the developed world where the 'thin ideal' prevails. Migrants from the developing world seem to be at particular risk. Also at risk are those living in urban areas and undertaking life pursuits where body image concerns predominate, for example, competitive gymnastics and fashion modelling.

In all eating disorders there is an increased genetic heritability and frequency of a family history. A family history of 'leanness' may be associated with anorexia nervosa and a personal or family history of obesity with bulimic eating disorders. Early menarche (controlling for body weight) also increases risk. Also likely important are epigenetic changes to DNA structure that are not encoded by the DNA sequence itself but which nonetheless result in enduring changes in gene expression and which are transmitted to subsequent generations. These can occur following periods of food deprivation (e.g. the Dutch starvation in World War 2), food repletion, or severe environmental stress (Campbell et al., 2011).

Psychological factors include a 'milieu' of weight concern in formative developmental years and specific personality traits, mostly notably low self-esteem (all eating disorders) and high levels of clinical perfectionism for those with anorexia nervosa, and impulsivity for bulimic disorders. Adverse experiences including emotional and sexual child abuse increase personal vulnerability, most likely through impeding a robust sense of self-worth and adaptive coping. The eating disorder then provides a sense of improved self-esteem and self-control for the individual (Stice, 2002).

Aim and scope

This clinical guideline will aim to provide guidance in the clinical treatment of people with eating disorders, namely anorexia nervosa, bulimia nervosa, binge eating disorder, ARFID. The clinical practice guideline (CPG) may have clinical utility to corresponding forms of other specified or not specified eating disorders that fail to meet DSM or ICD diagnostic criteria for anorexia nervosa, bulimia nervosa, binge eating disorder or ARFID. It will focus on two age groups in anorexia nervosa: (1) adults and older adolescents (18 years and above) and (2) children and adolescents living at home, as treatment and outcomes differ between these groups. One special population, people who are obese or overweight with an eating disorder, will also be addressed.[2]

There is much more space given to anorexia nervosa in this CPG than other disorders. This does not reflect the prevalence of disorders but rather the added complexities of assessment and management for anorexia nervosa compared to bulimia nervosa and binge eating disorder, and the paucity of knowledge in ARFID. In contrast to bulimia nervosa and binge eating disorder, anorexia nervosa presents more frequently in children and adolescents as well as in adults, it is more likely to become severe and enduring and has more extensive medical co-morbidities. We decided not to separate the two disorders of recurrent binge eating (bulimia nervosa and binge eating disorder) as there is much overlap in assessment and treatment approaches and evidence for a transdiagnostic approach.

Method

This guideline was developed as part of the Royal Australian and New Zealand College of Psychiatrists, Clinical Practice Guidelines Project 2013–2014. It was developed in accordance with best practice as outlined by the National Health and Medical Research Council (NHMRC, 2007).

The literature review focused on recent systematic reviews that would include relevant treatment trials since the RANZCP guidelines were written and subsequently reviewed and updated for the consumer guidelines in 2009 (Beumont et al., 2003; RANZCP, 2004; RANZCP, 2009). A comprehensive literature review was thus conducted with dates 2008–2013 to systematically identify and synthesise all studies that were potentially relevant to the guideline. The search was undertaken using PubMed and the search terms ‘anorexia nervosa’ OR ‘bulimia nervosa’ AND ‘treatment guidelines’ OR ‘systematic review’ OR meta-analysis’. Reference lists of identified systematic reviews were also searched for relevant empirical studies on which the CPG recommendations are based. Forty-nine papers were generated and inspected for relevance and quality (including level of evidence grade according to NHMRC categories). Twenty-seven potentially relevant systematic reviews and empirical trials were reviewed by each of two members of the working group for inclusion, and 21 papers were included (members being ineligible to review literature that they had authored or co-authored themselves). Inclusion criteria were reaching a gradable level of evidence according to NHMRC categories of at least level III or higher. Five papers (three from expert reviewers, one from a working group member and one by a member of the public who was consulted) had also been identified by members of the working group and reviewed according to the same process. The results of the search are depicted in Figure 1.

Evidence based recommendations (EBR) were formulated after appraising the evidence using the NHMRC levels of evidence ratings (see Table 2). Where evidence was weak or lacking, consensus based recommendations (CBR) have been formulated. A consensus based recommendation is the lowest level of evidence. It is the consensus of a group of experts in the field and is informed by their agreement as a group according to their collective clinical and research knowledge and experience. In this process level IV articles were considered where higher level evidence was lacking and they informed the CBR.

A series of drafts were then prepared and refined by the working group. The final draft was then reviewed by national and international expert advisers, professional bodies (medicine, psychology, dietetics, nursing, social work and occupational therapy) and special groups (consumer, carer, Aboriginal and Torres Strait Islander, Māori and Pacific and migrant) prior to

an extensive community consultation process. A full list of people and groups consulted and tables of included and excluded studies are available on the RANZCP web site.

General principles of treatment for all eating disorders

Person-centred informed decision-making. Safe and empirically supported treatment options based on available research and expert consensus should be discussed with the individual and their family. These options should be centred wherever possible on an informed decision made with the person and (where appropriate) their family. For children and adolescents the decision balance will be age appropriate and will involve their parents or legally appointed guardian.

Involving family and significant others. Unless there are contraindications or the individual is opposed, family or significant others should be enlisted as partners in the assessment and treatment process. Given the considerable burden on family members it is important that the family is provided with appropriate support and information.

Recovery-oriented practice. Care for people with eating disorders should be provided within a framework that supports the values of recovery-oriented care (Australian Health Ministers Advisory Council, 2013). Recovery-oriented practice encapsulates mental health care that:

- recognises and embraces the possibilities for recovery and wellbeing created by the inherent strength and capacity of all people experiencing mental health issues
- maximises self-determination and self-management of mental health and wellbeing
- assists families to understand the challenges and opportunities arising from their family member's experiences
- provides evidence-informed treatment, therapy, rehabilitation and psychosocial support that helps people to achieve the best outcomes for their mental health, physical health and wellbeing
- works in partnership with consumer organisations and a broad cross-section of services and community groups
- embraces and supports the development of new models of peer-run programs and services
- maximises choice
- supports positive risk-taking
- recognises the dignity of risk, i.e. the individual's right to make treatment choices that the treating health care team might not see as being the most effective decision
- takes into account medico-legal requirements and duty of care
- promotes safety.

Least restrictive treatment context. Where possible, treatment should be offered in the setting that is least restrictive and best suited to the individual's needs and preferences.

Many people with eating disorders are difficult to engage as they are ambivalent, or fear giving up their eating disorder behaviours, or deny the illness or the seriousness of their symptoms. Offering options and control can help with therapeutic engagement. Legislation in both New Zealand and Australia allows for involuntary assessment or treatment if a person with anorexia nervosa has impaired decision-making capacity, and is unable or unwilling to consent to interventions required to preserve life. Although involuntary treatment may provide the opportunity to prevent fatal complications, the potential adverse effects on therapeutic alliance needs to be considered (Carney et al., 2007). The short-term weight gain response of involuntary patients with anorexia nervosa has been shown to be comparable to those admitted voluntarily (Watson et al., 2000). Many of those who are treated on an involuntary basis later agree that treatment was necessary and remain therapeutically engaged (Guarda et al., 2007; Tan et al., 2010; Watson et al., 2000).

Multidisciplinary approach. Expert consensus and clinical cohort studies (e.g. NICE, 2004b) support a multidisciplinary approach to ensure that the individual gets access to the combined medical, dietetic (Dietitians Association of Australia, 2012) and psychological interventions required to maximise the chances of a full recovery. Ideally, team members will have specialist knowledge, skills and experience in the area of eating disorders, and be situated in the same location or at least in places easily accessible for those being treated and their families. Although team members may have differing perspectives, a united approach in delivering treatment is critical. The general practitioner is often in the best position to be the key coordinating clinician, especially if the treating specialists are not co-located.

Stepped and seamless care. Ideally, a range of options including outpatient, intensive outpatient with meal support, day program, and inpatient treatment should be available. As many people will not be seen by a specialist service, specialists should build strong links with primary care, general hospital and community providers in order to facilitate access and smooth transitions of care between general practice, emergency departments, medical wards, mental health settings, private clinicians and specialist services (House et al., 2012).

A dimensional and culturally informed approach to diagnosis and treatment. Establishing the presence of the core syndrome of an eating disorder is crucial in terms of informing treatment, but significant symptom variability occurs within and between individual experiences of anorexia nervosa, bulimia nervosa and binge eating disorder and other eating disorders. A rigid approach to diagnosis should be avoided (Pike, 2013). Empirically supported definitions of severity are still in development (Maguire et al., 2008) and although physical measures (e.g. BMI and more specific indicators of malnutrition such as amenorrhoea, hypotension, bradycardia, hypothermia and neutropaenia) are commonly used as markers of severity, psychological symptoms and clinical history should also inform severity formulations (Maguire et al., 2012). Comparative studies (EBR III) highlight possible cultural and ethnic variability in the presentation of anorexia nervosa or other eating disorders (Soh et al., 2006). There are insufficient studies on gender differences to inform the need for major differences in assessment approaches or treatment delivery for males (Murray and Touyz, 2013).

Indigenous care. Clinicians must demonstrate the appropriate knowledge, skills, awareness, and attitudes ('cultural competence') when working with people with eating disorders from indigenous and other cultural backgrounds. Assessment and treatment of eating disorders in Aboriginal and Torres Strait Islander and Māori and Pacific peoples should ascribe to the broader principles of mental health care as recommended in the RANZCP online training guide (RANZCP, 2012).

Approaches to care of people of Aboriginal and Torres Strait Islander background should be informed by an understanding of their history and culture even though there is little written about specific approaches to treatment for those who suffer from an eating disorder. However, epidemiologic evidence indicates that Aboriginal and Torres Strait Islander people are no less likely, and may be more likely, to experience an eating disorder as non-indigenous Australians (Hay and Carriage, 2012). In particular, poor nutrition and health-related consequences such as Type II diabetes are well recognised problems in this population. The reasons for this are complex, but the loss of traditional values and practices in food choices and replacement with foods of less nutritional value may be part of a broader process of cultural dispossession combined with social disadvantage. In addition, Aboriginal and Torres Strait Islander adolescents do have body image concerns which appear to focus around a desired 'muscular' shape (Cinelli and O'Dea, 2009) and may be less concerned about being slim or losing weight (McCabe et al., 2005; Ricciardelli et al., 2004).

Epidemiological data are scarce in Māori and Pacific peoples, with older data suggesting a lifetime prevalence of 3.1% (0.7% anorexia nervosa; 2.4% bulimia nervosa) and 3.9% respectively (Baxter et al., 2006). However, there are high levels of obesity in both Māori and Pacific peoples compared with the New Zealand adult population overall (Ministry of Health, 2012a, 2012b). Thus, whilst the prevalence of binge eating disorder is unknown, because of an associated risk with obesity it is possible that Māori and Pacific peoples are at increased risk of binge eating disorder.

In New Zealand 'cultural competence' explicitly includes application of te Tiriti o Waitangi (the Treaty of Waitangi) principles of partnership, participation and protection. This may require different assessment practices, for example the presence of wider whānau (family) at consultations, styles of treatment engagement and the communication of treatment information. These competencies are further outlined by the Medical Council of New Zealand and the New Zealand Psychologists Board (MCNZ, 2006a, 2006b, 2010; NZPB, 2011). In every aspect, working with different cultural groups requires respect for different world views, values and meanings. Clinicians should seek cultural advice and leadership from cultural support staff/whānau advisers to maximise engagement and therapeutic alliance.

Section one: anorexia nervosa

Anorexia nervosa in adults

Introduction

The following section outlines core assessment and treatment guidelines for adults with anorexia nervosa. There is a lack of high quality evidence to guide the clinician in the treatment of adults who have anorexia nervosa. Methodologically robust studies are small in number and inconclusive, meaning that conflicting results are common. Many studies are limited by methodological issues including small samples, low quality design, and short follow-up periods (Watson et al., 2010). There have however been improvements in the evidence base for treatments for adults with anorexia nervosa since the 2003 RANZCP Clinical Practice Guidelines (Beumont et al., 2003; Watson and Bulik, 2012). Clinical practice is as well best informed by considering recent systematic reviews and accessing empirically investigated treatments in conjunction with consensus opinions of experts in the field (Beumont et al., 2003; RANZCP, 2004; Watson et al., 2010; Yager et al., 2006).

Assessment

A comprehensive assessment of the individual and their circumstances should be undertaken to confirm the diagnosis of anorexia nervosa and any comorbid psychiatric or medical diagnoses, to evaluate medical and psychiatric risks, and to develop a biopsychosocial formulation. Collecting assessment information is an ongoing task as clinical issues and priorities unfold throughout treatment. Comprehensive initial assessment of adults should include the following components:

- Collating a thorough history including the various symptoms of anorexia nervosa which include but are not limited to: dietary restriction; weight loss; inability to restore weight; body image disturbance; fears about weight gain; binging; purging; excessive exercise; early satiety; constipation; and the use of laxatives, diuretics, or medications to lose or maintain low weight (APA, 2013). Other symptoms may include disturbed eating behaviours, e.g. eating apart from others and ritualistic patterns of eating such as prolonged meal times and division of food into very small pieces (Wilson et al., 1985). It is important to accurately assess nutritional and fluid intake, with specific enquiries made as to the adequacy of main meals and snacks consumed. Where possible, collateral sources such as family members and other clinicians involved in the person's care should be utilised. The perspective of others is especially important given that symptom minimisation, poor insight or genuinely poor understandings of the seriousness of symptoms are common aspects of anorexia nervosa (APA, 2013).
- Investigating any medical complications and the current level of medical risk. This is essential and should include a brief physical examination including measurement of weight, height, calculation of BMI, seated and standing pulse rate to detect resting bradycardia and/or tachycardia on minimal exertion due to cardiac deconditioning, blood pressure (seated and standing) and temperature. These findings are needed to determine if immediate hospital admission is required (see Table 3). Investigations should include serum biochemistry to detect hypokalaemia, metabolic alkalosis or acidosis, hypoglycaemia, hypophosphataemia, and hypomagnesaemia, serum liver

function tests, serum prealbumin levels and a full blood examination looking for evidence of starvation-induced bone marrow suppression such as neutropaenia and an electrocardiogram (ECG). A bone mineral density scan should be performed routinely if the person has been underweight for six months or longer with or without amenorrhea and thereafter every two years whilst still struggling with an eating disorder (Mehler et al., 2011). The assessment should also include any history of fainting, light-headedness, palpitations, chest pain, shortness of breath, ankle swelling, weakness, tiredness and amenorrhoea or irregular menses

- Assessing psychiatric comorbidity, e.g. anxiety, depression, substance misuse, suicidality, personality disorders, anxiety disorders and deliberate self-harm. Comorbidity in people with anorexia nervosa is common and therefore assessment for such should be routine. Lifetime prevalence of comorbidity has been reported ranging from 55% in community adolescent samples to 96% in adult samples (Godart et al., 2007; Madden et al., 2009; Milos et al., 2003; Swanson et al., 2011). All forms of anxiety disorder may occur and in one study the most common was social phobia (42%), followed by post-traumatic stress disorder (26%) and generalised anxiety disorder (23%) (Swinbourne et al., 2012). Furthermore, social anxiety is not only related to eating in public but may pre-date the onset of the eating disorder. Rates of comorbidity are similar in men and women (Raevuori et al., 2009). However, clinicians should be aware that depression, obsessional thinking, anxiety and other psychiatric symptoms can represent the reversible effects of starvation on the brain (Keys et al., 1950).
- Assessing cognitive changes due to starvation such as slowed thought processing, impaired short-term memory, reduced cognitive flexibility and concentration and attention difficulties (Hatch et al., 2010). Whilst brain imaging is not routine, these problems reflect consistent findings of reduced grey matter volumes that often do not reverse following weight recovery (Phillipou et al., 2014).
- Considering possible predisposing and precipitating factors including a family history of eating disorders, early attachment and developmental difficulties, premorbid obesity, interpersonal problems, and dieting or other causes of rapid weight loss (Mitchison and Hay, 2014; Stice, 2002). Rapid weight loss from any cause, including physical illness, can trigger cognitive changes including obsessive thinking about food, in turn precipitating and perpetuating the symptoms of anorexia nervosa (Keys et al., 1950).

Treatment

Setting priorities. Thorough assessment should lead to a working diagnosis or diagnoses, risk assessment and case formulation setting immediate treatment priorities. The case formulation should include preliminary hypotheses about predisposing, precipitating and maintaining factors, as well as noting the individual's strengths and protective factors. Given the typically prolonged time between onset and presentation, and the diverse pathways into anorexia nervosa, early firm statements about causal factors pertaining to an individual should be avoided. It is generally agreed that treatment priorities should be set as follows: engagement, medical stabilisation, reversal of the cognitive effects of starvation, and provision of structured psychological treatment. These are discussed in more detail below.

Engagement. Engaging with the individual with anorexia nervosa and their family at the first appointment can be challenging as there is typically extreme anxiety at the prospect of increased nutrition and weight gain, which are essential for recovery. To enable therapeutic engagement it is crucial that the clinician take a non-judgemental, inclusive, empathetic and non-threatening stance. Although lacking level I empirical evidence, sensible practices for improving engagement have included psychoeducation, enlisting the support of the family (Treasure et al., 2007), motivational interviewing around what the individual themselves reports as important, and appealing to the 'healthy' part of the person (Nordbø et al., 2008; Vitousek et al., 1998).

Medical stabilisation. Admission to hospital is indicated if the person is at imminent risk of serious medical complications, or if outpatient treatment is not working (Beumont et al., 2003). Indicators of high medical risk requiring consideration for admission (see Table 3) include any one of the following: heart rate <40 bpm or tachycardia on standing due to cardiac deconditioning with >20 bpm increase in heart rate, blood pressure <90/60 mm Hg or with >20 mm Hg drop on standing, hypokalaemia, hypoglycaemia, hypophosphataemia, temperature <35.5°C, or BMI < 14 kg/m^2. It is important to note that patients can report feeling well even when the risk of cardiac arrest is high. Admission is also indicated if there is rapid weight loss, several days of no oral intake, supervision required for every meal, uncontrolled purging or exercise, or suicidality.

Ideally, whenever possible and practicable, people with anorexia nervosa requiring admission should be admitted to a specialist eating disorders unit. There is debate about rates of weight gain in inpatient settings with recommended rates ranging from 500–1,400g/week (NICE, 2004b; Yager et al., 2006). There is evidence that where weight gain is the key prioritised treatment goal, inpatient treatment is superior to less intense forms of treatment due to the faster weight gain in those settings (Hartmann et al., 2011). However, inpatient beds are unfortunately scarce and can be difficult to access. In such situations, another model that has been effective in managing acute problems is admission to general medical or psychiatric beds, with support provided to generalist clinicians by a specialist eating disorder consultation-liaison service (EDOS, 2011). Where patients are at very high medical risk (e.g. with BMI < 12 or significant medical complications), they will need to be admitted to a medical setting with input from psychiatry consultation-liaison services until medically stabilised, before being transferred to a psychiatric or eating disorders specialist unit for ongoing nutritional rehabilitation and psychiatric treatment. Discharge from hospital should only occur when the person is medically stabilised, has had enough nutrition to reverse any cognitive effects of starvation so that she or he can benefit from outpatient or day patient psychotherapy (often several weeks of nutrition are required to achieve this), has had trials of leave to demonstrate that she or he can eat outside hospital, and has a direct link in with appropriate outpatient monitoring, support and treatment (EDOS, 2011).

Following discharge to less intense treatment settings, there is insufficient evidence to point to the best means of maintaining weight (Hartmann et al., 2011). One small randomised control trial found a manualised cognitive behavioural therapy for anorexia nervosa (CBT-AN) was superior to nutritional counselling and usual care (all treatment including nutritional counselling delivered by psychologists) in preventing relapse (Pike et al., 2003). Nutritional counselling alone should not be the sole treatment for people with anorexia nervosa; a multidisciplinary approach is more supported (Dietitians Association Australia, 2003) and

there is strong clinical consensus that continuity of treatment should be provided. For people with severe and enduring eating disorders, a more flexible and patient-centred approach may need to be taken when considering criteria for admission and discharge (see separate section on severe and enduring eating disorders in this clinical practice guideline).

Medical complications and their treatment. Table 4 lists the common medical complications and their management. If the medical complication is secondary to malnutrition, effective treatments must always include regular and adequate nutrition. It is important to ensure that prescribed nutrition is being consumed by the individual, either through supervision and support during mealtimes by staff in hospital or family/carers out of hospital; or if the person is well enough, through self-recording of oral intake discussed at outpatient treatment sessions. Similarly, if medical complications are due to purging or other behaviours, measures need to be taken to address these.

Refeeding syndrome. Refeeding syndrome is a serious and potentially fatal medical complication of aggressive refeeding of an individual who has been malnourished for a lengthy period. Refeeding syndrome is understood to be due to the switch from fasting gluconeogenesis to carbohydrate-induced insulin release triggering rapid intracellular uptake of potassium, phosphate and magnesium into cells to metabolise carbohydrates (Kohn et al., 2011). This, on top of already low body stores of such electrolytes due to starvation, can lead to rapid onset of hypophosphataemia, hypomagnesia and hypokalaemia. In addition, insulin-triggered rebound hypoglycaemia can occur, exacerbated by the fact that such patients have depleted glycogen stores. The risk factors for refeeding syndrome include the degree of malnutrition and adaptation to this state, the levels of serum minerals and electrolytes such as phosphate and potassium and the rate of provision of carbohydrate in relation to other nutrients (Gentile et al., 2010; Kohn et al., 2011; Mehler et al., 2010; O'Connor and Goldin, 2011; Ornstein et al., 2003).

There is a wide range of opinion as to ideal starting doses of nutrition for adults with anorexia nervosa, with often little evidence to support the varied opinions (Gaudiani et al., 2012; Katzman, 2012; Kohn et al., 2011). Traditionally, it has been thought that the risk of refeeding syndrome can be reduced by 'starting low' and 'going slow' with nutrition, and monitoring serum phosphate, potassium and magnesium daily for the first 1–2 weeks of refeeding, and replacing these electrolytes immediately if they fall below normal range (Beumont et al., 2003; NICE, 2004b; Yager et al., 2006).

All authors agree on the importance of regularly monitoring and replacing phosphate, potassium and magnesium. However, traditional recommendations for refeeding designed to prevent refeeding syndrome are now seen by many to be too conservative, and unnecessarily put the severely malnourished person at risk of 'underfeeding syndrome' and further medical deterioration. Findings from case series studies range from those reporting large numbers of adolescents being fed on relatively high initial rates of up to 8400kJ of low-carbohydrate continuous nasogastric feeds with supplemental phosphate without causing refeeding syndrome (Kohn et al., 2011; Whitelaw et al., 2010), to those of severely malnourished adults reporting that 45% of participants developed significant refeeding-induced hypophosphataemia with much lower mean initial refeeding doses of 4000kJ/day (Gaudiani et al., 2012). Thus, refeeding syndrome has been observed even with very low initial feeding doses, and initial dose has not been shown to be a predictor of refeeding

hypophosphataemia or refeeding syndrome (Gaudiani et al., 2012; O'Connor and Goldin, 2011). It is unclear whether the recent literature supporting safe use of higher refeeding doses in adolescents (for more information, see the section in these guidelines on treating anorexia nervosa in children and adolescents) is applicable to adults who may have been more severely malnourished for much longer periods, theoretically putting them at higher risk of refeeding syndrome.

In light of the conflicting and inadequate literature, the CPG group recommend taking a 'middle path' with adults, commencing refeeding at 6000kJ/day. This should be increased by 2000kJ/day every 2–3 days until an adequate intake to meet the person's needs for weight restoration is reached. This diet should be supplemented by phosphate at 500mg twice daily and thiamine at least 100mg daily for the first week, and thereafter as clinically indicated for people at high risk of refeeding syndrome (e.g. BMI <13). For people at high risk of refeeding syndrome, commencing with continuous nasogastric feeding with low-carbohydrate preparations (i.e. 40–50% of energy from carbohydrates) seems prudent to avoid triggering postprandial rebound hypoglycaemia due to insulin secretion in people with inadequate glycogen stores. The most important aspects of preventing refeeding syndrome are a heightened physician awareness of the syndrome, and regular monitoring of the person's clinical status, including physical observations and biochemical monitoring, especially to guide phosphate prophylaxis or supplementation.

Refeeding protocols should, however, be individualised where necessary to minimise both the risk of refeeding syndrome and complications due to underfeeding, and involve the input of a dietician experienced in the treatment of eating disorders. Methods of nutritional provision include supervised meals, high energy high protein oral liquid supplements and nasogastric feeding. On very rare occasions where the above methods are unable to be utilised, parenteral nutrition may be indicated. The least intrusive and most normal method of nutrition that can be reliably provided should be used.

Monitoring progress and reviewing priorities. In assessing whether an individual's nutritional heath has been adequately restored, weight is only an approximate indicator. Other indicators of normal physiological functioning should be considered, including normal blood glucose levels, reversal of hypotension and bradycardia, normal blood cell counts with bone marrow suppression reversed, return of menstruation and normal cognitive functioning.

Assessing psychological progress is more difficult given that many features of anorexia nervosa involve internalised symptoms and behavioural deficits. However, such assessment should include monitoring dietary intake, compensatory behaviours and body image disturbance and dissatisfaction. Absence of progress in treatment after reasonable trial periods should prompt a treatment review and consideration for changing interventions and/or increasing treatment intensity.

Specific treatments. Providing psychoeducation, support and building a therapeutic relationship are all crucial activities at all stages of treatment. The more intense structured psychological therapies should generally be initiated only after the individual is sufficiently stabilised and cognitively improved from the acute effects of starvation. It is important that the treatment plan is individualised and addresses any comorbid conditions.

Individual therapies. There are major shortcomings in the literature, making any robust and direct comparison between commonly used psychological treatments (e.g. cognitive behaviour therapy, interpersonal psychotherapy, or psychodynamic psychotherapy) very difficult. These limitations include small numbers of trials and lack of statistical power.

Cognitive behavioural therapy (CBT) and its many forms, for example CBT-Enhanced (Fairburn, 2008), are frequently recommended approaches for anorexia nervosa. Common to these approaches are the activities of directly challenging anorexia-related behaviours, cognitions and patterns of thinking, especially symptoms that maintain the disorder. In inpatient settings, less restrictive behavioural approaches are likely to be more effective than strict regimens which are often perceived by the individual as punitive and demeaning (Touyz et al., 1984). Despite the popularity of CBT and improving evidence of its effectiveness, further investigation is required (Bulik et al., 2007; Hay, 2013b; Zipfel et al., 2014). There is no empirical evidence that guided self-help CBT (CBT-GSH) is a useful treatment for anorexia nervosa, with some concluding that it is contraindicated (Wilson and Zandberg, 2012).

Specialist supportive clinical management (SSCM) (McIntosh et al., 2006) has been shown in one trial to be more effective than CBT or interpersonal therapy (McIntosh et al., 2005). SSCM has as its primary focus resumption of normal eating and the restoration of weight, but it also allows a flexible approach to addressing life issues impacting on the eating disorder.

The Maudsley model of anorexia nervosa treatment for adults (MANTRA), not to be confused with the 'Maudsley' model of family based therapy, is a recently developed manualised individual therapy for adults with anorexia nervosa, drawing on a range of approaches including motivational interviewing, cognitive remediation and flexible involvement of carers. It aims to address the obsessional and anxious/avoidant traits that are proposed as being central to the maintenance of the illness (Schmidt et al., 2012). In a randomised controlled trial (RCT) it was, however, shown to be no more effective than SSCM, with recovery rates low in both arms of the trial (Schmidt et al., 2012).

Motivation-based therapies (motivational interviewing, motivational enhancement etc.), either as the main treatment or in conjunction with another therapy, have been adapted to eating disorders (Casasnovas et al., 2007; Nordbø et al., 2008; Treasure et al., 2007) including anorexia nervosa. Meaningful engagement in therapy is a crucial component in all treatments for anorexia nervosa, as are techniques to enhance change. Recent critical reviews question whether purely motivation-based therapies improve treatment efficacy in anorexia nervosa (Knowles et al., 2012; Waller, 2012). This does not dismiss the crucial importance of challenging resistance to change and targeting behavioural changes alongside wider psychological changes: these processes underpin most psychotherapies.

Other individual approaches that may be helpful in adults with anorexia nervosa include interpersonal psychotherapy, cognitive analytic therapy, focal psychoanalytic and other psychodynamic therapies (Watson, 2010; Zipfel, 2014), although again the level of evidence for each of these is modest, and generally limited to a very small number of trials. Interpretation of findings where specific psychological therapies are compared with other therapies is hampered by methodological problems (Hartmann et al., 2011).

Web-based therapies for anorexia nervosa have not been sufficiently investigated by EBR II research (Aardoom et al., 2013). A single RCT has shown that a CBT-based program

delivered on the internet may reduce the risk of relapse after hospitalisation (Fichter et al., 2012). It is advised that any treatment via the internet poses unique risks in providing responsible care. There is increased likelihood of miscommunications and there are significant regulatory issues if the practice occurs across different jurisdictions. Given the very limited evidence of effectiveness, these additional factors contribute to a high degree of caution if using internet-based therapies to treat anorexia nervosa.

Family therapies. There is moderate research-based evidence for family therapies in younger people with anorexia nervosa up to their late teens, living with family and with an illness duration of less than three years, with evidence that family therapy is more effective than individual treatment (Russell et al., 1987). Whilst family therapy approaches vary in their focus and etiological stances, a common theme is the involvement of family in treatment to help recovery especially in regard to interventions to restore weight.

Recent evidence reviews have reported that family therapy and a specific form of family therapy termed 'family based treatment' (FBT) remains the most well-studied treatment for young people with anorexia nervosa, and has been associated with persistent positive outcomes on physical and psychological parameters (Keel and Haedt, 2008; NEDC, 2010; Watson and Bulik, 2012); however, views are mixed (Couturier et al., 2013; Fisher et al., 2010; Strober, 2014). One of these reviews (Couturier et al., 2013) found that although FBT was not superior to individual treatment at the end of treatment, there did appear to be significant benefits at the 6–12 month follow-up. It is important to note that FBT has not been shown to be an effective treatment for anorexia nervosa in adults older than 18 years.

There is wider clinical consensus that families play an important role in assessment and overall treatment processes for younger people. Thus, unless contraindicated, family functioning should be assessed and appropriate support provided to manage the burden to families of anorexia nervosa. Other promising models of family interventions have been developed for adults, such as skills-based training for family members (Treasure et al., 2007) and couples work with partners (Bulik et al., 2011), but these are yet to be evaluated in well-designed studies. Further research and guidance with regards to family therapy in children and adolescents is discussed later in this guideline.

Pharmacotherapy. Recent systematic reviews of RCTs and meta-analyses of the pharmacological treatment of anorexia nervosa suggest weak evidence for the use of any psychotropic agents (Aigner et al., 2011; Flament et al., 2012; Hay and Claudino, 2012; Kishi et al., 2012). Prescribing for comorbid conditions (e.g. anxiety or mood disorders) is best left until it is clear that such symptoms are not simply secondary to starvation, although low doses of antipsychotics such as olanzapine may be helpful when patients are severely anxious and demonstrate obsessive eating-related ruminations, but more trials are needed (Hay and Claudino, 2012). Caution is required for any psychotropic medication, as physical problems secondary to anorexia nervosa may place individuals at greater risk of adverse side effects. Evidence for the effectiveness of medications to reverse bone density loss is lacking (Mehler et al., 2011).

Other treatments. A variety of other treatments have been proposed as primary treatments or as an adjunct to treatment of anorexia nervosa, but not established because of inadequate

evidence to support their routine use. Zinc deficiency is common in anorexia nervosa and may be associated with dermatological change (Kim et al., 2010; Lask et al., 1993). In a single small double-blind trial, zinc supplementation was associated with a more rapid rate of body mass increase (Birmingham et al., 1994). Zinc supplementation is, however, not a routine component of therapy, because assessment of zinc status is difficult, the purported benefit limited, and deficiency will correct with general nutritional improvement (Lask et al., 1993).

Clinical observations and animal studies of decreased thermogenic activity with application of external heat have led to the incorporation of warming as a component of some treatment programs (Bergh et al., 2002; Birmingham et al., 2004; Gutierrez and Vazquez, 2001). A controlled trial of warming for three hours a day failed to demonstrate any advantage for weight gain compared with 'treatment as usual'. Biofeedback focused on satiety and rate of eating underpins the Mandometer, a computer-based device that attempts to retrain eating patterns, has support from small observational studies and a small RCT compared to a wait-list control group (Bergh et al., 1996, 2002), but not from another larger controlled study (van Elburg et al., 2012). Recently, stereotactic surgery and deep brain stimulation have been considered for the management of refractory anorexia nervosa (Lipsman et al., 2013b). A pilot study of six adult patients with chronic refractory anorexia nervosa underwent implantation of electrodes to the sub-callosal cingulate region of their brain and then long-term deep brain stimulation (Lipsman et al., 2013a). While there may be some benefits to this approach, assessment has been inadequate and the wider response has been cautious (Hutton, 2013; Treasure and Schmidt, 2013).

Relapse prevention. Prospective and retrospective studies show varying rates of relapse, but it is generally agreed that the rates are substantial. A recent prospective study (Carter et al., 2012) showed a relapse rate of 41%, with the highest risk for relapse within 4–9 months after treatment. There is insufficient evidence about treatments to reduce relapse, but well-designed studies are consistent in finding that those with anorexia nervosa-binge purge subtype have higher rates of relapse, meaning that these individuals should receive more intense follow-up.

Outcomes and prognosis. Steinhausen (2002) reported that 70% of a multicentre European cohort with anorexia nervosa had fully recovered at follow-up at a mean of 6.4 years, and around 75% had no other evidence of psychiatric illness. Strober and colleagues documented in detail the follow-up of a cohort of 95 individuals with anorexia nervosa followed longitudinally. Eighty-seven percent recovered at least partially (loss of three of four diagnostic criteria of anorexia nervosa) at a mean of 59 months from assessment, and 76% experienced full recovery at a mean of 79 months. In an adolescent cohort, it was reported that recovery was still occurring 11 years after initial assessment (Strober et al., 1997). Steinhausen also reported lower mortality rates than 'all-age' data, with a 2.9% crude mortality rate in an adolescent European cohort (Steinhausen et al., 2003).

Summary of recommendations with level of evidence base (EBR) or consensus (CBR)

Recommendation	Grade
Assessment of adults with anorexia nervosa • Be person-centred and culturally informed in assessment practices. • Involve family and significant others unless there are clear contraindications. • Take a multidisciplinary approach. • Use a dimensional approach to the illness, i.e. tailor management based on stage of illness severity and symptom profile. • Conduct detailed assessments of core symptomatology including restriction methods, psychological symptoms related to fear of weight gain, weight loss, drive for thinness, and body image disturbance/ dissatisfaction, establishing both severity and (where possible) duration of illness. • Conduct detailed evaluations for any comorbid psychiatric diagnoses. • Conduct detailed physical, medical, and laboratory examinations, thereby setting priorities for any specific medical interventions.	EBR III
Treatment of adults with anorexia nervosa • Treat in the least restrictive environment possible. Many people can be treated in outpatient care (EBR II). • Provide 'stepped and seamless care' options, with smooth transition between services (including between primary care, general hospital settings and other community services). • Admit to hospital when indicated for those at high risk of life-threatening medical complications, extremely low weights, and other uncontrolled symptoms. • Use refeeding and weight gain regimes that minimise the risk of refeeding syndrome, 'underfeeding' and other medical complications arising from increased nutritional intake. • Take a multi-axial approach to assessing treatment progress, including considering nutritional, medical and psychological aspects. • Provide psychoeducation, support and a therapeutic relationship at all stages of treatment and initiate more intense psychological therapies after the person is sufficiently medically stabilised and cognitively improved from the effects of starvation. • Initiate longer-term follow-up as recovery rates are low and relapse rates are high. This will limit the need for re-intensified treatment.	EBR III

Summary of recommendations with level of evidence base (EBR) or consensus (CBR) (continued)

Recommendation	Grade
Specific pharmacological treatments • There is only weak evidence for pharmacological treatment of anorexia nervosa. Low-dose antipsychotics such as olanzapine may be useful in reducing anxiety and obsessive thinking, but results of trials are mixed and such individuals are at greater risk of adverse side effects (EBR I). Exercise caution in prescribing psychotropic medication for severe comorbid conditions/symptoms until it is clear that such symptoms are not secondary to starvation.	EBR III
Specific psychological treatments • Psychological therapy is considered essential, but there is limited high quality evidence to direct the best choice of therapy modality. • There is modest evidence that family based therapies are effective for younger people (up to the age of 18) living with families. • Specialist therapist-led manualised based approaches show the most promising evidence base, and as such should be first-line options.	EBR I
Future research • Future research should include methodological designs that are robust and can overcome past problems by recruiting larger samples and enabling longer follow-up periods.	EBR I

Anorexia nervosa in adolescents and children

Introduction

Anorexia nervosa is the third most common chronic disorder affecting adolescent girls, with a mean mortality rate of 5% in adults and 2% in adolescents (Steinhausen et al., 2002). The mortality rate is up to 18 times greater than in non-affected women aged 15–24 years (Steinhausen et al., 2003). The peak age of onset for anorexia nervosa is 15 to 19 years, accounting for approximately half of all presentations (73.9 to 270 females per 100,000 person years and 6.4 to 15.7 males per 100,000 person years) with nearly all people with anorexia nervosa presenting between 10 and 29 years (Herpertz-Dahlmann, 2009). Individuals between 10 and 14 years account for approximately one in five new presentations of anorexia nervosa (Keski-Rahkonen et al., 2007; Lucas et al., 1999; Raevuori et al., 2009). The incidence of early onset (children aged 5–13 years) eating disorders in Australia is estimated to be 1.4–2.8 per 100,000 children (Madden et al., 2009).

Lifetime prevalence rates for anorexia nervosa are higher in younger women compared with older age women, with rates consistently higher in more recent studies. This is consistent with the findings of Lucas et al. (1999), who demonstrated that the incidence of anorexia nervosa in 15 to 24 year old women has increased over the past 50 years. In the 10 to 14 year old age group, rates of anorexia nervosa also appear to have increased since the 1960s (Lucas et al., 1999), though the limited number of studies that have looked at this group make it difficult to be definitive. What can be clearly stated about this younger age group is that males make up a far greater proportion of those with anorexia nervosa, making up between one in six and one in four presentations (Madden et al., 2009; Nicholls et al., 2011b) compared with between one in eight and one in 15 presentations in adults (Hoek and van Hoeken, 2003; Lucas et al., 1999; Miller and Golden, 2010; Preti et al., 2009).

While two thirds of children aged less than 12 years with weight loss due to an eating disorder present with similar psychological symptoms to older adolescents and adults with anorexia nervosa, there are also differences. Children in this age group are less likely to report fear of weight gain or fatness (Lask and Bryant-Waugh, 1992; Madden et al., 2009; Nicholls et al., 2011b; Walker et al., In Press), more likely to fail to appreciate the severity of their illness (Fisher et al., 2001), more likely to present with non-specific somatic symptoms (Blitzer et al., 1961; Madden et al., 2009; Nicholls et al., 2011b), more likely to be diagnosed with other specified or unspecified feeding or eating disorder or ARFID, more likely to be boys, less likely to report vomiting or laxative abuse, more likely to have lost weight more rapidly and more likely to have a lower percent ideal body weight (%IBW) than older individuals with anorexia nervosa (Madden et al., 2009; Nicholls et al., 2011b; Peebles et al., 2006; Walker et al., In Press).

Complications from malnutrition and compensatory behaviours associated with anorexia nervosa include growth retardation, osteoporosis, infertility and changes in brain structure (Katzman, 2005) as well as psychological complications including depression, anxiety, obsessive compulsive disorder and cognitive impairment (Hatch et al., 2010). These complications are greatest during early adolescence due to disruption of critical periods of physical, psychological and social development (Golden et al., 2003; Katzman, 2005).

Assessment

There is limited evidence to guide the assessment of children and adolescents with anorexia nervosa with all recommendations based on expert consensus. At assessment, every child or adolescent suspected of having anorexia nervosa needs a comprehensive review of psychological and physiological signs and symptoms. Ideally assessments should be multidisciplinary and include professionals with expertise in psychiatric diagnosis, medicine and dietetics. Assessment should involve both children and their families or carers unless this is contraindicated due to safety concerns such as abuse or domestic violence (Mariano et al., 2013). Assessments in children and adolescents should be developmentally informed.

Psychological assessment should include a review of both eating disorder symptoms and comorbid psychiatric symptoms. The most common comorbid illnesses in adolescents with anorexia nervosa are anxiety disorders, including obsessive compulsive disorder (OCD) and major depressive disorder (MDD) (Milos et al., 2003; Steinhausen, 2002; Swanson et al., 2011). As in adult care, minimising mortality associated with anorexia nervosa is key

to initial medical care. Some people will require admission for medical compromise (see Tables 3, 4 and 5). Medical instability is the key indicator for acute hospitalisation.

Treatment

Current evidence supports outpatient care as the first-line treatment in adolescent anorexia nervosa, and there is a growing body of evidence to guide such care, particularly the role of family treatment (Lock, 2011; Smith and Cook-Cottone, 2011). There are, however, few studies into the role of inpatient treatment in anorexia nervosa. Hospitalisation of adolescents with anorexia nervosa for the management of acute medical instability (e.g. hypothermia, hypotension, electrolyte abnormalities and cardiac arrhythmias) is thought to be essential in preventing mortality associated with anorexia nervosa (Golden et al., 2003; Katzman, 2005).

Treatment, especially of children and adolescents with more severe disease, should be multidisciplinary, and include focused psychological therapy of the eating disorder and comorbid psychological problems. It should typically include psychoeducation of families, nutritional and medical therapy (at times pharmacotherapy) and may require case management involving schools and other agencies.

A total of nine RCTs have looked at treatment of anorexia nervosa exclusively in adolescents, and 12 RCTs have included adolescents (see Table 6). (Ball and Mitchell, 2004; Crisp et al., 1991; Eisler et al., 2000; Godart et al., 2012; Gowers et al., 2007; le Grange et al., 1992; Lock et al., 2005, 2010; Rhodes et al., 2008; Robin et al., 1994, 1999; Russell et al., 1987). All 12 of these studies included an outpatient intervention and all but one of these trials (Gowers et al., 2007) included an evaluation of some form of family intervention, including four trials that have compared different structures or doses of family interventions (Eisler et al., 2000; le Grange et al., 1992; Lock et al., 2005; Rhodes et al., 2008). Eight of the trials have looked at individual psychological interventions in anorexia nervosa, five in a direct comparison with family treatment (Ball and Mitchell, 2004; Lock et al., 2010; Robin et al., 1994, 1999; Russell et al., 1987), one comparing individual treatment with individual treatment augmented with family treatment (Godart et al., 2012) and two trials comparing individual outpatient treatment to inpatient treatment (Crisp et al., 1991; Gowers et al., 2007).

Psychological treatment

Family therapy. There have been 11 RCTs in adolescent anorexia nervosa that have included family interventions, though of these interventions only the one created by investigators from the Maudsley Hospital in the 1980s (Family Based Treatment – FBT) has been systematically investigated (Lock, 2011). Based on the outcomes of these RCTs and a recently published systematic review (Hay, 2013b), there is a clear and growing body of evidence that supports the efficacy of family treatment in adolescent anorexia nervosa, in particular family treatment that focuses on eating disorder behaviours and weight gain. Of these treatments FBT has been the most extensively studied and has not only demonstrated efficacy in the treatment of adolescent anorexia nervosa but also superiority to some types of individual therapy. There is a general consensus that FBT is now the first-line treatment for adolescents with anorexia nervosa who are aged less than

19 years and have a duration of illness of less than three years (le Grange et al., 2010; Lock, 2011; Russell et al., 1987).

Individual therapy. A total of eight RCTs in adolescent anorexia nervosa have included individual therapy interventions, including five that have compared individual therapy with family treatment. The three largest of these trials compared family treatment with ego orientated individual therapy (EOIT) or a modification of it called adolescent focused therapy (AFT). Both therapies are psycho-dynamically informed, individual, adolescent psychotherapies focusing on issues of adolescent development including autonomy, self-efficacy, individuation, assertiveness and psychological barriers to eating (Lock et al., 2010; Robin et al., 1995, 1999). While family treatment led to significantly higher weight gain and menstruation at treatment completion and improved remission rates 12 months after treatment completion in comparison to EOIT/AFT (Lock et al., 2010), individual treatment did lead to improvements in weight, menstruation and eating disorder pathology. In the two remaining RCTs comparing family and individual treatment the individual interventions included a supportive or 'treatment as usual' (TAU) intervention (Russell et al., 1987) and an eating disorder specific CBT intervention (Ball and Mitchell, 2004). This second study was hampered by small numbers and demonstrated no difference between CBT and family therapy. A recently published case series has suggested that CBT-E (enhanced cognitive behavioural therapy) may be effective in adolescent anorexia nervosa (Dalle Grave et al., 2013).

TAU has been examined in three RCTs. It has been described as supportive, educational and problem centred, focusing on both eating disorder specific issues and those related issues considered to prolong eating disorder behaviours. In the two studies comparing TAU with either FBT or TAU and family treatment, outcomes from TAU were inferior (Godart et al., 2012; Russell et al., 1987). In the third trial, outcomes from TAU were equivalent to specialist inpatient care (Crisp et al., 1991).

Positive prognostic factors for FBT are early weight gain (approximately 2kg in the first four weeks), while individuals with more severe eating disorder symptoms (assessed using the eating disorder examination (Fairburn, 2008)) and/or comorbid OCD appear to have a poorer outcome or are more likely to need additional sessions of FBT (Lock et al., 2005). In addition, high expressed emotion families were shown to do better with separated FBT (Eisler et al., 2000).

There is little evidence to guide clinicians as to which adolescents may do poorly with family therapy and better with an individual approach. Lock et al. (2010) have emphasised the important of compliance. Thus, a relative contraindication to family therapy is inability on the part of the family to commit to the treatment for whatever reason, including parental illness (Lock et al., 2010). While there is clearly much need for further studies of individual therapy in adolescents, particularly CBT, evidence to date suggests that in those adolescents and their families who do not respond to, or are unable to engage in, FBT, options for individual interventions that may be considered would include AFT and eating disorder specific CBT, with little evidence to support TAU.

Inpatient treatment. Hospitalisation of adolescents with anorexia nervosa for the management of acute medical instability (e.g. hypothermia, hypotension, electrolyte

abnormalities and cardiac arrhythmias) is thought to be essential in preventing associated mortality (Golden et al., 2003; Katzman, 2005). However, the benefits of inpatient weight restoration and the assumption that hospital is the best venue for refeeding once medical stability has been achieved remain unsupported by current evidence.

There is little evidence to guide the role of inpatient care in adolescent anorexia nervosa. Expert consensus currently recommends outpatient therapy as the first-line treatment (NICE, 2004b). This position is supported by two RCTs comparing inpatient treatment for anorexia nervosa with a number of individual outpatient interventions (Crisp et al., 1991; Gowers et al., 2007). In both of these trials, adolescents with anorexia nervosa were admitted to psychiatric units with experience in treating eating disorders, though not restricted to the treatment of individuals with eating disorders. In both trials there was no significant difference in outcomes between inpatient treatment and outpatient individual therapy, though Gowers et al. (2010) reported improved treatment adherence and cost effectiveness with outpatient treatment. Patient satisfaction was highest with specialist treatment, either inpatient or outpatient (Gowers et al., 2010). Previous findings have suggested that outpatient care costs approximately 10% of the cost of inpatient care (Katzman et al., 2000).

Pharmacotherapy. There is insufficient evidence to recommend psychotropic medication in adolescents with anorexia nervosa. The use of anxiolytic or antidepressant medications to relieve symptoms should be done with caution.

Nutritional and medical treatment. There is a need to formulate appropriate nutritional goals for weight regain. There are, however, widespread views and practices in relation to weight goals in treatment. A UK and European survey of services identified a 24kg range of target-weights for a 14 year old girl of average height (Roots et al., 2006), albeit that prediction of physiological 'normality' is imprecise. BMI centiles can be utilised to predict the weight at which endocrinological normality will be achieved, but they need to be interpreted in the light of other physical assessments. Golden recommends a 'target-weight' between the 14th and 39th BMI percentile for age (Golden et al., 2008). Key and colleagues (2002) have promoted the use of pelvic ultrasound demonstration of ovarian follicles as an indicator of normal weight. This is likely to be achieved between the 13th and 30th BMI centile (Allan et al., 2010; Key et al., 2002; Madden et al., 2009). It is essential to note that as recommended healthy weight for height changes with age, BMI centile charts for children and adolescents must be utilised when determining weight goals in treatment. Charts are freely available from the World Health Organization and Centers for Disease Control and Prevention websites (CDC, 2013; WHO, 2013), as are the freely available computer program EpiInfo (CDC, 2008) and AnthroPlus (WHO, 2007), which can be used for calculating age related centiles.

Nutritional therapy in children and adolescents may be provided using regular food or special supplements and delivered orally or via nasogastric tubes to ensure timely provision of adequate nutrients (Rigaud et al., 2007; Zuercher et al., 2003). Gastrostomy feeding and parenteral nutrition have been utilised but should not be part of routine therapy (Diamanti et al., 2008; Findlay et al., 2011; Melchior and Corcos, 2009; Silber, 2008).

As with adults, initiation of nutritional therapy in significantly malnourished adolescents has risks, and should be undertaken carefully, by experienced clinicians who are cognisant

of the risk of refeeding syndrome (MARSIPAN, 2011). While there is disagreement and lack of clear evidence regarding the optimal rate of feeding (orally or by nasogastric tube), it is generally accepted that for medically unstable adolescents the process should proceed cautiously, that 'full feeds' for longer-term weight recovery should be achieved within 5–7 days of initiation, usually with the use of nasogastric tube feeding to ensure that nutrients are delivered, and that the risk of hypoglycaemia and electrolyte shifts is minimised (Kohn et al., 2011; MARSIPAN, 2011; O'Connor and Goldin, 2011). Many groups routinely supplement with phosphate and thiamine during this period (Kohn et al., 2011; MARSIPAN, 2011). Monitoring of serum electrolytes and minerals is important during initiation of feeding.[3]

Nutritional therapy needs to continue after the achievement of a healthy weight and discharge. This needs to involve regular monitoring of nutritional status (anthropometry, assessment of physiological function through measuring temperature, pulse, blood pressure and capillary refill, as well as intermittent measurement of biochemical parameters such as hormonal profiles and vitamin D). This should involve an experienced dietician who can assess nutrient intake and aid in setting appropriate dietary goals (MARSIPAN, 2011).

Sustained malnutrition in childhood and adolescence may be associated with a range of complications such as growth failure, pubertal delay, osteopaenia and osteoporosis, and in the longer-term increased risk of obesity, hypertension and heart disease are common sequelae. In order to minimise these risks long-term maintenance of healthy weight is important. Osteopaenia and osteoporosis are most likely to develop in girls who become malnourished early in pubertal development, and those with prolonged malnutrition and amenorrhoea (Swenne and Stridsberg, 2012; Turner et al., 2001). Worryingly, recovery from anorexia nervosa does not ensure resolution of osteopaenia (Wentz et al., 2007). It has been recognised that restoration of normal hormonal function via restoration and maintenance of normal weight is the best way of dealing with this problem (Misra and Klibanski, 2011). The common practice of prescription of the oral contraceptive pill in malnourished adolescent girls with amenorrhoea is not recommended because it does not improve bone density, and may provide false reassurance about physiological normality (Golden et al., 2002). Recently, use of physiological levels of oestrogen (via a hormonal patch) and progesterone in an RCT was associated with improvement in bone density, and may have a place in therapy (Misra and Klibanski, 2011; Misra et al., 2011).

Outcomes and prognosis. A small Swedish cohort of patients with adolescent-onset anorexia nervosa who were followed for 18 years experienced full recovery in 88%, despite more than a third having a persisting identifiable psychiatric disorder (Wentz et al., 2007). The outcomes for young onset eating disorders appear generally better than for older adolescent and adult onset eating disorders.

Transitioning from child and adolescent services into adult streams can be a potentially stressful and destabilising time for adolescents and their families or carers. Careful planning and appropriate levels of support are necessary to ensure this changeover proceeds smoothly.

Summary of recommendations

Recommendation	Grade
Outpatient treatment is the first-line treatment in adolescent anorexia nervosa	EBR I
For most children and adolescents with anorexia nervosa, family based therapy (FBT) or an alternate family therapy is the treatment of choice	EBR I
Individual therapy should be considered in older adolescents with anorexia nervosa where family therapy is inappropriate or not suitable	EBR II
Options for individual therapy include adolescent focused therapy	EBR II
Options for individual therapy include CBT	EBR III
'Treatment as usual' is not supported in adolescent anorexia nervosa	EBR II
Use anxiolytic or antidepressant or other medications with caution	CBR
Selective serotonin reuptake inhibitors (SSRIs) are not indicated in the acute or maintenance stages of anorexia nervosa	EBR I

Severe and long-standing anorexia nervosa

Introduction

People with severe and long-standing anorexia nervosa[4] have one of the most challenging disorders in mental health care (Strober et al., 2010; Wonderlich et al., 2012). They have the highest mortality rate of any mental illness with a marked reduction in life expectancy (Steinhausen et al., 2002; Arcelus et al., 2011) and impose a heavy burden on health and other public services. Furthermore, they are often under or unemployed, on sickness benefits, suffer multiple medical complications (renal, liver, cardiac failure and osteoporosis), have repeated admissions to general and specialist medical facilities and are frequent users of primary care services with considerable strain on carers and families.

Robinson (2009) has argued that those with a severe and enduring eating disorder (SEED) need to be considered as having a serious illness which comprises not only psychiatric and medical sequelae but family, social and occupational complications as well. He conducted a series of qualitative studies in which he found that people with SEED scored similarly to severely depressed people on quality of life measures. More worrisome was the finding that life skill scores were on a par with people with schizophrenia. As a result he advocates a psychiatric rehabilitation model that comprises long-term follow-up, crisis intervention, specific psychological interventions, and attention to substance misuse. He also includes basic self-care needs which pay attention to nutrition, housing, financial issues, recreational activities as well as occupational ones (Robinson, 2009).

Evidence of efficacy for treatment approaches for people with severe and enduring anorexia nervosa (SE-AN) is very limited (Hay et al., 2012). To date, there has only been one RCT (Touyz et al., 2013) that specifically tested two psychological treatments for such individuals. Touyz and colleagues (2013) compared two standard treatments (SSCM and CBT) which were modified for those patients who suffered from a profound and persistent disorder. The findings suggest that CBT for severe and enduring (SE) illness was superior in reducing core symptoms at follow-up, but that both CBT-SE and SSCM-SE contributed to improvements over time in health-related quality of life, body weight, depression and motivation to change. These findings should not only provide hope for those suffering from severe and enduring anorexia nervosa but also stimulate interest in developing new psychosocial treatments.

Clinical and research implications

People with severe and enduring anorexia nervosa require a special treatment paradigm as they have usually experienced multiple treatment failures and present with a myriad of mental health and medical problems (Wonderlich et al., 2012). Goals of therapy need to be reconceptualised. The general clinical wisdom to date has been to reduce the focus on changing eating disorder symptoms and instead work collaboratively with the individual in a measured manner to reduce harm, maintain symptom stability and in particular enhance their quality of life. Because of their history of negative treatment experiences and repeated treatment failures, both the clinician and patient often share the experience of hopelessness and despair about the likelihood of meaningful change.

Treatment

Paradigm of management for severe and long-standing eating disorders. Adapted from approaches discussed by Strober et al. (2010), Williams et al. (2010), and Wonderlich et al. (2012).

It is important to collaboratively agree and articulate goals with the patient and (where appropriate) significant others, so as to create an environment of support and comfort. The individual is best served by a safe and secure treatment strategy that allows them to feel contained but yet allows for very gradual change. Elements of this framework include the following:

- A prolonged period of assessment allowing the development of a shared understanding of the maintaining factors for their eating disorder and the identification of simple achievable goals that are embarked upon using extremely small steps.
- Focus on improved adaptive function as a primary goal. Restoration of a normal weight or BMI may not be a primary focus of treatment unless desired by the individual. Refeeding is a collaborative enterprise so as to avoid unnecessary distress and further evasion or avoidance of therapy.
- Changes to eating behaviours that improve nutrition and that are emphasised. This is done cognisant that most people with severe and long-standing anorexia nervosa can increase their caloric content to 1200 kilocalories per day without resultant weight gain or loss due to reduced energy metabolism or adaptations to starvation, although most people would lose weight on 1200 kilocalories. Careful encouragement so that any endeavours in this regard are recognised and the fear involved understood. There should be no reproach if aborted as this challenge can easily be visited again.

- Assessment and encouragement of improved interpersonal function and social or other activity that enables the experience of feelings of pleasure or mastery. This can also stimulate the individual's cognitive function. Independence and autonomy are actively fostered.
- Careful monitoring of physical health by an empathic medical practitioner with decisions being made in a supportive and respectful manner, maintaining safety and avoiding crises wherever possible. Short-term admissions for medical stabilisation can, however, be life-saving.
- Inclusion of meetings with family members and pertinent others providing education and ongoing support, with an aim to minimise anger and negative affect displayed towards the person with anorexia nervosa. It is also very important to give permission to loved ones to take leave of absence.
- Some patients benefit from multidisciplinary case management services offered through public hospitals or others who work with other long-term psychiatric patients.

Hospitalisation and partial hospitalisation. Many people with long-standing disorders have multiple previous negative experiences of inpatient care (La Puma et al., 2009). Hospitalisation should be in order to achieve realistic, collaboratively agreed goals of care or for achievement of medical rescue based on achieving well defined medical parameters such as systolic BP above 90mmHg, pulse rate above 50 bpm, normal white cell count or albumin, etc. General inpatient psychiatric hospitals tend to be ill suited for this type of patient. Refeeding, if required, should be undertaken by a medical team with both the knowledge and experience of treating such patients (George et al., 2004). George et al. have reported a pilot day hospital program designed specifically for those with severe and enduring anorexia nervosa. Such programs that cater specifically for the needs of people with severe and enduring symptomatology are not only able to retain them in longer-term treatment but result in clinically significant changes, for example unexpected but encouraging requests to be transferred to a traditional day program as greater changes in weight and lifestyle are desired.

Countertransference. Treating someone with severe and enduring anorexia nervosa is an entirely different experience to more conventional treatment and the work can be long-term and not immediately rewarding. However, clinicians who work with people who have long-standing eating disorders consistently report in the literature the importance of never giving up hope or expectation of improvement (Theander, 1985).

In the view of the authors of this CPG, no therapist should ever be placed in a position to take on people who have long-standing eating disorders unless there is a clear will to want to do so, as this often turns into a formidable task with the therapist's patience, anxieties and energies challenged, especially when the individual may be close to death. Outpatient teams usually comprise a psychiatrist and/or clinical psychologist, physician and dietitian. It is an absolute imperative that there is regular communication amongst team members and any medical and/or non-medical decisions are negotiated carefully with the individual. Therapists are reminded that the words of Tom Main in 'The ailment' (1957) remain relevant today: '*The sufferer who frustrates a keen therapist by failing to improve is always in danger of meeting primitive human behaviour disguised as treatment*' (Main, 1957: 9).

Summary of recommendations

Recommendation	Grade
Maintain realistic hope and expectations for improvement.	EBR II
Take a harm minimisation approach to nutrition, medical complications and weight control behaviours.	EBR II
Focus on supporting functions, relationships and quality of life.	EBR II
Collaboratively set achievable eating and health-related goals and be clear with the individual and family what the goals of treatment are.	EBR III
Reserve hospitalisation for medical rescue, management of psychiatric risk.	EBR II
Be prepared to treat comorbidity to improve quality of life.	EBR III
Have appropriate monitoring and management of medical and psychiatric risk.	EBR III
Communicate regularly with all team members.	EBR III
Meet with family members and relevant others on an ‘as-needs’ basis.	EBR III

Section two: bulimia nervosa and binge eating disorder

Introduction

This section addresses management guidelines for the major eating disorders found in those who are not underweight, namely bulimia nervosa and binge eating disorder. In the DSM-5 (APA, 2013), those who do not meet full diagnostic criteria for bulimia nervosa or binge eating disorder because of low frequency and/or duration of behaviours may be categorised under ‘other specified’ or if there is another reason for not meeting criteria (e.g. ‘binge’ episodes are not objectively large) the diagnosis may be ‘unspecified feeding or eating disorder’. Whilst there is little evidence base for these related disorders it is likely that strategies effective for the full disorder may also be effective for sub-threshold disorders.

Several systematic reviews published in the past decade are in agreement on the evidence base for psychosocial and pharmacological treatments in bulimia nervosa and binge eating disorder and where more research is required (Aigner et al., 2011; Bulik et al., 2007; Hay, 2013b; Hay and Claudino, 2012; Hay et al., 2009; NICE, 2004b; Wilson and Zandberg, 2012).

Assessment

Assessment of people with bulimia nervosa or binge eating disorder should include inquiry into characteristic eating disorder: (a) behaviours, namely binge eating (uncontrolled episodes of overeating large amounts of food), weight control behaviours that may or may not be compensatory for binge eating (self-induced vomiting, laxative, and/or diuretic

misuse), dietary restriction and/or fasting, compulsive or driven exercise and others such as insulin misuse in diabetic patients or misuse of diet pills or illicit stimulant drugs such as methamphetamine; and (b) cognitions of weight and/or shape overvaluation, and body image and eating preoccupations. People should be assessed for a past history of other eating disorders, especially anorexia nervosa, as this may be associated with increased likelihood of relapse and a poorer outcome in some (but not all) studies (Eckert et al., 1995; Goldschmidt et al., 2013; Mitchison et al., 2013; Vaz-Leal et al., 2011). Other common psychiatric co-morbidities are anxiety and mood disorder(s), impulse control and substance use disorder (Hudson et al., 2007; Lacey and Evans, 1986).

Physical examination is important as there is evidence of an increased risk of medical co-morbidities including, but not exclusive to, those associated with obesity, notably Type II diabetes, mellitus and hypertension (Kessler et al., 2013). Assessment should include measurement of weight, height, pulse rate and blood pressure and calculation of BMI. Serum biochemistry should be done to check for hypokalaemia and dehydration (effects of purging behaviours). Other assessments such as random glucose and cardiovascular examination and ECG should be done as medically indicated. Where primary psychological treatment is provided by a therapist without medical training, a general practitioner will need to assist with medical assessment and/or ongoing care.

Treatment

Psychological therapies. First-line treatment for bulimia nervosa and binge eating disorder in adults is an individual psychological therapy. The best evidence for such therapy is for CBT. CBT has been found to be superior consistently to wait-list control and most other psychological therapies for bulimia nervosa (NICE, 2004b). The evidence is weaker due to fewer trials in binge eating disorder where behavioural weight loss management is also effective in the short (Hay, 2013b; Hay et al., 2009) but not longer-term (Wilson et al., 2010).

A specific transdiagnostic enhanced therapy (CBT-E) developed by Fairburn (Fairburn, 2008) has been found more efficacious than other psychological approaches (Fairburn et al., 2009), although the specificity of CBT-E requires more evidence (Spielmans et al., 2013). As CBT-E is a well delineated and manualised form of CBT it is described here in detail. However, in accordance with evidence based practice, clinicians may apply variations of CBT and/or use other evidence based psychological therapies according to their expertise and individual preference. CBT/CBT-E has four well defined stages over 20 weeks. It begins with psychoeducation and a CBT informed formulation of the processes maintaining the person's disorder, and uses it to identify problems to be targeted in therapy. This is followed by the introduction of monitoring of key behaviours, establishment of regular meals and snacks, and within session weighing (sessions 1–7 over one month). The second stage (sessions 8 and 9, weeks 5 and 6) is a 'taking stock', or reflection and review phase with revisiting and modification of the formulation as appropriate. The third stage (sessions 10–17, weeks 7–14) is a personalised program where the main mechanisms maintaining the eating disorder are addressed. This includes the utilisation of behavioural experiments to reduce problematic behaviours, particularly those associated with weight/shape overvaluation such as body checking, and an additional module addressing a core maintaining factor, namely mood intolerance. Stage 4 (sessions 18–20, weeks 15–20) looks to the future, ensuring improvements are maintained and includes relapse prevention. A broad version

(CBT-Eb) has been developed to address additional core maintaining factors with three optional modules addressing interpersonal deficits, clinical perfectionism and low self-esteem if applicable. CBT-Eb has been found to have an advantage over the original 'focused' CBT-Ef for people with comorbid personality disorder or other complex psychopathology (Fairburn et al., 2009).

Self-help and scalability of CBT. Where access to a therapist is delayed or there are costs or other barriers, CBT can be provided as a first-step, or stand-alone therapy in guided self-help form. An example of such an evidence based self-help book that has been evaluated for delivery within 10 half-hour session times by Australian general practitioners (Banasiak et al., 2005) is *Bulimia Nervosa and Binge Eating: A Guide to Recovery* (Cooper, 1995). Pure or unguided self-help may be effective in binge eating disorder; however, it has poorer outcomes compared to guided self-help CBT or specialist provided CBT in bulimia nervosa (Hay, 2013b).

Wilson and Zandberg's (2012) systematic review similarly supported self-help CBT as an effective, accessible and time and cost-efficient alternative to specialist delivery of CBT. Furthermore, it has been translated into delivery via telemedicine and the internet. They noted, however, that most CBT-guided self-help books have not kept up to date with developments in CBT such as CBT-Eb (see above).

Other psychological therapies. There is a small and weak evidence base for interpersonal psychotherapy and dialectical behaviour therapy in both bulimia nervosa and binge eating disorder, and mindfulness in binge eating disorder (Kristeller and Wolever, 2011). Where therapists have expertise in these therapies and not in CBT and a CBT-trained therapist is not accessible then it may be appropriate to use either of these for adults. Findings are mixed for FBT in older adolescents or adults and, unlike in anorexia nervosa, FBT would not be first-line in bulimia nervosa or binge eating disorder (le Grange et al., 2007; Schmidt et al., 2007).

Pharmacotherapy. RCTs and meta-analysis have found that tricyclic antidepressants may be efficacious for people with bulimia nervosa (Flament et al., 2012; Hay and Claudino, 2012) but adverse effects limit clinical utility. In contrast, high dose fluoxetine (60mg/day) is effective for people with bulimia nervosa and this or other SSRI antidepressants are effective for both bulimia nervosa and binge eating disorder. The antiepileptic topiramate also is effective in both conditions and is associated with weight loss. However, topiramate may cause problematic side effects such as paresthesias and taste perversion (Arbaizar et al., 2008; Hay and Claudino, 2012).

Where psychological therapy is not available, antidepressants or antiepileptic medication such as topiramate[5] may be used (Flament et al., 2012; Hay and Claudino, 2012). However, trials of drug alone treatments have seldom followed participants up in the long-term, and therefore how long the medication should be continued for is unclear. High attrition rates and binge eating abstinence rates have consistently been found to be lower for drug alone treatments than when combined with CBT (Flament et al., 2012; Hay and Claudino, 2012). Trials in bulimia nervosa and binge eating disorder (Flament et al., 2012; Hay and Claudino, 2012) find an additive benefit for combined psychological and pharmacological treatment,

but findings are inconsistent. Thus, when people have limited response to psychotherapy alone, or they have a comorbid mood disorder such as depression, pharmacotherapy may have a role as an adjunctive treatment.

Indicators for admission. The majority of people with bulimia nervosa may be treated as outpatients. Although evidence is lacking, people who are not responding to outpatient care or otherwise have an increased risk (e.g. because of suicidality or pregnancy) may benefit from the increased intensity of therapy and eating supervision available in an inpatient or day patient unit.

Medical assessment. Most medical problems in people with bulimia nervosa occur as a result of purging behaviours. Table 4 lists these and their management. A dental evaluation should be considered if self-induced vomiting has been a prominent symptom. An increasing problem is comorbid weight disorder and metabolic syndrome, both of which may require further medical assessment and treatment.

Management of weight disorder

Many people who have a binge eating disorder and increasing numbers with bulimia nervosa are also obese with consequential medical complications. Behavioural weight loss therapy for those with bulimia nervosa may be as effective as CBT in reducing binge eating and more effective in attaining weight loss in the short-term, but not the longer-term (Wilson et al., 2010). Similarly, topiramate and/or orlistat may aid weight loss and binge eating in the short-term (Arbaizar et al., 2008; Golay et al., 2005; Grilo et al., 2005).

Where comorbid obesity is problematic some people may benefit from weight loss management strategies but evidence is weak for any specific approach (Bulik et al., 2012).

Outcomes for bulimia nervosa and binge eating disorder

Most people with bulimia nervosa, binge eating disorder or other specified feeding or eating disorders (OSFED) experience a good outcome in long-term follow-up studies, with 50% or more free of symptoms at five years or more (Fairburn et al., 2000; Steinhausen and Weber, 2009). Steinhausen and Weber conducted a quantitative analysis of outcome data from 79 studies of bulimia nervosa (Steinhausen and Weber, 2009). He reported a recovery rate of 45%, whilst 27% of patients improved considerably and 23% had a chronic protracted course and a crude mortality rate of 0.32%. There was a 10–32% mean rate of crossover to other eating disorder diagnoses, most commonly to OSFED, followed by anorexia nervosa. A low rate of conversion to binge eating disorder may have been partially because the term had not been introduced when many of the older outcome measures were performed. Childhood obesity, substance use disorder and having a personality disturbance have most consistently been poor predictors of outcomes in bulimia nervosa, although it has been difficult to establish such predictors across studies (NICE, 2004a; Steinhausen and Weber, 2009). The long delays from illness onset to presentation likely contribute to poorer outcomes but there is, to our knowledge, no direct evidence for this.

Summary of recommendations

Recommendation	Grade
During the assessment of adults with possible binge eating disorder or bulimia nervosa: • Take a history enquiring into any binge eating, dietary restriction and/or fasting, compulsive or driven exercise, or additional weight control behaviours. • Assess for cognitions of weight and/or shape overvaluation, and body image and eating preoccupations. • Enquire about any past history of eating disorders, or other psychiatric co-morbidities. • Conduct a physical examination, including measurement of weight, height, BMI calculation, pulse rate, and blood pressure. Consider cardiovascular examination as clinically indicated. • Arrange for serum biochemistry. Consider random glucose and ECG as indicated medically. • Consider involvement of a general practitioner and/or dentist as appropriate.	CBR
Consider admission to an inpatient or day program unit where there is increased risk of non-response to outpatient/community based care.	CBR
First-line treatment for bulimia nervosa and binge eating disorder in adults is an individual psychological therapy; the best evidence is for therapist-led CBT and a specific enhanced form, CBT-E focused, has been found to be more efficacious than some other psychological approaches. There is also evidence for CBT adapted for internet delivery, or in guided self-help form.	EBR I
Consider topiramate or orlistat for those with comorbid obesity, the latter for the effect of weight loss.	EBR II
Where psychological therapy is not available, evidence supports pharmacological treatment.	EBR I
High dose fluoxetine has the strongest evidence base for bulimia nervosa; other selective serotonin reuptake inhibitors are also effective in both bulimia nervosa and binge eating disorder.	EBR I
Monitor adverse effects of any antiepileptic or antidepressants used and modify use as required.	EBR II
Consider pharmacotherapy as an adjunctive treatment, since an additive benefit has been shown for combined psychological and pharmacological therapy.	EBR I
Further research is required into CBT regarding both the specificity of CBT-E, and other forms of delivery. RCTs of alternate treatment approaches, longer-term studies and the best management of comorbid obesity are also required.	EBR I

Section three: avoidant/restrictive food intake disorder (ARFID)

Introduction

ARFID (APA, 2013) is a new disorder to DSM-5. It replaces and extends the DSM-IV diagnosis of feeding disorder of infancy and early childhood as well as DSM-IV somatoform disorders that were characterised by phobic food avoidance. Due to general paucity of data and absence of published data concerning this condition in older adolescents or adults, consideration of ARFID in this guideline is confined to the following brief overview.

Overview

The key diagnostic features of ARFID are restriction of food intake accompanied by one of the following: significant weight loss; significant nutritional deficiency; marked interference with social functioning; or dependence on enteral feeds or oral supplements, in the absence of body image concerns. It may occur at any age. Data from three recently published studies on early onset eating disorder has shown that between 21.2% and 35.2% of children aged 12 years and under presenting with weight loss and deliberate food avoidance do not report abnormal body image or fear of weight gain. These children presented with similar physical complications of their malnutrition and similar rates of psychiatric comorbidity to children meeting diagnostic criteria for anorexia nervosa. To date there have been no published studies to guide appropriate treatment interventions or inform prognosis for this group (Madden et al., 2009; Nicholls et al., 2011a; Pinhas et al., 2011).

Section four: future research in treating eating disorders

The most important challenge for future research is the elucidation of assessment and treatment of the newly introduced disorders, especially ARFID. Second, although CBT is a first-line therapy for people with bulimia nervosa, further evaluation of CBT and alternate therapies is needed. This is particularly needed for anorexia nervosa (where in adults there is no clear first-line psychological therapy) and those with additional complex problems such as borderline personality disorder for which broader treatment approaches have been found to be associated with improved outcomes (Fairburn et al., 2009). More randomised controlled trials of approaches with an emerging evidence base, for example acceptance and commitment therapy (ACT) and psychodynamic therapy, and novel biological treatments such as neuromodulation and deep brain stimulation, are also needed.

From a psychopharmacological perspective, greater clarification of the neurobiological basis of eating disorders and mechanisms of action of treatments may help elucidate other treatment options, for example in bulimia nervosa antidepressants appear to have actions on satiety separate to their effects on mood modulation. In addition, data on the combination or augmentation of psychological and pharmacological approaches may guide further management of those who do not respond to first-line treatments.

More information on the long-term efficacy of treatments would be gained by longer-term trials and post-treatment follow-up, since many studies last less than six months. Inclusion and subgroup analyses of more heterogeneous and clinically complex patients (e.g. those

with severe co-morbidities, as seen clinically) would expand the data on effectiveness of what treatments work and for whom.

In bulimia nervosa even after treatment is successful in reducing behaviours such as binge eating and purging, abstinence rates may remain low. Since the long-term outcome is likely better if abstinence is achieved, improvement of such rates is important. Further identification of features differentiating those who achieve remission from those who remain symptomatic may clarify factors moderating outcome.

Further research is also needed into non-specialist therapist-guided self-help, given that in comparison to therapist-led CBT, adherence to therapy and outcomes may not be as good, especially in treatment of bulimia nervosa; more specificity about the provision of such guidance would also be helpful. In binge eating disorder associated with obesity, energy restriction has, in the short-term, been associated with weight loss; however, longer-term studies of weight reduction management in this population are required.

Future studies should also address specific treatment needs and approaches as they may apply to groups underrepresented in current research. This includes, but is not limited to males and to Aboriginal and Torres Strait Islander peoples and Māori.

Conclusion

Assessment and management of people with anorexia nervosa should be multidisciplinary and include specific specialist psychological therapies and family based treatments in younger people. Recommendations for treatment in this CPG were based on evidence (see Table 7) of variable levels. The evidence base for therapies is stronger in bulimia nervosa and binge eating disorder where a specific transdiagnostic CBT has a high level of evidence. CBT can also be provided in less intensive guided self-help and online forms for less severe eating disorders. The majority of people can be treated as an outpatient with inpatient or day patient care needed for more severe illness, and particularly low weight people with anorexia nervosa. In the absence of evidence, trials evaluating treatments for ARFID are urgently needed to guide clinical practice.

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Notes

1. At the time of writing the other major international diagnostic scheme, the World Health Organization's international classification of diseases and related health problems (ICD), was under revision. The 10th revision of the ICD (ICD-10) and previous DSM-IV schemes used similar diagnostic terms and the same numerical systems. The ICD-11 also proposes introducing binge eating disorder and ARFID. However, there is potential for confusion with the DSM-5 using some ICD-10 terms, e.g. 'atypical' anorexia nervosa and bulimia nervosa, with different criteria, and the 11th revision of the ICD may remove the requirement of an objectively large amount in the criterion for binge eating episodes (Al-Adawi et al., 2013).

2. The guideline will address the most common feeding and eating disorders but it does not cover Pica UFED, OSFED or rumination disorder. It does not address general management of obesity, other disorders of body image such as body dysmorphic disorder, subclinical problems of disordered eating or body dissatisfaction or the economic costs of eating disorders and their treatment.

3. At the time of writing, refeeding practices are currently under review in the *Journal of Adolescent Health*.

4. There is no agreed definition about how many years constitutes 'long-standing'; however, most researchers agree that it is at least several years (Tierney and Fox, 2009).

5. The use of topiramate for weight loss was approved in 2012 by the United States Food and Drug Administration but is not yet approved for this use in New Zealand or Australia.

References

Aardoom JJ, Dingemans AE, Spinhoven P, et al. (2013) Treating eating disorders over the internet: A systematic review and future research directions. *International Journal of Eating Disorders*. 46: 539–552.

Aigner M, Treasure J, Kaye W, et al. (2011) World Federation of Societies of Biological Psychiatry (WFSBP) guidelines for the pharmacological treatment of eating disorders. *World Journal of Biological Psychiatry* 12: 400–443.

Al-Adawi S, Bax B, Bryant-Waugh R, et al. (2013) Revision of ICD – Status update on feeding and eating disorders. *Advances in Eating Disorders* 1: 10–20.

Allan R, Sharma R, Sangani B, et al. (2010) Predicting the weight gain required for recovery from anorexia nervosa with pelvic ultrasonography: An evidence-based approach. *European Eating Disorders Review* 18: 43–48.

APA (2013) *Diagnostic and Statistical Manual of Mental Disorders Fifth Edition*. Washington DC: American Psychiatric Publishing.

Arbaizar B, Gómez-Acebo I and Llorca J (2008) Efficacy of topiramate in bulimia nervosa and binge-eating disorder: A systematic review. *General Hospital Psychiatry* 30: 471–475.

Arcelus J, Mitchell AJ, Wales J, et al. (2011) Mortality rates in patients with anorexia nervosa and other eating disorders: A meta-analysis of 36 studies. *Archives of General Psychiatry* 68: 724–731.

Australian Health Ministers Advisory Council (2013) A national framework for recovery-oriented mental health services. *Guide for practitioners and providers*. Available at: http://www.health.gov.au/internet/main/publishing.nsf/Content/mental-pubs-n-recovgde (accessed 7 October 2014).

Bahia A, Mascolo M, Gaudiani JL, et al. PseudoBartter syndrome in eating disorders. *International Journal of Eating Disorders* 45: 150–153.

Ball J and Mitchell P (2004) A randomized controlled study of cognitive behavior therapy and behavioral family therapy for anorexia nervosa patients. *Brunner-Mazel Eating Disorders Monograph Series* 12: 303–14.

Banasiak SJ, Paxton SJ and Hay PJ (2005) Guided self-help for bulimia nervosa in primary care: A randomised controlled trial. *Psychological Medicine* 35, 1283–1294.

Baxter J, Kingi TK, Tapsell R, et al. (2006) Prevalence of mental disorders among Māori in Te Rau Hinengaro: The New Zealand mental health survey. *Australian and New Zealand Journal of Psychiatry* 40: 914–923.

Bergh C, Brodin U, Lindberg G, et al. (2002) Randomized controlled trial of a treatment for anorexia and bulimia nervosa. *Proceedings of the National Academy of Sciences* 99: 9486–9491.

Bergh C, Eklund S, Eriksson M, et al. (1996) A new treatment of anorexia nervosa. *The Lancet* 348: 611–612.

Beumont P, Hay P and Beumont R (2003) Summary Australian and New Zealand clinical practice guideline for the management of anorexia nervosa (2003). *Australasian Psychiatry* 11: 129–133.

Birmingham CL, Goldner EM and Bakan R (1994) Controlled trial of zinc supplementation in anorexia nervosa. *International Journal of Eating Disorders* 15: 251–255.

Birmingham CL, Gutierrez E, Jonat L, et al. (2004) Randomized controlled trial of warming in anorexia nervosa. *International Journal of Eating Disorders* 35: 234–238.

Blitzer JR, Rollins N and Blackwell A (1961) Children who starve themselves: Anorexia nervosa. *Psychosomatic Medicine* 23: 369–383.

Bruch H (1978) *The Golden Cage*. Cambridge, Mass: Harvard University Press.

Bulik CM, Baucom DH, Kirby JS, et al. (2011) Uniting couples (in the treatment of) anorexia nervosa (UCAN). *International Journal of Eating Disorders* 44: 19–28.

Bulik CM, Berkman ND, Brownley KA, et al. (2007) Anorexia nervosa treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders* 40: 310–320.

Bulik CM, Marcus MD, Zerwas S, et al. (2012) The changing "weightscape" of bulimia nervosa. *American Journal of Psychiatry* 169: 1031–1036.

Butterfly Foundation (2012) *Paying the Price: The Economic and Social Impact of Eating Disorders in Australia*. Sydney: The Butterfly Foundation. Available at: http://thebutterflyfoundation.org.au/wp-content/uploads/2012/12/Butterfly_Report.pdf (accessed 7 October 2014).

Campbell IC, Mill J, Uher R, et al. (2011) Eating disorders, gene–environment interactions and epigenetics. *Neuroscience & Biobehavioral Reviews* 35: 784–793.

Carney T, Tait D and Touyz S (2007) Coercion is coercion? Reflections on trends in the use of compulsion in treating anorexia nervosa. *Australasian Psychiatry* 15: 390–395.

Carter JC, Mercer-Lynn KB, Norwood SJ, et al. (2012) A prospective study of predictors of relapse in anorexia nervosa: Implications for relapse prevention. *Psychiatry Research* 200: 518–523.

Casasnovas C, Fernández-Aranda F, Granero R, et al. (2007) Motivation to change in eating disorders: Clinical and therapeutic implications. *European Eating Disorders Review* 15: 449–456.

CDC (2008) *Centers for Disease Control and Prevention. EpiInfo 7*. Available at: www.cdc.gov/epiinfo/ (accessed 8 November 2013).

CDC (2013) *Centers for Disease Control and Prevention*. Available at: www.cdc.gov/ (accessed 8 November 2013).

Cinelli RL and O'dea JA (2009) Body image and obesity among Australian adolescents from indigenous and Anglo-European backgrounds: Implications for health promotion and obesity prevention among Aboriginal youth. *Health Education Research* 24: 1059–1068.

Cooper PJ (1995) *Bulimia Nervosa & Binge-Eating: A Guide to Recovery*. New York: New York University Press.

Couturier J, Kimber M and Szatmari P (2013) Efficacy of family-based treatment for adolescents with eating disorders: A systematic review and meta-analysis. *International Journal of Eating Disorders* 46: 3–11.

Crisp AH, Norton K, Gowers S, et al. (1991) A controlled study of the effect of therapies aimed at adolescent and family psychopathology in anorexia nervosa. *British Journal of Psychiatry* 159: 325–333.

Dalle Grave R, Calug S, Doll H, et al. (2013) Enhanced cognitive behaviour therapy for adolescents with anorexia nervosa: An alternative to family therapy? *Behaviour Research & Therapy* 51: 9–12.

Diamanti A, Basso M, Castro M, et al. (2008) Clinical efficacy and safety of parenteral nutrition in adolescent girls with anorexia nervosa. *Journal of Adolescent Health* 42: 111–118.

Dietitians Association Australia (2003) *Practice Recommendations for the Nutritional Management of Anorexia Nervosa in Adults*. Available at: daa.asn.au (accessed 18 December 2013).

Dietitians Association Of Australia (2012) Role statement for accredited practising dietitians practising in the area of nutrition support.

Eckert ED, Halmi K, Marchi P, et al. (1995) Ten-year follow-up of anorexia nervosa: Clinical course and outcome. *Psychological Medicine* 25: 143–156.

EDOS (Eating Disorders Outreach Service) (2011) *Access Pathways to Queensland Health Adult Inpatient Beds for Patients with Eating Disorders*. State of Queensland: Queensland Health.

Eisler I, Dare C, Hodes M, et al. (2000) Family therapy for adolescent anorexia nervosa: The results of a controlled comparison of two family interventions. *Journal of Child Psychology and Psychiatry* 41: 727–736.

Fairburn CG (2008) *Cognitive Behavior Therapy and Eating Disorders*. New York: Guilford Press.

Fairburn CG, Cooper Z, Doll HA, et al. (2000) The natural course of bulimia nervosa and binge eating disorder in young women. *Archives of General Psychiatry* 57: 659–665.

Fairburn CG, Cooper Z, Doll HA, et al. (2009) Transdiagnostic cognitive-behavioral therapy for patients with eating disorders: A two-site trial with 60-week follow-up. *The American Journal of Psychiatry* 166: 311–319.

Favaro A, Ferrara S and Santonastaso P (2003) The spectrum of eating disorders in young women: A prevalence study in a general population sample. *Psychosomatic Medicine* 65: 701–708.

Fichter MM, Quadflieg N, Nisslmüller K, et al. (2012) Does internet-based prevention reduce the risk of relapse for anorexia nervosa? *Behaviour Research and Therapy* 50: 180–190.

Findlay SM, Toews H and Grant C (2011) Use of gastrostomy tubes in children and adolescents with eating disorders and related illnesses. *Journal of Adolescent Health* 48: 625–629.

Fisher CA, Hetrick SE and Rushford N (2010) Family therapy for anorexia nervosa. *The Cochrane Database of Systematic Reviews* 4: CD004780.

Fisher M, Schneider M, Burns J, et al. (2001) Differences between adolescents and young adults at presentation to an eating disorders program. *Journal of Adolescent Health* 28: 222–227.

Flament MF, Bissada H and Spettigue W (2012) Evidence-based pharmacotherapy of eating disorders. *International Journal of Neuro-Psychopharmacology* 15: 189–207.

Gaudiani JL, Sabel AL, Mascolo M, et al. (2012) Severe anorexia nervosa: Outcomes from a medical stabilization unit. *International Journal of Eating Disorders* 45: 85–92.

Gentile MG, Pastorelli P, Ciceri R, et al. (2010) Specialized refeeding treatment for anorexia nervosa patients suffering from extreme undernutrition. *Clinical Nutrition* 29: 627–632.

George L, Thornton C, Touyz SW, et al. (2004) Motivational enhancement and schema-focused cognitive behaviour therapy in the treatment of chronic eating disorders. *Clinical Psychologist* 8: 81–85.

Godart N, Berrthoz S, Curt F, et al. (2012) A randomized controlled trial of adjunctive family therapy and treatment as usual following inpatient treatment for anorexia nervosa adolescents. *PLoS ONE [Electronic Resource]* 7: 1–9.

Godart NT, Perdereau F, Rein Z, et al. (2007) Comorbidity studies of eating disorders and mood disorders. Critical review of the literature. *Journal of Affective Disorders* 97: 37–49.

Golay A, Laurent-Jaccard A, Habicht F, et al. (2005) Effect of orlistat in obese patients with binge eating disorder. *Obesity Research* 13: 1701–1708.

Golden NH, Jacobson MS, Sterling WM, et al. (2008) Treatment goal weight in adolescents with anorexia nervosa: Use of BMI percentiles. *International Journal of Eating Disorders* 41: 301–306.

Golden NH, Katzman DK, Kreipe RE, et al. (2003) Eating disorders in adolescents: Position paper of the Society for Adolescent Medicine. *Journal of Adolescent Health* 33: 496–503.

Golden NH, Lanzkowsky L, Schebendach J, et al. (2002) The effect of estrogen-progestin treatment on bone mineral density in anorexia nervosa. *Journal of Pediatric and Adolescent Gynecology* 15: 135–143.

Goldschmidt AB, Peterson CB, Wonderlich SA, et al. (2013) Trait-level and momentary correlates of bulimia nervosa with a history of anorexia nervosa. *International Journal of Eating Disorders*. 46: 140–46.

Gowers SG, Clark A, Roberts C, et al. (2007) Clinical effectiveness of treatments for anorexia nervosa in adolescents: Randomised controlled trial. *British Journal of Psychiatry* 191: 427–435.

Gowers SG, Clark AF, Roberts C, et al. (2010) A randomised controlled multicentre trial of treatments for adolescent anorexia nervosa including assessment of cost-effectiveness and patient acceptability – The TOuCAN trial. *Health Technology Assessment* 14: 1–98.

Grilo CM, Masheb RM and Salant SL (2005) Cognitive behavioral therapy guided self-help and orlistat for the treatment of binge eating disorder: A randomized, double-blind, placebo-controlled trial. *Biological Psychiatry* 57: 1193–1201.

Guarda A, Pinto A, Coughlin J, et al. (2007) Perceived coercion and change in perceived need for admission in patients hospitalized for eating disorders. *American Journal of Psychiatry* 164: 108–114.

Gull WW (1874) Anorexia nervosa. *Transactions of the Clinical Society of London* 7: 22–28.

Gutierrez E and Vazquez R (2001) Heat in the treatment of patients with anorexia nervosa. *Eating and Weight Disorders: EWD* 6: 49–52.

Hart S, Abraham S, Franklin RC, et al. (2011a) Hypoglycaemia following a mixed meal in eating disorder patients. *Postgraduate Medical Journal* 87: 405–409.

Hart LM, Granillo MT, Jorm AF, et al. (2011b) Unmet need for treatment in the eating disorders: A systematic review of eating disorder specific treatment seeking among community cases. *Clinical Psychology Review* 31: 727–735.

Hartmann A, Weber S, Herpertz S, et al. (2011) Psychological treatment for anorexia nervosa: A meta-analysis of standardized mean change. *Psychotherapy and Psychosomatics* 80: 216–226.

Hatch A, Madden S, Kohn M, et al. (2010) Anorexia nervosa: Towards an integrative neuroscience model. *European Eating Disorders Review* 18: 165–179.

Hay PJ (2013a) What is an eating disorder? Implications for current and future criteria. *Australian and New Zealand Journal of Psychiatry* 47: 208–211.

Hay P (2013b) A systematic review of evidence for psychological treatments in eating disorders: 2005–2012. *International Journal of Eating Disorders* 46: 462–469.

Hay PJ and Carriage C (2012) Eating disorder features in indigenous Aboriginal and Torres Strait Islander Australian Peoples. *BMC Public Health* 12: 233.

Hay PJ and Claudino AM (2012) Clinical psychopharmacology of eating disorders: A research update. *International Journal of Neuro-Psychopharmacology* 15: 209–222.

Hay P, Bacaltchuk J, Stefano S, et al. (2009) Psychological treatments for bulimia nervosa and binging. *The Cochrane Database of Systematic Reviews* 4: CD000562.

Hay PJ, Mond J, Buttner P, et al. (2008) Eating disorder behaviors are increasing: findings from two sequential community surveys in South Australia. *PloS ONE* 3: e1541.

Hay PJ, Touyz S and Sud R (2012) Treatment for severe and enduring anorexia nervosa: A review. *Australian and New Zealand Journal of Psychiatry* 46: 1136–1144.

Herpertz-Dahlmann B (2009) Adolescent eating disorders: Definitions, symptomatology, epidemiology and comorbidity. *Child and Adolescent Psychiatric Clinics of North America* 18: 31–47.

Hoek HW and Van Hoeken D (2003) Review of the prevalence and incidence of eating disorders. *International Journal of Eating Disorders* 34: 383–396.

House J, Schmidt U, Craig M, et al. (2012) Comparison of specialist and nonspecialist care pathways for adolescents with anorexia nervosa and related eating disorders. *International Journal of Eating Disorders* 45: 949–956.

Hudson JI, Hiripi E, Pope HG, et al. (2007) The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry* 61: 348–358.

Hutton P (2013) Deep brain stimulation for anorexia nervosa. *The Lancet* 382: 305–306.

Jenkins PE, Hoste RR, Meyer C, et al. (2011) Eating disorders and quality of life: A review of the literature. *Clinical Psychology Review* 31: 113–121.

Katzman DK (2005) Medical complications in adolescents with anorexia nervosa: A review of the literature. *International Journal of Eating Disorders* 37: S52–S59; S87–S89.

Katzman DK (2012) Refeeding hospitalized adolescents with anorexia nervosa: Is "start low, advance slow" urban legend or evidence based? *Journal of Adolescent Health* 50: 1–2.

Katzman DK, Golden NH, Neumark-Sztainer D, et al. (2000) From prevention to prognosis: Clinical research update on adolescent eating disorders. *Pediatric Research* 47: 709–712.

Keel PK and Haedt A (2008) Evidence-based psychosocial treatments for eating problems and eating disorders. *Journal of Clinical Child & Adolescent Psychology* 37: 39–61.

Keski-Rahkonen A, Hoek HW, Susser ES, et al. (2007) Epidemiology and course of anorexia nervosa in the community. *American Journal of Psychiatry* 164: 1259–1265.

Kessler RC, Berglund PA, Chiu WT, et al. (2013) The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health surveys. *Biological Psychiatry* 73: 904–914.

Key A, Mason H, Allan R, et al. (2002) Restoration of ovarian and uterine maturity in adolescents with anorexia nervosa. *International Journal of Eating Disorders* 32: 319–325.

Keys A, Brožek J, Henschel A, et al. (1950) *The biology of human starvation (2 vols)*. Minneapolis: University of Minnesota Press.

Kim ST, Kang JS, Baek JW, et al. (2010) Acrodermatitis enteropathica with anorexia nervosa. *The Journal of Dermatology* 37: 726–729.

Kishi T, Kafantaris V, Sunday S, et al. (2012) Are antipsychotics effective for the treatment of anorexia nervosa? Results from a systematic review and meta-analysis. *The Journal of Clinical Psychiatry* 73: e757–e766.

Knowles L, Anokhina A and Serpell L (2012) Motivational interventions in the eating disorders: What is the evidence? *International Journal of Eating Disorders* 46: 97–107.

Kohn MR, Madden S and Clarke SD (2011) Refeeding in anorexia nervosa: Increased safety and efficiency through understanding the pathophysiology of protein calorie malnutrition. *Current Opinion in Pediatrics* 23: 390–394.

Kristeller JL and Wolever RQ (2011) Mindfulness-based eating awareness training for treating binge eating disorder: The conceptual foundation. *Eating Disorders* 19: 49–61.

La Puma M, Touyz S, Dean H, et al. (2009) To hospitalize or not to hospitalize patients with anorexia nervosa. In: Interventions for body image and eating disorders: Evidence and practice. Paxton SJ and Hay PJ (eds). Melbourne: IP Communications, pp. 95–118.

Lacey JH and Evans C (1986) The impulsivist: A multi-impulsive personality disorder. *British Journal of Addiction* 81: 641–649.

Lasegue C (1873) On hysterical anorexia. *Medical Times and Gazette* 2: 2: 265–266, 367–369.

Lask B and Bryant-Waugh R (1992) Early-onset anorexia nervosa and related eating disorders. *Journal of Child Psychology and Psychiatry and Allied Disciplines* 33: 281–300.

Lask B, Fosson A, Rolfe U, et al. (1993) Zinc deficiency and childhood-onset anorexia nervosa. *Journal of Clinical Psychiatry* 54: 63–66.

Le Grange D, Crosby RD, Rathouz PJ, et al. (2007) A randomized controlled comparison of family-based treatment and supportive psychotherapy for adolescent bulimia nervosa. *Archives of General Psychiatry* 64: 1049–1056.

Le Grange D, Eisler I, Dare C, et al. (1992) Evaluation of family treatments in adolescent anorexia nervosa: A pilot study. *International Journal of Eating Disorders* 12: 347–357.

Le Grange D, Lock J, Loeb K, et al. (2010) Academy for Eating Disorders position paper: The role of the family in eating disorders. *International Journal of Eating Disorders* 43: 1–5.

Lewinsohn PM, Striegel-Moore RH and Seeley JR (2000) Epidemiology and natural course of eating disorders in young women from adolescence to young adulthood. *Journal of the American Academy of Child & Adolescent Psychiatry* 39: 1284–1292.

Lipsman N, Woodside DB, Giacobbe P, et al. (2013a) Subcallosal cingulate deep brain stimulation for treatment-refractory anorexia nervosa: A phase 1 pilot trial. *The Lancet* 381: 1361–1370.

Lipsman N, Woodside DB, Giacobbe P, et al. (2013b) Neurosurgical treatment of anorexia nervosa: Review of the literature from leucotomy to deep brain stimulation. *European Eating Disorders Review* 21: 428–435.

Lock J (2011) Evaluation of family treatment models for eating disorders. *Current Opinion in Psychiatry* 24: 274–279.

Lock J, Agras WS, Bryson S, et al. (2005) A comparison of short- and long-term family therapy for adolescent anorexia nervosa. *Journal of the American Academy of Child & Adolescent Psychiatry* 44: 632–639.

Lock J, Le Grange D, Agras WS, et al. (2010) Randomized clinical trial comparing family-based treatment with adolescent-focused individual therapy for adolescents with anorexia nervosa. *Archives of General Psychiatry* 67: 1025–1032.

Lucas AR, Crowson CS, O'Fallon WM, et al. (1999) The ups and downs of anorexia nervosa. *International Journal of Eating Disorders* 26: 397–405.

McCabe MP, Ricciardelli L, Mellor D, et al. (2005) Media influences on body image and disordered eating among indigenous adolescent Australians. *Adolescence* 40: 115–127.

McIntosh VV, Jordan J, Carter FA, et al. (2005) Three psychotherapies for anorexia nervosa: A randomized, controlled trial. *American Journal of Psychiatry* 162: 741–747.

McIntosh VV, Jordan J, Luty SE, et al. (2006) Specialist supportive clinical management for anorexia nervosa. *International Journal of Eating Disorders* 39: 625–632.

Madden S, Morris A, Zurynski YA, et al. (2009) Burden of eating disorders in 5–13-year-old children in Australia. *The Medical Journal of Australia* 190: 410–414.

Maguire S, Le Grange D, Surgenor L, et al. (2008) Staging anorexia nervosa: Conceptualizing illness severity. *Early Intervention in Psychiatry* 2: 3–10.

Maguire S, Touyz S, Surgenor L, et al. (2012) The clinician administered staging instrument for anorexia nervosa: Development and psychometric properties. *International Journal of Eating Disorders* 45: 390–399.

Main TF (1957) The ailment. *British Journal of Medical Psychology* 30: 129–145.

Mariano P, Watson HJ, Leach DJ, et al. (2013) Parent–child concordance in reporting of child eating disorder pathology as assessed by the eating disorder examination. *International Journal of Eating Disorders*. 46: 617–625.

MARSIPAN (2011) Junior MARSIPAN: Management of really sick patients under 18 with anorexia nervosa (College Report CR168). *Royal College of Psychiatrists*.

MCNZ (Medical Council of New Zealand) (2006a) Statement of best practice when providing care to Māori patients and their whānau. Available at: www.mcnz.org.nz/ (accessed 7 October 2013).

MCNZ (Medical Council of New Zealand) (2006b) Statement of cultural competence. Available at: www.mcnz.org.nz/ (accessed 7 October 2013).

MCNZ (Medical Council of New Zealand) (2010) Best health outcomes for Pacific Peoples: Practice implications. Available at: www.mcnz.org.nz (accessed 7 October 2013).

Mehler PS, Cleary BS and Gaudiani JL (2011) Osteoporosis in anorexia nervosa. *Eating Disorders* 19: 194–202.

Mehler PS, Winkelman AB, Andersen DM, et al. (2010) Nutritional rehabilitation: Practical guidelines for refeeding the anorectic patient. *Journal of Nutrition and Metabolism* 1: 1–7.

Melchior J-C and Corcos M (2009) Parenteral nutrition and anorexia nervosa: Is it useful, is it ethical? *Journal of Adolescent Health* 44: 410–411.

Miller CA and Golden NH (2010) An introduction to eating disorders: Clinical presentation, epidemiology, and prognosis. *Nutrition in Clinical Practice* 25: 110–115.

Milos GF, Spindler AM, Buddeberg C, et al. (2003) Axes I and II comorbidity and treatment experiences in eating disorder subjects. *Psychotherapy & Psychosomatics* 72: 276–285.

Ministry of Health (2012a) The health of Maori adults and children: Key findings from the 2011/12 New Zealand Health Survey. Available at: www.health.govt.nz/ (accessed 9 October 2013).

Ministry of Health (2012b) The health of Pacific adults and children: Key findings from the 2011/12 New Zealand Health Survey. Available at: www.health.govt.nz/ (accessed 9 October 2013).

Misra M, Katzman D, Miller KK, et al. (2011) Physiologic estrogen replacement increases bone density in adolescent girls with anorexia nervosa. *Journal of Bone and Mineral Research* 26: 2430–2438.

Misra M and Klibanski A (2011) Bone health in anorexia nervosa. *Current Opinion in Endocrinology, Diabetes, and Obesity* 18: 376–382.

Mitchison D, Hay P, Mond J, et al. (2013) Self-reported history of anorexia nervosa and current quality of life: Findings from a community-based study. *Quality of Life Research* 22: 273–281.

Mitchison D, Hay P, Slewa-Younan S, et al. (2012) Time trends in population prevalence of eating disorder behaviors and their relationship to quality of life. *PloS ONE* 7: e48450.

Mitchison D and Hay PJ (2014) The epidemiology of eating disorders: Genetic, environmental, and societal factors. *Clinical Epidemiology* 6: 89–97.

Moher D, Liberati A, Tetzlaff J, et al. (2009) Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. *Annals of internal medicine* 151: 264–269.

Mond JM, Hay P, Rodgers B, et al. (2012) Quality of life impairment in a community sample of women with eating disorders. *Australian and New Zealand Journal of Psychiatry* 46: 561–568.

Murray SB and Touyz SW (2013) Muscle dysmorphia: Towards a diagnostic consensus. *Australian and New Zealand Journal of Psychiatry* 47: 206–207.

NEDC (2010) *The National Eating Disorders Collaboration: Eating disorders prevention, treatment and management: An evidence review*. Commonwealth Department of Health and Aging. Available at: http://www.nedc.com.au/files/pdfs/NEDC_Evidence%20Review_Final.pdf (accessed 7 October 2014).

NHMRC (2007) NHMRC standards and procedures for externally developed guidelines Available at: www.nhmrc.gov.au/guidelines/information-guideline-developers (accessed 8 January 2013).

NICE (National Institute for Clinical Excellence) (2004a) Eating disorders: Anorexia nervosa, bulimia nervosa and related eating disorders. Understanding NICE guidance: A guide for people with eating disorders, their advocates and carers, and the public.

NICE (National Institute for Clinical Excellence) (2004b) Eating disorders: Core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related eating disorders. In: NICE (ed). *National Clinical Practice Guideline CG9*. London: British Psyhcological Society and Gaskell.Available at: http://www.nice.org.uk/guidance/cg9 (accessed 7 October 2014).

Nicholls D, Hudson L and Mahomed F (2011a) Managing anorexia nervosa. *Archives of Disease in Childhood* 96: 977–982.

Nicholls DE, Lynn R and Viner RM (2011b) Childhood eating disorders: British national surveillance study. *British Journal of Psychiatry* 198: 295–301.

Nordbø RH, Gulliksen KS, Espeset E, et al. (2008) Expanding the concept of motivation to change: The content of patients' wish to recover from anorexia nervosa. *International Journal of Eating Disorders* 41: 635–642.

NZPB (New Zealand Psychologists Board) (2011) Core competencies for psychologists practicing within the "psychologist" scope of practice. Available at: www.psychologistsboard.org.nz/ (accessed 7 October 2013).

Oakley Browne MA, Wells JE, Scott KM, et al. (2006) Lifetime prevalence and projected lifetime risk of DSM-IV disorders in Te Rau Hinengaro: The New Zealand mental health survey. *Australian and New Zealand Journal of Psychiatry* 40: 865–874.

O'Connor G and Goldin J (2011) The refeeding syndrome and glucose load. *International Journal of Eating Disorders* 44: 182–185.

Ornstein RM, Golden NH, Jacobson MS, et al. (2003) Hypophosphatemia during nutritional rehabilitation in anorexia nervosa: Implications for refeeding and monitoring. *Journal of Adolescent Health* 32: 83–88.

Peebles R, Wilson JL and Lock JD (2006) How do children with eating disorders differ from adolescents with eating disorders at initial evaluation? *Journal of Adolescent Health* 39: 800–805.

Phillipou A, Rossell SL and Castle DJ (2014) The neurobiology of anorexia nervosa: A systematic review. *Australian and New Zealand Journal of Psychiatry* 48: 128–152.

Pike KM (2013) Classification, culture, and complexity: A global look at the diagnosis of eating disorders: Commentary on Wildes and Marcus: Incorporating dimensions into the classification of eating disorders. *International Journal of Eating Disorders* 46: 408–411.

Pike KM, Walsh BT, Vitousek K, et al. (2003) Cognitive behavior therapy in the posthospitalization treatment of anorexia nervosa. *American Journal of Psychiatry* 160: 2046–2049.

Pinhas L, Morris A, Crosby RD, et al. (2011) Incidence and age-specific presentation of restrictive eating disorders in children: A Canadian paediatric surveillance program study. *Archives of Pediatrics & Adolescent Medicine* 165: 895–899.

Preti A, Girolamo GD, Vilagut G, et al. (2009) The epidemiology of eating disorders in six European countries: Results of the ESEMeD-WMH project. *Journal of Psychiatric Research* 43: 1125–1132.

Raevuori A, Hoek HW, Susser E, et al. (2009) Epidemiology of anorexia nervosa in men: A nationwide study of Finnish twins. *PLoS ONE* 4: e4402.

RANZCP Clinical Practice Guidelines Team for Anorexia Nervosa (2004) *Australian and New Zealand clinical practice guidelines for the treatment of anorexia nervosa. Australian and New Zealand Journal of Psychiatry* 38: 659–670.

RANZCP (2009) Anorexia nervosa: Australian treatment guide for consumers and carers: Royal Australian and New Zealand College Of Psychiatrists (ed). Melbourne. Available at: https://www.ranzcp.org/Files/Resources/Publications/CPG/Australian_Versions/aus_anorexia_nervosa-pdf.aspx (accessed 7 October 2014).

RANZCP (2012) Australian Indigenous Mental Health. Available at: indigenous.ranzcp.org/index.php (accessed 7 October 2013).

Rhodes P, Baillee A, Brown J, et al. (2008) Can parent-to-parent consultation improve the effectiveness of the Maudsley model of family-based treatment for anorexia nervosa? A randomized control trial. *Journal of Family Therapy* 30: 96–108.

Ricciardelli LA, Mccabe MP, Ball K, et al. (2004) Sociocultural influences on body image concerns and body change strategies among Indigenous and non-Indigenous Australian adolescent girls and boys. *Sex Roles* 51: 731–741.

Rigaud D, Brondel L, Poupard AT, et al. (2007) A randomized trial on the efficacy of a 2-month tube feeding regimen in anorexia nervosa: A 1-year follow-up study. *Clinical Nutrition* 26: 421–429.

Robin AL, Siegel PT, Koepke T, et al. (1994) Family therapy versus individual therapy for adolescent females with anorexia nervosa. *Journal of Developmental & Behavioral Pediatrics* 15: 111–116.

Robin AL, Siegel PT and Moye A (1995) Family versus individual therapy for anorexia: Impact on family conflict. *International Journal of Eating Disorders* 17: 313–322.

Robin AL, Siegel PT, Moye AW, et al. (1999) A controlled comparison of family versus individual therapy for adolescents with anorexia nervosa. *Journal of the American Academy of Child & Adolescent Psychiatry* 38: 1482–1489.

Robinson P (2009) *Severe and Enduring Eating Disorder (SEED): Management of Complex Presentations of Anorexia and Bulimia Nervosa*. Chichester, UK; Malden, MA: Wiley-Blackwell.

Roots P, Hawker J and Gowers S (2006) The use of target weights in the inpatient treatment of adolescent anorexia nervosa. *European Eating Disorders Review* 14: 323–328.

Russell GF, Szmukler GI, Dare C, et al. (1987) An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry* 44: 1047–1056.

Russell J (2013) What is an eating disorder? The importance of nourishment comtrolling behaviours in the service of self-worth. *Australian and New Zealand Journal of Psychiatry* 47: 581–582.

Schmidt U, Lee S, Beecham J, et al. (2007) A randomized controlled trial of family therapy and cognitive behavior therapy guided self-care for adolescents with bulimia nervosa and related disorders. *American Journal of Psychiatry* 164: 591–598.

Schmidt U, Oldershaw A, Jichi F, et al. (2012) Out-patient psychological therapies for adults with anorexia nervosa: Randomised controlled trial. *The British Journal of Psychiatry* 201: 392–399.

Silber TJ (2008) A change of paradigm in the treatment of anorexia nervosa? Not so soon. *Journal of Adolescent Health* 42: 109–110.

Silverman JA (1983) Richard Morton, 1637–1698, limner of anorexia nervosa: His life and times. *JAMA: The Journal of the American Medical Association* 250: 2830–2832.

Smink FR, Van Hoeken D and Hoek HW (2012) Epidemiology of eating disorders: Incidence, prevalence and mortality rates. *Current Psychiatry Reports* 14: 406–414.

Smith A and Cook-Cottone C (2011) A review of family therapy as an effective intervention for anorexia nervosa in adolescents. *Journal of Clinical Psychology in Medical Settings* 18: 323–334.

Soh NL, Touyz SW and Surgenor LJ (2006) Eating and body image disturbances across cultures: A review. *European Eating Disorders Review* 14: 54–65.

Spielmans GI, Benish S, Marin C, et al. (2013) Specificity of psychological treatments for bulimia nervosa and binge eating disorder? A meta-analysis of direct comparisons. *Clinical Psychology Review* 33: 460–469.

Steinhausen H-C (2002) The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry* 159: 1284–1293.

Steinhausen H-C and Weber S (2009) The outcome of bulimia nervosa: Findings from one-quarter century of research. *American Journal of Psychiatry* 166: 1331–1341.

Steinhausen HC, Boyadjieva S, Griogoroiu-Serbanescu M, et al. (2003) The outcome of adolescent eating disorders: Findings from an international collaborative study. *European Child & Adolescent Psychiatry* 12 Suppl 1: 191–198.

Stice E (2002) Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin* 128: 825–848.

Stice E, Marti CN and Rohde P (2013) Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *Journal of Abnormal Psychology* 122: 445–457.

Striegel-Moore RH, Dohm FA, Kraemer HC, et al. (2003) Eating disorders in white and black women. *American Journal of Psychiatry* 160: 1326–1331.

Strober M (2014) Family-based treatment is overvalued: reply to Lock and Le Grange. *Advances in Eating Disorders: Theory, Research and Practice*. Epub ahead of print 7 April 2014, DOI: 10.1080/21662630.2014.898395.

Strober M, Freeman R and Morrell W (1997) The long-term course of severe anorexia nervosa in adolescents: Survival analysis of recovery, relapse, and outcome predictors over 10–15 years in a prospective study. *International Journal of Eating Disorders* 22: 339–360.

Strober M, Grilo C and Mitchell J (2010) The chronically ill patient with anorexia nervosa: Development, phenomenology, and therapeutic considerations. In: Grilo C and Mitchell JE (eds) *The Treatment of Eating Disorders: A Clinical Handbook*. New York: Guilford Press, pp.225–237.

Swanson SA, Crow SJ, Le Grange D, et al. (2011) Prevalence and correlates of eating disorders in adolescents. Results from the national comorbidity survey replication adolescent supplement. *Archives of General Psychiatry* 68: 714–723.

Swenne I and Stridsberg M (2012) Bone metabolism markers in adolescent girls with eating disorders and weight loss: Effects of growth, weight trend, developmental and menstrual status. *Archives of Osteoporosis* 7: 125–133.

Swinbourne J, Hunt C, Abbott M, et al. (2012) The comorbidity between eating disorders and anxiety disorders: Prevalence in an eating disorder sample and anxiety disorder sample. *Australian and New Zealand Journal of Psychiatry* 46: 118–131.

Tan JO, Stewart A, Fitzpatrick R, et al. (2010) Attitudes of patients with anorexia nervosa to compulsory treatment and coercion. *International Journal of Law and Psychiatry* 33: 13–19.

Theander S (1985) Outcome and prognosis in anorexia nervosa and bulimia: Some results of previous investigations, compared with those of a Swedish long-term study. *Journal of Psychiatric Research* 19: 493–508.

Tierney S and Fox JR (2009) Chronic anorexia nervosa: A Delphi study to explore practitioners' views. *International Journal of Eating Disorders* 42: 62–67.

Touyz S, Beumont P, Glaun D, et al. (1984) A comparison of lenient and strict operant conditioning programmes in refeeding patients with anorexia nervosa. *The British Journal of Psychiatry* 144: 517–520.

Touyz S, Le Grange D, Lacey H, et al. (2013) Treating severe and enduring anorexia nervosa: A randomized controlled trial. *Psychological Medicine* 43: 2501–2511.

Treasure J and Schmidt U (2013) DBS for treatment-refractory anorexia nervosa. *The Lancet* 381: 1338–1339.

Treasure J, Smith G and Crane A (2007) *Skills-Based Learning for Caring for a Loved One with an Eating Disorder: The New Maudsley Method*. London; New York: Routledge.

Turner J, Bulsara M, Mcdermott B, et al. (2001) Predictors of low bone density in young adolescent females with anorexia nervosa and other dieting disorders. *International Journal of Eating Disorders* 30: 245–251.

Van Elburg AA, Hillebrand JJ, Huyser C, et al. (2012) Mandometer treatment not superior to treatment as usual for anorexia nervosa. *International Journal of Eating Disorders* 45: 193–201.

Vaz-Leal FJ, Santos LR, García-Herráiz MA, et al. (2011) Bulimia nervosa with history of anorexia nervosa: Could the clinical subtype of anorexia have implications for clinical status and treatment response? *International Journal of Eating Disorders* 44: 212–219.

Vitousek K, Watson S and Wilson GT (1998) Enhancing motivation for change in treatment-resistant eating disorders. *Clinical Psychology Review* 18: 391–420.

Wade TD, Bergin JL, Tiggemann M, et al. (2006) Prevalence and long-term course of lifetime eating disorders in an adult Australian twin cohort. *Australian and New Zealand Journal of Psychiatry* 40: 121–128.

Walker T, Watson HJ, Leach DJ, et al. (2014) Comparative study of children and adolescents referred for eating disorder treatment at a specialist tertiary setting. *International Journal of Eating Disorders*.47: 47–54.

Waller G (2012) The myths of motivation: Time for a fresh look at some received wisdom in the eating disorders? *International Journal of Eating Disorders* 45: 1–16.

Watson HER, Dreher C, Steele A, et al. (2010) Eating disorders prevention, treatment & management: An evidence review. The National Eating Disorders Collaboration.

Watson HJ and Bulik CM (2012) Update on the treatment of anorexia nervosa: Review of clinical trials, practice guidelines and emerging interventions. *Psychological Medicine* 43: 2477–2500.

Watson TL, Bowers WA and Andersen AE (2000) Involuntary treatment of eating disorders. *American Journal of Psychiatry* 157: 1806–1810.

Wells JE, Browne MAO, Scott KM, et al. (2006) Prevalence, interference with life and severity of 12 month DSM-IV disorders in Te Rau Hinengaro: The New Zealand mental health survey. *Australian and New Zealand Journal of Psychiatry* 40: 845–854.

Wentz E, Mellström D, Gillberg IC, et al. (2007) Brief report: Decreased bone mineral density as a long-term complication of teenage-onset anorexia nervosa. *European Eating Disorders Review* 15: 290–295.

Whitelaw M, Gilbertson H, Lam PY, et al. (2010) Does aggressive refeeding in hospitalized adolescents with anorexia nervosa result in increased hypophosphatemia? *Journal of Adolescent Health* 46: 577–582.

WHO (World Health Organization) (2007) Growth reference data for 5–19 years. Available at: http://www.who.int/growthref/en/ (accessed 8 November 2013).

WHO (World Health Organization) (2013) WHO Child Growth Standards. Available at: http://www.who.int/en/ (accessed 8 November 2013).

Williams KD, Dobney T and Geller J (2010) Setting the eating disorder aside: An alternative model of care. *European Eating Disorders Review* 18: 90–96.

Wilson AJ, Touyz SW, O'Connor M, et al. (1985) Correcting the eating disorder in anorexia nervosa. *Journal of Psychiatric Research* 19: 449–451.

Wilson GT and Zandberg LJ (2012) Cognitive-behavioral guided self-help for eating disorders: Effectiveness and scalability. *Clinical Psychology Review* 32: 343–357.

Wilson GT, Wilfley DE, Agras WS, et al. (2010) Psychological treatments of binge eating disorder. *Archives of General Psychiatry* 67: 94–101.

Wonderlich S, Mitchell JE, Crosby RD, et al. (2012) Minimizing and treating chronicity in the eating disorders: A clinical overview. *International Journal of Eating Disorders* 45: 467–475.

Yager J, Devlin M, Halmi K, et al. (2006) American Psychiatric Association practice guideline for the treatment of patients with eating disorders. *American Journal of Psychiatry* 163(7 Suppl): 4–54.

Zipfel S, Wild B, Groß G, et al. (2014) Focal psychodynamic therapy, cognitive behaviour therapy, and optimised treatment as usual in outpatients with anorexia nervosa (ANTOP study): Randomised controlled trial. *The Lancet* 383: 127–137.

Zuercher JN, Cumella EJ, Woods BK, et al. (2003) Efficacy of voluntary nasogastric tube feeding in female inpatients with anorexia nervosa. *Journal of Parenteral and Enteral Nutrition* 27: 268–276.Table 1. Comparative clinical features of DSM-5 eating disorder diagnostic groups.

Tables and Figures

Figure 1. PRISMA flow diagram.
From: (Moher et al., 2009). For more information visit www.prisma-statement.org.

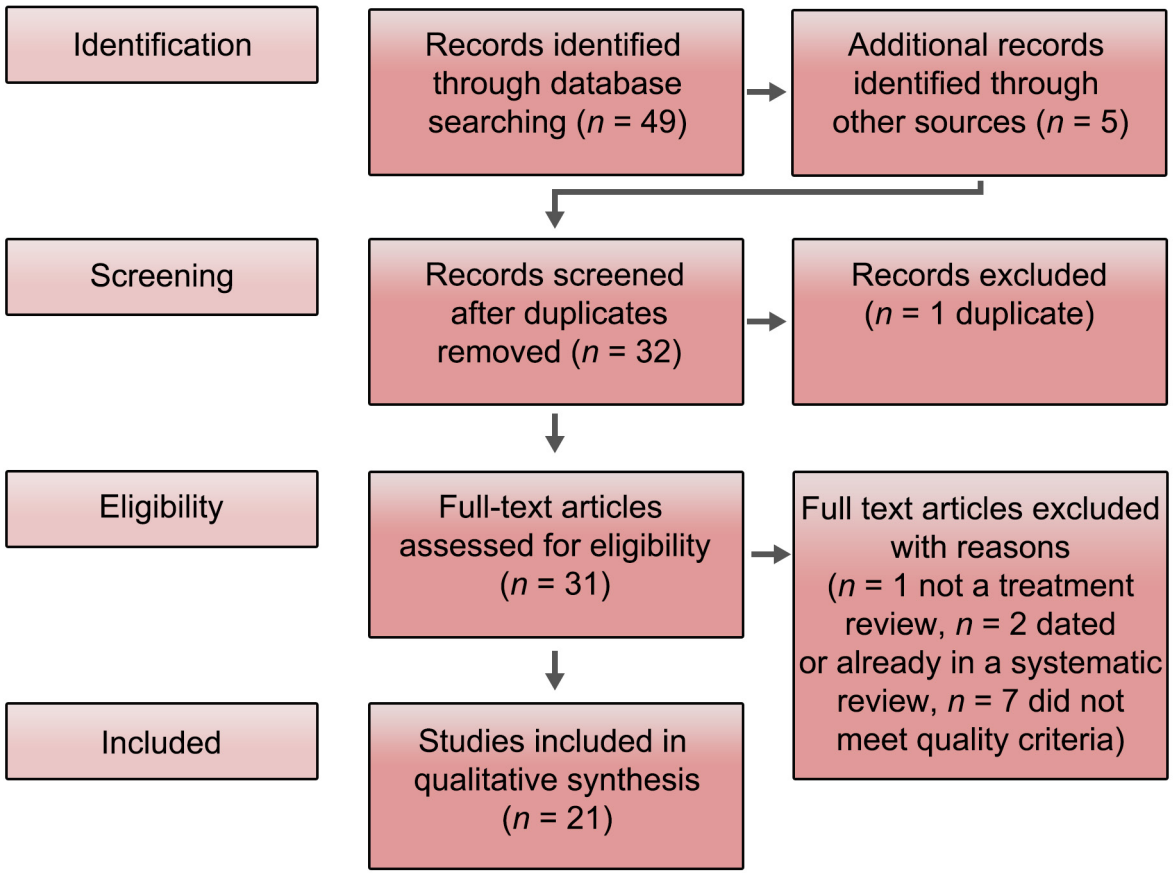

Table 1. Comparative clinical features of DSM-5 eating disorder diagnostic groups.

	Anorexia nervosa (AN)	Atypical anorexia nervosa[1]	Bulimia nervosa (BN)	Binge eating disorder	Avoidant/ restrictive food intake disorder	Purging disorder
Overvaluation of weight/shape	Required	Required	Required	May occur	Not required	May occur
Fear of fatness and/or behaviour preventing weight gain	Required	Required	May occur	Uncommon	No fear of fatness but food is restricted	May occur
Underweight	Required	Not present	NA	NA	May occur	May occur
Unmet nutritional and/or energy needs	Required	Required	May occur	NA	Required	May occur
Overweight	NA	May occur	May occur	Not required but is common	NA	May occur
Regular (weekly) binge eating	May occur	May occur	Required	Required with distress and 3/5 descriptors	NA	Absent
Regular (weekly) compensatory behaviours	May occur	May occur	Required	Do not occur	NA	Regular purging required, not compensatory

Table 1. Comparative clinical features of DSM-5 eating disorder diagnostic groups. (continued)

	Anorexia nervosa (AN)	Atypical anorexia nervosa[1]	Bulimia nervosa (BN)	Binge eating disorder	Avoidant/ restrictive food intake disorder	Purging disorder
AN not concurrent	NA	NA	Required	Required and no BN	Required and no BN	Not meeting full criteria for AN or ARFID
Subtypes	Restricting or binge purging	None	None	None	None	NA
Remission specifier	Partial/full	None	Partial/full	Partial/full	In remission	NA, is a subtype of OSFED
Severity specifier	BMI scale	None	Frequency of compensatory behaviours	Frequency of binge eating	None	None

Table 2. National Health and Medical Research Council designations of level of evidence based recommendation (EBR).

Level	Intervention	Diagnostic accuracy
EBR I	A systematic review of level II studies	A systematic review of level II studies
EBR II	A randomised controlled trial	A study of test accuracy with: an independent, blinded comparison with a valid reference standard, among consecutive persons with a defined clinical presentation
EBR III-1	A pseudo-randomised controlled trial (i.e. alternate allocation or some other method)	A study of test accuracy with: an independent, blinded comparison with a valid reference standard, among non-consecutive persons with a defined clinical presentation
EBR III-2	A comparative study with concurrent controls: • Non-randomised, experimental trial • Cohort study • Case-control study • Interrupted time series with a control group	A comparison with reference standard that does not meet the criteria required for level II and III-1 evidence
EBR III-3	A comparative study without concurrent controls: • Historical control study • Two or more single arm studies • Interrupted time series without a parallel control group	Diagnostic case-control study
EBR IV	Case series with either post-test or pre-test/post-test outcomes	Study of diagnostic yield (no reference standard)

Table 3. Indicators for consideration for psychiatric and medical admission for adults.

	Psychiatric admission indicated[a]	Medical admission indicated[b]
Weight	Body mass index (BMI) <14	BMI <12
Rapid weight loss	1kg per week over several weeks or grossly inadequate nutritional intake (<100kcal daily) or continued weight loss despite community treatment	
Systolic BP	<90 mmHg	<80 mmHg
Postural BP	>10 mmHg drop with standing	>20 mmHg drop with standing
Heart rate		≤40 bpm or > 120 bpm or postural tachycardia > 20/min
Temperature	<35.5°c or cold/blue extremities	<35°c or cold/blue extremities
12-lead ECG		Any arrhythmia including QTc prolongation, non-specific ST or T-wave changes including inversion or biphasic waves
Blood sugar	Below normal range*	< 2.5 mmol/L
Sodium	<130 mmol/L*	<125 mmol/L
Potassium	Below normal range*	<3.0 mmol/L
Magnesium		Below normal range*
Phosphate		Below normal range*

Table 3. Indicators for consideration for psychiatric and medical admission for adults.(continued)

	Psychiatric admission indicated[a]	Medical admission indicated[b]
eGFR		<60ml/min/1.73m^2 or rapidly dropping (25% drop within a week)
Albumin	Below normal range	<30 g/L
Liver enzymes	Mildly elevated	Markedly elevated (AST or ALD >500)[*]
Neutrophils	<1.5 × 10^9/L	<1.0 × 10^9/L
Risk assessment	Suicidal ideation Active self-harm Moderate to high agitation and distress	

**Please note, 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*

[a]Patients who are not as unwell as indicated above may still require admission to a psychiatric or other inpatient facility.

[b]Medical admission refers to admission to a medical ward, short stay medical assessment unit or similar.

Table 4. Physical and laboratory findings and their management.

System	Physical/lab findings	Action/investigation
Cardiac	• Bradycardia and/or hypotension and/or tachycardia and/or prolonged QT interval and/or arrhythmias[a]	• ECG • Cardiac monitoring • Cardiology consultation • Nutritional assessment/resuscitation • Re-hydration: preferential use of oral fluids because of risk of cardiac failure, note glucose based solutions may increase risk of refeeding syndrome
Core body temperature	• Hypothermia (may mask serious infection)	• Monitor; warm with external heat, nutrition
Endocrine	• Hypoglycaemia[b] • Poor metabolic control in co-existent Type I diabetes • Amenorrhoea • Secondary hyperaldosteronism[c]	• If in first week of refeeding, give thiamine; ensure adequate, steady carbohydrate supply and monitor blood glucose levels • Specialist management of diabetes • Nutritional restoration until menstruation returns[g] • Provision of very slow IV fluids
Fluid and electrolyte changes	• Hypokalaemia, hypochloraemia, metabolic alkalosis[c] • Hypophosphataemia (frequently emerges during refeeding) • Hypomagnesaemia[c] • Hyponatraemia	• Suspect purging, careful K+ replacement: best orally and correct alkalosis first, monitor closely • Phosphate Sandoz 500mg bd then recheck phosphate level, keep replacing until normal[e] • Replace magnesium • Suspect fluid loading, or over drinking as part of weight loss behaviours. 1.5 litre/day fluid restriction. Monitor in all patients
Haemato-logical	• Anaemia[d] • Neutropaenia	• Monitor in all patients. Consider iron level and stores of B_{12} and folate. Replace as necessary,[f] • Improve nutrition

Table 4. Physical and laboratory findings and their management. (continued)

System	Physical/lab findings	Action/investigation
Gastro-intestinal	• Severe acute pancreatitis[c,i] • Parotid and salivary gland hypertrophy[c] • Reduced gastric motility (and early satiety) • Mallory-Weiss tears, ruptures[c] • Oesophagitis • Constipation • Raised liver enzymes and low albumin	• Bowel rest, nasogastric suction and IV fluid replacement • Nil specific • Smaller but more frequent meals may be preferred • Urgent surgical referral • Consider proton pump inhibitor for severe symptoms – symptomatic relief for mild symptoms • Reassure, increase nutrition, stool softeners (do not use stimulant laxatives such as senna) • Monitor/improve nutrition
Skin/bone	• Osteopaenia, stress fractures • Brittle hair, hair loss, lanugo hair • Dorsal hand abrasions, facial purpura, conjunctival haemorrhage[c]	• Monitor bone density, nutritional restoration until menstruation returns, calcium[h] and Vitamin D, specialist referral • No specific treatment • No specific treatment
Dental	• Erosions and perimylolysis	• Dental referral

[a]Cardiac arrhythmia is a common cause of death.

[b]Hypoglycaemia in the first weeks is generally post prandial and occurs several hours after refeeding, hence some units preferentially use nasogastric feeding (Hart et al., 2011a).

[c]Complications caused by purging behaviours as well as starvation (Bahia et al., 2012).

[d]May be normocytic and normochronic, as characteristic of nutritional deficiency, but microcytic (iron-deficiency) is increasing as more people choose vegetarianism. Copper deficiency may also play a role.

[e]For patients at risk of refeeding syndrome (e.g. first 7–10 days of inpatient refeeding) prophylactic phosphate is recommended.

[f]Iron injections should not be given to the medically compromised patient as it is potentially hepatotoxic. Oral replacement is preferred.

[g]Oral contraceptives are not effective in restoring bone health.

[h]Phosphate required to prevent or treat refeeding syndrome should take precedence over calcium. Calcium should not be given at the same time as phosphate.

[i]Mild acute pancreatitis is almost universal and not an indication for the proposed intervention.

Sources: Eating Disorders Outreach Service, Queensland (EDOS, 2011) Royal Australian and New Zealand College of Psychiatrists (RANZCP, 2004).

Table 5. Guidelines for inpatient admission for children.

	Indicators for admission[a] and specialist consultation	
Medical status[b]	Heart rate	<50 bpm
	Cardiac arrhythmia	
	Postural tachycardia	> 20/min
	Blood pressure	<80/50 mm
	Postural hypotension	>20 mm
	QTc	>450 msec
	Temperature	<35.5°C
	Hypokalaemia	
	Neutropaenia	
Weight	***Children*** < 75% of expected body weight or rapid weight loss	

NB: These are a guide only and do not replace the need for individual clinical judgement.

[a]For children, admission would generally be to a medical ward.

[b]People may also require admission for:

- Uncontrolled eating disorder behaviour.
- Failure to respond to outpatient treatment.
- Severe psychiatric comorbidity.

Table 6. Randomised controlled psychotherapy trials in adolescent anorexia nervosa.

Study	*N*	Mean Age (Yrs)	Treatments	Results
1. Russell et al., 1987	21	16.6	Family based treatment (FBT) vs individual supportive therapy	FBT produced significantly superior outcome to individual treatment in a subset of 21 patients with anorexia nervosa of less than 3 years duration and onset prior to 19 years on Morgan Russell criteria at 12 month (60% vs 9%) and 5 year follow-up (90% vs 36%).
2. Crisp et al., 1991	90	21.7	Treatment as usual (TAU) with or without family treatment sessions vs group therapy vs inpatient treatment vs single assessment	No significant difference in outcomes between active treatment arms with all active treatments demonstrating significantly superior outcomes to a single assessment only.
3. le Grange et al., 1992	18	15.3	Conjoint family based treatment (CFT) vs separated family based treatment (SFT)	No significant difference in outcomes between CFT and SFT.
4. Robin et al., 1994[a]	24	14.7	Behavioural systems family therapy (BSFT) vs ego orientated individual therapy (EOIT)	BSFT produced significantly greater weight gain and higher rates of return of menstruation at the end of treatment. There were no differences in 12 month outcomes.

Table 6. Randomised controlled psychotherapy trials in adolescent anorexia nervosa. (continued)

Study	*N*	Mean Age (Yrs)	Treatments	Results
5. Robin et al., 1999[a]	37	14.2	BSFT vs EOIT	BSFT produced significantly greater weight gain and higher rates of return of menstruation at the end of treatment. There were no differences in 12 month outcomes.
6. Eisler et al., 2000	40	15.5	CFT vs SFT	No significant difference in outcomes between CFT and SFT on global outcomes, though families with high levels of expressed maternal criticism did better with SFT.
7. Ball and Mitchell, 2004	25	18.5	BSFT vs cognitive behavioural therapy (CBT)	No significant difference in outcomes between BSFT and CBT.
8. Lock et al., 2005	86	15.2	20 session FBT vs 10 session FBT	No significant difference in outcomes between the short and longer duration FBT, though post hoc analysis suggests individuals with severe obsessive compulsive eating disorder symptoms from non-intact families do better with longer treatment.
9. Gowers et al., 2007	167	14.9	Multidisciplinary inpatient psychiatric treatment vs specialist outpatient eating disorder treatment (CBT, parental counselling, dietary consultation, multimodal feedback) vs TAU in community mental health service	No significant differences in outcomes between the three interventions. Outpatient treatment more cost effective with higher treatment adherence. Increased parental satisfaction with specialist eating disorder treatment.

Table 6. Randomised controlled psychotherapy trials in adolescent anorexia nervosa. (continued)

Study	*N*	Mean Age (Yrs)	Treatments	Results
10. Rhodes et al., 2008	20	14.0	FBT vs FBT with parent to parent consultation	No significant differences in outcomes between the two treatment arms, though qualitative analysis suggested parents felt empowered and less alone in the parent to parent consultation arm.
11. Lock et al., 2010	121	14.4	FBT vs adolescent focused therapy (AFT)	FBT led to significantly greater weight gain and significantly greater reduction in the global eating disorder examination score at the end of treatment. No difference in remission rates at the end of treatment, though FBT demonstrated significantly higher remission rates at 6 and 12 month follow-up (49% vs 23%).
12. Godart et al., 2012	60	16.6	TAU and adjunctive relationship focused family therapy vs TAU	Adjunctive family therapy and TAU produced significantly superior outcomes to TAU using Morgan Russell criteria (40.0% vs 17.2% good outcome).

[a]The later trial by Robin and colleagues was an extension of the first and overlapped in design and participants.

Table 7. Role and evidence base support for specific treatments in eating disorders.

	Treatment	Indication	Grade
Anorexia nervosa	Family therapy	Improved eating disorder symptoms and weight	Level I – family based treatment
	Individual psychotherapy	Improved eating disorder symptoms and weight	Level I
	Antipsychotic medication	Improved weight gain	Level I – inconsistent support Level II – olanzapine, amisulpride only
		Reduced eating disorder symptoms	Level I – not supportive
		Improved mood	Level I – mixed support
		Reduced anxiety or ruminations	Level I – inconsistent support
	Antidepressant medication	Improved weight gain or relapse prevention	No robust and/or conflicting evidence
Bulimia nervosa	Individual psychotherapy	Reduction in binge eating & reduced eating disorder psychopathology	Level I – cognitive behaviour therapy especially CBT-E Level I – interpersonal psychotherapy Level II – dialectical behaviour therapy – weaker evidence
	Family therapy	Reduction in binge eating & reduced eating disorder psychopathology	Level II – conflicting evidence

Table 7. Role and evidence base support for specific treatments in eating disorders. (continued)

	Treatment	Indication	Grade
	Antidepressant medication	Reduction in binge eating especially when combined with psychotherapy	Level I – tricyclics, mono-amine oxidase inhibitors, selective serotonin reuptake inhibitors
			Level III – other antidepressant classes
		Relapse prevention	Level II – conflicting evidence, high attrition
	Anticonvulsants	Reduction in binge eating and purging Improved health-related quality of life	Level I – topiramate Level II – topiramate
Binge eating disorder	Individual psychotherapy	Reduction in binge eating & reduced eating disorder psychopathology	Level I – cognitive behaviour therapy especially CBT-E Level I – guided and pure self-help CBT Level I – interpersonal psychotherapy
	Behaviour weight loss therapy	Reduced binge eating and weight loss	Level II – dialectical behaviour therapy – weaker evidence Level I – but long-term effects unclear
	Antidepressant medication	Reduction in binge eating especially when combined with psychotherapy	Level I – SSRIs Level II – atomoxetine Level III – other antidepressant classes
	Mood stabilising medication	Reduced binge eating & improved weight loss in obese	Level I – topiramate Level II – zonisamide but problematic adverse effects

Austin Health

ACED Referral

U.R Number .....

Surname .....

Given Name(s) .....

Date of Birth .....

AFFIX PATIENT LABEL HERE

FAH067084

Referral Source

Dr.....

Address.....

Phone..... Fax.....

Provider No.....

Email.....

Signature.....

Date of Referral.....

Physical Parameters

Weight..... kg Height.....cm

Temperature.....°C

Lying pulse..... Lying BP.....

Standing pulse..... Standing BP.....

Menstrual status.....

Patient Address.....

.....

Home Phone.....

Mobile.....Gender.....

Medicare No.....

Diagnostics – Please attach results of the following investigations:

☐ ECG ☐ FBE ☐ UEC ☐ LFTs ☐ Ca, Mg, PO4

☐ Random glucose ☐ TFTs ☐ Iron studies

Medications

.....

.....

.....

.....

Recent Weight Trajectory

Current Eating Disorder Symptoms

Physical Symptoms including Syncope

Co-morbid Mental and Physical health diagnoses

Current Risk Issues

Family Situation

Treating Team (if applicable)

Please return completed forms to the Paediatric Eating Disorder Service for triage
Fax to 03 9496 5386
Email – Paediatriceatingdisorders@austin.org.au. Telephone – 03 9496 5000 pager 5515

Adolescent & Child Eating Disorder (ACED) Referral

C1.10

ST VINCENT'S HOSPITAL
MELBOURNE
A FACILITY OF ST VINCENT'S HEALTH AUSTRALIA

Austin Health

Body Image and Eating Disorders Treatment and Recovery Service (BETRS)

A joint mental health initiative of St Vincent's Melbourne and Austin Health

St Vincent's Hospital (Melbourne) Ltd
ABN 22 052 110 755

All correspondence and enquiries to:
BETRS
Email: betrs@svhm.org.au
Web: www.betrs.org.au
General enquiries: (03) 9231 5700
Clinical Intake Service: (03) 9231 5718

Rear of Caritas Christi Hospice
104 Studley Park Road
Kew VIC 3101
Tel: (03) 9231 5700

Austin Hospital
Acute Psychiatric Unit
PO Box 5555
Heidelberg VIC 3084
Tel: (03) 9496 6407

Thank you for providing a referral to the Body Image and Eating Disorders Treatment and Recovery Service (BETRS).

The following information is useful to include, along with anything else you feel it would be helpful for us to know:

- Consumer name, date of birth, address and telephone number
- Current eating disorder symptoms (restriction/ bingeing/ compensatory behaviours etc.)
- Current weight and height
- Recent weight trajectory
- History of eating disorder symptoms
- Co-morbid mental & physical health diagnoses
- Current risk issues
- Current medication
- Current treating team
- GP details (if not referrer)

Medical parameters

- blood pressure
- pulse
- temperature
- recent pathology (see below)
- ECG

Blood tests for Eating Disorders
Full blood count, Electrolytes, Urea, Creatinine
Calcium, Magnesium, Phosphate
Fasting/random blood glucose
Liver function tests
Thyroid function tests

Alternatively, please feel free to contact us on our intake line to discuss your referral further.

We can be contacted on 9231 5718 between 9:30am and 11:30am, Monday to Friday.

Please fax referrals to: 9231 5701

Eating disorders diagnoses for <18 years

Anorexia Nervosa (AN)

Restriction of energy intake relative to an individual's requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory and health status. Disturbance of body image, an intense fear of gaining weight, lack of recognition of the seriousness of the illness and/or behaviours that interfere with weight gain are also present.

Bulimia Nervosa (BN)

Binge eating (eating a large amount of food in a relatively short period of time with a concomitant sense of loss of control) with purging/compensatory behaviour (e.g. self-induced vomiting, laxative or diuretic abuse, insulin misuse, excessive exercise, diet pills) once a week or more for at least 3 months. Disturbance of body image, an intense fear of gaining weight and lack of recognition of the seriousness of the illness may also be present.

Avoidant / Restrictive Food Intake Disorder (ARFID)

Significant weight loss (or failure to achieve expected weight gain or faltering growth in children and adolescents), nutritional deficiency, dependence on nutritional supplement or marked interference with psychosocial functioning due to caloric and/or nutrient restriction, but without weight or shape concerns.

Binge Eating Disorder (BED)

Binge eating, in the absence of compensatory behaviour, once a week for at least 3 months. Binge eating episodes are associated with eating rapidly, when not hungry, until extreme fullness, and/or associated with depression, shame or guilt.

Other Specified Feeding and Eating Disorder (OSFED)

An eating disorder that does not meet full criteria for one of the above categories, but has specific disordered eating behaviours such as restricting intake, purging and/or binge eating as key features. This category includes Atypical Anorexia Nervosa, where all of the criteria for anorexia nervosa are met, except that despite significant weight loss, the individual's weight is within or above the normal range.

Unspecified Feeding or Eating Disorder (UFED)

Eating disorder behaviours are present, but they are not specified by the care provider.

Note that in children and adolescents, failure to gain expected weight or height, and/or delayed or interrupted pubertal development, should be investigated for the possibility of an eating disorder.

Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition
http://www.aedweb.org/images/updatedmedicalcareguidelines/AED-Medical-Care-Guidelines_English_02.28.17_NEW.pdf

Eating disorders diagnoses for >18 years

Anorexia Nervosa (AN)

Restriction of energy intake relative to an individual's requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory and health status. Disturbance of body image, an intense fear of gaining weight, lack of recognition of the seriousness of the illness and/or behaviours that interfere with weight gain are also present.

Bulimia Nervosa (BN)

Binge eating (eating a large amount of food in a relatively short period of time with a concomitant sense of loss of control) with purging/compensatory behaviour (e.g. self-induced vomiting, laxative or diuretic abuse, insulin misuse, excessive exercise, diet pills) once a week or more for at least 3 months. Disturbance of body image, an intense fear of gaining weight and lack of recognition of the seriousness of the illness may also be present.

Avoidant / Restrictive Food Intake Disorder (ARFID)

Significant weight loss, nutritional deficiency, dependence on nutritional supplement or marked interference with psychosocial functioning due to caloric and/or nutrient restriction, but without weight or shape concerns.

Binge Eating Disorder (BED)

Binge eating, in the absence of compensatory behaviour, once a week for at least 3 months. Binge eating episodes are associated with eating rapidly, when not hungry, until extreme fullness, and/or associated with depression, shame or guilt.

Other Specified Feeding and Eating Disorder (OSFED)

An eating disorder that does not meet full criteria for one of the above categories, but has specific disordered eating behaviours such as restricting intake, purging and/or binge eating as key features. This category includes Atypical Anorexia Nervosa, where all of the criteria for anorexia nervosa are met, except that despite significant weight loss, the individual's weight is within or above the normal range.

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Eating disorder behaviours are present, but they are not specified by the care provider.

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Guidance for clinical evaluation of children and adolescents with a possible eating disorder

Eating disorders are serious mental illnesses with significant medical and psychiatric sequelae, and have the highest mortality rate of any psychiatric disorder. They can impair the workings of all organ systems of the body, as well as school and family functioning. Individuals with an eating disorder may not recognize the seriousness of their illness and/or may be ambivalent regarding treatment and recovery. Early identification and timely intervention is crucial, as shorter duration of illness is associated with increased rates of recovery and greatly improved patient outcomes.

Initial evaluation is aimed at establishing the patient's diagnoses, excluding alternative causes for the clinical features present, performing a comprehensive nutritional and psychosocial assessment, appraising for medical complications, with determination of the severity of malnutrition, and overall medical and psychiatric risks for the young person. It is essential that in children and adolescents, failure to gain expected weight or height, and/or delayed or interrupted pubertal development, be investigated for the possibility of an underlying eating disorder.

Management is based on an evidence-based, developmentally appropriate and multidisciplinary team based approach, with mental health, medical and dietetic input recommended to optimise patient care and outcomes.

Comprehensive assessment includes:

History

- History of weight loss or change and how it was achieved. Reviewing past photos can additionally assist in appraising premorbid growth and development.
- Growth and feelings regarding weight & shape as a child and teenager.
- Body image – including importance of weight and shape on self-evaluation, body checking, fear of being fat / weight gain. Patient's desired weight.
- Nutritional history including typical food and fluid intake over a 24-hour period detailing quantity and variety of foods and fluids consumed, restriction of specific foods or food groups, calorie counting, usual shopping and cooking arrangements, family meals.
- Abnormal eating behaviours including meal time rituals, difficulties with eating in social settings.
- Compensatory behaviours and their frequency - fasting or dieting, self- induced vomiting, exercise, laxative, diuretic and insulin misuse, use of diet pills and/or other over-the-counter supplements. Last dental check.
- Exercise type, frequency, duration and intent (for enjoyment / compulsion / manage weight).
- Physical symptoms (e.g. palpitations, dizziness, syncope, dyspnoea, nausea, abdominal discomfort, early satiety, bloating, reflux, constipation, cold intolerance, insomnia, weakness and lethargy).
- Menstrual history, including presence of amenorrhoea, oligomenorrhoea, use of hormonal contraceptives.
- School and academic functioning.
- Developmental history, including temperament, attachment, relationships and early feeding difficulties.

- Mental health history including symptoms of mood, anxiety and substance use disorders, difficulties with concentration and memory.
- History of trauma (physical, sexual or emotional).
- Individual assessment including mental state examination, risk assessment including risk of self-harm and suicide.
- Family history including symptoms or diagnoses of eating disorders, obesity, mood & anxiety disorders, and substance use disorders.
- Family assessment including parenting style and consistencies, strengths and resources, impact of the illness, current level of concern, understanding of eating disorders and treatment and available social supports.

Examination

- Measure and plot weight (post void and in light clothing), height, BMI, mid parental height
- Lying and standing pulse rate and blood pressure
- Temperature
- Hydration
- Lanugo, alopecia, oedema, hypercarotenemia, skin and nail health
- Stigmata of purging - dorsal finger abrasions, parotidomegaly, oral and dental health
- Tanner staging
- Muscle size and strength

Investigations

- ECG – including rate, rhythm, QT_c
- Bloods
  - Full blood examination, electrolytes, calcium, magnesium, phosphate, liver function tests, thyroid function (TSH, FT3, FT4), iron studies, amylase, lipase, active vitamin B12, folate, vitamin D, glucose, zinc, ESR, coeliac screen, FSH / LH / oestradiol or testosterone, IGF1, IGF BP3.
- Radiology
  - Bone age x-ray and bone densitometry

It is vital to note that biochemical markers are often not an accurate indicator of the severity of malnutrition and degree of medical stability.

Criteria for hospitalisation <18 years

Inpatient treatment is advised for children and adolescents with an eating disorder with the following:

- **Cardiac and hemodynamic instability:**
  - Bradycardia <50bpm
  - Hypotension <80/50
  - Orthostasis
    - postural systolic hypotension >20mmHg
    - postural tachycardia >30bpm
    - recurrent syncope
  - Cardiac arrhythmia
  - ECG abnormality (e.g. prolonged QTc > 450msec)
  - Cardiac failure
  - Hypothermia <35.5 degrees Celsius
- **Other acute medical complication of malnutrition:**
  - Electrolyte abnormality (e.g. hypokalaemia, hyponatraemia, hypophosphataemia)
  - Hypoglycaemia
  - Dehydration
  - Other – e.g. pancreatitis
- **Acute food and fluid refusal for >48hrs**
- **Weight ≤ 75% median BMI for age and sex**
- **Rapid weight loss (>500g-1kg per week over consecutive weeks)**
- **Arrested growth and development**
- **Co-morbid psychiatric or medical illness precluding community treatment (e.g. severe depression, type 1 diabetes mellitus)**
- **Other psychiatric:**
  - Acute risk of suicide, severe self-harm
  - Uncontrolled eating disorder behaviours
- **Consider hospitalisation if:**
  - Inadequate response to community treatment
  - Limited social supports

ACED Multidisciplinary Clinical Review

Date
Attended by

Patient since ……..
☐ 1mo review ☐ 3mo ☐ 6mo ☐ 12mo ☐ other

Case overview and issues

Weight
Δwt
Height date
BMI z score

EBW %EBW
TW %TW

LMP

Physical stability

Psych Mx Meds
Rx modality
☐ FBT
☐ Other……

Last DEXA due

Last bloods due

Risk issues

Impressions and aims

Treatment plan / recommendations

Next scheduled appointments

Next ACED clinical review

Clinician
Name:
Designation:
Signature:

Psychiatrist / Paediatrician / Manager / Review Clinician
Name:
Designation:
Signature:

ACED Clinical Communication

Date

To:	**From:**
Name	Name
Practice	Practice
Address	Address
Phone	Phone
Fax	Fax
e-mail	e-mail

Regarding patient:
Name
UR
DOB
Address
Carers
Contact numbers

Key Issues	
	Weight Recent changes in wt Height date measured BMI Expected body wt %EBW Target wt %TW Last menstrual period Pulse lying standing BP lying standing Temperature Last DEXA due Last bloods due
Mental health management	Medications

Current eating disorder symptoms

ACED Clinical Communication

Family situation
School situation
Current risk issues
Other
Impressions
Treatment plan / recommendations
Next scheduled appointments

Girls 2-18 years

Pfizer Australia Pty Ltd
ABN 50 008 422 348
38-42 Wharf Road
West Ryde NSW 2114

Endorsed By Australasian Paediatric Endocrine Group

Surname

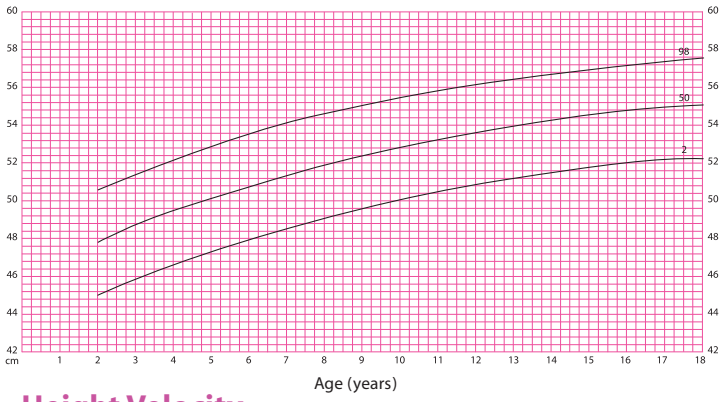
Given Names

Identification No.

Date of Birth

Head Circumference

Measuring Technique: The tape should be placed over the eyebrows, above the ears and over the most prominent part of the occiput taking a direct route. A paper tape is preferable to plastic, which stretches unacceptably under tension. The maximum measurement should be recorded to the nearest 0.1 cm.



Data Source: 2-5 yr: Jones, D.L. 1973, *NSW Health Communication Publication*. 5-18 yr: Nellhaus, G. 1968, *Pediatrics*, 41:106-114.

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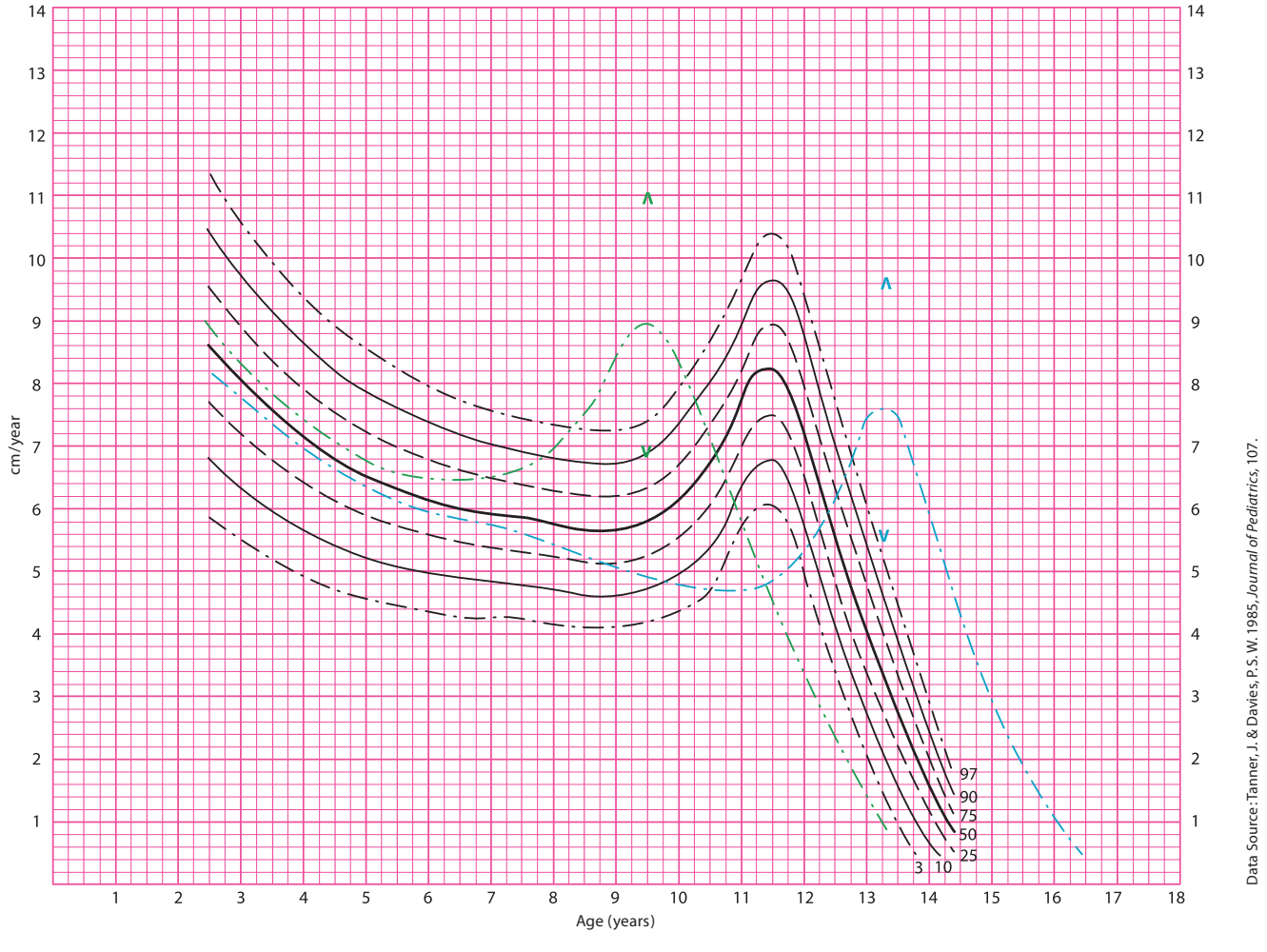
Centiles for girls maturing at average time — 97, 50, 3

97 and 3 centiles at peak height velocity for
Early (+ 2SD) maturers
Late (- 2SD) maturers



Data Source: Tanner, J. & Davies, P.S. W. 1985, *Journal of Pediatrics*, 107.

Height Percentile

Mother's Height

Father's Height

Simplified Calculation of Body Surface Area (BSA)

$$BSA\ (m^2) = \sqrt{\frac{Ht\ (cm) \times Wt\ (kg)}{3600}}$$

Reference: Mosteller, R. D. 1987, 'Simplified calculation of body surface area', *N. Engl. J. Med.*, 317:1098.

Supine Length (recommended up to the age of 3 so that there is overlap with standing height at 2 to 3) is taken on a flat surface, with the child lying on her back. One observer holds the child's head in contact with a board at the top of the table and another straightens the legs and turns the feet upward to be at right angles to the legs and brings a sliding board in contact with the child's heels.

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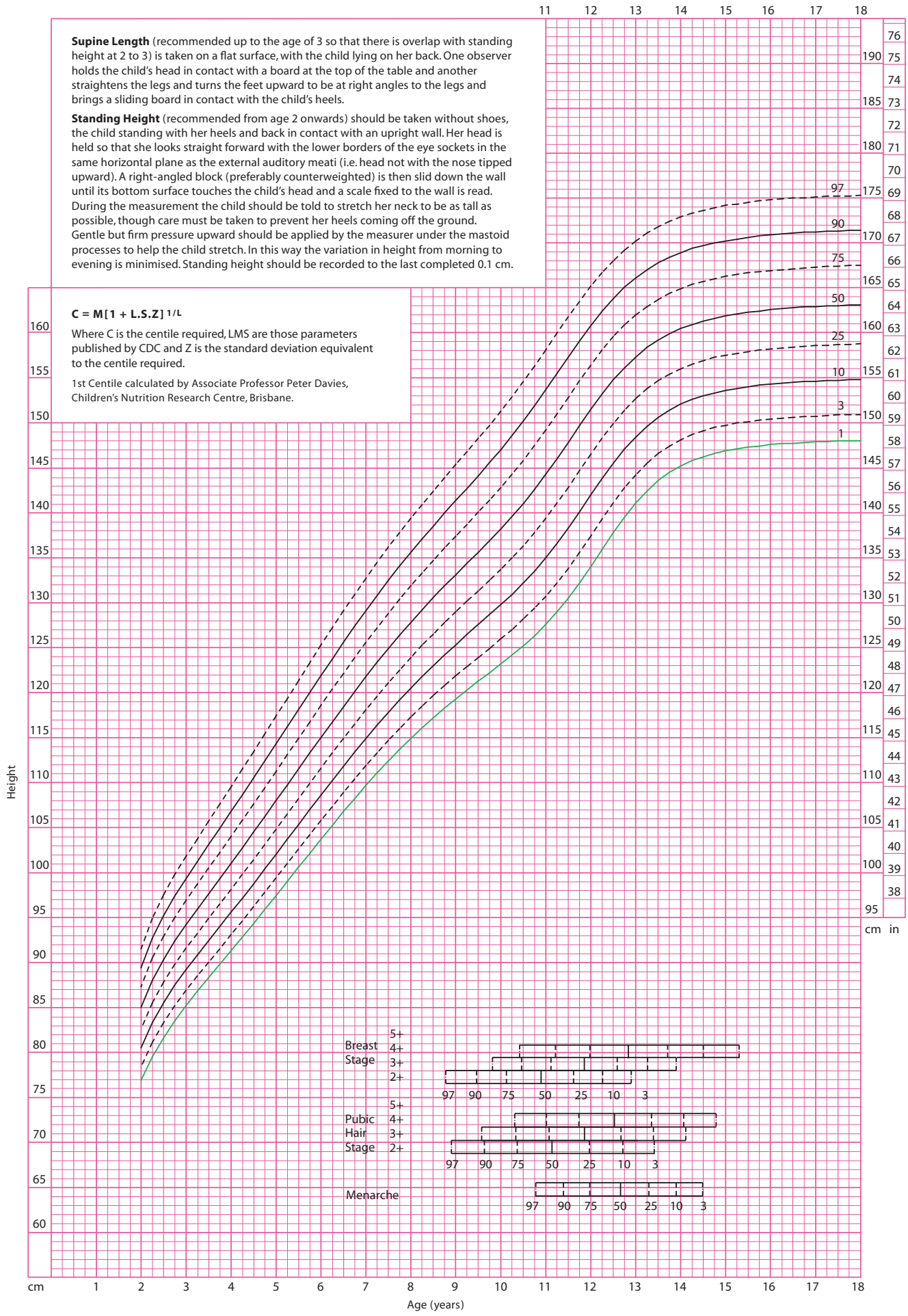
$$C = M[1 + L.S.Z]^{1/L}$$

Where C is the centile required, LMS are those parameters published by CDC and Z is the standard deviation equivalent to the centile required.

1st Centile calculated by Associate Professor Peter Davies, Children's Nutrition Research Centre, Brisbane.

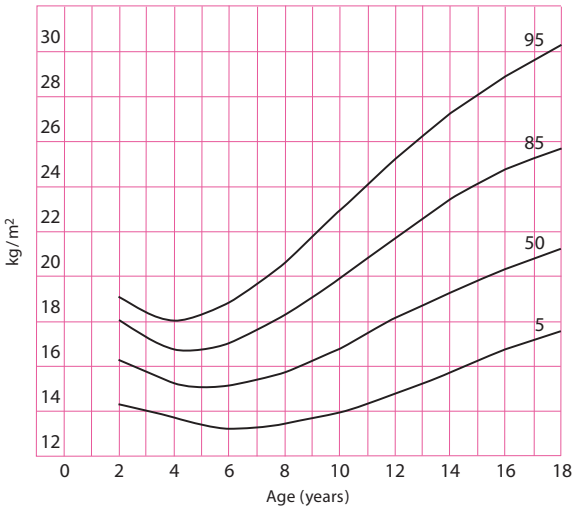


Data Source: www.cdc.gov/nchs/about/major/nhanes/growthcharts/datafiles.htm

Weight Percentile

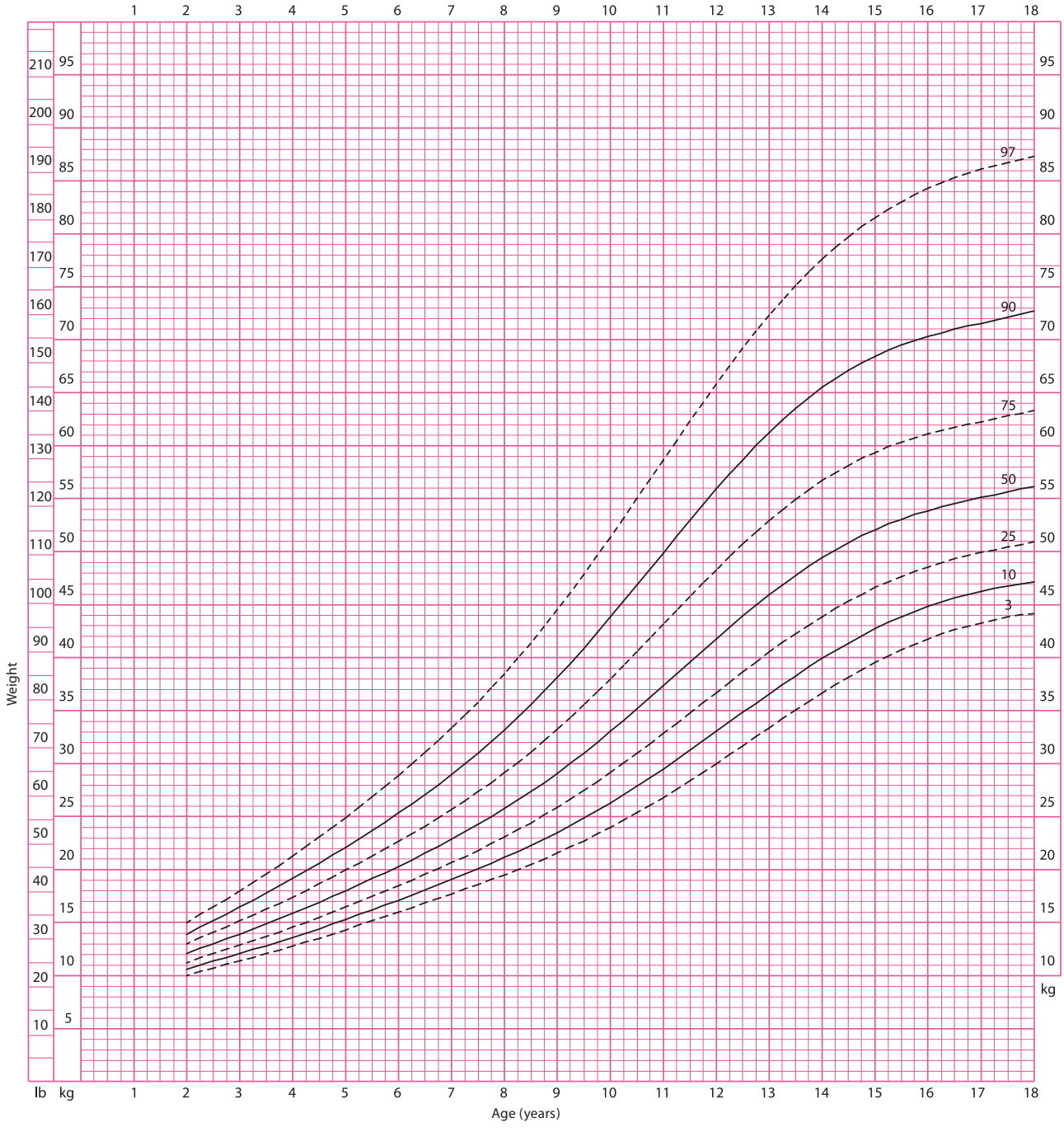
Weight should be taken in the nude, or as near thereto as possible. If a surgical gown or minimum underclothing (vest and pants) is worn, then its estimated weight (about 0.1 kg) must be subtracted before weight is recorded. Weights are conventionally recorded to the last completed 0.1 kg above the age of six months. The bladder should be empty.

Body-Mass Index



Data Source: www.cdc.gov/nchs/about/major/nhanes/growthcharts/datafiles.htm

					PUBERTAL STAGES		
DATE	AGE	HEIGHT	WEIGHT	HEAD CIRCUM.	BREAST	PUBIC HAIR	MENARCHE



Data Source: www.cdc.gov/nchs/about/major/nhanes/growthcharts/datafiles.htm

Girls 2 -18 years

Stages of Puberty

Ages of attainment of successive stages of pubertal sexual development are given in the Height Percentile chart.
The stage Pubic Hair 2+ represents the state of a child who shows the pubic hair appearance stage 2 but not stage 3 (see below).

The centiles for age at which this state is normally seen are given, the 97th centile being considered as the early limit, the 3rd centile as the late limit. The child's puberty stages may be plotted at successive ages (Tanner. 1962, *Growth at Adolescence*, 2nd edn).

Pubic Hair Development

Stage 1. Pre-adolescent. The vellus over the pubes is not further developed than that over the abdominal wall, i.e. no pubic hair.

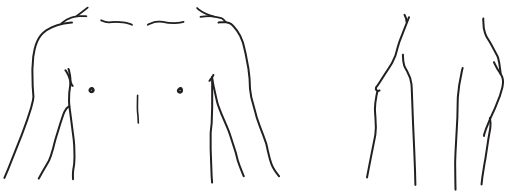
Stage 2. Sparse growth of long, slightly pigmented downy hair, straight or slightly curled, chiefly along labia.

Stage 3. Considerably darker, coarser and more curled. The hair spreads sparsely over the junction of the pubes.

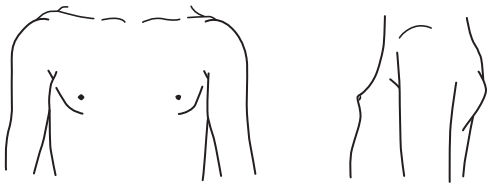
Stage 4. Hair now adult in type, but area covered is still considerably smaller than in the adult. No spread to the medial surface of thighs.

Stage 5. Adult in quantity and type with distribution of the horizontal (or classically 'feminine') pattern. Spread to medial surface of thighs but not up linea alba or elsewhere above the base of the inverse triangle (spread up linea alba occurs late and is rated stage 6).

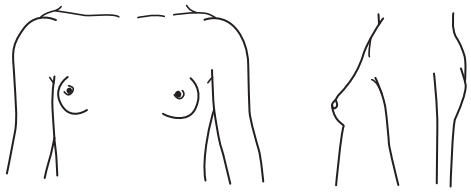
Breast Development Stages



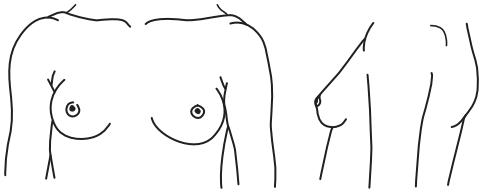
Stage 1. Prepubertal



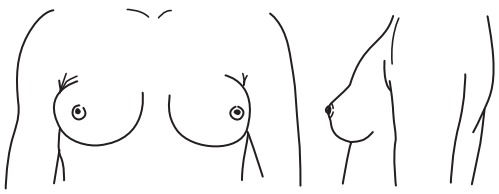
Stage 2. Elevation of breasts and papilla



Stage 3. Further elevation and areola but no separation of contours

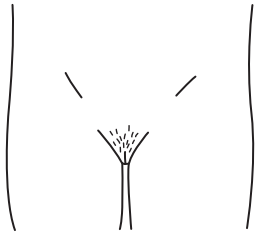


Stage 4. Areola and papilla form a secondary mound above level of the breast



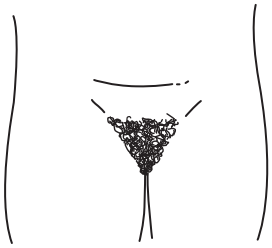
Stage 5. Areola recesses to the general contour of the breast

Pubic Hair Stages



Stage 2

Stage 3



Stage 4

Stage 5

1841.04/08
25060210

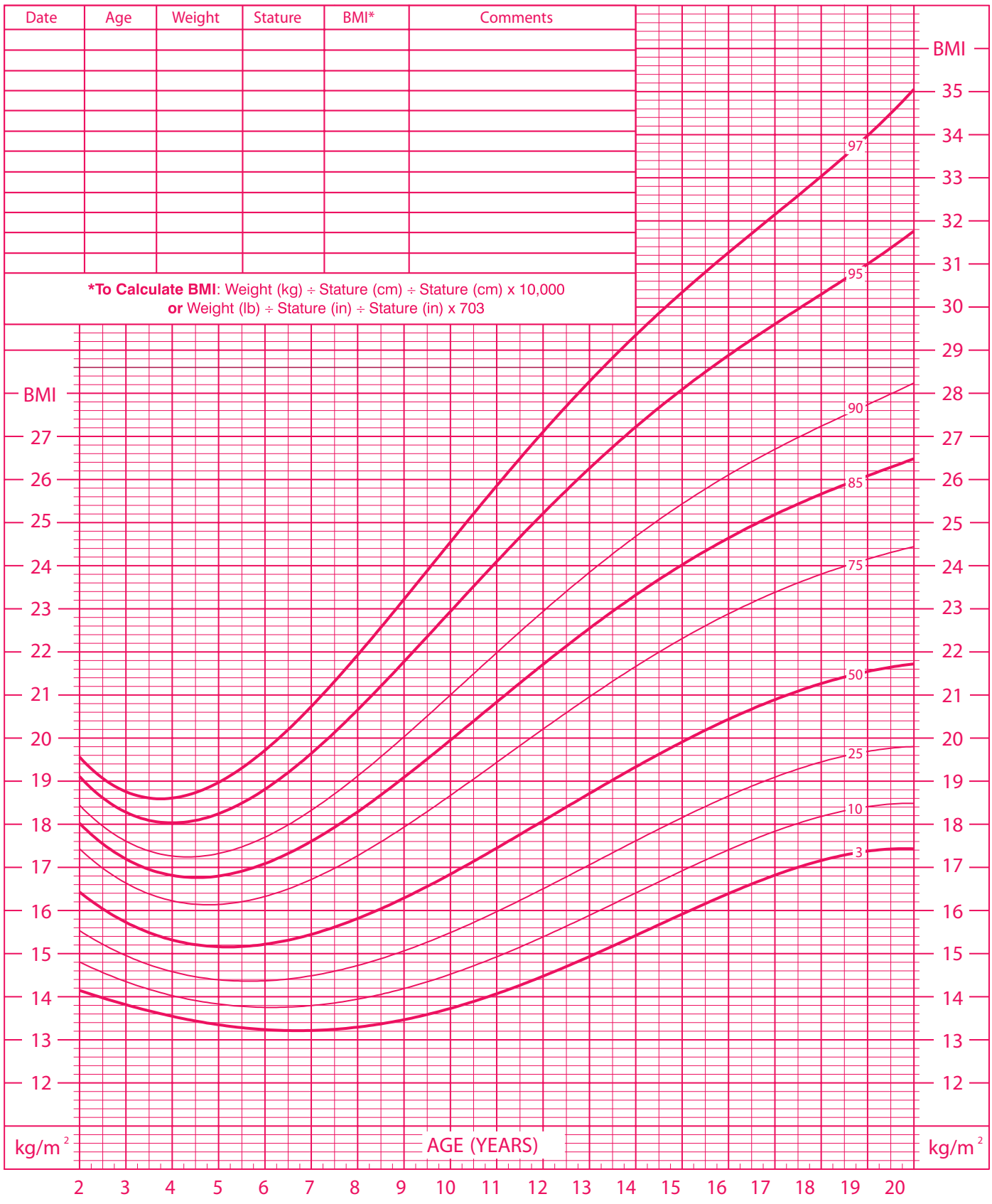
2 to 20 years: Girls
Body mass index-for-age percentiles

NAME _____

RECORD # _____

Date	Age	Weight	Stature	BMI*	Comments

***To Calculate BMI**: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 10/16/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

SAFER • HEALTHIER • PEOPLE™

Boys 2-18 years

Pfizer Australia Pty Ltd
ABN 50 008 422 348
38-42 Wharf Road
West Ryde NSW 2114

Endorsed By Australasian Paediatric Endocrine Group

Surname

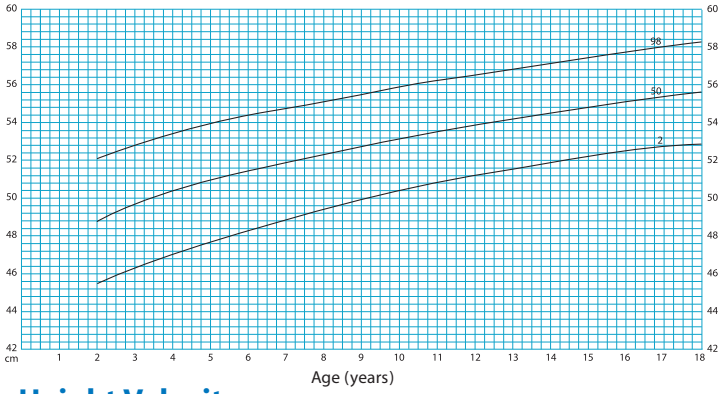
Given Names

Identification No.

Date of Birth

Head Circumference

Measuring Technique: The tape should be placed over the eyebrows, above the ears and over the most prominent part of the occiput taking a direct route. A paper tape is preferable to plastic, which stretches unacceptably under tension. The maximum measurement should be recorded to the nearest 0.1 cm.



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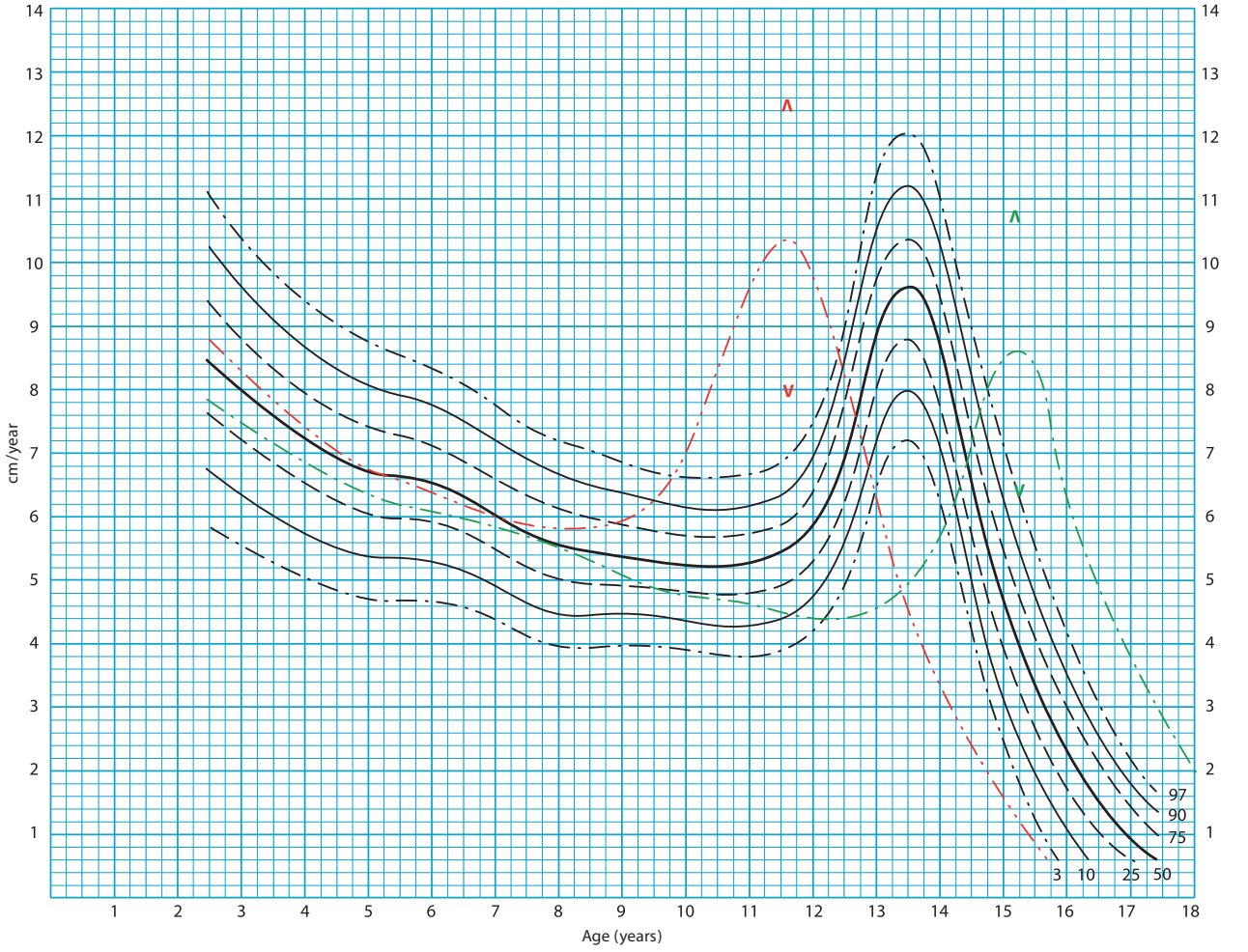
Centiles of a whole year velocity for maturers at average time — 97, 50, 3

97 and 3 centile at peak height velocity for
Early (+ 2SD) maturers
Late (- 2SD) maturers



Data Source: Tanner, J. & Davies, P.S. W. 1985, *Journal of Pediatrics*, 107.

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Father's Height

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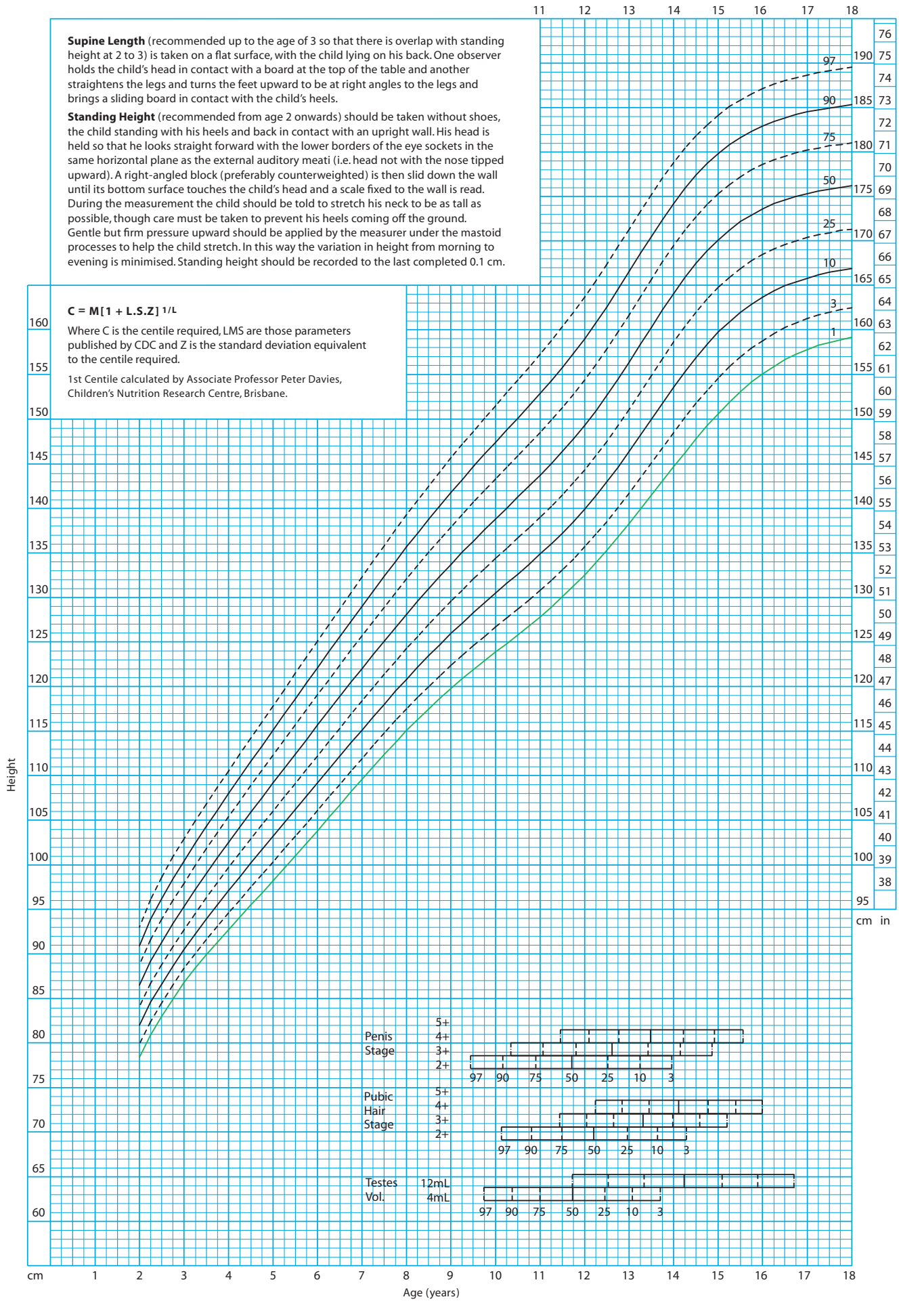

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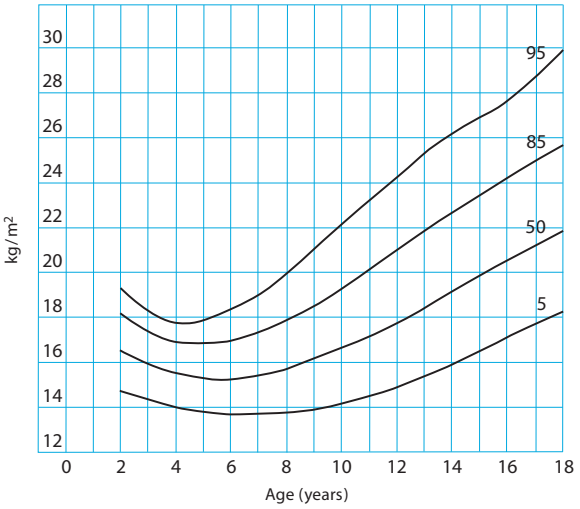

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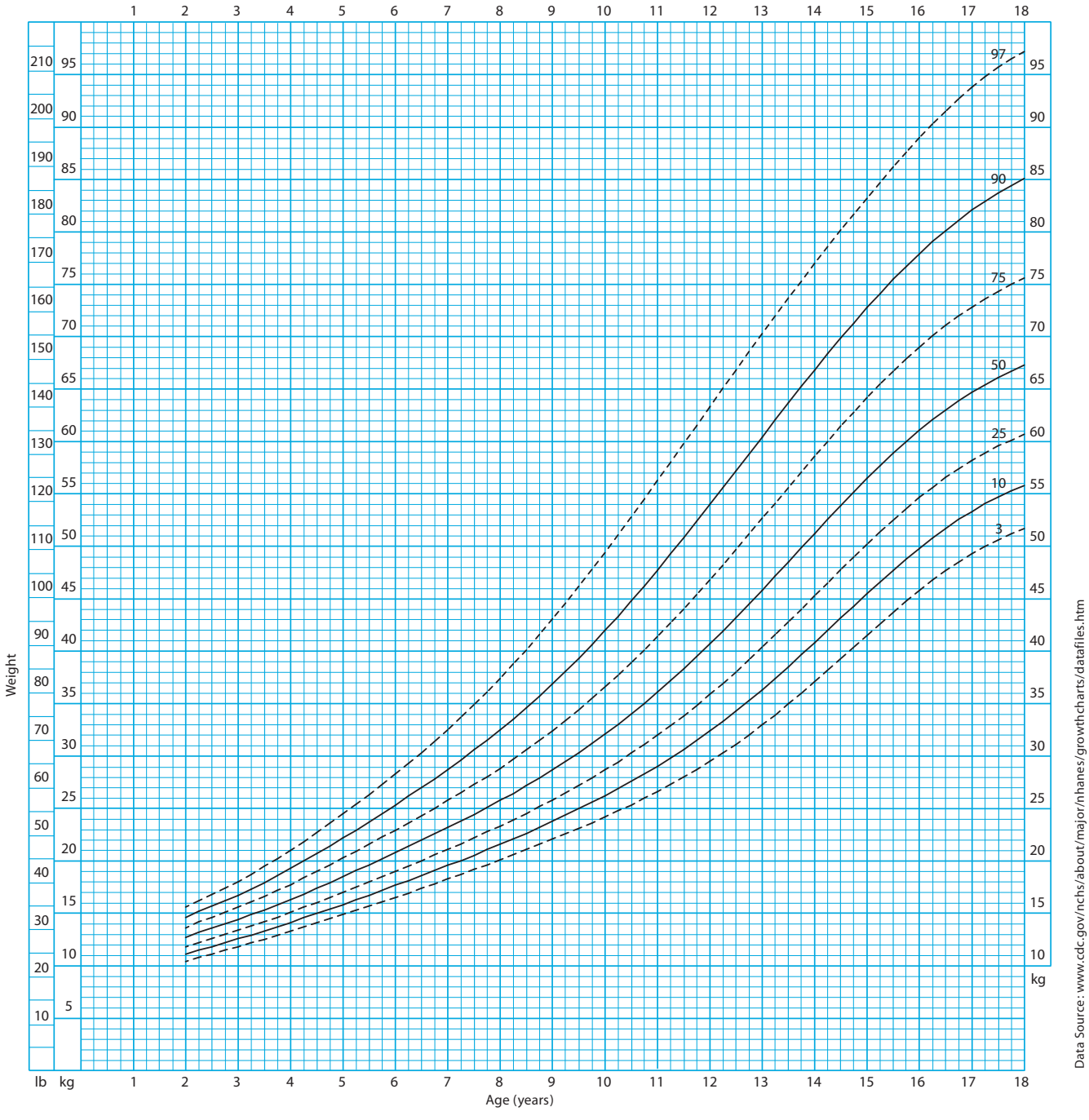
					PUBERTAL STAGES			
DATE	AGE	HEIGHT	WEIGHT	HEAD CIRCUM.	GENITAL	PUBIC HAIR	TESTES L	TESTES R



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Boys 2-18 years

Stages of Puberty

Ages of attainment of successive stages of pubertal sexual development are given in the Height Percentile chart.
The stage Pubic Hair 2+ represents the state of a child who shows the pubic hair appearance stage 2 but not stage 3 (see below).

The centiles for age at which this state is normally seen are given, the 97th centile being considered as the early limit, the 3rd centile as the late limit. The child's puberty stages may be plotted at successive ages (Tanner. 1962, *Growth at Adolescence*, 2nd edn). Testis sizes are judged by comparison with the Prader orchidometer (Zachmann, Prader, Kind, Haflinger & Budliger. 1974, *Helv. Paed. Acta*. 29, 61-72).

Genital (Penis) Development

Stage 1. Pre-adolescent. Testes, scrotum and penis are of about the same size and proportion as in early childhood.

Stage 2. Enlargement of scrotum and testes. Skin of scrotum reddens and changes in texture. Little or no enlargement of penis at this stage.

Stage 3. Enlargement of the penis which occurs at first mainly in length. Further growth of the testes and scrotum.

Stage 4. Increased size of penis with growth in breadth and development of glans. Testes and scrotum larger; scrotal skin darkened.

Stage 5. Genitalia adult in size and shape.

Pubic Hair Development

Stage 1. Pre-adolescent. The vellus over the pubes is not further developed than that over the abdominal wall, i.e. no pubic hair.

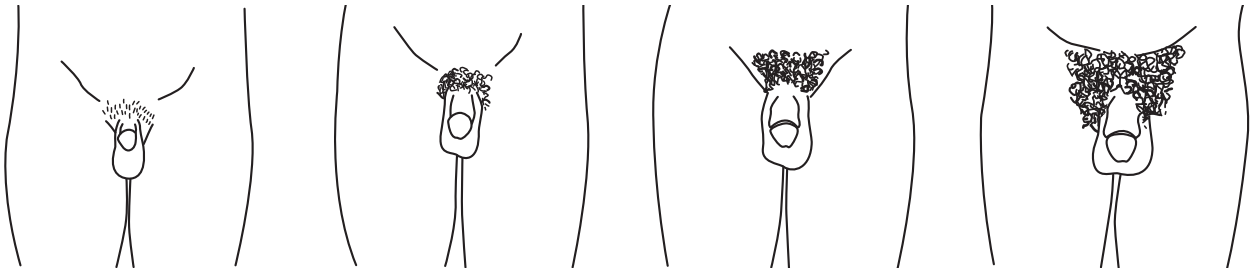
Stage 2. Sparse growth of long, slightly pigmented downy hair, straight or slightly curled at the base of the penis.

Stage 3. Considerably darker, coarser and more curled. The hair spreads sparsely over the junction of the pubes.

Stage 4. Hair now adult in type, but area covered is still considerably smaller than in the adult. No spread to the medial surface of thighs.

Stage 5. Adult in quantity and type with distribution of the horizontal (or classically 'feminine') pattern. Spread to medial surface of thighs but not up linea alba or elsewhere above the base of the inverse triangle (spread up linea alba occurs late and is rated stage 6).

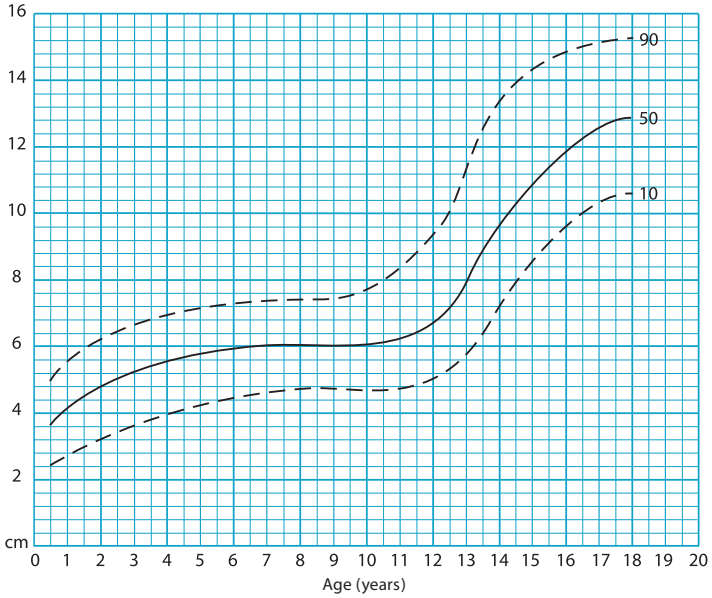
Genital and Pubic Hair Development Stages



Stage 2 **Stage 3** **Stage 4** **Stage 5**

Stretched Penile Length

Measured from the pubo-penile skin junction to the tip of the glans (Shonfeld & Beebe. 1942, *Journal of Urology,* 48, 759-777).



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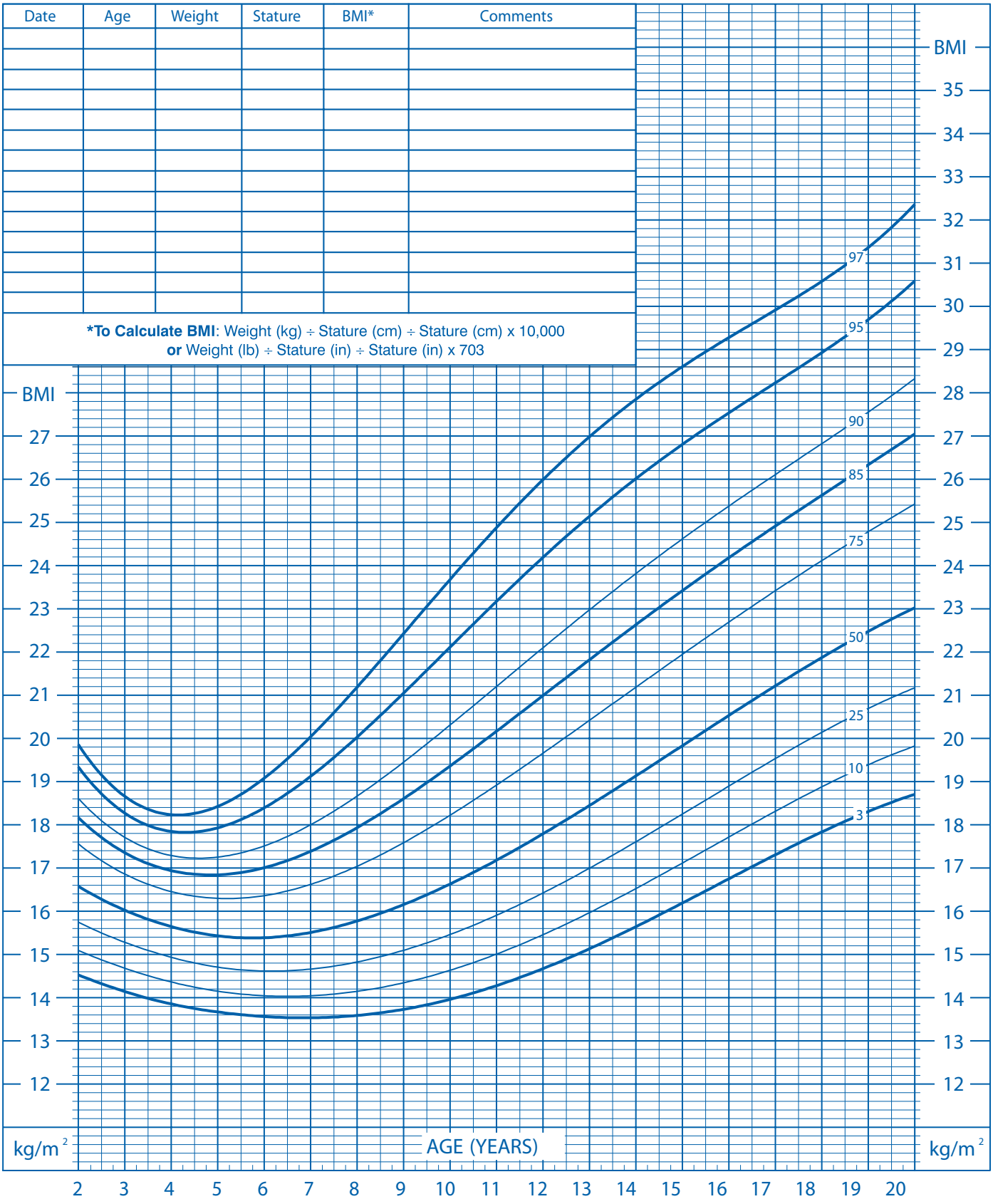
2 to 20 years: Boys
Body mass index-for-age percentiles

NAME _____

RECORD # _____

Date	Age	Weight	Stature	BMI*	Comments

***To Calculate BMI**: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 10/16/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

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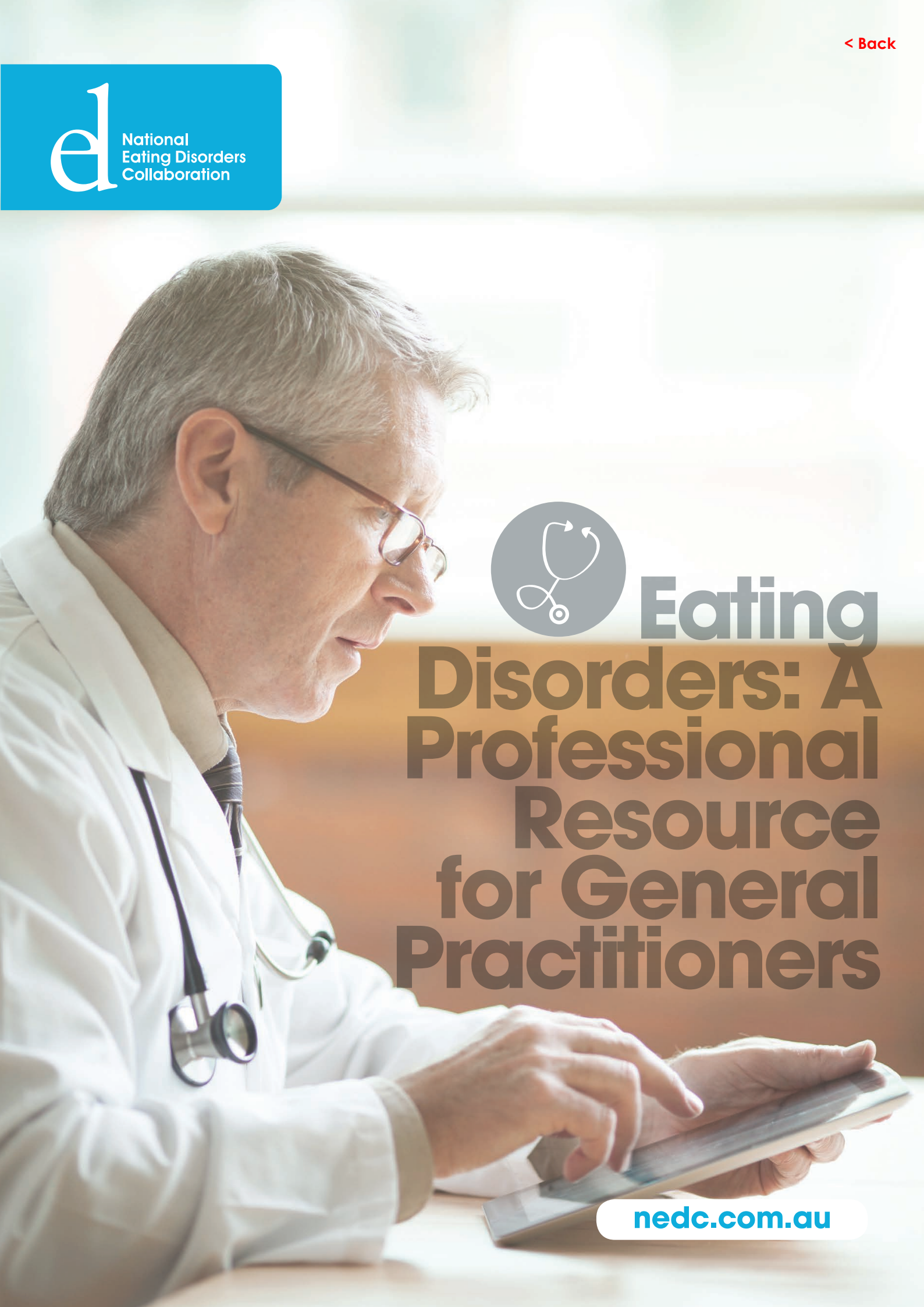

Eating Disorders: A Professional Resource for General Practitioners

nedc.com.au

Eating Disorders: A Professional Resource for General Practitioners

A professional resource developed by the National Eating Disorders Collaboration

General information about Eating Disorders

Eating disorders are serious mental illnesses; they are not a lifestyle choice or a diet gone 'too far.'

Eating disorders are associated with significant physical complications and increased mortality. The mortality rate for people with eating disorders is the highest of all psychiatric illnesses, and over 12 times higher than that for people without eating disorders. Eating disorders occur in people of all genders, ages and backgrounds. About one in 20 Australians has an eating disorder and the rate in the Australian population is increasing.

There are four specified eating disorders defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-5); Anorexia Nervosa, Bulimia Nervosa, Binge Eating Disorder and Other Specified Feeding and Eating Disorder (OSFED).

Eating disorders defy classification solely as mental illnesses as they not only involve considerable psychological impairment and distress, but they are also associated with major wide-ranging and serious medical complications, which can affect every major organ of the body.

As a GP you are likely to be one of the first health professionals a person with an eating disorder will come in contact with. A GP's role in the treatment of eating disorders can encompass prevention, identification, medical management in a primary care setting and referral.

Step 1: Screening and assessment

Early intervention depends on early detection of symptoms. There is an average delay of approximately 4 years between the start of disordered eating symptoms and first treatment, and this delay can be 10 or more years. A reduction of this delay can result in improved health and quality of life.

The delay in the commencement of treatment can be for a number of reasons. We do know the majority of people with eating disorders have contact with health professionals. They present with apparently unrelated complaints and do not disclose their eating problems. While people may not volunteer information about their eating problem, asking questions and allowing the person to see that their eating habits are important may offer a non-judgemental environment for them to start seeking help.

For many people with eating disorders, their first attempt at seeking treatment is a test of attitudes and responses. If the first help seeking is a positive experience then the person is more likely to engage successfully with future treatment.

Common health presentations include:

- emotional problems
- weight loss
- gastro-intestinal problems
- infertility issues
- injuries caused by overexercising
- fainting or dizziness
- feeling fatigued or not sleeping well
- feeling cold most of the time regardless of the weather
- swelling around the cheeks or jaw, calluses on knuckles, damage to teeth and bad breath (signs of vomiting).

The full list of the physical, psychological and behavioural warning signs of an eating disorder is available under the Mental Health First Aid Guidelines: **http://www.mhfa.com.au/Guidelines.shtml**.

Screening questions

Screening questions help to initiate a disclosure which may then lead to earlier access to treatment for individuals with eating disorders (Gilbert et al., 2012). Screening for eating disorders involves asking a small number of evidence based questions posed on an opportunistic basis when the patient presents for other reasons (e.g. weight related concerns, depression or anxiety). The questionnaires do not diagnose eating disorders but detect the possible presence of an eating disorder and identify when a more detailed assessment is warranted.

SCOFF

S – Do you make yourself Sick because you feel uncomfortably full?
C – Do you worry you have lost Control over how much you eat?
O – Have you recently lost more than 6.35 kg in a three-month period?
F – Do you believe yourself to be Fat when others say you are too thin?
F – Would you say Food dominates your life?

An answer of 'yes' to two or more questions indicates the need for a more comprehensive assessment.
A further two questions have been shown to indicate a high sensitivity and specificity for bulimia nervosa.

1. Are you satisfied with your eating patterns?
2. Do you ever eat in secret?

Eating Disorder Screen for Primary Care (ESP)

- Are you satisfied with your eating patterns? (A "no" to this question is classified as an abnormal response).
- Do you ever eat in secret? (A "yes" to this and all other questions is classified as an abnormal response).
- Does your weight affect the way you feel about yourself?
- Have any members of your family suffered with an eating disorder?
- Do you currently suffer with or have you ever suffered in the past with an eating disorder?

Cotton, Ball & Robinson, 2003 found that the best individual screening questions are:

- Does your weight affect the way you feel about yourself?
- Are you satisfied with your eating patterns?

Screening for high risk groups

Eating disorders occur in both males and females; in children, adolescents, adults and older adults; across all socio-economic groups and cultural backgrounds. Within this broad demographic however there are some groups with a particularly high level of risk. Based on the known risk factors for eating disorders, high risk groups who may benefit from screening for eating disorders include:

Adolescents: The peak period for the onset of eating disorders is between the ages of 12 and 25 years, with a median age of around 18 years.

Women, particularly during key transition periods: from school to adult life, pregnancy and menopause. Targeting preventive interventions at women with high weight and shape concerns, a history of critical comments about eating weight and shape, and a history of depression may reduce the risk for eating disorders.

Women with Polycystic Ovary Syndrome or Diabetes: Adolescents with diabetes may have a 2.4-fold higher risk of developing an eating disorder, particularly Bulimia Nervosa and binge eating, than their peers without diabetes. Polycystic Ovary Syndrome is associated with body dissatisfaction and eating disorders. Screening for abnormal eating patterns is recommended.

Athletes: People engaged in competitive fitness, dance and other physical activities where body shape may be perceived as affecting performance have a high level of risk of eating disorders.

People with a family history of eating disorders: There is evidence that eating disorders have a genetic basis and people who have family members with an eating disorder may be at higher risk of developing an eating disorder themselves.

People seeking help for weight loss: Eating disorders almost invariably occur in people who have engaged in dieting or disordered eating.

Eating disorders in males

Population studies have suggested that males make up approximately 25% of people with Anorexia or Bulimia and 40% of people with Binge Eating Disorder. In a recent study lifetime prevalence for anorexia nervosa in adolescents aged 13 – 18 years found no difference between males and females.

One unique difference between males and females with eating disorders is that men more typically engage in compulsive exercise as a compensatory behaviour, often with the aim of achieving a more muscular, and not just slender, body type. Compulsive exercise describes a rigid, driven urge to exercise, which is a serious health concern.

The Compulsive Exercise Test (CET) is a screening questionnaire of 24 questions (1 page) which asks people to rate their own behaviour and emotions in relation to exercise.

The CET can be used with adolescents. You can download the CET at **www.lboro.ac.uk/media/wwwlboroacuk/content/ssehs/downloads/compulsive-exercise-test.pdf** Physical assessment can also be useful in the identification of compulsive exercise.

Risk Assessment

Patients must be screened for physical health risks and risk of suicide. Medical stabilization, where required, must be provided before or simultaneously with other interventions. Eating disorders can impair a person's insight and ability to make informed decisions. Decisions regarding treatment must always take into consideration the person's capacity to make decisions for their own safety.

When taking the patients general history and conducting physical examinations assess their general state (eg. well/unwell), alertness/somnolence, height and weight history, disproportion in weight for height (>1standard deviation apart), menstruation pattern/menstrual history, hydration (tongue, lips, skin, sunken eyes), ketones on breath, deep, irregular, sighing, breathing seen in ketoacidosis, temperature <36°C, pulse rate <60 beats per min, regular or irregular, BP – lying and standing (postural drop in BP > 20mmHg), limbs – peripheral circulation, cold peripheries, ankle oedema, abdomen scaphoid, symptoms of electrolyte disturbance (thirst, dizziness, fluid retention, swelling of arms and legs, weakness and lethargy, muscle twitches and spasms) and alkaline urinary pH. More information about medical assessments can be found in the resource 'Eating Disorders: An Information Pack for General Practitioners' produced by The Eating Disorders Association Inc (Qld) at **http://eda.org.au/wp-content/uploads/Complete-GP-Information-Kit-2013.pdf**

Step 2: Referral to appropriate services

The eating disorders treatment team requires a multi-disciplinary approach to address the physical components of the illness, the eating behaviours, the psychological thought processes, and the social and work needs of the person. Members of the multidisciplinary team will vary depending on the needs of the patient but a minimum team for safe interventions will include both physical and psychological disciplines. GPs should be aware of the risks of rapid deterioration of health in people with Eating Disorders and should consider the impact of very low BMI on cognition and the role of mental health legislation and compulsory treatment for some patients. More information about treatment and recovery including treatment approaches for specific disorders and complementary treatment approaches can be found on the NEDC's treatment and recovery page.

A 'complete' team could include a range of the following professionals depending on individual needs and treatment plans:

- GP or paediatrician
- Psychiatrist
- Dietitian
- Psychologist
- Psychiatric Nurse
- Nurse Practitioner
- Family therapist
- Specialists working with co-morbid conditions
- Social worker
- Occupational therapist
- Physiotherapist
- Educator
- Community support organisation
- School counsellor

Specialist interventions may also be required by some patients to prevent or treat a wide range of physical health conditions including gastrointestinal disorders, malnutrition, osteoporosis, damage to teeth, infection, cardiac complications, kidney failure, menstrual problems and treatment of comorbid conditions such as diabetes. Some patients may require referral to a Hospital Emergency Department or an Eating Disorders specialist. More information about treatment options including inpatient treatments, outpatient treatments, day programs, community based support and rural options can be found on the NEDC's treatment options page. Criteria for admitting a patient with an eating disorder to hospital can be found in the resource 'Eating Disorders: An Information Pack for General Practitioners' produced by The Eating Disorders Association Inc (Qld) at **http://eda.org.au/wp-content/uploads/Complete-GP-Information-Kit-2013.pdf**

Step 3: Ongoing treatment and management

A flowchart depicting a management plan for eating disorders can be found in the resource 'Eating Disorders: An Information Pack for General Practitioners' produced by The Eating Disorders Association Inc (Qld) at **http://eda.org.au/wp-content/uploads/Complete-GP-Information-Kit-2013.pdf**

Important points to consider in the treatment and management of an eating disorder include:

Develop and implement a treatment plan by

- Working with the patient to identify their strengths and resources for goal setting
- Remembering general medical care is critical with regular assessment of physical health risks
- Actively promoting normal physical growth in children and adolescents as a priority of care
- Applying strategies to reduce the risk of re-feeding syndrome (more information available at http://eda.org.au/wp-content/uploads/Complete-GP-Information-Kit-2013.pdf)
- Appointing a case coordinator to coordinate the individual care plan by acting as a principle point of contact, coordinating shared information, and facilitating collaboration to support the implementation of complex care plans (this may be the GP)
- Monitoring the patients progress and measuring treatment outcomes
- Supporting transfer between services and providing appropriate follow-up of the patient

Work collaboratively with patients and their families by

- Providing information, education and support for the patient and their family

- Including the patients family in assessment, engagement, treatment and recovery support
- Engaging the patient, and where possible their family, in collaborative decision making to enhance motivation for change
- Identifying and responding to engagement difficulties and ambivalence about treatment
- Modelling an understanding and supportive attitude
- Being aware of your personal attitudes, values and beliefs (e.g. re: body shape) so that you can manage countertransference or collusion with the patient

Support recovery by:

- Valuing the role of recovery support in reducing the risk of relapse and recurrence
- Utilising community based support services and resources

Clinical Guidelines

There are a number of established treatment guidelines, which may be helpful including: NICE, AMA and RANZCP. When determining which guidelines to utilise, the date of publication should be taken into consideration and the guidelines should be interpreted in the context of emerging evidence on effective and safe treatment approaches, the clinical environment, the individual patient and the National Standards Schema for the treatment of eating disorders.

Criteria for Recovery (Bardone-Cone, et al., 2010)

1. Diagnosis – no longer meeting diagnostic criteria
2. Behaviour – no longer engaging in eating disorder behaviours
3. Physical health – weight within healthy BMI range
4. Psychological – positive attitudes to one's self, food, the body, expression of emotions and social interaction
5. Practical – quality of life including capacity for engagement in work or education, and leisure

Relapse prevention refers to strategies to reduce the risk of relapse after treatment and interventions, and responses to early signs of recurring illness for people who have already experienced an eating disorder. Some experience of relapse (or 'lapse') is common during and immediately post treatment and with timely intervention and support contributes to the learning and self-discovery process of recovery. More information about relapse and recurrence including risk factors for relapse can be found on the NEDC's relapse and recurrence page: **www.nedc.com.au/relapse-and-recurrence**

Where to find more information

A more comprehensive resource is available for GPs from The Eating Disorders Association Inc (Qld) at **http://eda.org.au/wp-content/uploads/Complete-GP-Information-Kit-2013.pdf**

Clinical resources for health professionals

To access clinical resources for health professionals working with eating disorders visit **http://ceed.org.au/clinical-resources/ and http://cedd.org.au/health-professionals/resources-clinical-guidance/**

Website Resources for GP's

The National Eating Disorders Collaboration (NEDC) has a number of resources available through their website which may meet the professional needs of GPs with eating disorders patients: **www.nedc.com.au/health-professionals**

Mental Health First Aid

For professionals who do not have a background in working with people with eating disorders, the Mental Health First Aid guidelines may provide a useful starting place to support recognition and safe responses to people who are developing or experiencing an eating disorder. The guidelines provide an evidence based set of general recommendations about how you can help someone who is developing or experiencing an eating disorder: **www.mhfa.com.au/cms/**

Latest Research and Resources

NEDC provides a single gateway through which healthcare providers can access the latest evidence-based information and resources on the prevention, identification, early intervention and management of eating disorders here: **www.nedc.com.au/research-resources**

Professional Development

Within Australia there are opportunities for professionals to advance their knowledge and expertise in the field of eating disorders.
Professional development: **www.nedc.com.au/proffesional-develop**
Upcoming events: **http://member.nedc.com.au/events/event_list.asp**

Eating Disorders Awareness for Professionals

Appropriate messages can be combined with effective engagement strategies to help health service providers and other professionals educate the community about eating disorders. The NEDC website provides further information on how to communicate about eating disorders, breaking down barriers, preventing eating disorders and eating disorders and obesity: **www.nedc.com.au/eating-disorders-awareness**

National Support Line: 1800 ED HOPE (1800 33 4673)

References

Key references

American Psychiatric Association. (2013). Diagnostic and Statistical Manual of Mental Disorders (DSM-5), 5th ed. American Psychiatric Association: Washington DC, USA.

Bardone-Cone, A. M., Schaefer, L. M., Maldonado, C. R., Fitzsimmons, E. E., Harney, M. B., Lawson, M. A., Robinson, D. P., Tosh, A. & Smith, R. (2010). Aspects of Self-concept and Eating Disorder Recovery: What Does the Sense of Self Look Like When an Individual Recovers From an Eating Disorder? Journal of Social and Clinical Psychology, 29(7), 821-846.

Cotton, M.A., Ball, C. & Robinson, P. (2003). Four Simple Questions Can Help Screen for Eating Disorders. Journal of General Internal Medicine, 18(1), 53-56.

Gilbert, N., Arcelus, J., Cashmore, R., Thompson, B., Langham, C. & Meyer, C. (2012). Should I Ask About Eating? Patients' Disclosure of Eating Disorder Symptoms and Help-seeking Behaviour. European Eating Disorders Review, 20(1), 80-85.

Luck, A. J., MorganLuck, J. F., Reid, F., O'Brien, A., Brunton, J., Price, C., Perry, L., & Lacey, J. H. (2002). The SCOFF Questionnaire and Clinical Interview for Eating Disorders in General Practice: Comparative Study. BMJ, 325(7367), 755-756.

Other useful references

American Psychiatric Association. (1994). Diagnostic and Statistical Manual of Mental Disorders (DSM-4), 4th ed. American Psychiatric Association: Washington DC, USA.

de la Rie, S., Noordenbos, G., Donker, M. & van Furth, E. (2006). Evaluating The Treatment of Eating Disorders from the Patient's Perspective. International Journal of Eating Disorders, 39(8), 667–676.

Evans, E. J., Hay, P. J., Mond, J., Paxton, S. J., Quirk, F., Rodgers, B., Jhajj, A. K. & Sawoniewska, M.A. (2011). Barriers to Help Seeking in Young Women with Eating Disorders: a Qualitative Exploration in a Longitudinal Community Survey. Eating Disorders: The Journal of Treatment & Prevention, 19 (3), 270-285.

Fursland, A., Allen, K. L., Watson, H. & Byrne, S. M. (2010). Eating Disorders – Not Just an Adolescent Issue? Australia and New Zealand Academy of Eating Disorders (ANZAED) 8th Annual Conference, Auckland, New Zealand.

Goodwin, H., Haycraft, E., Taranis, L. & Meyer, C. (2011). Psychometric Evaluation of the Compulsive Exercise Test (CET) in an Adolescent Population: Links with Eating Psychopathology. European Eating Disorders Review. Special Issue: Special edition on compulsive exercise, 19(3), 269-279.

Hart, L. M., Jorm, A. F., Paxton, S. J., Kelly, C. M. & Kitchener, B. A. (2009). First Aid for Eating Disorders. Eating Disorders: The Journal of Treatment & Prevention, 17(5), 357-384.

Hay, P. J., Mond, J., Buttner, P. & Darby, A. (2008). Eating Disorder Behaviors Are Increasing: Findings from Two Sequential Community Surveys in South Australia. PLoS ONE, 3(2), e1541.

Hill, L. S., Reid, F., Morgan, J. F. & Lacey, J. (2010). SCOFF, The Development of an Eating Disorder Screening Questionnaire. International Journal of Eating Disorders, 43(4), 344-351.

Himelein, M. J. & Thatcher, S. (2006). Polycystic Ovary Syndrome and Mental Health: A Review. Obstetrical and Gynecological Survey, 61(11), 723-32.

Hudson, J. I., Hiripi, E., Pope Jr, H. G. & Kessler, R. C. (2007). The Prevalence and Correlates of Eating Disorders in the National Comorbidity Survey Replication. Biological Psychiatry, 61(3), 348-358.

Jacobi, C. & Fittig, E. (2010). Psychosocial Risk Factors for Eating Disorders. In Agras, W.S. (Ed.), Oxford Handbook of Eating Disorders. Oxford University Press: New York, USA.

Jacobi, C., Fittig, E., Bryson, S. W., Wilfley, D., Kraemer, H. C. & Taylor, C. B. (2011). Who is Really at Risk? Identifying Risk Factors for Subthreshold and Full Syndrome Eating Disorders in a High-risk Sample. Psychological Medicine, 41, 1939-1949.

Jones, W. & Morgan, J. (2010). Eating Disorders in Men: A Review of the Literature. Journal of Public Mental Health, 9 (2), 23 – 31.

Mangweth-Matzek, B., Rupp, CI., Hausmann, A., Assmayr, K., Mariacher, E.,

Kemmler, G., Whitworth, A. B. & Biebl, W. (2006). Never Too Old for Eating Disorders or Body Dissatisfaction: A Community Study of Elderly Women. International Journal of Eating Disorders, 39(7), 583-586.

Newton, M. S. & Chizawsky, L. L. K. (2006). Treating Vulnerable Populations: The Case of Eating Disorders During Pregnancy. Journal of Psychosomatic Obstetrics & Gynecology, 27(1), 5-7.

Pereira, R. F. & Alvarenga, M. (2007). Disordered Eating: Identifying, Treating, Preventing, and Differentiating It From Eating Disorders. Diabetes Spectrum, 20(3), 141-148.

Sullivan, P. F. (1995). Mortality in Anorexia Nervosa. The American Journal of Psychiatry, 152(7), 1073-1073-1074.

Swanson, S. A., Crow, S. J., Le Grange, D., Swendsen, J. & Merikangas, K. R. (2011). Prevalence and Correlates of Eating Disorders in Adolescents Results from the National Comorbidity Survey Replication Adolescent Supplement. Archives of General Psychiatry, 68 (7), 714-723.

Taranis, L., Touyz, S. & Meyer, C. (2011). Disordered Eating and Exercise: Development and Preliminary Validation of the Compulsive Exercise Test (CET). European Eating Disorders Review, 19(3), 256-68.

National
Eating Disorders
Collaboration

nedc.com.au

The National Eating Disorders Collaboration is a collaboration of people and organisations with expertise in the field of eating disorders, individuals from a range of healthcare and research sectors and people with a lived experience of an eating disorder.

Through the contribution of its members, the NEDC has the resources to lead the way in addressing eating disorders in Australia.

nedc.com.au brings research, experise and evidence from leaders in the field together in one place.

Become a member

We welcome individuals and organisations to become members of the NEDC. As a member you can get involved in one of the working groups and contribute to project deliverables. You will also be informed on collaboration activities and receive access to the members only area of the website. Join the collaboration: **www.nedc.com.au/become-a-member**

Sign up for the NEDC Monthly e-Bulletin

If you would like to keep up to date with what is happening in the wider eating disorders sector including the latest evidence based research on eating disorders you can register receive our monthly e-Bulletin. Subscribe to the e-Bulletin: **www.nedc.com.au/subscribe**

Join our e-Network

If you are an eating disorders expert or other professional with an interest in eating disorders and would like to connect to other professionals about this issue please join our Clinician's e-Network. Find out more about the e-Network: **www.nedc.com.au/e-network**

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For a downloadable copy of this resource visit:
www.nedc.com.au/health-professionals

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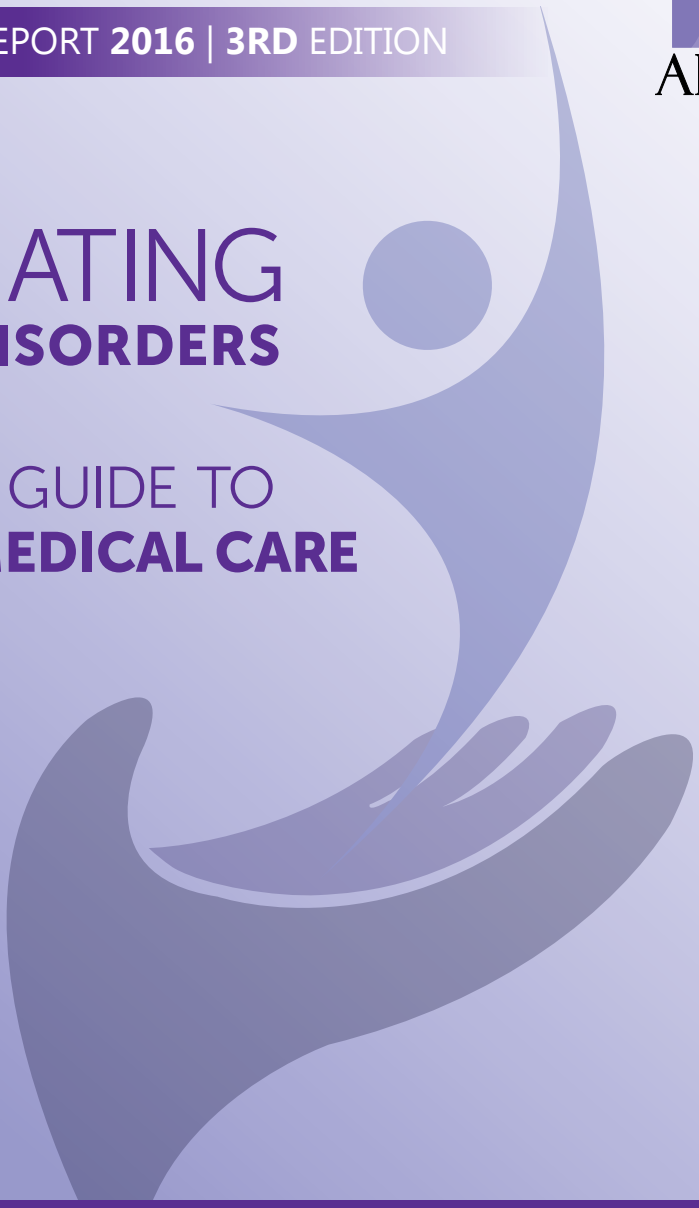
EATING DISORDERS

A GUIDE TO MEDICAL CARE



Critical Points for Early Recognition & Medical Risk Management in the Care of Individuals with Eating Disorders

DISCLAIMER: This document, created by the Academy for Eating Disorders' Medical Care Standards Committee, is intended as a resource to promote recognition and prevention of medical morbidity and mortality associated with eating disorders. It is not a comprehensive clinical guide. Every attempt was made to provide information based on the best available research and current best practices. For further resources, practice guidelines and bibliography visit: **www.aedweb.org** and **www.aedweb.org/Medical_Care_Standards**

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EATING DISORDERS: A GUIDE TO MEDICAL CARE

TABLE OF CONTENTS

AED

KEY GUIDELINES

All eating disorders (EDs) are serious mental illnesses with significant, life-threatening medical and psychiatric morbidity and mortality, regardless of an individual's weight. Patients with EDs have the highest mortality rates of any psychiatric disorder. The risk of premature death is 6-12 times higher in women with Anorexia Nervosa (AN) as compared to the general population, adjusting for age.

Early recognition and timely intervention, based on a developmentally appropriate, evidence-based, multidisciplinary team approach (medical, psychological & nutritional) is the ideal standard of care, whenever possible. Members of the multidisciplinary team may vary and will depend upon the needs of the patient and the availability of these team members in the patient's community. In communities where resources are lacking, clinicians, therapists, and dietitians are encouraged to consult with the Academy for Eating Disorders (AED) and/or ED experts in their respective fields of practice.

EATING DISORDERS

For the purpose of this document, we will focus on the most common EDs including:

1. **Anorexia Nervosa (AN):** Restriction of energy intake relative to an individual's requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory and health status. Disturbance of body image, an intense fear of gaining weight, lack of recognition of the seriousness of the illness and/or behaviors that interfere with weight gain are also present.
2. **Bulimia Nervosa (BN):** Binge eating (eating a large amount of food in a relatively short period of time with a concomitant sense of loss of control) with purging/compensatory behavior (e.g. self-induced vomiting, laxative or diuretic abuse, insulin misuse, excessive exercise, diet pills) once a week or more for at least 3 months. Disturbance of body image, an intense fear of gaining weight and lack of recognition of the seriousness of the illness may also be present.

3. **Binge Eating Disorder (BED):** Binge eating, in the absence of compensatory behavior, once a week for at least 3 months. Binge eating episodes are associated with eating rapidly, when not hungry, until extreme fullness, and/or associated with depression, shame or guilt.
4. **Other Specified Feeding and Eating Disorder (OSFED):** An ED that does not meet full criteria for one of the above categories, but has specific disordered eating behaviors such as restricting intake, purging and/or binge eating as key features.
5. **Unspecified Feeding or Eating Disorder (UFED):** ED behaviors are present, but they are not specified by the care provider.
6. **Avoidant/Restrictive Food Intake Disorder (ARFID):** Significant weight loss, nutritional deficiency, dependence on nutritional supplement or marked interference with psychosocial functioning due to caloric and/or nutrient restriction, but without weight or shape concerns.

Consult **www.aed.org**, DSM-5 or ICD-10 for full diagnostic descriptions.

IMPORTANT FACTS ABOUT EATING DISORDERS

- **All** EDs are serious disorders with life-threatening physical and psychological complications.
- EDs do not discriminate. They can affect individuals of all ages, genders, ethnicities, socioeconomic backgrounds, and with a variety of body shapes, weights and sizes.
- Weight is not the only clinical marker of an ED. People who are at low, normal or high weights can have an ED and individuals at any weight may be malnourished and/or engaging in unhealthy weight control practices.
- Individuals with an ED may not recognize the seriousness of their illness and/or may be ambivalent about changing their eating or other behaviors.
- All instances of precipitous weight loss or gain in otherwise healthy individuals should be investigated for the possibility of an ED as rapid weight fluctuations can be a potential marker of an ED.
- In children and adolescents, failure to gain expected weight or height, and/or delayed or interrupted pubertal development, should be investigated for the possibility of an ED.

- All EDs can be associated with serious medical complications affecting every organ system of the body.
- The medical consequences of EDs can go unrecognized, even by an experienced clinician.

PRESENTING SIGNS AND SYMPTOMS

Individuals with EDs may present in a variety of ways. In addition to the cognitive and behavioral signs that characterize EDs, the following physical signs and symptoms can occur in patients with an ED as a consequence of restricting food or fluid intake, nutritional deficiencies, binge-eating, and inappropriate compensatory behaviors, such as purging. However, it is important to remember that a life-threatening ED may occur without obvious physical signs or symptoms.

GENERAL:

- Marked weight loss, gain, fluctuations or unexplained change in growth curve or body mass index (BMI) percentiles in a child or adolescent who is still growing and developing
- Cold intolerance
- Weakness
- Fatigue or lethargy
- Presyncope (dizziness)
- Syncope (fainting)
- Hot flashes, sweating episodes

ORAL AND DENTAL:

- Oral trauma/lacerations
- Perimyolysis (dental erosion on posterior tooth surfaces) and dental caries (cavities)
- Parotid (salivary) gland enlargement

CARDIORESPIRATORY:

- Chest pain
- Heart palpitations
- Orthostatic tachycardia/ hypotension (low blood pressure)
- Dyspnea (shortness of breath)
- Edema (swelling)

GASTROINTESTINAL:

- Epigastric discomfort
- Abdominal bloating
- Early satiety (fullness)
- Gastroesophageal reflux (heartburn)
- Hematemesis (blood in vomit)
- Hemorrhoids and rectal prolapse
- Constipation

ENDOCRINE

- Amenorrhea or oligomenorrhea (absent or irregular menses)
- Low sex drive
- Stress fractures
- Low bone mineral density
- Infertility

NEUROPSYCHIATRIC

- Depressive/Anxious/Obsessive/Compulsive symptoms and behaviors
- Memory loss
- Poor concentration
- Insomnia
- Self-harm
- Suicidal thoughts, plans or attempts
- Seizures

DERMATOLOGIC

- Lanugo hair (fine hair growth on the body and face)
- Hair loss
- Carotenoderma (yellowish discoloration of skin)
- Russell's sign (calluses or scars on the back of the hand from self-induced vomiting)
- Poor wound healing
- Dry brittle hair and nails

EARLY RECOGNITION

Consider evaluating an individual for an ED who presents with any of the following:

- Precipitous weight changes (significant weight lost or gained) or fluctuations
- Sudden changes in eating behaviors (new vegetarianism/veganism, gluten-free, lactose-free, elimination of certain foods or food groups, eating only "healthy" foods, uncontrolled binge eating)
- Sudden changes in exercise patterns, excessive or compulsive exercise or involvement in extreme physical training
- Body image disturbance, the desire to lose weight despite low or normative weight, or extreme dieting behavior regardless of weight
- Abdominal complaints in the context of weight loss behaviors
- Electrolyte abnormalities without an identified medical cause (especially hypokalemia, hypochloremia, or elevated bicarbonate)
- Hypoglycemia

- Bradycardia
- Amenorrhea or menstrual irregularities
- Unexplained infertility
- Type 1 diabetes mellitus with poor glucose control or recurrent diabetic ketoacidosis (DKA) with or without weight loss
- Use of compensatory behaviors (i.e., self-induced vomiting, laxative abuse, dieting, fasting or excessive exercise) to influence weight after eating or binge eating
- Inappropriate use of appetite suppressants, caffeine, diuretics, laxatives, enemas, ipecac, artificial sweeteners, sugar-free gum, prescription medications that affect weight (insulin, thyroid medications, psychostimulants, or street drugs) or nutritional supplements marketed for weight loss

Malnutrition is a serious medical condition that requires urgent attention. It can occur in patients engaging in disordered eating behaviors, regardless of weight status. Individuals with continued restrictive eating behaviors, binge eating or purging, despite efforts to redirect their behavior, require immediate intervention.

COMPREHENSIVE ASSESSMENT

COMPLETE HISTORY TO INCLUDE:

- Rate and amount of weight loss/change in past six months
- Nutritional history to include dietary intake (quantity and variety of foods consumed), restriction of specific foods or food groups (such as fats or carbohydrates)
- Compensatory behaviors and their frequency (fasting or dieting, self-induced vomiting, exercise, laxative, diuretic or ipecac abuse, insulin misuse, use of diet pills and/or other over-the-counter supplements)
- Exercise (frequency, duration and intensity). Is the exercise excessive, compulsive or rigid or is it used to manage weight?
- Menstrual history (menarche, last menstrual period, regularity, oral contraceptive use)
- Current medications including any supplements & alternative medications
- Family history including symptoms or diagnosis of EDs, obesity, mood & anxiety disorders, substance use disorders
- Psychiatric history including symptoms of mood, anxiety and substance use disorders
- History of trauma (physical, sexual or emotional)
- Growth history (obtain past growth charts whenever possible)

PHYSICAL EXAMINATION TO INCLUDE:

- Measurement of height, weight, and determination of body mass index (BMI = weight (kg)/height (m^2)); record weight, height and BMI on growth charts for children and adolescents
- Lying and standing heart rate and blood pressure
- Oral temperature

INITIAL DIAGNOSTIC EVALUATION:

- Laboratory and other diagnostic studies recommended for consideration in evaluating a patient with an ED, along with potential corresponding abnormalities seen in patients with EDs, are outlined in the following chart.
- It is important to note that laboratory studies may be normal even with significant malnutrition.

DIAGNOSTIC TESTS INDICATED FOR ALL PATIENTS WITH A SUSPECTED ED	
BASIC TESTS	**POTENTIAL ABNORMAL FINDINGS AND CAUSES**
Complete blood count	Leukopenia, anemia, or thrombocytopenia
Comprehensive panel to include electrolytes, renal function tests and liver enzymes	Glucose: ↓ *poor nutrition* Sodium: ↓ *water loading or laxatives* Potassium: ↓ *vomiting, laxatives, diuretics* Chloride: ↓ *vomiting, laxatives* Blood bicarbonate: ↑ *vomiting* ↓ *laxatives* Blood urea nitrogen: ↑ *dehydration* Creatinine: ↑ *dehydration, renal dysfunction, muscle wasting* Calcium: slightly ↓ *poor nutrition at the expense of bone* Phosphate: ↓ *poor nutrition* Magnesium: ↓ *poor nutrition, laxative use* Total protein/albumin: ↑ *in early malnutrition at the expense of muscle mass,* ↓ *in later malnutrition* Prealbumin: ↓ *in protein-calorie malnutrition* Aspartate aminotransaminase (AST), Alanine aminotransaminase (ALT): ↑ *starvation*
Electrocardiogram (ECG)	Bradycardia (low heart rate), prolonged QTc (>450msec), other arrhythmias
ADDITIONAL DIAGNOSTIC TESTS TO CONSIDER	
ADDITIONAL TESTS	**POTENTIAL ABNORMAL FINDINGS**
Leptin level	Leptin: ↓ *in malutrition*
Thyroid stimulating hormone (TSH), thyroxine (T4)	TSH: ↓ *or normal* T4: ↓ *or normal euthyroid sick syndrome*
Pancreatic enzymes (amylase and lipase)	Amylase: ↑ *vomiting, pancreatitis* Lipase: ↑ *pancreatitis*
Gonadotropins (LH and FSH) and sex steroids (estradiol and testosterone)	LH, FSH, estradiol (women) and testosterone (men) levels: ↓ *or normal*

Erythrocyte sedimentation rate (ESR)	ESR: ↓ *starvation or* ↑ *inflammation*
Dual Energy X-ray Absorptiometry (DEXA)	Patients with EDs are at risk of low *bone mineral density (BMD)*. There is no evidence that hormone replacement therapy (estrogen/progesterone in females or testosterone in males) improves BMD. Nutritional rehabilitation, weight recovery, and normalization of endogenous sex steroid production are the treatments of choice.

CRITERIA FOR HOSPITALIZATION FOR ACUTE MEDICAL STABILIZATION

PRESENCE OF ONE OR MORE OF THE FOLLOWING:

1. ≤ 75% median BMI for age, sex, and height
2. Hypoglycemia
3. Electrolyte disturbance (hypokalemia, hyponatremia, hypophosphatemia and/or metabolic acidosis or alkalosis)
4. ECG abnormalities (e.g., prolonged QTc > 450, bradycardia, other arrhythmias)
5. Hemodynamic instability
   - Bradycardia
   - Hypotension
   - Hypothermia
6. Orthostasis
7. Acute medical complications of malnutrition (e.g., syncope, seizures, cardiac failure, pancreatitis, etc.)
8. Comorbid psychiatric or medical condition that prohibits or limits appropriate outpatient treatment (e.g., severe depression, suicidal ideation, obsessive compulsive disorder, type 1 diabetes mellitus)
9. Uncertainty of the diagnosis of an ED

CRITERIA FOR HOSPITALIZATION FOR ACUTE PSYCHIATRIC STABILIZATION

PRESENCE OF ONE OR MORE OF THE FOLLOWING:

1. Acute food refusal
2. Suicidal thoughts or behaviors
3. Other significant psychiatric comorbidity that interferes with ED treatment (anxiety, depression, obsessive compulsive disorder)

OTHER CONSIDERATIONS REGARDING HOSPITALIZATION:

1. Failure of outpatient treatment
2. Uncontrollable binge eating and/or purging by any means
3. Inadequate social support and/or follow up medical or psychiatric care

REFEEDING SYNDROME

Refeeding syndrome describes the clinical and metabolic derangements that can occur during refeeding (orally, enterally, or parenterally) of a malnourished patient. The clinical features of refeeding syndrome include edema, cardiac and/or respiratory failure, gastrointestinal problems, profound muscle weakness, delirium and, in extreme cases, death. Laboratory abnormalities may include hypophosphatemia (most significant), hypoglycemia, hypokalemia, hypomagnesemia and hyponatremia. Refeeding syndrome can occur in patients of any age and weight, and is a potentially fatal condition requiring specialized care on an inpatient unit.

RISK FACTORS FOR REFEEDING SYNDROME INCLUDE:

- The degree of malnutrition at presentation (< 70% median BMI in adolescents, BMI <15 most at risk in adults)
- Patients who are chronically undernourished and those who have had little or no energy intake for more than 10 days
- History of refeeding syndrome
- Patients with rapid or profound weight loss, including those who present at any weight after rapid weight loss (> 10-15% of total body mass in 3-6 months)

IMPORTANT – Consider initiating refeeding in an inpatient setting if one or more risk factors for refeeding are present. Ideally patients should be admitted to a hospital that has access to, or onsite ED specialist support.

- Patients with significant alcohol intake (these patients are also at risk for the development of Wernicke's encephalopathy with refeeding. Prior to refeeding they should receive thiamine and folate supplementation)
- Post-bariatric surgery patients with significant weight loss (increased risk with electrolyte losses from malabsorption)
- Patients with a history of diuretic, laxative or insulin misuse
- Patients with abnormal electrolytes prior to refeeding

HOW TO PREVENT AND MANAGE REFEEDING SYNDROME:

- Know the signs, symptoms and risk factors for refeeding syndrome.
- Whenever possible, refer patients at risk for refeeding syndrome to physicians with expertise in medical and behavioral management of EDs and/or admit to an inpatient medical or med-psych unit with this expertise.
- Serum electrolytes (sodium, potassium, phosphorous and magnesium) and glucose should be checked prior to initiating refeeding. Be aware that these may be normal prior to refeeding. Phosphorus will reach its lowest point 3-7 days after initiation of nutritional rehabilitation.
- While treating a patient on an inpatient unit, monitor serum electrolytes and glucose frequently (at least daily if significantly abnormal) during early refeeding until stabilized (at least 72 hours).
- Aggressively replete all electrolyte deficiencies. Oral repletion is preferable but IV supplementation may be necessary. It is not necessary to correct fluid and electrolyte imbalance before initiating feeding. With careful monitoring, this can be safely achieved simultaneously.
- Start a multivitamin daily prior to initiating and throughout refeeding. Add thiamine supplementation in severely malnourished patients due to the risk of Wernicke's encephalopathy.
- In consultation with a nutrition specialist with expertise in refeeding patients with ED, adjust rate of refeeding according to the age, developmental stage, and degree of malnourishment.

- Monitor fluid replacement to avoid overload. The preferred rehydration route is oral. In cases where IV fluid is deemed necessary, it is best to avoid large fluid boluses. Replace losses slowly instead with continuous IVF at low rates (e.g., 50-75 cc/hour for adult patients or ½ normal maintenance in children).
- Closely monitor vital signs and cardiac and mental status of all patients during refeeding.
- Monitor blood glucose frequently. Postprandial glucose is often low in severely starved patients with AN.

UNDERFEEDING

Underfeeding due to overly cautious rates of refeeding can lead to further weight loss, and may be associated with a worse prognosis, slower response to treatment, and even death in seriously malnourished patients.

METHODS OF REFEEDING:

- "Start low and go slow" methods of refeeding have recently been challenged and more rapid refeeding with close medical monitoring is now preferred during **inpatient** treatment. For instance, an adult with an ED who is significantly malnourished, and has had very low intake prior to hospitalization, might be safely started at approximately 1600 kcal/day and increased by 300 kcal/day every 2-3 days until consistent weight gain of at least 2-4 lb (1-2 kg)/week is achieved.
- Most patients will require high calorie intake (3500-4000 kcal/day) to achieve consistent weight gain once medically stabilized beyond the initial stages of refeeding. This may be initiated as an inpatient and continued as an outpatient (once the patient is medically stabilized) until complete weight restoration is achieved. At this time, a reassessment of nutritional needs should be performed for weight maintenance and/or growth.
- Children/adolescents and their families may need to be reminded that they are in a state of growth and development. Treatment goal weights and nutritional needs will change with time as children and adolescents continue to grow and develop.
- Oral refeeding is always preferred. Supplemental enteral feeds may be indicated when rates of weight gain are low (<2 lb/week) or access to an expert behavioral refeeding program is limited. Parenteral feeding is not recommended and should only be used in patients without a functional gut.

INFORMATION FOR MEDICAL SPECIALTY PROVIDERS

Individuals with EDs are frequently referred to specialty providers for evaluation of medical complaints. It is important that every provider, regardless of specialty, recognize the signs and symptoms consistent with an ED, manage complications appropriately, and know when to refer a patient for specialized ED evaluation and treatment and/or when to refer for hospital admission due to significant medical compromise. Collaborative care with an ED specialist is in the best interest of all patients.

FOR ALL PROVIDERS: Please maintain a high index of suspicion for EDs, especially in high-risk patients. Keep in mind that EDs may be present in patients of any age, race, gender or size. Screen and refer to specialty care as indicated. A validated screening tool such as the SCOFF (see Appendix 1) may be used in identifying adult patients who would benefit from further evaluation for an ED.

CARDIOLOGY

- Bradycardia is a physiologic, adaptive response to starvation and is the most common arrhythmia in patients with EDs. Bradycardia should not be automatically attributed to athleticism or training in patients who are underweight, who have experienced rapid weight loss, or who have inadequate nutritional intake for their level of activity.
- Cardiologists should consult with ED specialists if they are considering an ED, or evaluating a known patient with an ED. Collaborative care helps put the patient's diagnosis and clinical presentation into an appropriate context.
- Arrhythmias due to electrolyte abnormalities are a common cause of death in patients with EDs.

EMERGENCY MEDICINE

- Patients with EDs present to emergency departments at rates higher than individuals without EDs for a variety of complaints.
- An emergency department visit may be the first interaction with healthcare providers for a patient with an ED. The severity and treatment resistance of EDs increase with length of illness. The sooner a patient receives appropriate treatment, the more likely they are to fully recover from their illness. A positive clinician-patient interaction in the emergency department, and referral to appropriate ED specialty care may help to significantly reduce the length and severity of illness.

- Consult with ED specialists if unclear about the appropriate disposition and follow-up.
- Avoid overly aggressive fluid resuscitation in patients with EDs as this may precipitate volume overload and heart failure. Use low volume continuous IV fluid rather than large boluses to rehydrate.
- Avoid **excess** glucose administration in the emergency department as this may precipitate refeeding syndrome. If blood glucose is low, continuous infusion of D5 is preferred to administration of boluses of D50.

ENDOCRINOLOGY

- Adolescent females with Type I diabetes are at increased risk of EDs. They may underdose insulin and may suffer increased long-term complications of diabetes, including early death. Poor glucose control and/or frequent episodes of DKA in any diabetic patient should prompt evaluation for an ED.
- Consider Euthyroid Sick Syndrome (ESS) in a low weight patient with abnormal thyroid studies. Thyroid hormone supplementation is not required for ESS and will resolve with weight restoration.

GASTROENTEROLOGY

- GI complaints such as constipation, abdominal pain, nausea, hematemesis, frequent heartburn, and early satiety are amongst the most common physical symptoms in persons with EDs.
- Slightly elevated aminotransferases are also frequently seen in patients with EDs.
- These signs and symptoms often prompt referral to a gastroenterologist. Symptomatic treatment for GI symptoms may be initiated. It is important to note that most GI symptoms improve or resolve with resolution of the ED.

OBSTETRICS AND GYNECOLOGY

- Amenorrhea or oligomenorrhea without other identified cause should prompt evaluation for an ED.
- Oral contraceptive pills (OCPs) for treatment of amenorrhea or oligomenorrhea are not indicated for most patients with ED who do not otherwise require contraception.
- There is no current evidence to support the use of OCPs for treatment of low bone mineral density in a low weight patient with amenorrhea. Weight restoration and resumption of menses is the treatment of choice.
- Although individuals with EDs may have suppressed ovarian function, pregnancy can still occur.
- Infertility may also be a presenting complaint in patients with EDs. Assisted reproductive technology (ART) is contraindicated in low weight patients with EDs. These patients are at increased risk of miscarriage, intrauterine growth retardation, low birth weight and other pregnancy and birth complications with the use of ART.

PSYCHIATRY

- Patients with EDs have high rates of comorbidity with other psychiatric disorders including depression, anxiety, obsessive compulsive disorder, post-traumatic stress disorder, self-harm behaviors and substance use, and are at high risk of suicide.
- Patients with EDs may report symptoms of depression or other mental illness without recognizing or revealing ED thoughts or behaviors.
- Patients with EDs have the highest mortality rates of any psychiatric disorder. Their identification and appropriate treatment by an ED specialist is imperative.

PEDIATRICS

- Children and adolescents presenting with failure to thrive, fussy or selective eating, gastrointestinal symptoms (e.g., nausea, loss of appetite, constipation), unexplained weight loss, lack of weight gain or delayed growth and development should be evaluated for a possible ED.
- Carefully consider a parent's concerns about any change in their child's eating behaviors, mood, weight or growth.

TIMELY INTERVENTIONS

1. **Patients with EDs may not acknowledge that they are ill, and/or they may be ambivalent about accepting treatment.**

   This is a symptom of their illness. Patients may minimize, rationalize, or hide ED symptoms and/or behaviors. Their persuasive rationality and competence in other areas of life can disguise the severity of their illness. Outside support and assistance with decision-making will likely be necessary regardless of age.

2. **Parents/guardians are the frontline help-seekers for children, adolescents, and young adults with EDs.**

   Trust their concerns. Even a single consultation about a child's eating behavior or weight/shape concerns is a strong predictor of the presence or potential development of an ED.

3. **Diffuse blame.**

   Help families understand that they did not cause the illness; neither did their child/family member choose to have it. This recognition facilitates acceptance of the diagnosis, referral, treatment, and minimizes undue stigma associated with having the illness.

4. **Monitor physical health including vital signs and laboratory tests.**

   Patients with an ED should be regularly monitored for acute and chronic medical complications. Assessments should be interpreted in the context of physiological adaptation to malnutrition and purging behavior. Clinicians need to remember that physical exam and laboratory tests may be normal even in the presence of a life-threatening ED.

5. **Psychiatric risk.**

   Always assess for psychiatric risk, including suicidal and self-harm thoughts, plans and/or intent. Up to 1/2 of deaths related to EDs are due to suicide.

GOALS OF TREATMENT

It is important to note that full resolution of symptoms may take an extended period of treatment. Psychological symptoms may transiently increase with initial treatment and improvement in physical health. Recognize that EDs are not merely fads, phases, or lifestyle choices. They are biologically-based, heritable disorders. People do not choose to have EDs and they can recover fully from them.

Medical stabilization, nutritional rehabilitation to achieve weight restoration, management of refeeding and its potential complications, and interruption of purging/compensatory behaviors should be the immediate goals of treatment for all patients with EDs. Additional psychological and other therapeutic goals can be addressed in parallel when appropriate.

MEDICAL STABILIZATION—*AS PRESENTED ABOVE*

- Management of acute and chronic medical comorbidities and complications
- Includes resumption of menses (where appropriate)

NUTRITIONAL REHABILITATION

- Weight restoration
- Restore meal patterns that promote health and social connections

NORMALIZATION OF EATING BEHAVIOR

- Cessation of restrictive or binge eating and/or purging behaviors
- Elimination of disordered or ritualistic eating behaviors

PSYCHOSOCIAL STABILIZATION

- Evaluation and treatment of any comorbid psychological diagnoses
- Re-establishment of appropriate social engagement
- Improvement in psychological symptoms associated with ED
- Improved body image

ONGOING MANAGEMENT

Following initial stabilization, ongoing evidence-based treatment delivered by healthcare professionals with expertise in the care of patients with EDs is essential for full recovery. Optimal care includes a multidisciplinary team approach by ED specialists including medical, psychological, nutritional, and psychopharmacologic services. Families (i.e., parents, spouses, partners) should be included in ED treatment whenever possible.

In low weight patients with ED, restoration of an appropriate, healthy weight will significantly improve their physical, psychological, social, and emotional functioning. Failure to fully restore weight correlates with poor outcomes, and maintenance of a healthy weight strongly correlates with improved outcomes.

For full recovery from an ED, however, weight restoration alone is not sufficient for full recovery. It is equally important that distorted body image and other ED thoughts/behaviors, psychological comorbidities and any social or functional impairments be addressed by qualified professionals during the treatment of patients with EDs.

APPENDIX 1: An example of a validated screening tool for eating disorders—The SCOFF.* Other screening tools are available.

- **S** Do you make yourself **S**ick because you feel uncomfortably full?
- **C** Do you worry you have lost **C**ontrol over how much you eat?
- **O** Have you recently lost more than **O**ne stone (6.35 kg or 14 lb) in a three-month period?
- **F** Do you believe yourself to be **F**at when others say you are too thin?
- **F** Would you say **F**ood dominates your life?

*Two or more positive responses on the SCOFF indicates a possible ED and should prompt referral for further evaluation.

NOTES

NOTES

REFERENCES

- Garber AK, Sawyer SM, Golden NH, Guarda AS, Katzman DK, Kohn MR, Le Grange D, Madden S, Whitelaw M, Redgrave GW. (2016). A Systematic Review of Approaches to Refeeding in Patients with Anorexia Nervosa. ***International Journal of Eating Disorders***. 49(3), 293-310.
- Mond JM, Myers TC, Crosby RD, Hay PJ, Rodgers B, Morgan JF, Lacey JH, Mitchell JE. (2008). Screening for Eating Disorders in Primary Care: EDE-Q versus SCOFF. ***Behaviour Research and Therapy***. 46, 612–622.
- Sachs K, Andersen D, Sommer J, Winkelman A, Mehler PS. (2015). Avoiding Medical Complications During the Refeeding of Patients with Anorexia Nervosa. ***Eating Disorders***. 23(5), 411-421.
- The Society for Adolescent Health and Medicine. (2015). Position Paper of the Society for Adolescent Health and Medicine: Medical Management of Restrictive Eating Disorders in Adolescents and Young Adults. ***Journal of Adolescent Health***. 56(1), 121–125.
- The Society for Adolescent Health and Medicine. (2014). Refeeding Hypophosphatemia in Hospitalized Adolescents with Anorexia Nervosa: A Position Statement of the Society for Adolescent Health and Medicine. ***Journal of Adolescent Health***. 55(3), 455-457.

For further references and information about the diagnosis and treatment of EDs visit:

www.aedweb.org

ABOUT THE ACADEMY FOR EATING DISORDERS (AED):
The AED is a global multidisciplinary professional association committed to leadership in promoting EDs research, education, treatment, and prevention. The AED provides cutting-edge professional training and education, inspires new developments in the field of EDs, and is the international source for state-of-the-art information on EDs.

JOIN THE AED: Become a member of a global community dedicated to ED research, treatment, education, and prevention. Join online at: **www.aedweb.org**

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