Clinical Practice Guideline for Eating Disorders

NOTE:

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

The recommendations included should be considered with caution taking into account that it is pending evaluate its validity.

CLINICAL PRACTICE GUIDELINES IN THE NHS.
MINISTRY OF HEALTHCARE AND CONSUMER AFFAIRS

GOBIERNO DE ESPAÑA
MINISTERIO DE SANIDAD Y CONSUMO
Plan de Calidad para el Sistema Nacional de Salud
Generalitat de Catalunya
Departament de Salut

CLINICAL PRACTICE GUIDELINE FOR EATING DISORDERS

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Generalitat de Catalunya
Departament de Salut

This clinical practice guideline (CPG) is an aid for decision-making in health care. It is not in any way an obliged requirement to adhere to every aspect of this CPG and it does not replace the clinical judgement of health care professionals.

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Table of Contents

Presentation 9
Authors and Collaborations 11
Key Questions 13
CPG Recommendations 17

1. Introduction 33
2. Scope and Objectives 39
3. Methodology 43
4. Definition and Classification of Eating Disorders 47
5. Prevention of Eating Disorders 55
6. Detection of Eating Disorders 63
7. Diagnosis of Eating Disorders 73
8. Interventions at the Different Levels of Care in the Management of Eating Disorders 81
9. Treatment of Eating Disorders 91
10. Assessment of Eating Disorders 179
11. Prognosis of Eating Disorders 191
12. Legal Aspects Concerning Individuals with Eating Disorders in Spain 195
13. Detection, Diagnosis and Treatment Strategies for Eating Disorders 201
14. Dissemination and Implementation 215
15. Recommendations for Future Research 219

ANNEXES
Annex 1. Levels of Evidence and Grades of Recommendation 221

Annex 2. Clinical Chapters 223
Annex 2.1. Spanish version of the SCOFF survey 223
Annex 2.2. Spanish version of the EAT-40 224
Annex 2.3. Spanish version of the EAT-26 226
Annex 2.4. Spanish version of the ChEAT 227
Annex 2.5. Spanish version of the BULIT 228
Annex 2.6. Spanish version of the BITE 234

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Annex 2.7. Diagnostic Criteria for Eating Disorders 236
Annex 2.8. Spanish version of the EDE-12 semi-structured interview 242
Annex 2.9. Incorrect Ideas about Weight and Health 242
Annex 2.10. Description of Proposed Indicators 243

Annex 3. Information for Patients with Eating Disorders and their Families 247
Annex 3.1. Patient Information 247
Annex 3.2. Support Associations for Patients with Eating Disorders and their Families 252

Annex 4. Glossary 253

Annex 5. Abbreviations 261

Annex 6. Others 267
Annex 6.1. Protocols, Recommendations, Therapeutic Orientations and Guidelines for Eating Disorders 267
Annex 6.2. Results of the Search, Selection and Quality Assessment of Evidence based on the stages performed 269
Annex 6.3. Description of the NICE'S CPG 270
Annex 6.4. Description of the AHRQ'S SRSE 272

References 275

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Presentation

Health care practice is becoming more and more complex due to multiple factors, the most relevant being the exponential increase of scientific information.

To ensure that clinical decisions are appropriate, efficient and safe, health care professionals must constantly update their knowledge, an objective that entails great dedication and effort.

In the year 2003, the National Health System's Interterritorial Council created the Health Guide (GuíaSalud) project, which aims ultimately to improve evidence-based clinical decision-making by means of training activities and the configuration of a Clinical Practice Guidelines (CPG) register in the NHS. Since then, the Health Guide project has assessed dozens of CPGs in accordance with explicit criteria generated by its scientific committee, registered these CPGs and disseminated them throughout the Internet. In early 2006, the Directorate General of the National Health System Quality Agency elaborated the Quality Plan for the National Health System, a plan that encompasses twelve strategies. The objective of this Plan is to increase cohesion of the NHS and aid in guaranteeing maximum quality health care to all citizens, regardless of their place of residence. As part of the plan, the development of eight CPGs on prevalent pathologies related with health strategies was assigned to different agencies and experts groups. This guide on eating disorders is the result of this assignment.

Additionally, the establishment of a common CPG development methodology for the NHS was assigned to CPG experts groups in our country, resulting in a collective effort of consensus and coordination amongst them.

In 2007, the Health Guide project was renovated by creating the Clinical Practice Guideline Library. This project thoroughly covers the elaboration of CPGs and includes other services and products of evidence-based medicine. It also aims to favour the implementation and assessment of the use of CPGs in the National Health System.

Eating disorders, anorexia nervosa and bulimia nervosa, as well as other similar clinical pictures, are disorders of multifactorial ethiopathogeny that have been a great challenge for public health care in the last decades. Sociocultural factors that can lead to eating disorders, as well as the serious physical, social and psychological sequelae that these disorders entail have caused great social alarm. Eating disorders are diseases that not only involve the affected individual, but also the family and closest environment, and even health care and education professionals who are directly or indirectly involved, and who have no access to guides to address these disorders successfully.

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This CPG aims to provide the population and health care and education professionals with a useful instrument to address the most basic aspects of the disease, especially those concerning prevention and treatment. Understanding and assessing these diseases, identifying them and assessing their risk potential, as well as presenting therapeutic objectives, and deciding on the best site for treatment and providing help to families, are tasks that can be tackled from different professional settings with an undeniable benefit for patients and family members. Such is the role that this evidence-based guide aims to exercise, and which is the result of the work performed by a group of professionals involved in the field of eating disorders and experts on CPG methodology.

This CPG has been revised by Spanish eating disorders experts and is endorsed by Spanish patient associations and scientific societies involved in the management of these patients.

Pablo Rivero
General Director
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AEETCA is the only scientific society specifically dedicated to eating disorders

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Declaration of Interests:

All members of the working group, as well as the individuals who have collaborated in the development of this guide (experts on eating disorders, representatives from different associations, scientific societies, federations and external reviewers), have carried out the declaration of conflict of interests by completing a form designed to this end.

None of the participants have declared having a conflict of interest related with eating disorders.

This guide is editorially independent from the funding organisation.

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Key Questions

Definition and Classification of Eating Disorders

1. How are eating disorders defined and classified? What are the shared and specific clinical features of each type?

2. Ethiopathogeny of eating disorders: What are the main risk factors?

3. What are the most frequent co morbidities of eating disorders?

Prevention of Eating Disorders

4. What is the efficacy of primary care interventions in avoiding eating disorders? Are there any negative effects?

Detection of Eating Disorders

5. What screening instruments are useful to identify cases of eating disorders?

Diagnosis of Eating Disorders

6. What clinical criteria are useful to diagnose eating disorders?

7. How are eating disorders diagnosed?

8. What is the differential diagnosis of eating disorders?

Interventions at the Different Levels of Care in the Management of Eating Disorders

9. What are the primary care (PC) and specialised care interventions for eating disorders? Other resources?

10. In eating disorders**,** what clinical criteria may be useful to assess referral amongst the health care resources available in the NHS?

11. In eating disorders, what clinical criteria may be useful to assess inpatient care (complete hospitalisation) in the healthcare resources available in the NHS?

12. In eating disorders, what clinical criteria may be useful to assess discharge in the healthcare resources available in the NHS?

Treatment of Eating Disorders

13. What is the efficacy and safety of re-nutrition in patients with eating disorders?

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14. What is the efficacy and safety of nutritional counselling in patients with eating disorders?

15. What is the efficacy and safety of cognitive-behavioural therapy in patients with eating disorders?

16. What is the efficacy and safety of self-help and guided self-help in patients with eating disorders?

17. What is the efficacy and safety of interpersonal therapy in patients with eating disorders?

18. What is the efficacy and safety of family therapy (systemic or not) in patients with eating disorders?

19. What is the efficacy and safety of psychodynamic therapy in patients with eating disorders?

20. What is the efficacy and safety of behavioural therapy in patients with eating disorders?

21. What is the efficacy and safety of antidepressants in patients with eating disorders?

22. What is the efficacy and safety of antipsychotic drugs in patients with eating disorders?

23. What is the efficacy and safety of appetite stimulants in patients with anorexia nervosa (AN)?

24. What is the efficacy and safety of opioid antagonists in patients with eating disorders?

25. What is the efficacy and safety of other psychoactive drugs in patients with eating disorders?

26. What is the efficacy and safety of combined interventions in patients with eating disorders?

27. What is the treatment for eating disorders that occur with comorbidities?

28. How are chronic cases of eating disorders treated?

29. What is the treatment for eating disorders in special situations such as pregnancy and delivery?

Assessment of Eating Disorders

30. What tools are useful to assess the symptoms and behaviour of eating disorders?

31. What tools are useful for the psychopathological assessment of eating disorders?

Prognosis of Eating Disorders

32. What is the prognosis of eating disorders?

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33. Are there prognostic factors for eating disorders?

Legal Aspects Concerning Patients with Eating Disorders in Spain

34. What legal procedure must be followed when a patient with an eating disorder refuses to receive treatment?

35. Is the informed consent of a minor with an eating disorder legally valid?

36. In the case of a minor with an eating disorder, what is the legal solution to the dilemma stemming from the responsibility of confidentiality, respect of autonomy and obligations towards the minor's parents or legal guardians?

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CPG Recommendations

In this section recommendations are presented following the guide's structure. Chapters 1,2 and 3 of the CPG include an introduction, scope and objectives, and methodology, respectively. Chapter 4 covers eating disorders and Chapter 11 addresses prognosis. All these chapters are descriptive and thus no recommendations have been formulated for clinical practice. Chapter 5, which covers prevention, is the first to provide recommendations. This section's abbreviations can be found at the end.

Grade of recommendation: A, B, C o D, depending on whether evidence quality is very high, high, moderate or low.

✓ Good clinical practice: recommendation based on the working group's consensuses. (Please refer to Annex 1).

5. Primary Prevention of Eating Disorders (Question 5.1.)

✓	5.1.	Sample, format and design characteristics of eating disorder prevention programmes that have shown greater efficacy should be considered the model for future programmes.
✓	5.2.	In the design of universal eating disorder prevention strategies, it must be taken into account that expected behavioural changes in children and adolescents without these types of problems might differ from those of high-risk populations.
✓	5.3.	Messages on measures that indirectly protect individuals from eating disorders should be passed on to the family and adolescent: following a healthy diet and eating at least one meal at home with the family, facilitating communication and improving self-esteem, avoiding family conversations from compulsively turning to eating and image and avoiding jokes and disapproval regarding the body, weight or eating manner of children and adolescents.

6. Detection of Eating Disorders (Question 6.1.)

D	6.1.	Target groups for screening should include young people with low body mass index (BMI) compared to age-based reference values, patients consulting with weight concerns without being overweight or people who are overweight, women with menstrual disorders or amenorrhoea, patients with gastrointestinal symptoms, patients with signs of starvation or repeated vomiting, and children with delayed or stunted growth, children, adolescents and young adults who perform sports that entail a risk of developing an eating disorder (athletics, dance, synchronised swimming, etc.).

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D	6.2.	In anorexia nervosa (AN), weight and BMI are not considered the only indicators of physical risk.
D	6.3.	Early detection and intervention in individuals presenting weight loss are important to prevent severe emaciation.
D	6.4.	In the case of suspected AN, attention should be paid to overall clinical assessment (repeated over time), including rate of weight loss, growth curve in children, objective physical signs and appropriate laboratory tests.
✓	6.5.	It is recommended to use questionnaires adapted and validated in the Spanish population for the detection of eating disorder cases (screening). The use of the following tools is recommended: Eating disorders in general: SCOFF (for individuals aged 11 years and over) AN: EAT-40, EAT-26 and ChEAT (the latter for individuals aged between 8 and 12 years) Bulimia nervosa (BN): BULIT, BULIT-R and BITE (the three for individuals aged 12-13 years and over)
✓	6.6.	Adequate training of PC physicians is considered essential for early detection and diagnosis of eating disorders to ensure prompt treatment or referral, when deemed necessary.
✓	6.7.	Due to the low frequency of visits during childhood and adolescence, it is recommended to take advantage of any opportunity to provide comprehensive management and to detect eating disorder risk habits and cases. Eating disorder risk behaviour, such as repeated vomiting, can be detected at dental check-ups.
✓	6.8.	When interviewing a patient with a suspected eating disorder, especially if the suspected disorder is AN, it is important to take into account the patient's lack of awareness of the disease, the tendency to deny the disorder and the scarce motivation to change, these reactions being more pronounced in earlier stages of the disease.
✓	6.9.	It is recommended that different groups of professionals (teachers, school psychologists, chemists, nutritionists and dieticians, social workers, etc.) who may be in contact with at-risk population have adequate training and be able to act as eating disorder detection agents.

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7. Diagnosis of Eating Disorders (Questions 7.1.-7.3.)

✓	7.1.	It is recommended to follow the WHO's (ICD-10) and the APA's (DSM-IV o DSM-IV-TR) diagnostic criteria.
D	7.2.1.	Health care professionals should acknowledge that many patients with eating disorders are ambivalent regarding treatment due to the demands and challenges it entails.
D	7.2.2.	Patients and, when deemed necessary, carers should be provided with information and education regarding the nature, course and treatment of eating disorders.
D	7.2.3.	Families and carers may be informed of existing eating disorder associations and support groups.
✓	7.2.4.	It is recommended that the diagnosis of eating disorders include anamnesis, physical and psychopathological examinations and complementary explorations.
✓	7.2.5.	Diagnostic confirmation and therapeutic implications should be in the hands of psychiatrists and clinical psychologists.

8. Interventions at the Different Levels of Care (Questions 8.1.-8.4.)

D	8.1.	Individuals with eating disorders should be treated in the appropriate care level based on clinical criteria: outpatient care, day care (day hospital) and inpatient care (general or psychiatric hospital).
D	8.2.	Health care professionals without specialist experience in eating disorders or who are faced with uncertain situations should seek the advice of a trained specialist when emergency inpatient care is deemed the most appropriate option for a patient with an eating disorder.
D	8.3.	The majority of patients with BN can be treated on an outpatient basis. Inpatient care is indicated when there is risk of suicide, self-inflicted injuries and serious physical complications.
D	8.4.	Health care professionals should assess patients with eating disorders and osteoporosis and advise them to refrain from performing physical activities that may significantly increase the risk of fracture.
D	8.5.	The paediatrician and the family physician must be in charge of the management of eating disorders in children and adolescents. Growth and development must be closely monitored.

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D	8.6.	Primary care centres should offer monitoring and management of physical complications to patients with chronic AN and repeated therapeutic failures who do not wish to be treated by mental health services.
D	8.7.	Family members, especially siblings, should be included in the individualized treatment plan (ITP) of children and adolescents with eating disorders. The most common interventions involve sharing of information, advice on behavioural management of eating disorders and improving communication skills. The patient's motivation to change should be promoted by means of family intervention.
D	8.8.	Where inpatient care is required, it should be carried out within a reasonable distance to the patient's home to enable the involvement of relatives and carers in treatment, to enable the patient to maintain social and occupational links and to prevent difficulties between care levels. This is particularly important in the treatment of children and adolescents
D	8.9.	Patients with AN whose disorder has not improved with outpatient treatment must be referred to day patient treatment or inpatient treatment. For those who present a high risk of suicide or serious self-inflicted injuries, inpatient management is indicated.
D	8.10.	Inpatient treatment should be considered for patients with AN whose disorder is associated with high or moderate risk due to common disease or physical complications of AN.
D	8.11.	Patients with AN who require inpatient treatment should be admitted to a centre that ensures adequate re-nutrition, avoiding the re-feeding syndrome, with close physical monitoring (especially in the first few days), along with the appropriate psychological intervention.
D	8.12.	The family physician and paediatrician should take charge of the assessment and initial intervention of patients with eating disorders who attend primary care.
D	8.13.	When management is shared between primary and specialised care, there should be close collaboration between health care professionals, patients and relatives and carers.
✓	8.14.	Patients with confirmed diagnosis or clear suspicion of an eating disorder will be referred to different health care resources based on clinical and age criteria.

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✓	8.15.	Referral to adult or children mental health centres (CSMA/CSMIJ) by the family physician or paediatrician should consist of integrated care with shared responsibilities.
✓	8.16.	Cases referred to adult or children mental health centres (CSMA/CSMIJ) still require different levels to work together and short- and mid-term monitoring of patients, to avoid complications, recurrences and the onset of emotional disorders, and to detect changes in the patient's environment that could influence the disease.
✓	8.17.	The need to prescribe oestrogen treatment to prevent osteoporosis in girls and adolescents with AN should be carefully assessed, given that this medication can hide the presence of amenorrhoea.
✓	8.18.	In childhood, specific eating disorder treatment programmes designed for these ages will be required.

9. Treatment of Eating Disorders (Questions 9.1.-9.20.)

Medical Measures (Re-nutrition and Nutritional Counselling)

Re-nutrition (Question 9.1.)

Anorexia nervosa

D	9.1.1.1.	A physical exploration and in some cases oral multivitamin and/or mineral supplements are recommended, both in outpatient and inpatient care, for patients with AN who are in the stage of body weight restoration.
D	9.1.1.2.	Total parenteral nutrition should not be used in patients with AN unless the patient refuses nasogastric feeding and/or when there is gastrointestinal dysfunction.
✓	9.1.1.3.	Enteral or parenteral re-nutrition must be applied using strict medical criteria and its duration will depend on when the patient is able to resume oral feeding.

General Recommendations on Medical Measures (GM) for Eating Disorders (Questions 9.1.-9.2.)

Eating Disorders

✓	9.GM.01.	Nutritional support for patients with eating disorders will be selected based on the patient's degree of malnutrition and collaboration, and always with the psychiatrist's approval.

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✓	9.GM.02.	Before initiating artificial nutrition the patient's degree of collaboration must be assessed and an attempt must always be made to convince him/her of the benefits of natural feeding.
✓	9.GM.03.	In day hospitals, nutritional support for low-weight patients, where an oral diet is insufficient, can be supplemented with artificial nutrition (oral enteral nutrition). To ensure its intake, it must be administered during the day hospital's hours, providing supplementary energy ranging from 300 to 1,000 kcal/day.
✓	9.GM.04.	Oral nutritional support in eating disorder inpatients is deemed adequate (favourable progress) when a ponderal gain greater than 0.5 kg per week is produced, with up to 1 kg increments being the usual during that period. Sometimes, when the patient with moderate malnutrition resists resuming normal feeding, the diet can be reduced by 500-700 kcal and be supplemented by complementary oral enteral nutrition in the same amount, which must be administered after meals and not instead of meals.
✓	9.GM.05.	In the case of severe malnutrition, extreme starvation, poor progress or lack of cooperation of the patient in terms of eating, artificial nutrition treatment is indicated. If possible, an oral diet with or without oral enteral nutrition is always the first step, followed by a 3 to 6 day period to assess the degree of collaboration and medical-nutritional evolution.
✓	9.GM.06.	Regarding estimated energetic requirements, it is recommended that caloric needs at the beginning always be below the usual, that real weight, as opposed to ideal weight, is used to make the estimation and that in cases of severe malnutrition energetic requirements be 25 to 30 kcal/kg real weight or total kcal not higher than 1,000/day.

Anorexia nervosa

D	9.GM.1.	In feeding guidelines for children and adolescents with anorexia Nervosa, carers should be included in any dietary information, education and meal planning.
D	9.GM.2.	Feeding against the will of the patient should be used as a last resort in the management of AN.
D	9.GM.3.	Feeding against the will of the patient is an intervention that must be performed by experts in the management of eating disorders and related clinical complications.

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D	9.GM.4.	Legal requirements must be taken into account and complied with when deciding whether to feed a patient against his/her will.
D	9.GM.5.	Health care professionals must be careful with the healthy weight restoration process in children and adolescents with AN, administering the nutrients and energy required by providing an adequate diet in order to promote normal growth and development.

Bulimia nervosa

D	9.GM.6.	Patients with BN who frequently vomit and abuse laxatives can develop abnormalities in electrolyte balance.
D	9.GM.7.	When electrolyte imbalance is detected, in most cases elimination of the behaviour that caused it is sufficient to correct the problem. In a small number of cases, oral administration of electrolytes whose plasmatic levels are insufficient is necessary to restore normal levels, except in cases involving gastrointestinal absorption.
D	9.GM.8.	In the case of laxative misuse, patients with BN must be advised on how to decrease and stop abuse. This process must be carried out gradually. Patients must also be informed that the use of laxatives does not decrease nutrient absorption.
D	9.GM.9.	Patients who vomit habitually must have regular dental check-ups and be provided with dental hygiene advice.

Psychological Therapies for Eating Disorders
Cognitive-Behavioural therapy (Question 9.3.)

Bulimia nervosa

A	9.3.2.1.1.	CBT-BN is a specifically adapted form of CBT and it is recommended that 16 to 20 sessions are performed over 4 or 5 months of treatment.
B	9.3.2.1.2	Patients with BN who do not respond to or refuse to receive CBT treatment may be offered alternative psychological treatment.
D	9.3.2.1.3.	Adolescents with BN can be treated with CBT adapted to their age, level of development, and, if appropriate, the family's intervention can be incorporated.

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Binge-Eating Disorder

A	9.3.3.1.	A specifically adapted form of CBT can be offered to adults with binge-eating disorder (BED).

Self-Help (guided or not) (Question 9.4.)

Bulimia nervosa

B	9.4.1.1.1.	A possible first step in BN treatment is initiating a SH programme (guided or not).
B	9.4.1.1.2.	SH (guided or not) is sufficient only in a small number of patients with BN.

Interpersonal Therapy (Question 9.5.)

Bulimia nervosa

B	9.5.2.1.	IPT should be considered an alternative to CBT although patients should be informed that it requires 8 to 12 months to achieve results similar to those obtained with CBT.

Binge-Eating Disorder

B	9.5.3.1.	IPT-BED can be offered to patients with persistent BED.

Family Therapy (systemic or not) (Question 9.6)

Anorexia nervosa

B	9.6.1.1.1.	FT is indicated in children and adolescents with AN.
D	9.6.1.1.2.	Family members of children with AN and siblings and family members of adolescents with AN can be included in treatment, taking part in improving communication, supporting behavioural treatment and sharing therapeutic information.
D	9.6.1.1.3.	Children and adolescents with AN can be offered individual appointments with health care professionals, separate from those in which the family is involved.
D	9.6.1.1.4.	The effects of AN on siblings and other family members justifies their involvement in treatment.

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

General Recommendations for Psychological Therapy (GP) in Eating Disorders (GP) (Questions 9.3.-9.8.)

Anorexia nervosa

D	9.GP.1.	The psychological therapies to be assessed for eating disorders are: CBT, SFT, IPT, PDT and BT.
D	9.GP.2.	In the case of patients who require special care, the selection of the psychological treatment model that will be offered is even more important.
D	9.GP.3.	The objective of psychological treatment is to reduce risk, to encourage weight gain by means of a healthy diet, to reduce other symptoms related with eating disorders and to facilitate physical and psychological recovery.
D	9.GP.4.	Most psychological treatments for patients with AN can be performed on an outpatient basis (with physical monitoring) by professionals specialised in eating disorders.
D	9.GP.5.	The duration of psychological treatment should be of at least 6 months when performed on an outpatient basis (with physical monitoring) and 12 months for inpatients.
D	9.GP.6.	For patients with AN who have undergone outpatient psychological therapy but have not improved or have deteriorated, the indication of more intensive treatments (combined individual and family therapy, day or inpatient care) must be considered.
D	9.GP.7.	For inpatients with AN, a treatment programme aimed at suppressing symptoms and achieving normal weight should be established. Adequate physical monitoring is important during renutrition.
D	9.GP.8.	Psychological treatments must be aimed at modifying behavioural attitudes, attitudes related to weight and body shape and the fear of gaining weight.
D	9.GP.9.	The use of excessively rigid behaviour modification programmes is not recommended for inpatients with AN.
D	9.GP.10.	Following hospital discharge, patients with AN should be offered outpatient care that includes monitoring of normal weight restoration and psychological intervention that focuses on eating behaviour, attitudes to weight and shape and the fear of social response regarding weight gain, along with regular physical and psychological follow-up. Follow-up duration must be of at least 12 months.

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D	9.GP.11.	In children and adolescents with AN who require inpatient treatment and urgent weight restoration, age-related educational and social needs should be taken into account.

Binge-Eating Disorder

A	9.GP.12.	Patients must be informed that all psychological treatments have a limited effect on body weight.
B	9.GP.13.	A possible first step in the treatment of patients with BED is to encourage them to follow a SH programme (guided or not).
B	9.GP.14.	Health care professionals can consider providing BED patients with SH programmes (guided or not) that may yield positive results. However, this treatment is only effective in a limited number of patients with BED.
D	9.GP.15.	If there is a lack of evidence to guide the care of patients with EDNOS or BED, health care professionals are recommended to follow the eating disorder treatment that most resembles the eating disorder the patient presents.
D	9.GP.16.	When psychological treatments are performed on patients with BED, it may be necessary in some cases to treat comorbid obesity.
D	9.GP.17.	Adolescents with BED must be provided with psychological treatments adapted to their developmental stage.

Pharmacological Treatment of Eating Disorders
Antidepressants (Questions 9.9.)

Bulimia nervosa

B	9.9.2.1.1.	Patients should be informed that antidepressant treatment can reduce the frequency of binge-eating and purging episodes but effects are not immediate.
B	9.9.2.1.2.	In the treatment of BN, pharmacological treatments other than antidepressants are not recommended.
D	9.9.2.1.3.	The dose of fluoxetine used in patients with BN is greater than the dose used for treating depression (60 mg/day).
D	9.9.2.1.4.	Amongst SSRI antidepressants, fluoxetine is the first-choice drug for treatment of BN, in terms of acceptability, tolerability and symptom reduction.

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Binge-Eating Disorder

B	9.9.3.1.1.	SSRI antidepressant treatment can be offered to a patient with BED, regardless of whether he/she follows a guided SH programme or not.
B	9.9.3.1.2.	Patients must be informed that SSRI antidepressant treatment can reduce the frequency of binge-eating, but the duration of long-term effects is unknown. Antidepressant treatment may be beneficial for a small number of patients.

General Recommendations for Pharmacological Treatment (GPH) of Eating Disorders (Questions 9.9.-9.15.)

Anorexia nervosa

D	9.GPH1.	Pharmacological treatment is not recommended as the only primary treatment for patients with AN.
D	9.GPH.2.	Caution should be exercised when prescribing pharmacological treatment for patients with AN who have associated comorbidities such as obsessive-compulsive disorder (OCD) or depression.
D	9.GPH.3.	Given the risk of heart complications presented by patients with AN, prescription of drugs whose side effects may affect cardiac function must be avoided.
D	9.GPH.4.	If drugs with adverse cardiovascular effects are administered, ECG monitoring of patients should be carried out.
D	9.GPH.5.	All patients with AN must be warned of the adverse effects of pharmacological treatments.

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Binge-Eating Disorder

D	9.GPH.6.	In the absence of evidence to guide the management of BED, it is recommended that the clinician treat the patient based on the eating problem that most closely resembles the patient's eating disorder according to BN or AN guides.

Treatment of Eating Disorders in the Presence of Comorbidities (Question 9.18)

Eating Disorders with Organic Disorders

D	9.18.1.	Treatment of clinical and subclinical cases of eating disorders in patients with diabetes mellitus (DM) is essential given the increased risk in this group.
D	9.18.2.	Patients with Type 1 DM and an eating disorder must be monitored due to the high risk of developing retinopathy and other complications.
D	9.18.3.	Young people with type 1 DM and poor adherence to antidiabetic treatment should be assessed for the probable presence of an eating disorder.

Treatment of Chronic Eating Disorders (Question 9.19.)

✓	9.19.1.	The health care professional in charge of the management of chronic eating disorder cases should inform the patient on the possibility of recovery and advise him/her to see the specialist regularly regardless of the number of years elapsed and previous therapeutic failures.
✓	9.19.2.	It is necessary to have access to health care resources that are able to provide long-term treatments and follow-up on the evolution of chronic eating disorder cases, as well as to have social support to decrease future disability.

Treatment of Eating Disorders in Special Cases (Question 9.20.)

D	9.20.1.	Pregnant patients with AN, whether it is the first episode or a relapse, require intensive prenatal care with adequate nutrition and follow-up of foetal development.
D	9.20.2.	Pregnant women with eating disorders require careful follow-up throughout pregnancy and the postpartum period.

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10. Assessment of Eating Disorders (Questions 10.1.-10.2.)

D	10.1.1.	Assessment of patients with eating disorders should be comprehensive and include physical, psychological and social aspects, as well as a complete assessment of risk to self.
D	10.1.2.	The therapeutic process modifies the level of risk for the mental and physical health of patients with eating disorders, and thus should be monitored throughout treatment.
D	10.1.3.	Throughout treatment, health care professionals who evaluate children and adolescents with eating disorders should be alert to possible indicators of abuse (emotional, physical and sexual) to ensure an early response to this problem.
D	10.1.4.	Health care professionals who work with children and adolescents with eating disorders should familiarise themselves with national CPGs and current legislation regarding confidentiality.
✓	10.1.5.	It is recommended to use questionnaires adapted and validated in the Spanish population in the assessment of eating disorders. At present, the following specific instruments for eating disorders are recommended: EAT, EDI, BULIT, BITE, SCOFF, ACTA and ABOS (the selection of the version should be based on the patient's age and other application criteria). To assess aspects related with eating disorders, the following questionnaires are recommended: BSQ, BIA, BAT, BES and CIMEC (the selection of the version should be based on age and other application criteria).
✓	10.2.	The use of questionnaires adapted and validated in the Spanish population is recommended for the psychopathological assessment of eating disorders. At present, the following instruments for psychopathological assessment of eating disorders are suggested (version selection based on patient's age and other application criteria): Impulsiveness: BIS-11 Anxiety: STAI, HARS, CETA Depression: BDI, HAM-D, CDI Personality: MCMI-III, MACI, TCI-R, IPDE Obsessiveness: Y-BOCS

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11. Prognosis of Eating Disorders (chapter without recommendations)

12. Legal aspects concerning patients with eating disorders in Spain (questions 12.1.-12.2.)

12.1.	The use of the legal route is recommended in cases where the professional deems appropriate to safeguard the patient's health, while upholding his/her right to be listened to and properly informed on the process and medical and legal measures that will be applied. Clearly conveying the procedure is not only respectful to the right to information, but can also facilitate the cooperation and motivation of the patient and his/her environment in the procedure of complete hospitalisation (According to current legislation).
12.2.	One of the characteristic symptoms of eating disorders, and especially of AN, is the lack of awareness of the disease. The disease itself often entails a lack of sufficient judgement to provide valid and unmarred consent regarding treatment acceptance and decision. Hence, if a minor presenting AN and serious health risks refuses treatment, the use of appropriate legal and judicial routes should be employed. (According to current legislation).
12.3.	The necessary balance between different clashing rights requires the professional to observe and interpret the best solution in each case. However, it is always important to inform and listen carefully to both parties in order for them to understand the relationship between safeguarding health and the physician's decision. (According to current legislation).

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Abbreviations, Clinical Questions and Recommendations. (The complete list of abbreviations can be found in Annex 5)

ABOS: Anorectic Behaviour Observation Scale for parents/spouse

ACTA: Attitude Towards Change in Eating Disorders

AN: Anorexia nervosa

APA: American Psychiatric Association

BAT: Body Attitude Test

BED: Binge-Eating Disorder

BDI or Beck: Beck Depression Inventory

BES: Body Esteem Scale

BIA: Body Image Assessment

BIS-11: Barrat Impulsivity Scale

BITE: Bulimic Investigation Test of Edinburgh

BN: Bulimia nervosa

BMI: Body Mass Index

BPD Borderline Personality Disorder

BSQ: Body Shape Questionnaire

BT: Behavioural Therapy

BULIT: Bulimia test

BULIT-R: Revised version of BULIT

CBT: Cognitive-behavioural therapy

CBT-BN: Cognitive-behavioural therapy for bulimia nervosa

CBT-TA: Cognitive-behavioural therapy for binge-eating disorder

CDI: Children Depression Inventory

CETA: Assessment of anxiety disorders in children and adolescents

ChEAT: Children's Eating Attitude Test

CIMEC: Questionnaire on Influences on the Aesthetic Body Model (Body Shap Questionnaire (BSQ)

CPG: Clinical Practice Guideline

DM: Diabetes mellitus

DSM-IV-TR: Diagnostic and Statistical Manual of Mental Disorders, fourth edition- revised text

EAT (EAT-40): Eating Attitude Test

EAT-26: Short version of EAT-40

ECG: Electrocardiogram

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ED: Eating disorder

EDNOS: Eating disorder not otherwise specified (non-specific ED)

EDI: Eating Disorder Inventory

FT: Family therapy (non-specific)

GPH: General recommendations for pharmacological treatments

GM: General recommendations for medical measures

GP: General recommendations for psychological therapies

HAM-D: Hamilton Depression Scale

HARS: Hamilton Anxiety Scale

ICD-10: International Classification of Diseases, 10th edition

IPDE: International Personality Disorder Examination

IPT: Interpersonal therapy

IPT-BED: Interpersonal therapy for binge-eating disorder

ITP: Individualized Treatment Plan

Kcal.: Kilocalorie

Kg: Kilogram

MHC: Mental Health Centre

MHCFA Mental Health Centre for Adults

MGCFCA Mental Health Centre for Children and Adolescents

MACI: Millon Adolescent Clinical Inventory

MCMI-III: Millon Multiaxial Clinical Inventory-III

NHS: National Health System

OCD: Obsessive-Compulsive Disorder

PDT: Psychodynamic Therapy

PC: Primary care

SCOFF: Sick, Control, One, Fat, Food questionnaire

SH: Self-help

SSRI: Selective Serotonin Reuptake Inhibitor

STAI: State-Trait Anxiety Inventory

TCI-R: Temperament and Character Inventory-revised

Y-BOCS: Yale-Brown obsessive-compulsive scale

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1. Introduction

Background

The development of the evidence-based clinical practice guideline elaboration Programme for the NHS is being carried out 2ithin the framework of the development of the Quality Plan of the Ministry of Health and Consumer Affairs (MSC), through the National Health System (NHS)'s Quality Agency.

In the initial phase of this Programme (2006), the development of eight CPGs has been prioritised. A collaboration agreement has been established between the ISCIII and health technology assessment agencies and units and the Iberoamerican Cochrane Centre. The Health Sciences Institute of Aragón is in charge of the Programme's coordination activities.

In the bilateral agreement between the CAHTA of Catalonia and the ISCIII it was agreed to develop a CPG for eating disorders: anorexia nervosa (AN), bulimia nervosa (BN) and atypical or unspecified eating disorders (EDNOS), based on the best scientific evidence available, which would address the most important areas for the NHS, in a coordinated manner and with shared methodology which would be determined by a group of NHS professionals experienced in CPG development. These professionals have comprised the methodological group and the panel of collaborators of the CPG development programme[1].

Justification

In the last decades, eating disorders have gained increasing sociosanitary relevance due to their severity, complexity and difficulty in establishing a diagnosis and specific treatment. Eating disorders are pathologies of multifactorial ethiology where genetic, biological, personality, family and sociocultural factors converge, affecting mainly children, adolescents and young adults.

There are is no data available in Spain that analyses the economic burden of eating disorder treatments nor studies that assess the cost-effectiveness of different treatments. However, different studies conducted in countries of the European Union[2-7] indicate that direct costs (diagnosis, treatment and monitoring or follow-up) and especially indirect costs (economic losses derived from the disease that impact the patient and his/her social setting) entail a high economic burden and considerably decrease the quality of life of patients with eating disorders. According to a German study that was carried out in 2002, in the case of AN, average hospitalisation cost is 3.5 times higher than the general hospitalisation average[4].

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[(1)] N= 2,188 AN discharges, recorded during 2003 and 2004 in 156 health areas of 15 autonomous communities.

In 2002, Gowers SG, *et al.*[8] published the results of a survey conducted in 12 countries that were participating in the European project COST Action B6, which aimed to explore consensus and differences in the therapeutic approaches indicated for adolescents with AN (several different Spanish hospitals participated in this project). Results demonstrated significant agreement between the interviewed countries regarding the need to offer a wide array of services at the different levels of care. However, considerable differences were detected in terms of strategies, especially in hospital admission criteria, use of day centres/day hospitals, etc.

In the case of AN[1], according to the *Atlas of Variations in medical practice of the NHS,* where the variability in admissions into acute care public or publicly funded[9] hospitals, are described and mapped, a low hospitalisation incidence is observed in AN with, 0.32 admissions per 10,000 inhabitants and year. Hospitalisation rates for AN ranged between 0.08 –practically nil- and 1.47 admissions per 10,000 inhabitants and year. In terms of the weighted variation coefficient, the figures confirmed the wide variation among the different health areas in 70% of AN cases. After consideration of the variation randomised effect, the systematic component of variation of the health areas included in the percentiles 5 to 95 shows high variability (SCV_{5-95} = 0,26). The ample variation found may be related either to demand factors (different morbidity, socio-economic differences of the cities and towns) or to offer factors (practice style, healthcare resources, different policies for the development of mental care procedures, etc. The variability observed in hospitalisation rates among Spanish provinces is significant and accounts for a 40% of the variability in hospitalisation rates in cases of AN. Upon multilevel analysis, provincial level accounts for a larger variance proportion than in the region (regional level), reinforcing the hypothesis that the different provincial development of psychiatric models and services is behind the variations found. The remaining studied factors (age, gender, available income, educational level, predisposition to hospitalisation) do not appear to exert an influence upon the variability observed in hospitalisation rates in AN, save for the recorded unemployment that works in the opposite way: in areas with more recorded unemployment, the probability of hospitalisation is lower. No data are available for BN or eating disorder not otherwise specified (EDNOS-atypical eating disorders).

Different governmental Spanish and foreign institutions have published guides, recommendations and protocols on EDs in the last years, of which the following stand out:

• Osona (2008)[10] and the Gerona health region (2006)[11] from the Department of Health-Catalan Health Service
• semFYC (prevention activities and health promotion group) (2007)[12], (2005)[13].
• American Psychiatric Association (2006)[14].
• Action Manual of the Ministry of Health and Consumer Affairs on the scientific evidence regarding specialised nutritional support (2006)[15].
• General subdirectorate of Mental Health- Health Service of Murcia (2005)[16].
• Official College and Board of Physicians of Barcelona (2005)[17].
• Royal Australian and New Zealand College of Psychiatrists (2004)[18].
• National Health Institute (INSALUD, 2000)[19], (1995)[20].

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However, there is a need for a guide adapted to the Spanish population using the best possible methodology and based on the best available evidence.

Recently, other important actions related with eating disorders have been carried out in our context, such as: NAOS and PAOS programme for the prevention of obesity in children and a healthy diet; the Social Pact of the Madrid for the prevention of eating disorders; Image and Self-Esteem Foundation, and pacts between user federations (FEACAB and ADANER) and the MSC.

Magnitude of the problem

Estimations on the incidence and prevalence of eating disorders vary depending on the studied population and assessment tools employed. Therefore, in order to compare data from different national and international sources it is essential that the study design be the same. The study in "two phases" is the most appropriate methodology for the detection of cases in the community. The first phase consists of screening using self-report symptom questionnaires. In the second phase, assessment is carried out by means of clinical interviews that are conducted with individuals who obtained scores above the cut-off point in the screening questionnaire ("at risk" subjects), meaning only a subsample of the total initially screened sample is interviewed.

Within the studies that use correct two-phase methodology, very few perform random sampling of participants who score below the cut-off point of the screening questionnaire to interviews, which leads to a subestimation of the prevalence of eating disorders by not taking false negatives into account[21-23]. An additional problem is the use of different cut-off points in screening questionnaires.

In spite of these methodological difficulties, the increased prevalence of eating disorders is significant, especially in developed or developing countries, while it is nearly inexistent in third-world countries. Increased prevalence is attributable to increased incidence and duration and chronicity of these clinical pictures.

Based on two-phase studies conducted in Spain (Tables 1 and 2) on the highest risk population, women ranging from 12 to 21 years of age, a prevalence of 0.14% to 0.9% is obtained for AN, 0.41% to 2.9% for BN and 2.76% to 5.3% in the case of EDNOS. In total, we would be looking at an eating disorder prevalence of 4.1% to 6.41%. In the case of male adolescents, even though there are fewer studies available, a prevalence of 0% for AN, 0% to 0.36% for BN and 0.18% to 0.77% for EDNOS is obtained, with a total prevalence of 0.27% to 0.90%[21-29].

These numbers are similar to those presented in the NICE CPG (2004), where prevalence in of EDNOS, BN and AN in women ranges from 1% to 3.3%, 0.5% to 1.0% and 0.7%, respectively, and also to those in the Systematic Review of Scientific Evidence (SRSE) (2006) which reports prevalence in Western Europe and the United States (0.7% to 3% of EDNOS in the community, 1% of BN in women and 0.3% of AN in young women).

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Several studies on the incidence of eating disorders that have been published in North America and Europe present a 5- to 6-fold increase in incidence between 1960 and 1970. In 1998, a review presented by Pawluck, *et al*. on the general population of the United States reported that annual incidence of AN was 19 per 100,000 in women and 2 per 100,000 in males[30]. In the review presented by Hoek in 2003, incidence was 8 cases per 100,000 inhabitants for AN and 12 cases per 100,000 inhabitants for BN[32].

In a recent study in the United Kingdom (UK), incidence for AN in 2000 was 4.7 per 100,000 inhabitants (95% CI: 3.6 to 5.8) and 4.2 per 100,000 inhabitants in 1993 (95% CI: 3.4 to 5.0). In Holland, incidence of AN was 7.7 (95% CI: 5.9 to 10.0) per 100,000 inhabitants/year between 1995-1999 and 7.4 between 1985 and 1989[33]. In Navarra, a population survey in 1,076 13-year old girls was used to estimate an eating disorder incidence of 4.8% (95% CI: 2.84 to 6.82) in a period of 18 months, corresponding to: 0.3% AN (95% CI: 0.16 to 0.48); 0.3% BN (95% CI: 0.15 to 0.49) and 4.2% EDNOS (95% CI: 2.04 to 6.34)[34].

Incidence was greater in women aged 15 to 19 years: they constitute approximately 40% of identified cases in studies both in the US and Europe[30, 31, 33]. There are very few studies that report data on AN in prepubertal children or in adults[31]. There are also scarce studies that present incidence data of AN in men. Of all the aforementioned studies, we can conclude that incidence is lower than 1 per 100,000 inhabitants/year[33]. All these sources establish an eating disorder prevalence ratio of 1 to 9 in males versus women.

Table 1. Studies on the prevalence of eating disorders in adolescent females in Spain

Study	N	Age (years)	AN (%)	BN (%)	EDNOS (%)	EDs (%)
Madrid, 1997[24] Morandé G and Casas J.	723	15	0.69	1.24	2.76	4.69
Zaragoza, 1998[26] Ruiz P *et al.*	2,133	12-18	0.14	0.55	3.83	4.52
Navarra, 2000[27] Pérez-Gaspar M *et al.*	2,862	12-21	0.31	0.77	3.07	4.15
Reus, 2008[29] Olesti M *et al.*	551	12-21	0.9	2.9	5.3	9.1

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Table 2. **Studies on the prevalence of eating disorders in adolescent males and females in Spain**

Study	N	Age (years)	AN (%)	BN (%)	EDNOS (%)	EDs (%)
Madrid, 1999[25] Morandé G *et al.*	1,314	15	0.00 to 0.69	0.36 to 1.24	0.54 to 2.76	3.04 4.70 0.90
Valencia, 2003[21] Rojo L *et al.*	544	12-18	0.00 to 0.45	0.00 to 0.41	0.77 to 4.71	2.91 5.56 0.77
Ciudad Real, 2005[22] Rodríguez-Cano T *et al.*	1,766	12-15	0.00 to 0.17	0.00 to 1.38	0.60 to 4.86	3.71 6.41 0.60
Osona (Barcelona), 2006[28] Arrufat F	2,280	14-16	0.00 to 0.35	0.09 to 0.44	0.18 to 2.70	1.90 3.49 0.27
Madrid, 2007[23] Peláez MA *et al.*	1,545	12-21	0.00 to 0.33	0.16 to 2.29	0.48 to 2.72	3.43 5.34 0.64

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2. Scope and Objectives

Target Population

The CPG is focused on patients aged 8 years and older with the following diagnoses: AN, BN and eating disorders not otherwise specified (EDNOS). EDNOS include: binge-eating disorder (BED) and non-specific, incomplete or partial forms that do not satisfy all criteria for AN, BN and BED.

Although binge-eating disorder is the standard name, the truth is that several recurrent binge-eating episodes must take place to establish this diagnosis (amongst other manifestations). This guide refers to this disorder as binge-eating disorder.

The CPG also includes treatment of chronic eating disorder patients, refractory to treatment, who can be provided with tertiary prevention of the most serious symptoms and severe complications.

Comorbidities

The most frequent comorbidities which may require a different type of care have been included in the CPG:

- Mental: substance abuse, anxiety, obsessive-compulsive, personality, mood and impulse control disorders.

- Organic: diabetes mellitus, obesity, malabsorbtion syndromes and thyroid diseases.

Special situations

The approach to be employed in special situations such as pregnancy and delivery is also included.

Clinical setting

The CPG includes management provided in PC and specialised care. PC services are performed in primary care centres (PCC), the first level of access to health care. Patients with eating disorders receive specialised care, the second and third levels of access to health care, by means of inpatient management resources (psychiatric and general hospital), specialised outpatient consultations (adult and child/adolescent mental health centres/units, day hospitals for day care (partial hospitalisation) (specialised in eating disorders and for other general mental health disorders), emergency services and medical services of general hospitals. In general hospitals there are specific units specialised in eating disorders that include the three care levels. Other types of specific units are included: borderline personality disorder and toxicology units.

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Aspects included

The CPG includes the following aspects of eating disorders: prevention, detection, diagnosis, interventions at the different levels of care, treatment, assessment, prognosis and legal aspects.

Interventions

The CPG includes the following interventions for primary prevention of eating disorders: psychoeducational interventions, media literacy, social and political mobilization and activism (advocacy), dissonance-induction techniques, interventions focused on eliminating or reducing the risk factors of eating disorders or interventions to make the patient stronger (by developing stress coping skills).

Some of the outcome variables of primary prevention interventions are: incidence of eating disorders, BMI, internalisation of the thin-ideal, body dissatisfaction, anomalous diet, negative affects and eating disorders.

The CPG includes the following treatments:

- Medical measures: oral nutritional support, nutritional support with artificial nutrition (enteral oral [nasogastric tube] and parenteral intravenous) and nutritional counselling (NC). NC includes dietary counselling, nutritional counselling and/or nutritional therapy.

- Psychological therapies: cognitive-behavioural therapy (CBT), self-help (SH), guided self-help (GSH), interpersonal therapy (IPT), family therapy (systemic [SFT] or unspecified [FT]), psychodynamic therapy (PDT) and behavioural therapy (BT).

- Pharmacological treatments: antidepressants, antipsychotics, appetite stimulants, opioid antagonists and other psychoactive drugs (topiramate, lithium and atomoxetine).

- Combined interventions (psychological and pharmacological or more than one psychological intervention).

Clinically important treatment outcome variables according to the working group are: BMI, menstruation, pubertal development, reduction/elimination of binge-eating and purging, restoration of a healthy diet, absence of depression and psychosocial and interpersonal functioning. The latter two aspects are described in the questions regarding safety of interventions.
In some cases, recurrence or relapse results are described in the safety section, along with treatment withdrawls.

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Aspects not included in the CPG

The CPG does not include the following diagnoses related with eating disorders for several reasons:

- Orthorexia. A poorly defined and insufficiently studied syndromic spectrum that consists of extreme focus on eating healthy and contaminant-free foods. This disorder can be related to obsessive concerns about health, hypochondriac fears of diseases, and, in a certain way, to cultural attitudes linked to diet and food. Though it is true that people with orthorexia may present restrictive anomalies in their diets and ponderal losses, they cannot be considered atypical or incomplete cases of AN.

- Vigorexia (muscle dysmorphia). This disorder is characterized by excessive concern about obtaining body perfection by performing specific exercises. This extreme preoccupation entails significant dissatisfaction with one's own body image, excessive exercise, special diets and foods, to the point of generating dependence, as well as substance abuse[35]. At the moment it remains a vaguely defined disorder related with obsessiveness, perfectionism and dysmorphophobia[36-40].

- Night eating syndrome (nocturnal eaters). People with this disorder experience recurrent episodes of binge-eating during sleep. It is not defined whether these clinical pictures are due to an eating disorder or to a primary sleep disorder[41-44].

The CPG does not include patients under the age of 8 and, thus, diagnoses relating to eating disorders most common during those ages, such as swallowing phobia, selective eating and refusal to eat are not included. However, these disorders are not included either when they are observed in patients aged 8 and older for the following reasons:

- Food phobia (simple phobias). In some cases it may be an anxiety-related disorder, while in others it may be linked to hypochondria (fear of choking, swallowing phobia and death). Therefore, it does not seem to belong to the spectrum of eating disorders.

- Selective eating and refusal to eat. Both are eating disorders but lack the complete and characteristic symptomatology associated with AN and BN (cognitive disturbances, distorted body image, purging behaviours, etc).

This does not mean that when deciding on primary prevention policies no interventions should be carried out to address these behaviours, which could indeed be precursors of an eating disorder.

The CPG does not include exclusive interventions for comorbid conditions that may occur with eating disorders.

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Objectives

Main objective

To provide health care professionals responsible for the management of patients with eating disorders with a tool that enables them to make the best decisions to address the problems their care entails.

Secondary objectives

a) To help patients with eating disorders by providing them with useful information that will aid them in making decisions concerning their disease.

b) To inform families and carer on eating disorders and provide them with counselling and advice so they become actively involved in treatment.

c) To implement and develop health care quality indicators that enable the assessment of the clinical practice of recommendations presented in this CPG.

d) To establish recommendations for research on eating disorders that enable knowledge to grow.

e) To address confidentiality and informed consent issues of patients, especially in the case of minors under the age of 18, and to include legal procedures in the cases of complete hospitalisation (inpatient care) and involuntary treatment.

Main users

This CPG is aimed at professionals who are in direct contact with patients with eating disorders or who make decisions regarding the care of these patients (family physicians, paediatricians, psychiatrists, psychologists, nurses, dieticians, endocrinologists, pharmacists, gynaecologists, internal medicine physicians, odontologists, occupational therapists and social workers). It is also aimed at professionals pertaining to other fields who are in direct contact with patients with eating disorders (education, social services, media, justice).

The CPG's purpose is to serve as a tool for planning the integrated care of patients with eating disorders.

The CPG provides eating disorder patients with information that can also be used by family members and friends, as well as by the general population.

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3. Methodology

The methodology employed is described in the MSC's CPG development manual[1]. The steps followed have been:

– **Creation of the CPG working group** comprised of a clinical team, a technical team and two coordinators (clinical and technical). The clinical team consists of a group of health care professionals (psychology, psychiatry, nursing and nutrition and dietetics specialists) involved in the study and treatment of eating disorders and an attorney, who are carrying out their activity in Catalonia and are linked to the Master Plan of Mental Health and Addictions of the Catalan Department of Health, and representatives of Spanish scientific societies, associations or federations who are involved in the care of eating disorders. The technical team is composed of CAHTA members who have a wealth of experience and knowledge on the development of evidence-based CPGs and on critical assessment, and research support staff. The working group has relied on the participation of a group of collaborating experts from all over Spain selected by the clinical coordinator for his expertise in the matter, as well as another group of CAHTA members who have collaborated in some of the following activities: defining the scope, search, documental management, initial review of literature and internal review. Representatives of the societies, associations or federations involved in this project and expert collaborators have taken part in defining the scope and objectives of the CPG, in the formulation of key clinical questions and in the review of the guide's rough draft. The participants' declaration of conflict of interests can be found in the section "Authors and Collaborations".

– **Formulation of key clinical questions** in the following format: patient / intervention / comparison / *outcome* or result (PICO).

– **The search for scientific evidence** was structured in different stages:

1) Generic databases, meta-search engines and organizations that compile guidelines (National Guidelines Clearinghouse, National Electronic Library for Health, Tripdatabase, The Cochrane Library, Pubmed/Medline and BMJ Clinical Evidence) were consulted between 2003 and March 2007.

2) To complete the search, a manual search was performed for protocols, recommendations, narrative reviews, therapeutic orientations and guides on eating disorders elaborated by organisations pertaining to the health care administration, scientific societies, hospitals and other organizations of our setting. Some of these documents have inspired and served as a model for certain sections of this guide (see Annex 6.1.). This annex also lists documents created outside of Spain that have been excluded from the selection process due to low quality. However, some have been considered for the development of certain aspects of this CPG.

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3) To respond to those questions unanswered by the CPGs, SRSE and assessment reports (AR) included or to update them, a search for randomised controlled trials (RCTs) was performed in Pubmed/Medline between March 2007 and October 2007.

4) The search for CPG/SRSE/AR in Tripdatabase and Pubmed/Medline was also updated up to October 2007.

5) Additional searches were carried out in Pubmed/Medline and Scopus for primary prevention of eating disorders due to the limited information available in the documents included (until June 2008). The effect of primary prevention interventions for eating disorders has been assessed in RCT or in SRSE of RCT.

6) A search was also performed for cohort studies and prognosis of eating disorders in the Scopus and Psycinfo databases during the period spanning from 2000 to 2008.

7) The Ginebrina Foundation was also consulted for Medical Training and Research and the documents provided by the working group and the references of the documents included were reviewed.

– **Selection of Evidence.** The most relevant documents were selected by applying predefined inclusion and exclusion criteria:

- Inclusion criteria: guides, SRSE and ARs in certain languages (Spanish, Catalan, French, English and Italian) that dealt with the previously mentioned objectives. Minimum quality criteria were established for the guides, SRSE and ARs: the bibliographic bases consulted and/or the formulation process of recommendations (ad hoc defined criteria) had to be described.

- Exclusion criteria: documents/guides that were not original, unavailable (wrong reference or electronic address), not directly related with the proposed objectives, already included in the bibliography of other documents/guides or that didn't comply with minimum quality criteria.

Two independent reviewers examined the titles and/or summaries of the documents identified by the search strategy. If any of the inclusion criteria were not fulfilled, the document was excluded. If criteria were fulfilled, the complete document was requested and evaluated in order to decide whether it would be included or not. Discrepancies or doubts that arose during the process were resolved by consensus of the entire technical team.

– **Quality assessment of the scientific evidence.** Assessment of CPG quality was performed by a trained evaluator using the AGREE[45] instrument. Guides were considered of quality when they were classified as Recommended in the overall assessment. For SRSE/ARs and RCT, SIGN's methodology checklists were applied by an evaluator, following the recommendations established in the MSC's[1] CPG development manual. Classification of evidence has been carried out using the SIGN system (See Annex 1).

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– **Synthesis and analysis of the scientific evidence.** Different templates were used for information retrieval. Information regarding the main characteristics of the studies was obtained and then synthesised in evidence tables for a subsequent qualitative analysis. When the SRSE or CPGs reported the results of individual studies, these results were described in the section "scientific evidence".

In Annex 6.2 results of the CPG's search, selection and assessment of quality are described. In Annex 6.3. and Annex 6.4. NICE's CPG[30] and AHRQ's SRSE[31] are respectively described, representing the main scientific base on which this guide is founded.

– **Formulation of recommendations based on formal assessment or on SIGN's considered judgement.** The grading of recommendations has been performed using SIGN's system (See Annex 1). Recommendations pertaining to the NICE CPG have been considered by the working group and have been classified as: adopted (and, hence, accepted; they have simply been translated into Spanish) or adapted (and, hence, modified: changes have been made with the purpose of contextualising them to our setting). Controversial recommendations or those lacking in evidence have been resolved by the working group's consensus. The category of each recommendation appears in the chapters.

– **Description of psychological therapies.** Definitions are derived from the NICE[30] guide, from the SRSE where they have been assessed and from the working group itself (See Annex 4).

– **Description of drugs** (mechanism of action and approved indications in Spain) included in the CPG. The following websites have been consulted: Spanish Drug Agency (AGEMED) (https://agemed.es) and Vademecum (http://www.vademecum.es). It is recommended to read the technical chart of each drug before any therapeutic prescription given that the CPG only includes a very brief description of each drug and does not go into depth in terms of schemes, contraindications, etc. (See Annex 4).

– **Legal aspects.** To develop this chapter, aside from reviewing the current legislation in our country, several different articles and reference documents[17, 46-49] have been consulted.

– To develop **patient information** (See Annex 3.1.), a search has been performed for pamphlets and other documents containing information for the patient/carer both in printed and electronic formats. To this end, all documents identified in the websites of three relevant organizations (www.feacab.org, www.itacat.com and www.adaner.org) that comprise the majority of associations declared of public use at a national level through their different delegations and support groups and the material provided by the clinical coordinator have been reviewed.

Following the review of these documents, a content comparison table was elaborated from which the table of contents was developed. Once consensus of the final version had been reached by the working group and collaborating experts, the Association in Defence of Anorexia Nervosa and Bulimia Management (ADANER) and the Spanish Federation of Support Associations for Anorexia (FEACAB), which include most local organisations, carried out an external review, using a specifically designed questionnaire that inquired on the suitability of the information provided, the examples used, style and language, etc. Although this information is part of the CPG and must be delivered and explained to the patient/carer by health care professionals, we hope to edit individualised pamphlets that facilitate its dissemination.

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– **Formulation of research lines.** They have been developed from the research lines that are included in AHRQ 2006[31] (See chapter 15). G

– The **external reviewers of the CPG** are a group of experts in eating disorders (psychiatry, psychology and genetics specialists) selected for being heads of eating disorder units in Spanish hospitals and/or authors of relevant publications on the matter. Representatives from certain organizations who were unable to be a part of the CPG working group for a variety of reasons also served as external reviewers. The final version of the guide's text has been revised and approved by the group of authors.

– The **updating of this CPG** published in 2009 will be carried out after assessing new evidence that may arise in the next three years. Any changes made during this time will be reflected in the electronic format available at the GuíaSalud portal and the CAHTA website. To carry out guideline updating, the methodology proposed in the MSC's[1] CPG development manual will be applied.

– Resources containing detailed information on the CPG methodological process are available at the GuíaSalud portal (www.guiasalud.es).

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4. Definition and Classification of Eating Disorders

Key Questions:

4.1. How are eating disorders defined and classified? What are the shared and specific clinical features of each type?

4.2. Etiopathogeny of eating disorders: What are the main risk factors?

4.3. What are the most frequent comorbidities of eating disorders?

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4.1. How are eating disorders defined and classified? What are the shared and specific clinical features of each type?

Eating disorders are a group of mental disorders characterised by disordered eating behaviour and the development of behaviours aimed at managing weight. These behaviours lead to physical problems and deteriorated psychosocial functioning of the patient. Current classifications of eating disorders include AN, BN and other less specific disorders known as EDNOS (See chapter 7, "Diagnosis").

The first descriptions of AN date back to the 17th century, when Morton determined that the origin of this disorder, in contrast to other states of malnutrition, was a disturbance of the nervous system accompanied by sadness and pre-occupation. In the 19th century, it was described as an individual psychopathological picture similar to the one observed today, though it was believed to be a mood disease. Although the history of BN is much younger, the number of affected patients has been growing significantly in the past few years, possibly due to its less dramatic evolution and the ease with which affected individuals go undetected.

AN is an eating disorder that manifests itself as an uncontrollable desire to be thin, accompanied by the voluntary practice of procedures to achieve this goal: a strict, restrictive diet and purging behaviour (self-induced vomiting, laxative abuse, use of diuretics, etc). Despite gradual weight loss, patients present an extreme fear of becoming obese. They present body image distortion and an extreme pre-occupation with diet, figure and weight, and thus engage in food avoidance behaviour by means of compensatory actions to compensate for what they have ingested (extreme physical hyperactivity, purging behaviour, etc). Patients are not usually aware of the disease or the risks their behaviour entails. Their attention is focused on ponderal loss, leading to deficient nutritional states and ultimately to life-threatening risks. Usually there are previous personality traits that tend towards conformism, the need for approval, hyperresponsibility, perfectionism and poor response to internal needs.

BN is an eating disorder characterised by binge-eating episodes (voracious and uncontrolled eating), in which a large amount of food is consumed in a short period of time and usually in secret. Affected individuals attempt to counteract the effects of over-eating by means of self-induced vomiting and/or other purging methods (misuse of laxatives and diuretics, etc.) and physical hyperactivity. These individuals present pathological concern about weight and figure. BN does not necessarily lead to weight changes. Patients can present normal, low or excess weight. BN tends to be a hidden disorder given that it goes easily undetected and patients deal with feelings of shame and guilt. Patients usually seek help when the problem has progressed to advanced stages.

EDNOS are usually incomplete AN or BN pictures that do not constitute a complete picture. They are not, however, less serious. EDNOS include disorders such as the frequent use of inappropriate compensatory behaviour (after eating, chewing and spitting out small amounts of food) and recurrent compulsive eating episodes without compensatory behaviour. BED is a disorder that is currently in the study phase to determine if it is a disorder different from other EDNOS or simply a light form of BN. The main difference with BN is the absence of compensatory mechanisms, which eventually lead the patient to inevitably become overweight or obese.

What are the shared clinical features of AN and BN?

At a psychopathological level, AN and BN share an excessive pre-occupation with image and weight, which reaches irrational extremes in AN (not in BN). At a physical level, malnourishment and its potential complications are always present in AN and possible in the case of BN. There are also mixtures of anorexic and bulimic behaviour that are hard to differentiate, although ponderal loss and secondary malnutrition point to AN.

What are the specific clinical features of AN?

- Refusal to maintain normal body weight or to gain ponderal weight and distortion of body image: patients with AN are focused on their body weight, on the dread of gaining weight or becoming fat (a fear that is compounded as the patient loses weight) and on the desire to lose weight. An altered body experience is a core factor in the concept of eating disorders. Dissatisfaction with one's own body image is the main reason for weight loss, especially if it is associated with low self-esteem. It is a phobic fear of becoming fat and losing control over food. These ideas lead to behaviours aimed at achieving ponderal loss.

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– Other psychopathological disturbances: symptoms such as depressive mood, apathy, difficulty concentrating, anxiety, irritability, social alienation, loss of sexual drive, brooding and/or obsessive rituals regarding food are usually present.

– Physiological disturbances: as a consequence of ponderal loss there is malnutrition, leading to secondary disturbances, especially hormonal and metabolic[72].

– Amenorrhoea (primary or secondary): a characteristic symptom of the disease, it can appear in up to 70% of cases when there is significant ponderal loss. 20% of patients present amenorrhoea without prior detectable weight loss. This is due to hypogonadotrophic hypogonadism originated by a hypothalamic dysfunction that is considered to be primarily produced by a reduction of calorie intake and weight loss[73].

– Physical hyperactivity: Is usually present from the beginning of the disease. These individuals present two types of hyperactivity: deliberate physical exercise aimed at burning calories and losing weight, which can be practiced alone, presents obsessive characteristics and occurs only in a minority of patients, and involuntary hyperactivity secondary to malnutrition, which is an automatic response that manifests as persistent restlessness similar to that observed in laboratory animals subjected to hypocaloric intake.

What are the specific clinical features of BN?

– Loss of control over eating behaviour, which serves to understand recurrent binge-eating episodes. During these episodes, patients eat large amounts of food in a short period of time. The duration of these episodes can vary (approximately 2 hours), but is always within a 2-hour period; in fact, eating small amounts of food throughout the day is not considered a binge-eating episode, even though the episode does not have to occur in one place solely (for example, an individual may begin the episode in a restaurant and end it at home). Overall, even though the types of food eaten during the episode vary, in most cases it is sweets and high-calorie foods that are ingested, such as ice-cream or cake; quantity can also vary but may amount to several kilograms. Binge-eating episodes can occur at any time of the day but are more frequent at mid-afternoon and onward. They may be triggered by dysphoric moods, interpersonal difficulties, intense hunger or after restrictive diets or feelings related with weight, body figure or food. Episodes are accompanied by a feeling of losing control and can temporarily reduce dysphoria, but are always followed by feelings of guilt, self-contempt or depressive mood[74].

– Presence of compensatory mechanisms aimed at avoiding weight gain: 80% to 90% of patients engage in self-induced vomiting after the binge-eating episode. The immediate effect is relief of physical discomfort and decreased fear of gaining weight. The most frequent way of self-inducing vomiting is by inserting the hand to trigger the nauseous reflex. With time, it becomes easier to induce vomiting, and may be done by simply compressing the abdomen. Other mechanisms used to avoid weight gain are the misuse of laxatives and diuretics, the use of other anorexigenic drugs, excessive exercise or fasting. Both laxatives and diuretics cause dehydration and the resulting feeling of ponderal loss, but when their use is interrupted reflex fluid retention occurs and thus, their use is perpetuated.

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– Persistent preoccupation with weight and figure: it is morbid dread of becoming fat. Most BN symptoms are secondary to these beliefs and their modification is probably essential to achieve complete resolution of the disorder[75].

4.2. Ethiopathogeny of eating disorders: what are the main risk factors?

Like other mental disorders, eating disorders have a multiple and somewhat uncertain ethiology (yet unclear). According to studies, its ethiopathogeny involves several biological-genetic and vulnerability factors, psychological characteristics, sociocultural aspects and stressors. The specific impact of each one of them is yet to be determined.

Eating disorders tend to begin in adolescence, although we are seeing a gradual increase in the frequency of cases beginning in adulthood and childhood. These disorders usually affect the female population (for every 9 cases of eating disorders in women, there is 1 in men, approximately).

At present, several risk factors have been determined for these disorders. The designs of some of the studies used have been cross-sectional and enable us to establish associations and not causal relationships.

Biological factors

Research on biological factors has focused mainly on genetic factors and neurobiological disturbances.

Studies conducted on families show a higher frequency of eating disorders among relatives of individuals with eating disorders than among control subjects, leading to the conclusion that there must be family vulnerability to these disorders.

Case-control studies using molecular genetics have found a positive association between the presence of certain polymorphisms and a greater vulnerability to developing AN. The most studied are the serotonergic system (5-HT; regulates appetite, stress response, sexual behaviour, obsessive symptomatology, mood, etc), the dopaminergic system and neurotrophins[76] (especially BDNF, NTRK2[77] and NTRK3[78]).

Equally positive results have also been obtained in chromosomes 1, 2 and 13. However, these results are not yet conclusive. Genes seems to account for 60% to 70% of vulnerability in the case of AN[79]. In the case of BN, susceptibility is found in chromosome 10[80]. There is discrepancy regarding the early appearance of menarche as a risk factor in girls.

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Sociocultural factors

The studies identified the following sociocultural factors as risk factors for eating disorders: overprotective, rigid and demanding, conflictive and poorly cohesive family models[81, 82], destructured families (divorced parents), family history of mood disorders and obsessive-compulsive symptomatology[83], eating disorders (especially in mothers), atypical dieting and/or eating behaviour in the family (parents concerned about weight) [84, 85], obesity (especially in mothers) [85, 86], alcoholism (especially in fathers), inconsistent eating habits during childhood[81], careers and/or activities during childhood-adolescence that place too much emphasis on slimness and/or weight[87-89].

Psychological factors

The following psychological factors have been associated with eating disorders: mood disorders[90, 91], personality disorders[92], obsessive-compulsive disorders[93], impulse control disorder[94], following an anomalous and restrictive diet and pre-occupation with the body, personal history of eating difficulties[85], extreme rigidity, perfectionism, social alienation and low self-esteem[95].

Potentially stressful life events

Regarding the potentially stressful life events associated with eating disorders, the following stand out: sexual and/or physical abuse during childhood, criticism towards one's body and a history of life crisis[85].

According to a review of eating disorder risk factors, some of the previously mentioned factors have shown consistent results in the prediction of eating disorders, both in longitudinal and cross-sectional studies: gender, ethnicity (except Asians), eating problems and gastrointestinal disorders at early ages of childhood, sexual abuse and other adverse life experiences, low self-esteem, general psychiatric morbidity, high weight and bodily dissatisfaction and dieting[97].

Of all the different explanatory models of AN, Garner's (1993) [98] suggests that AN is the result of the interaction of three types of factors: predisposing, precipitating and perpetuating. Predisposing factors confer susceptibility to AN.

Some of these are determined by a genetic component, such as the female sex. Other predisposing factors are individual, family and cultural. Precipitating factors, such as dissatisfaction with body weight and shape, interact with predisposing factors in such a way that they condition affected individuals to the point where they decide they must lose weight and restrict eating. Once AN has initiated, gradual weight loss leads to complications derived from malnutrition. The disorder's multidimensional consequences (physical, psychological and social) are both perpetuating factors of the disorder and boosters of predisposing and precipitating factors.

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Current literature suggests that eating disorders are partially determined by both sociocultural[83, 84] and biological-genetic factors (the latter would explain 60%-70%)[99-101]. However, a part of the variance is not explained by any of these factors, leading to the performance of studies that assess the relevance of non-shared environmental factors which would explain why twins, who have been raised in a similar family environment, can differ in terms of eating behaviour, pathological in some cases and normal in others. Amongst these factors, the following would be included: parents treating each sibling differently, the subjects' personality and temperament, the subjects' relational style, experienced stressful situations and specific differential characteristics[81, 86]. Published scientific evidence shows that non-shared environmental factors are more relevant than shared factors. In this respect, 24% to 42% of variance in AN[102] and 17% to 46% of variance in BN[90] would be explained by the influence of non-shared environmental factors.

4.3. What are the most frequent comorbidities in eating disorders?

Comorbidity in eating disorders is common, both of mental and organic origin. The next section describes the most frequent associations. The therapeutic approach required in these cases will be addressed in the chapter dealing with treatment.

Substance-related disorders

Abuse of illegal substances and chemical dependence is common in eating disorders, especially in BN and similar clinical pictures. In the beginning the use of stimulants (amphetamines and cocaine) is related with attempts to decrease appetite, but later it is linked with impulsivity-associated BN pictures. Up to 40% of diagnosed patients (AN or BN) admit to abusing or having a dependence on alcohol or illegal substances[103-105].

Anxiety disorders

Anxiety is so present in eating disorders it is hard to decide if it is a specific clinical component of these disorders or if it is a comorbid condition. Eating disorders present specific phobia clinical pictures (phobia of certain foods, social phobia, etc.) and others such as claustrophobia or unrelated simple phobias. Panic attacks or anxiety crises present a similar situation. In some cases they are linked to eating disorders and in others they are truly comorbid. Studies report very varied anxiety prevalences that range between 10% and 40%, depending on the measurement tools and inclusion criteria used[106-109].

Obsessive-compulsive disorder (OCD)

Patients with eating disorders, especially AN, present significant prevalence of obsessive personality traits (See personality disorders). Many of these patients are rigid, strict, organized, responsible, constant, intransigent and intolerant, personality traits that predispose and

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accompany eating disorders. However, in a considerable number of patients, up to 40% of them diagnosed with AN, obsessive-compulsive disorder that meets comorbidity criteria is present[110-112].

Personality disorders

Approximately 30% of eating disorder cases present personality disorders[113-115]. There is a high prevalence of patients who meet BN criteria and present an associated personality disorder, especially borderline and histrionic.

Mood disorders

Depression is closely linked with eating disorders. It is hard to think of AN or BN without a depressive clinical picture. As in the case of anxiety, in some cases depression predisposes and in others it is linked with the clinical manifestation of eating disorders or presents itself as a comorbid condition. Prevalence of depression ranges between 40% and 80% and occurs more frequently in BN[116, 117].

Impulse control disorder

Some of the behavioural disturbances of eating disorders entail a loss of self-control (overeating, purging behaviour, self-aggression, etc.). Other impulse control disorders occur with eating disorders, such as kleptomania or trichotillomania[118, 119].

Diabetes mellitus

DM is present in the genesis and evolution of eating disorders and also in treatment, which will have to be adjusted to this physical condition. Studies show very varied prevalence rates. Between 0.5% and 7% of cases of AN and BN present Type 2 DM. This percentage reaches up to 20% in EDNOS. Up to 9% of obese diabetics present an eating disorder[120-122].

Type 1 DM is a risk factor for eating disorders (three times more risk in BN and two times more risk in subclinical EDNOS[120] than the population without type 1 DM). Type 2 DM is a risk factor for engaging in inadequate eating behaviours. When type 1 DM and an eating disorder coincide (OR: 4.8; 95% CI: 3.0 to 7.8) physical complication such as retinopathy increase. In another 4-year longitudinal study (N=91) on a cohort of patients with both pathologies (ED and type 1 DM), 60% were associated with retinopathy[123]. In a further study conducted by Nielsen, 2002[120], mortality in patients with type DM at 10 years follow-up was 2.2 per 1,000 inhabitants/year; in the AN population it was 7.3 and in the population with AN associated with type 1 DM it was 34.4.

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Obesity

Obesity as a risk factor is linked to AN and BN. It is also a mid-long term habitual state of BED and, in this case, obesity directly influences diagnosis and treatment. Up to 6% of obese children present BED[124-126].

Malabsorbtion syndromes

Especially in AN, malabsorbtion syndromes, gluten-intolerance or lactose-intolerance are risk factors for eating disorders, course and prognosis modifiers and pathologies that must be taken into account when planning a treatment diet aimed at ponderal recovery. There are no prevalence studies and the literature only yields descriptions and studies of alienated cases[127, 128].

Thyroid diseases

Thyroid diseases, both hyper and hypothyroidism, are relevant in the onset, course, prognosis and treatment of eating disorders. There are no prevalence studies, only case description studies[129-132].

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5. Prevention of Eating Disorders

Key Questions:

5.1. What is the efficacy of primary prevention interventions in avoiding eating disorders? Are there any negative effects?

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Primary prevention aims to limit the incidence of disease, in this case of eating disorders, by managing causes and exposure to risk factors or the patient's increased resistance to these. Clearly, the first step to establish primary prevention measures is to identify relevant exposures and assess their impact on the patient's and the population's risk of developing the disease.

Specific primary prevention interventions for the most frequent eating disorders can be classified in the following 5 types: psychoeducational, media literacy, dissonance-induction techniques, and interventions focused on eliminating eating disorder risk factors and strengthening the host.

In order to implement primary prevention measures there are two strategies, which often complement each other: the population strategy and the high-risk strategy. The population strategy is general and is aimed at the entire population with the objective of reducing its average risk. The high-risk strategy is individual and is aimed at those people who most need it, meaning the most vulnerable individuals or those presenting additional risk as a consequence of being exposed to certain risk factors. In the case of eating disorders, groups are considered of risk due to their age (adolescence), sex (female) or engagement in risk activities, be it as a hobby or at a professional level (gymnasts, athletes, models, ballerinas, skaters, elite athletes, etc.)[133].

5.1. What is the efficacy of primary prevention interventions in avoiding eating disorders? Are there any negative effects?

In order to respond to these questions, 4 high quality (1++) quantitative SRSE or metaanalysis (MA) on eating disorder prevention programmes have been identified.

Of the four MAs, one focuses the review on children and adolescents and only includes RCT[134]; another one focuses on reviewing RCT and quasi-experimental studies (Q-RCT) on Internet-based prevention programmes[135]; the others perform a general review of the topic, including RCT and quasi-experimental studies[136]. The potential iatrogenic effect (negative impact) of primary prevention programmes was another objective of two of these 4 MAs[134, 136].

The MA conducted by Stice E, *et al*. (2007) [137] includes all studies considered in the MA carried out by Newton MS and Ciliska D published in 2006[135]; 24/46 studies included in the MA conducted by Cororve FM, *et al*. (2006) [136]; 10/12 studies included in the fourth MA conducted

by Pratt BM and Woolfendern SR (Cochrane Review), which was published earlier (2002) [134].

The search for RCTs has identified eight studies that were either published after or not included in previous MAs[138-145]. The eight RCTs assess high-risk strategies given that primary prevention programmes are aimed at adolescents (men and women) [138]; at adolescent women with body dissatisfaction[141, 143, 145], at female university students with subclinical levels of eating pathology or who wish to improve their body image[139, 140, 144] and female athletes[142]. The most administered intervention type was psychoeducational[138-140, 145] (in three of the studies the Internet was used), followed by dissonance-induction techniques[143, 144], critical assessment of media content (media literacy) [141] and a specific programme called ATHENA (The Athletes Targeting Healthy Exercise and Nutrition Alternatives)[142].

The NICE CPG (2004)[30] did not address this aspect and only some protocols, recommendations and other documents on eating disorders elaborated by other organizations in our setting (See Annex 6.1.) tackled this aspect[10, 11, 13, 19, 146-151], even though only the PAPPS[12] group of experts formulates general recommendations.

In the following section, the scientific evidence on primary prevention programmes for eating disorders is described.

Scientific Evidence

In a RCT (Stice, 2008; United States) conducted on a sample of 481 adolescent women (mean age: 17 years) with body dissatisfaction, the following interventions were compared: dissonance-based thin-ideal internalisation reduction programme (group 1) vs. the healthy weight control programme (group 2) vs. expressive writing control condition (group 3) vs. assessment-only control condition (group 4). Group 1 showed significant reduction of thin-ideal internalisation, body dissatisfaction, negative affects and psychosocial impairment and lower risk for eating pathology onset at 2- and 3- year follow-up vs. control subjects. In group 1 there was a greater decrease in thin-ideal internalisation, body dissatisfaction and psychosocial incapacity when compared to group 3. Group 2 showed greater reduction in all results, less increases in weight and lower risk of eating pathology onset at 2- or 3- year follow-up vs. control subjects. Group 2 showed greater decrease in weight and in thin-ideal internalisation than group 3. **RCT 1++**

At 3-year follow-up, participants in the dissonance programme showed a 60% reduction in risk for eating pathology onset; healthy weight participants showed a 61% reduction; assessment-only controls showed a 55% reduction in risk of obesity onset. Therefore, the effect of such programmes is considered clinically important and enduring in time.

In a RCT (Jones M, 2008) [138] a population of adolescents (73 females and 22 males) from public schools in the US at risk for overweight was studied, and a specific 16-week Internet-facilitated intervention (StudentBodies2-BED)(N=52, 38m; 14v) was compared to a wait list (N=53, 35m; 18v) with 9-month follow-up. The specific programme's content combines psychoeducational and behavioural interventions **RCT 1++**

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(causal factors). Participants in the experimental group presented significant changes at baseline BMI ($p<0.01$) and at follow-up ($p<0.05$) and significant subjective and objective reduction of binge-eating both after the intervention ($p<0.01$) and during follow-up ($p<0.05$) *vs*. the control group. Results suggest that an Internet-based intervention programme is moderately effective at the short-term reduction and maintenance of body weight and in reducing binge-eating. This study also demonstrates that weight maintenance and reduction of psychological disturbances caused by eating disorders can be easily achieved when using easily disseminated, Internet-based programmes.

In a RCT (Becker, 2008) [144] a population comprised of 188 female university students (age range: 18-21 years) who had participated in a specific programme on body image but who did not have an eating disorder was studied. Cognitive dissonance moderated by trained peer leaders (N=88) was compared to mobilisation and social and political activism (*media advocacy*) (N=85) with 8-month follow-up. Results indicated that both interventions reduce thin-ideal internalisation, body dissatisfaction, dietary restraint and bulimic pathology at 8 months, although higher and lower risk groups responded differently. Both interventions were effective in the high-risk group. However, only the dissonance-based technique was effective in lower risk participants.

RCT 1+

Overall, both interventions yielded better results in the high-risk group, whereas cognitive dissonance only seems to benefit the lower risk group. Further studies are needed to corroborate the viability of using trained peer leaders in dissonance-based prevention programmes.

In an RCT (Heinicke, 2007) [145] a population of 83 female adolescents (mean age: 14.4 years) from schools in Australia who self-identified as having eating and body image problems was studied. The programme (My Body, My Life: Body Image Program for Adolescent Girls) (N=40) was compared to the control group (N=43) at 2- and 6-months follow-up. This programme consists of 6 weekly group sessions delivered via the Internet during 6 weeks and a SH manual (psychoeducational content), coordinated by a trained therapist. Out of those who completed the programme (28 and 26 of the experimental and control groups, respectively), clinically significant improvements were reported in the experimental group in terms of body dissatisfaction, disordered eating and depression at the end of the intervention ($p\leq0.002$). At two months follow-up, clinically significant improvements were obtained in the scores of different specific eating disorder outcome measures and other related measures (also at 6 months follow-up).

RCT 1++

This Internet-delivered programme was well-accepted and obtained good results in improving body dissatisfaction and eating problems.

In an RCT (Jacobi, 2007) [140] performed on a population comprised of 100 female university students in Germany (age range: 18-29 years) who wished to improve their body image, an 8-week Internet-based psychoeducational prevention programme (German adapted version of Stanford's original programme) (N=50) and a wait-list (N=50) were compared, with follow-up at 3 months. The experimental group obtained better results than the control group in all measures. Participants in the

RCT 1++

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experimental group presented sustained effects in terms of their knowledge regarding a healthy diet and exercise, a reduction in their wish to be thin and decreased disordered eating at 3 months follow-up.

The experimental programme was also effective in the group of women who were at risk, resulting in favourable changes in the majority of variables. This study demonstrates the benefits of using an Internet-based prevention programme and proves that transcultural adaptation can be successful.

Based on the results of this MA, 51% of eating disorder prevention programmes reduce eating disorder risk and 29% reduce current or future eating pathology. These overall percentages are favourable when compared to results obtained in other public health programmes (21% obesity prevention and 22% HIV prevention). **MA of RCT and Q-RCT 1++**

The effect of prevention programmes was greater if the following characteristics were satisfied: selected strategies aimed at high-risk populations (versus universal), aimed exclusively at women (versus both sexes), offered to participants over 15 years of age (versus younger people), interactive formats (versus didactic programmes), delivered by trained professionals (versus endogenous providers, such as teachers or educators), with multiple sessions (versus single session), including contents on body acceptance or the use of induction-dissonance techniques (versus psychoeducational or sociocultural interventions), assessed in trials using validated measures and, hence, more sensitive in determining the effect of interventions (only this type of measures should be used) and with shorter follow-up periods.

In an RCT (Brien, 2006) [139] a population of 24 university females from Canada of several different origins (58% Caucasian, 19% Hispanic and the rest of other origins), presenting subclinical levels of eating pathology, was studied. An 8-week psychoeducational intervention (N=13) was compared to self-monitoring control (N=11). Participants in the experimental group showed improvement in the scores of different specific questionnaires on eating disorders and other outcome measures that were significantly different from those observed in the control group. **RCA 1+**

A psychoeducational intervention can alter the subclinical levels of eating pathology in female university students from different cultures.

In an RCT (Wilksch, 2006) [141] a population of young adolescent (mean age: 13.8 years) students in Australia (N=100 women and 137 males) was studied to compare one media literacy lesson vs. 6 lessons in the control group. Following the intervention, males had significantly lower values in 4/5 subscales of the questionnaire used to measure media internalisation, whereas females had significantly lower values in one subscale. Higher baseline levels of dietary restraint, reading/buying magazines and a perception of sociocultural pressure predict lesser reductions in males' scores, whereas depression predicts lesser reductions in females. Males must be included in eating disorder prevention programmes and media literacy is a promising approach to primary prevention of eating disorders. **RCA 1+**

In the only RCT (Elliot, 2006) [142] identified in a population of 1,179 female athletes studying in schools in the US (mean age: 14.6 years), the ATHENA programme (N=457) was compared to a control group (standard treatments) (N=471). ATHENA **RCA 1++**

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is a programme that promotes a healthy diet and physical exercise as alternatives to eliminate harmful practices. It consists of group sessions with educational material. Significant reductions were observed in the experimental group versus the control group in terms of behaviours related with eating disorders and in the use of weight reduction drugs ($p<0.05$). Athletes obtained positive changes in strength-training self-efficacy ($p<0.005$) and in healthy dieting practices ($p<0.001$). The components of the ATHENA programme were significantly modified in the right direction: mood ($p<0.005$), refusal skills ($p<0.05$), belief in the media ($p<0.005$), perception of close friends' body shaping drug use ($p<0.001$).

The ATHENA programme significantly modified risk factors by reducing consumption and delaying the use of pills to lose weight, amphetamines and other body-shaping substances, such as anabolic steroids and other sports supplements.

The second meta-analytical review conducted by Newton MS and Ciliska (2006) [135] included 5 studies, 4 of which were RCTs and one quasi-experimental. All were carried out in California on a sample of women and using the same Internet-based programme vs. control group (wait-list). The prevention programme used (Student Bodies) includes lectures and reflexions, a magazine on body image via the Internet and an Internet-based asynchronous discussion group. Conceptually, this programme is comprehensive given that it addresses many of the factors that are involved in eating pathology (cognitive/affective factors, psychological factors, peer/sociocultural rules and behavioural factors). While carrying out the MA, none of the results studied evidenced statistically significant differences and, thus, conclusions could not be issued on the effectiveness of this programme. The authors mention the small sample size as a possible reason for this.

MA of RCT and Q-RCT 1++

In yet another identified MA, in this case conducted by Cororve FM, et al. (2006) [136] 46 studies (32 published and 14 unpublished) were finally included. The interventions assessed in the different studies were classified as purely psychoeducational, CBT-based psychoeducational, or purely interactive/non psychoeducational. There were no differences between the types of interventions. There were no significant different between purely psychoeducational and CBT-based psychoeducational interventions regarding dieting practices, internalisation and body dissatisfaction. Prevention programmes had the greatest beneficial effect on the acquisition of knowledge. The effect was also positive in other outcome variables, indicating an improvement in the symptoms of general eating pathology, dieting behaviour and internalisation of the thin-ideal. Body dissatisfaction also improved, but the effect was not sustained at follow-up.

MA of RCT 1 ++

No harmful effects related to including eating disorder information in the content of the interventions were observed.

In the MA (Cochrane Review) published in 2002 by Pratt BM and Woolfenden SR[134] 12 RCTs were included. Based on the programme's content there were 4 types of intervention: a) knowledge on eating disorders; b) eating attitudes and behaviours and adolescent issues; c) media literacy and mobilisation and social and political activism

MA of RCT 1++

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(advocacy); and d) interventions related with self-esteem. 2 of the programmes based on media literacy and mobilisation and social and political activism (advocacy) indicate a reduction in the internalisation or acceptance of societal ideals relating to appearance at 3- to 6-month follow-up (SMD:-0.28; 95% CI: -0.51 to -0.05). There is sufficient evidence to support the effect of the 5 programmes that include interventions on eating attitudes and behaviours and other adolescent issues. There is also sufficient evidence to support the effect of two of the programmes designed to improve self-esteem.

There is not enough evidence to indicate that harm resulted from any of the programmes included in this SRSE.

Evidence Summary

MA[137] 1++	This MA identifies the characteristics of eating disorder prevention programmes that produce the greatest effects. Programmes that are selected, interactive, multisession, offered solely to women, offered to people over 15 years of age, delivered by trained professionals, that incorporate contents related with body acceptance and induction-dissonance techniques, that assess effects using validated measures, that do no include psychoeducational contents and have shorter follow-up periods produce greater effects.
MA[137] 1++	There are several eating disorder prevention programmes that have also been proven to be effective when delivered by teachers under valid ecological conditions.
MA[137] 1++	Some of the eating disorder prevention programs have had an effect on both these disorders and obesity, which is promising from the point of view of public health.
MA[135] 1++	There is no consistent evidence regarding the impact of Internet-based prevention strategies on eating disorder symptomatology and on the factors that contribute to the development of these disorders.
MA[136] 1++	Prevention programmes had a greater effect on improving knowledge and a lesser effect on reducing incorrect behaviours and beliefs regarding eating.
MA[136] 1++	Studies aimed at high-risk populations produced greater benefits than those performed on the general population (universal strategies).
MA[136] 1++	The results did not confirm the iatrogenic effects of including psychoeducational content on eating disorders in prevention programmes.

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MA[134] 1++	No definitive conclusion was reached regarding the effectiveness of eating disorder prevention programmes, both aimed at the general population (universal strategies) and at high-risk populations (selective strategy), in children and adolescents.
MA[134] 1++	There is not sufficient evidence to suggest that any of the interventions included in the MAs have a short-term negative impact (harmful effects).

Recommendations

✓	5.1.	Sample, format and design characteristics of eating disorder programmes that have demonstrated the highest efficacy should be considered the model for future programmes.
✓	5.2.	In the design of universal eating disorder prevention strategies it must be taken into account that expected behaviour and attitude changes in children and adolescents without these types of problems may differ from those of higher risk populations.
✓	5.3.	Messages on measures that indirectly protect individuals from eating disorders should be passed on to the family and adolescent: following a healthy diet and eating at least one meal at home with the family, facilitating communication and improving self-esteem, avoiding family conversations from compulsively turning to eating and image and avoiding jokes and disapproval regarding the body, weight or eating manner of children and adolescents.

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6. Detection of Eating Disorders

Key Question:

6.1. What screening instruments are useful to identify eating disorder cases?

6.1. What screening instruments are useful to identify eating disorder cases?

Eating disorders represent the third most common chronic disease in the adolescent patient population. Additionally, the three types of eating disorders (AN, BN and EDNOS) are amongst the three most common psychiatric diagnoses in adolescent females. Delayed identification of eating disorder patients leads to higher morbidity due to delayed treatment, and hence, worse prognosis. It is important to identify people at high risk of developing an eating disorder in order to tackle the disease at early stages and carry out an early intervention. To achieve this goal, involvement of primary care physicians is essential to detect symptoms and signs of suspected eating disorders.

There are few studies on the detection and diagnosis of eating disorders in PC. Data point to a situation of subdiagnosis, due to several reasons: scarce awareness of professionals regarding this issue, the lack of real time resulting from health care pressure that impedes providing better, more comprehensive care and preventive activities aimed at these patients, the low attendance of adolescents to primary care practices and their lack of "disease awareness"[152].

Given these circumstances, the appropriate use of a brief and validated screening instruments during healthy children/adolescent consultations and those prior to performance of sports would provide a good opportunity to apply prevention programmes for eating disorders, smoking, alcohol, drugs and safe sex in an integrated manner within a structured health maintenance programme from a family medicine approach.

There has been some confusion regarding the use of screening instruments. They are inefficient to establish an eating disorder diagnosis, but are useful tools for a quick initial assessment aimed at ruling out suspicious symptoms in the first phase of the two stage screening process, in which those patients who obtain high scores are newly assessed to determine if they fulfil formal diagnostic criteria.

To identify potential cases of eating disorders, several self-report screening questionnaires that enable systematic assessment of eating behaviour have been designed. All of these include questions regarding personal eating and dieting habits, weight, exercise, menstruation, body shape perception, self-image, self-esteem, drug use, relationship with the family and others, among other topics, given that most of the time patients with incipient eating disorders go to the doctor due to other symptomatology, such as weight loss, amenorrhoea, depression, irritability, etc. Therefore, it is important to ask questions about these aspects.

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Of all the proposed criteria and recommendations for the assessment of the screening instruments, some authors determine their use/relevance, development and psychometric properties and external validity to be the most important. Taking these considerations into account, and based on the results of an SRSE published by Jacobi, *et al*. (2004) [153], only a few of the self-report instruments labelled as screening instruments for eating disorders fulfil the aforementioned criteria.

Of the screening instruments for identification of potential cases of eating disorders, four fulfilled the criteria established by the previous SRSE: BET (*Branched Eating Disorders Test*) [154], EDDS (*Eating Disorder Diagnostic Scale*) [155], SED (*Survey for Eating Disorders*) [158] and SCOFF (*Sick, Control, One, Fat, Food questionnaire*) [157]. Only the latter has an adapted version validated in our setting. At present there are no screening instruments that can differentiate between complete and partial eating disorders.

Only one screening instrument for AN, the EAT-40 and its versions (EAT-26 and ChEAT), has high sensitivity and specificity, but its positive predictive value (PPV) is low for identifying AN cases in the population. There are no questionnaires for screening of partial or subclinical cases of AN.

Of the three screening tools for BN: two-item screen[158], only BULIT and BITE have sensitivity and specificity values for BN. However, the specificity of these questionnaires to differentiate between BN and partial cases of BN and other eating disorders has not been clearly ascertained and population data are scarce.

The following section describes the tools used for detecting eating disorder cases that meet all recommended criteria to achieve this goal:

SCOFF Survey
Sick, Control, One, Fat, Food questionnaire.
Morgan J, *et al*., 1999.

The SCOFF is an eating disorder screening tool that consists of five yes/no questions that assess the loss of control over eating, purging and body dissatisfaction (See Annex 2.1), thus enabling its application in primary care. Scores range from 0 to 5 points (No=0 and Yes=1). A total score of 2 or more points identifies people at risk of having an eating disorder (AN sensitivity: 100%; BN: 100%; AN and BN specificity: 85% and 80%, respectively; false positive rate 7.3% for AN and 8% for BN) [157]. Independent studies performed in primary care indicate sensitivity values that range between 78% and 85% and specificity values that range between 88% and 90%, with only affirmative answers[159, 160]. These are excellent results, especially because the questionnaire is so brief. The reliability of the instrument when self-administered (written) or when administered by the physician (oral) was also assessed and results only evidenced minimal differences in SCOFF's detection ability. However, the authors suggest that self-report responses may be more honest given that the patient's confrontation with the interviewer is reduced[161].

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Spanish version

There is an adapted Spanish version that has been validated in our setting by García Campayo J, *et al*., 2004[162] for early detection of eating disorders in primary care (women aged between 14 and 55 years with a cut-off point of 2 or more); sensitivity was 98% [95% CI: 93.5 to 99.5] and specificity was 94% [95% CI: 86.4% to 98.5%]. For each specific eating disorder, sensitivities for 94% specificity were as follows: BN, 98%; AN, 93%; and EDNOS, 100%. Limitations relating to the adaptation of item 1 have been identified. It is recommended to review these aspects before administering the Spanish version of SCOFF.

A Catalan version has also been adapted and validated in our setting (SCOFF-c) by Muro-Sans P, *et al*., 2008 in a community sample comprised of Spanish adolescents (51% males and 49% females; mean age=14 years; SD=1.31)[163]. In this study sensitivity was 73% (95% CI: 63.2 to 82.9) and specificity was 94% (95% CI: 74.9 to 80.5). One of the possible reasons for low sensitivity is sample characteristics (Barcelona youth who were recruited in PC).

Of the numerous eating disorder screening questionnaires, results indicate the SCOFF survey can be a useful questionnaire given that it enables quick and easy primary care detection of eating disorder risk groups in the community[164]. The SCOFF survey has been adopted as the standard screening instrument in the UK[30]. Its characteristics also seem useful in monitoring the course of treatment (See Chapter 10, "Assessment").

EAT
Eating Attitudes Test. DM Garner and PE Garfinkel, 1979.

The EAT was designed for the assessment of disordered eating attitudes, especially those related with fear of gaining weight, the impulse to lose weight and the presence of restrictive eating patterns. Its intention was to devise an instrument that was easy to use and easy to correct and that was sensitive to symptomatic changes throughout time. The EAT is a self-report tool comprised of 40 items (EAT-40). Each item is valued in a 6-point Likert scale that ranges from "never" to "always". Scores range from 0 to 120. It is a valid and reliable questionnaire that has been widely used in the assessment of eating disorders. With the 30 point or more cut-off point in a group of AN patients and a control group, sensitivity was 100% and specificity was 84.7%, with a PPV of 78.5% and a false-positive rate of 9.8%[165].

Other studies had revealed that the EAT test can be useful for the detection of AN cases that have not been previously diagnosed or to identify current or incipient cases of AN in high risk populations (ballerinas, model trainees, for example) with sensitivities, specificities and PPVs that range between 75% and 91.7%, 66.1% to 75% and 16% to 18.8%, respectively, with false-positive rates between 23.2% and 31.7%[97]. Of all the different instruments that have been developed since the 70s up until today, the EAT-40 has been the most widely endorsed for the detection of eating disorders in the general population, and it is an instrument that seems valid to identify current or incipient cases of AN and BN, given its easy application, high reliability, sensitivity and transcultural validity.

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Spanish version of the EAT-40

In Spain, the adapted version of the EAT-40 that was validated in our setting was carried out by Castro J, *et al*. 1991 in an AN group and healthy control group[166] (See Annex 2.2). Using the 30-point cut-off point recommended by the original authors, sensitivity was 68% and specificity was 86%. Among the possible causes that may explain differences with the original version, the fact that Spanish anorexic patients were younger must be highlighted. When it was validated in a sample of 18-year old women using the questionnaire by Castro J, *et al*. and the 30-point cut-off point, sensitivity was 75%, specificity was 97.1% and PPV was 36%[167].

In Navarra, another study to validate the adaptation by Castro J, *et al*. after an 18-month follow-up was performed on a representative sample of adolescent students who did not attend the doctor's office but came from the general population instead, once prevalent cases of eating disorders were excluded[168]. With a cut-off point of 20 points, better diagnostic prediction was obtained (73% sensitivity and 85% specificity). PPV was 20% and the negative predictive value (NPV) was 98%.

From these results we can conclude that the EAT-40 is an adequate questionnaire for early detection of eating disorders in the general population, even if its PPV is low. However, despite using screening instruments it is always necessary to conduct individual interviews to confirm diagnoses of eating disorders. The fact that eating disorders are presented in a grading and severity continuum makes it essential to have a procedure that enables the detection of early signs to ensure a prompt intervention.

EAT-26 (Abbreviated version of the EAT-40)

Garner DM, *et al*., 1982 created the 26-item version of the EAT-40 by performing a factor analysis of the latter. The EAT-26 is highly predictive of the complete version (r=0.89). It consists of the first 26 items of the EAT-40, configuring three subscales: diet, bulimia and concern over eating and oral control. It is assessed using the same 6-poing Likert scale as the EAT-40. Answer scores range from 0 to 78. It is a self-report questionnaire[169]. Using a cut-off point of 22 points or more, the range of sensitivities, specificities and PPVs was 65.1% to 88.9%, 96.1% to 97.7% and 44.4% to 46.2%, respectively, the false-positive rates ranging from 2.7% to 3.8%[97].

Spanish version of the EAT-26

The Spanish validation of the EAT-26 in our setting was carried out by Gandarillas A, *et al.,* 2003 in a community setting on a female student population (15-18 years) (See Annex 2.3.). The psychometric characteristics of this questionnaire are similar to those described by their authors. For a 20-point or more cut-off point, sensitivity is 59%, specificity is 93%, PPV is 23%, NPV is 99% and the percentage of correctly classified subjected is 92%[170, 171]. As a screening questionnaire it is useful to differentiate eating disorder cases from the normal population, although it is important to mention the scarce PPV, given the low prevalence of the problem[170, 171].

When the cut-off point is lower (10 points or more), the EAT-26 presents 90% sensitivity,

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75% specificity, 11% PPV, 99.5% NPV and a percentage of correctly classified subjects of 76%[170, 171].

The MSC already recommended its use as a screening tool back in 1995[20] because the EAT-26 was able to distinguish between AN patients and normal population and between BN patients and normal population, but not between restrictive AN and BN, establishing a cut-off point of <20 points in women, >30 in high risk population and >50 in clinical population[20].

ChEAT (Child version of the EAT-26; Children Eating Attitudes Test)

Maloney MJ, et al., developed the ChEAT 1988, with the aim of detecting comprehension problems in children. When these problems were resolved by substituting certain words with simpler synonyms, validity and reliability results in a sample of children between the ages of 8 and 13 years were comparable to those published for adults (EAT-26), making the ChEAT a self-report questionnaire that can be administered starting at 8 years of age (it does require a 5th grade reading level in order to answer), which can aid in the assessment of concern over food, eating models and attitudes regarding food at these ages. A total score of 20 points in the upper scale would indicate the possible presence of an eating disorder[172].

Spanish version of the ChEAT (Children's Eating Attitudes Test)

The Spanish adaptation and preliminary validation of the ChEAT has been carried out by de Gracia M, *et al*., 2008 on a sample of girls and boys between the ages of 8 and 12 years. Reliability and validity results of the Spanish adaptation are analogous to the original study[173] (See Annex 2.4.).

The Catalan version of the ChEAT has also been adapted and validated in our setting in a sample of students (5th and 6th grades). Results indicate that the Catalan version is reliable. However, it is recommended that the cut-off point be lower than that established by the original authors, as was already suggested by the authors of the Spanish adaptation, given that it increases the number of subjects at risk of developing an eating disorder that may be detected at school[174].

BULIT Bulimia Test
Bulimia Test. MC Smith and MH Thelen, 1984.

The BULIT was designed with the objective of addressing certain needs detected in the assessment of BN, such as distinguishing BN patients from people without eating disorder problems; BN patients from patients with other eating disorders, and BN sub-groups based on specific criteria. It consists of 32 items (plus four informative type items relating to laxative abuse, use of diuretics, as well as amenorrhoea) that are distributed in five dimensions (binge-eating episodes or lack of control over meals, discomfort, vomiting, type of food and weight fluctuation). Each item is scored using a 5-point Likert scale (ranging from 1 to 5). In several items the most symptomatic answer is presented at the end rather than at the beginning to prevent a bias in the response, due to presentation order. The sum of all items (except the purely informative ones) leads to an overall score ranging from 32 to 160 (a higher score indicates greater intensity of bulimic symptomatology). Likewise, the sum of items corresponding to each

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of the five dimension results in the overall scores for each one of these dimensions. An overall score and five scores corresponding to each dimension are therefore obtained. Data derived from the original version indicates that it is a reliable, valid and objective instrument to identify individuals with bulimic symptoms, confirming its use to detect those individuals who present or who are at risk of developing BN in the general population[175]. With a cut-off point of 102 points or more, sensitivity, specificity, PPV and NPV were 95%, 98%, 91% and 99%, respectively.

Spanish version of the BULIT

In Spain, the adapted version of the BULIT validated in our setting was developed by AJ Vázquez, *et al*., 2007 in a group of people, mainly females between the ages of 13 and 54 years, who attended mental health centres[176] (See Annex 2.5.). Results solidly support the reliability and validity of the Spanish version of the BULIT, highlighting its use to identify BN cases, as well as to quantify the severity of bulimic symptoms. With a cut-off point of 88, it leads to a 90% correct classification of individuals with BN, and a 100% correct classification of individuals without eating disorders, data that firmly endorse its use as a screening instrument.

BULIT-R (Revised version of the BULIT)

Later, the revised version of the BULIT (BULIT-R), developed by Thelen, *et al*., 1991 was obtained, its most important contribution being the adaptation to DSM-III-R and later DSM-IV criteria[177]. It is comprised of 36 items, even though only 28 are used to determine the final score that ranges between 28 and 140 points. The estimated administration time of the instrument is 10 minutes. It is highly correlated with the original version (r=0.99). In a sample of nursing students and with a cut-off point of 104 points or more, sensitivity, specificity, PPV and NPV of the BULIT-R was high: 80%, 99.5%, 80% and 99.5%, respectively[178].

Spanish version of the BULIT-R

In Spain, the adapted version of BULIT-R validated in our setting was carried out by MN Berrios-Hernández, *et al*., 2007[179].

BITE Bulimia Investigatory Test Edinburgh. M Henderson and CPL Freeman, 1987.

The BITE is a self-report questionnaire completed in 10 minutes or less that is designed to identify subjects with bulimic symptoms (BN or BED). It consists of 36 items that configure two subscales: the symptoms scale (assesses the number and degree of existing symptoms; 30 items; highest score: 30; cut-off point: 20 points or more) and the severity scale (provides a disorder severity index based on the frequency with which pathological behaviours take place; 6 items; highest score: 39; cut-off point: 5 points or more). A total score of 25 or more points indicates the presence of a serious eating disorder. In the sample of bulimic women and control group the cut-off points used were as follows: >25 for the complete questionnaire, >20 for the symptoms subscale and/or >5 points for the severity scale. With these cut-off points, the BITE demonstrated perfect sensitivity, specificity and PPV (100%, 100% and 100%, respectively), even though data on its use in the population are not known[180].

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Spanish version of the BITE

The adapted version validated in our setting was developed by T Rivas, *et al*., 2004[181] (See Annex 2.6.).

The Spanish version of BITE was administered to a sample of adolescents aged between 12 and 21 years from different schools. Using cut-off points based on DSM-IV criteria for BN, high specificity and a much lower sensitivity than that found in clinical samples were obtained. Furthermore, the scores in the BN group were higher than in other eating disorders and in the group without eating disorders. Therefore, this instrument can be used for early detection of individuals who may present an eating disorder in the general population. It is also used to assess disease intensity and response to treatment.

Recommendations

D	6.1.	Target groups for screening should include young people with low body mass index (BMI) compared to age-based reference values, patients consulting with weight concerns without being overweight or people who are overweight, women with menstrual disorders or amenorrhoea, patients with gastrointestinal symptoms, patients with signs of starvation or repeated vomiting, and children with delayed or stunted growth, children, adolescents and young adults who perform sports that entail a risk of developing an eating disorder (athletics, dance, synchronised swimming, etc.). (Adapted from recommendation 5.2.5.3 of the NICE CPG).
D	6.2.	In AN, weight and BMI are not considered the only indicators of physical risk. (Adapted from recommendation 5.2.5.6 of the NICE CPG).
D	6.3.	Early identification and intervention of individuals presenting weight loss are important to prevent the development of severe emaciation. (Adapted from recommendation 6.6.1.2 of the NICE CPG).
D	6.4.	In the case of suspected AN, attention should be paid to overall clinical assessment (repeated over time), including rate of weight loss, growth curve in children, objective physical signs and appropriate laboratory tests. (Adopted from recommendation 5.2.5.7 of the NICE CPG).

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✓	6.5.	It is recommended to use questionnaires adapted and validated in the Spanish population for the detection of eating disorder cases (screening). The use of the following tools is recommended: Eating disorders in general: SCOFF (for individuals aged 11 years and over) AN: EAT-40, EAT-26 and ChEAT (the latter for individuals aged between 8 and 12 years) Bulimia nervosa (BN): BULIT, BULIT-R and BITE (all for individuals aged 12-13 years and over)
✓	6.6.	Adequate training of PC physicians is considered essential for early detection and diagnosis of eating disorders to ensure prompt treatment, or referral, when deemed necessary.
✓	6.7.	Due to the low frequency of consultations during childhood and adolescence, it is recommended to take advantage of any opportunity to provide comprehensive care and to detect eating disorder risk habits and cases. Eating disorder risk behaviour, such as repeated vomiting, can be detected at dental check-ups.
✓	6.8.	When interviewing a patient with a suspected eating disorder, especially if the suspected disorder is AN, it is important to take into account the patient's lack of awareness of the disease, the tendency to deny the disorder and the scarce motivation to change, this being more pronounced in earlier stages of the disease.
✓	6.9.	It is recommended that different groups of professionals (teachers, school psychologists, chemists, nutritionists and dieticians, social workers, etc.) who may be in contact with at-risk population have adequate training and be able to act as eating disorder detection agents.

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7. Diagnosis of Eating Disorders

Key Questions:

7.1. What clinical criteria are useful to diagnose eating disorders?

7.2. How are eating disorders diagnosed?

7.3. What is the differential diagnosis of eating disorders?

7.1. What clinical criteria are useful to diagnose eating disorders?

Clinical criteria for the diagnosis of AN and BN are well defined in the International Statistical Classification of Diseases and related health problems, tenth edition (ICD-10) of the World Health Organisation (WHO, 1992) [182] and in the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV) and text revision (DSM-IV-TR) of the American Psychiatric Association (APA, 1994[183] and APA, 2000[184], respectively).

The DSM-IV/DSM-IV-TR classifies mixed and partial forms of eating disorders, such as EDNOS, where BED is included, while the ICD-10 refers to them as atypical AN, atypical BN or unspecified eating disorders.

The DSM-IV/DSM-IV/TR, in contrast to the ICD-10, differentiates between two types of AN (restricting and compulsive/purging) and two types of BN (purging and non-purging) depending on the predominant behaviour.

The Spanish versions of these classification systems have been subsequently edited: ICD-10 (2000) [185] and DSM-IV-TR (2008) [186]. In the latter, the equivalent between classifications is presented (DSM-IV-TR, ICD-9 and ICD-10). In Annex 2.7. diagnostic criteria according to both classifications are described.

Recommendation

✓	7.1.	It is recommended to follow the diagnostic criteria of the WHO (ICD-10) and the APA (DSM-IV or DSM-IV-TR).

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7.2. How are eating disorders classified?

The diagnosis of eating disorders is based on a clinical interview complemented by complementary physical, psychopathological and behavioural examinations aimed at assessing the existence of physical, emotional, behavioural and cognitive disturbances. Diagnosis has good validity and reliability.

Anamnesis

When an eating disorder is suspected, both a personal and family complete clinical history must be performed. Given that patients are usually young adults and adolescents, these interviews must have certain specific characteristics to overcome the difficulties derived from a person's willingness to reveal his/her motives, symptoms and behaviour, sometimes an obstacle to making a diagnosis. Thus, the empathy, support and compromise that is perceived during the clinical interview will be essential in enabling the patient to reveal fears regarding weight gain, eating behaviour, purging behaviour and other disordered behaviour such as excessive exercise. When deemed appropriate, this clinical history must be accompanied by a corroborative account from parents or other relative.

After explaining the reason for the consultation, if patients are accompanied by their family, the latter must be asked to leave the room to generate a climate of privacy where the patient can freely respond to the questions asked (See chapter 12, "Legal Aspects").

The confidentiality of the medical act must always be emphasised, making it clear to the patient that none of what is discussed between him/her and the health care professional will be disclosed to parents or family members unless otherwise specified by the patient, except when there is risk to the patient's physical or psychological integrity (See chapter 12, "Legal Aspects"). The performance of blood work and other tests must be agreed upon with the patient. The health care professional will try to be perceived as somebody who looks after the patient's health, and not as an ally to the family. The patient's genogram and whom he/she lives with must be known. To this end, family and personal pathological history must be requested (especially if it relates to mental health: depressions, phobias, addictions, eating disorders, physical and psychological abuse).

During the anamnesis, questions will be asked regarding physical disorders (weight, skin and mucosa, menstruation), emotional disorders (anxiety, depression, social alienation, eating disorder triggers [see chapter 4, "Definition and Classification of Eating Disorders], sleep disorders), behavioural disorders (diets, exercise, binge-eating, extravagance with food, personal and family eating habits, purging behaviour, physical hyperactivity) and cognitive disorders (image distortion, disease awareness).

The semi-structured interviews most frequently used to diagnose eating disorders are[187]: section H (for eating disorders) of the diagnostic interview *Composite International Diagnostic Interview* (CID-I) developed by the WHO, 1990[188] and *Eating Disorders Examinations* (EDE) developed by Cooper and Fairburn, 1987[189].

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Although these two interviews are adapted to diagnostic criteria defined by the DSM-IV-TR and the ICD-10, there is consensus that the most reliable and best validated semistructured interview for the diagnosis of eating disorders is the EDE. The 12th edition of the EDE (EDE-12) developed by Fairburn and Cooper, 1993[190] has a Spanish version, adapted by MR Raich, 1994[191] and validated in our setting[192, 193] on a sample of 99 female university students, that has proven to be a reliable and valid tool. Another adaptation and validation of the EDE on a sample in our setting, performed by Robles, *et al*., 2006[194], has been identified, presenting appropriate psychometric qualities that are similar to those of the original version (See Annex 2.8., EDE-12).

There are preliminary results of the child version of the EDE-12 (ChEDE-12) on a sample of 15 children with AN and 15 with other eating disorders and two age-adjusted groups of 15 control subjects. Preliminary results indicate that the ChEDE-12 differentiates children with AN from children with other eating disorders and from control subjects[195]. There is no information regarding the adapted version validated in our setting in these ages.

During the anamnesis it may also be helpful to ask only the items relating to the diagnostic criteria of eating disorders described in the ICD-10 and DSM-IV-TR. This measure, which has been employed frequently in the literature, facilitates the diagnostic process, as well as the patient's participation, without compromising diagnostic efficacy. With this restriction, the duration of interviews is reduced to ten or fifteen minutes per person.

Informing the patient with an eating disorder

As usual, when informing the patient on his/her disease, care must be taken in how the message is conveyed, adjusting it to the patient's age, educational level and beliefs, amongst other aspects. To make this task easier for the health care professional, this CPG provides material aimed at children, adolescents and adults with eating disorders or who are at risk of developing them. This material may also be useful to family members, friends or people who interact with patients, as well as for the general population (See Annex 3.1.).

The diagnosis of an eating disorder must be communicated clearly, describing the important characteristics of this mental disorder. It is also helpful to explain the physical dimensions and disturbances it causes, as well as its evolution and prognosis.

The patient must not be made to feel guilty. However, it must be clearly explained that he/she is suffering from a disease, that it is treatable and curable but that his/her involvement is crucial for recovery.

Anamnesis and family awareness

It is very important to study the family environment and detect possible disturbances in its correct functioning (disorganised families, strict rules, etc.). Other family-related risk factors must also be assessed (mothers who are very critical of physical appearance, family conflicts, a history of weight loss diets in other family members, especially the mother, etc.) (See chapter 4, "Definition and Classification of Eating Disorders" and chapter 11, "Prognosis").

Communication with the family must be established to explain eating disorders, to emphasise that their active involvement in the patient's treatment and recovery process is crucial,

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to help them cope with the situation and to make them aware that positive changes in the family routine must be made (for example, strengthen the patient's maturation, autonomy and responsibility processes, by establishing rules and guidelines adjusted to the child's age, stimulate the expression of both positive and negative feelings). The patient information elaborated for this CPG can be useful (See Annex 3.1.).

Eating Disorders in Adults

When faced with potential cases of eating disorders in adults, clinical history questions should be adapted to a conventional interview with an adult, who is responsible for his/her acts and decisions, who meets with the health care professional alone most of the time, and where he/she usually prefers the family not to be involved.

The adult patient's economic independence enables easier access to harmful or toxic drugs, which is why the patient's personal toxicopharmacological background must be emphasised in order to detect abuse or misuse of substances.

In these cases, it is also more likely to find work-related, economic or social problems as maintaining factors or, sometimes, as eating disorder triggers.

It is important to highlight that there are also thin, non-pathological constitutions that are well-adjusted to development.

Physical exploration

Physical exploration is less useful than anamnesis in establishing diagnosis. However, complete physical exploration is crucial and must be aimed at assessing the patient's nutritional state and detecting possible secondary physical complications resulting from dietary restraints and/or purging behaviour that would determine the intervention of other specialists or not.

Data corresponding to vital signs (heart rate, blood pressure, axillary temperature and respiratory rhythm) will be collected; in patients with a certain degree of malnutrition these signs will be in the lower limits. For weight and size, the BMI (BMI=weight kg/size m^2) will be determined. If the BMI is >25 it is considered overweight; if it is between 25-18.5 is considered normal, and if it is <18.5 it is considered malnutrition (low weight). ICD-10 establishes a BMI<17.5 as a diagnostic criteria for AN. Patients with BN can present normal weight and even slight excess weight. In patients under the age of 18 years, the BMI must be calculated and compared to the percentiles pertaining to these ages[196].

A general examination of the different body systems will be performed with special attention paid to signs of malnutrition and the detection of growth or sexual development:

- Exploration of skin and mucosa and dental exploration. Hydration state of the skin and mucosa (skin fold), presence of carotenic pigmentation, presence of hypertrichosis, alopecia, brittle nails, skin injuries that indicate self-aggression (cuts, scratches, burns), presence of petechias, purpura, parotid hypertrophy and/or sub-maxillary glands, calluses or wounds on the back of the hand or on the fingers from induced vomiting (Russell's sign), chronic orodental or pharyngeal lesions,

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dental erosion, enamel demineralisation and cavities.

- Cardiocirculatory examination. An electrocardiogram must be performed on patients with AN and signs of malnutrition and also on patients with BN at risk of dyselectrolytemia. Bradycardia may be found. Hypopotasemia may cause a U-wave, T-wave flattening/inversion, premature ventricular contractions and ventricular arrhythmias, and hypomagnesemia can also produce arrhythmias.

- Respiratory examination. Possible opportunist infections. In patients who vomit, possible pneumothorax or even aspiration pneumonia.

- Neurological exploration. Detection of possible polyneuropathies secondary to vitamin deficiency, detection of neurological symptoms secondary to hypopotasemia and detection of aqueous intoxication

Psychopathological and behavioural examination

In the psychopathological and behavioural examination, different instrument can be administered with the objective of assessing eating behaviour and the psychopathological situation (impulsivity, anxiety, depression, personality and obsessiveness). (See chapter 10, "Assessment").

Complementary examinations

Based on the results of the physical exploration, further examinations that include laboratory tests and other explorations can be performed. However, an excess of examinations to confirm treatment may be counterproductive.

Laboratory tests

Blood work should include the following parameters: hemogram, glycaemia, total cholesterol, triglycerides, liver enzymes (AST, ALT and GGT), ions (K, Na, Cl, Ca, P and Mg), total proteins and albumin, creatinine and urea, TSH, free T3 and T4, coagulation (TP, TTPa), urine (sediment and osmolarity) and female hormone profile.

The analytical examination of eating disorders enables the ruling out of possible organic causes of weight loss and for the patient to pick up his/her results, making a new kind of weight management and more time for a detailed interview on losing weight possible.

A normal analysis can never rule out an eating disorder. In these cases, it can be counterproductive given that it can favour "false tranquillity" in the patient, family and non-specialised expert; it can become a positive reinforcement for the patient's attitude; or, the patient may even view the professional as an ally of the family.

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Other examinations

To ascertain whether there are bronchoaspirative lesions in patients who vomit a chest x-ray will be requested, as well as when other pathologies such as tuberculosis are suspected. Bone x-ray and densitometry enable the assessment of bone age in people in development and the presence of osteoporosis or osteopenia. Electrocardiography, amongst other tests, is indicated when there is suspicion of laxative abuse. The decision to perform more sophisticated examinations is left to consulting specialists.

Diagnostic difficulty is greater in cases where there are comorbidities. The most frequent comorbidities are: diabetes mellitus, obesity, malabsorbtion syndrome and thyroid diseases of organic origin and substance abuse and dependence, mood disorders, obsessive-compulsive disorders, personality disorders, as well as impulse control disorders.

Recommendations

D	7.2.1.	Health care professionals should acknowledge that may eating disorder patients are ambivalent regarding treatment due to the demands and challenges that it entails. (Adapted from recommendation 1.10.1.1 of the NICE CPG).
D	7.2.2.	Patients and, when deemed necessary, carers should be provided with information and education regarding the nature, course and treatment of eating disorders. (Adapted from recommendation 2.10.1.2 of the NICE CPG).
D	7.2.3.	Families and carers may be informed of existing eating disorder associations and support groups. (Adapted from recommendation 2.11.5.5 of the NICE CPG).
✓	7.2.4.	It is recommended that the diagnosis of eating disorders include anamnesis, physical and psychopathological examinations and complementary
✓	7.2.5.	Diagnostic confirmation and therapeutic implications should be in the hands of psychiatrists and clinical psychologists.

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7.3. Differential Diagnosis of Eating disorders

Despite the existence of certain well-defined diagnostic criteria for eating disorders, in clinical practice there are often difficulties relating to the differential diagnosis with other conditions that require rigorous clinical assessment.

In regards with AN, the differential diagnosis must be made with those pathologies that can present significant appetite loss and weight loss, even though the main features of AN, such as body image distortion, the desire to perpetuate weight loss and the fear of becoming fat, are not present in all cases:

- Mental disorders: depression, anxiety, psychotic disorders and substance abuse
- Diabetes mellitus
- Tuberculosis
- Hyperthyroidism
- Neoplasias of the central nervous system
- Less frequent: lymphomas, sarcoidosis, Addison's disease, celiac disease, superior mesenteric artery syndrome (it may sometimes be a complication of AN), AIDS, lactose intolerance, panhypopituitarism, etc.

The differential diagnosis of AN is especially indicated in adult patients, given the atypical age of onset of the disorder.

The differential diagnosis of BN is more limited than AN and includes organic conditions that present hyperphagia and weight gain:

- Diabetes mellitus
- Hypothyroidism
- Kleine-Levin Syndrome (idiopathic disorder that especially affects men aged 20-30 years and that presents with hypersomnia and hyperphagia).
- Hypothalamic lesions
- Tumours causing hyperphagia
- Major depression, atypical depression, borderline personality disorder (BPD).

The differential diagnosis of EDNOS must be performed with pathologies that may present with weight loss or gain and/or decreased or increased appetite. The differential trait of EDNOS is body-scheme distortion and excessive and irrational focus on weight and diet.

In BED, the differential diagnosis must be performed with the same pathologies described for BN and with clinical pictures of impulse control disturbance that may present with binge-eating episodes, such as those that occur in BPD.

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8. Interventions at the Different Levels of Care in the Management of Eating Disorders

Key Questions:

8.1. What are the primary care (PC) and specialised care interventions for eating disorders? Other resources?

8.2. In eating disorders, what clinical criteria may be useful to assess referral amongst the health care resources available in the NHS?

8.3. In eating disorders, what clinical criteria may be useful to assess complete hospitalisation (inpatient care) in healthcare resources available in the NHS?

8.4. In eating disorders, what clinical criteria may be useful to assess discharge in health care resources available in the NHS?

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

8.1. What are the primary care (PC) and specialised care interventions for eating disorders? Other resources?

The management model proposed, in which patients are referred from one management resource to another, is bound by protocols, recommendations and guidelines elaborated with clinical criteria for which there is little evidence that guides decisions regarding where to perform the intervention (See question 8.2. in this chapter).

8.1.1. Primary Care (PC) interventions

Primary care is carried out in primary care centres, the first level of access to health care. At this level interventions are focused on:

– Identifying individuals at risk of developing an eating disorder (See chapter 6, "Detection") and establishing an early diagnosis (See chapter 7, "Diagnosis").

– Deciding whether the disorder can be treated in the primary care centre or whether it must be referred to specialised care. In order to make this decision, the patient's type of eating disorder, age, level of risk, physical and psychological complications and preferences must be taken into account.

– Initiating nutritional treatment that includes the following objectives: restoring normal weight in the patient, correcting malnutrition, avoiding the refeeding syndrome, managing or curing medical complications, carrying out nutritional education with the objective of normalising altered dietary behaviour, both in the patient and in his/her family, and preventing and managing recurrences.

Initiating nutritional restoration by means of adequate renutrition or refeeding (See question 9.1. chapter 9, "Treatment") and performing nutritional education (nutritional counselling) (See question 9.2. chapter 9, "Treatment").

– Monitoring cases that are managed in primary care centres (in AN: the patient will be requested to record daily consumption, hyperactivity, laxative abuse and use of diuretics; he/she will be not be allowed to weigh him or herself and weight will be recorded weekly at the practice, results to which the patient will not have access; in BN: binge-eating episodes and self-induced vomiting, substance abuse and other behaviour disorders (impulse, etc.) will be recorded, as well as recurrence prevention.

– Conveying clear and true information regarding eating disorders to patients and relatives (See Annex 3.1.). Also detecting and correcting maladaptive ideas about weight and health (See Annex 2.9.).

– Carrying out interventions with affected families (See chapter 7, "Diagnosis").

– Managing physical complications (See chapter 9, "Treatment").

8.1.2. Specialised care interventions

Eating disorder patients are provided with specialised care, the second and third levels of access to health care, in the form of inpatient care resources (psychiatric and general hospital), specialised outpatient practices (mental health centres for adults and children), day hospitals for day care (centres specialised in eating disorders and general mental health centres), emergency services, medical services pertaining to general hospitals and specific units (for eating disorders, borderline personality disorder and toxicology).

– Assessment and diagnosis visits are performed at an outpatient level (CSMA and CSMIJ) after referral from PC. In the case of vital emergencies or autolytic risk that stand in the way of this assessment, the patient must be referred to internal medicine and psychiatry services and will be admitted if indicated by the physicians on-duty.

– The clinical history must update and complete what has been established in PC.

– After establishing a diagnosis, the mental health team will design an ITP which includes: a) definition of the problem based on the diagnosis and altered areas, b) formulation of psychotherapeutic objectives, c) election of treatment (psychological therapies, pharmacological treatments, medical measures and social interventions) (See the "treatment"

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section), and d) time-frame for therapeutic assessment. Following the therapeutic decision, the ITP will be presented to the patient and family, informing them on: a) health care professionals who will be involved in treatment, b) techniques that will be employed, c) duration, and d) time-frame for assessment.

The degree of nutritional deterioration, along with the presence or absence of complications, determine the selection of access and feeding route, as well as the location where nutritional follow-up must be performed.

The day care treatment programme includes meal monitoring, and patients must complete a survey on the foods they ingest in 24 hours, including on weekends when they are away from the centre. Each patient must be medically and nutritionally assessed at least once a week, weight and related medical symptoms must be monitored, requested blood work must be assessed if necessary, and the dietary survey and objectives must be reviewed. In inpatient care, nutritional support with artificial nutrition must be strictly monitored to avoid or manage the onset of the refeeding syndrome.

8.1.3. Other resources

Mutual help groups (MHG) are groups of people who meet voluntarily with the aim of helping each other. They are generally comprised of individuals who share the same problem or who find themselves in a similar difficult situation. The MHG emphasises personal interaction and each member's capacity to assume responsibilities. It tends to provide emotional help and promote values that help members strengthen their own sense of self. These groups provide assistance and emotional support to families and patients, facilitating the success of the corresponding therapy. Groups are guided by facilitators (people who have experienced the same problem or situation as the participants) and are periodically aided by a professional who supervises the intervention and provides instruments to improve group dynamics (See Annex 3.2. Support associations for patients with eating disorders and their families).

Counselling consists of performing a series of personal interviews with patients and relatives to inform and educate on the disease and its main health, family and social consequences, as well as to provide guidance on the current situation of health care, legal, economic and social resources, with the objective of reassuring and assisting the patient and/or family.

Day centres are public sociosanitary resources that accommodate patients with different long-term disorders, including cases of chronic eating disorders and cases with psychiatric comorbidity. These centres provide, amongst other activities and interventions, rehabilitation (tertiary prevention). In our setting there are no public day centres aimed exclusively at patients with eating disorders.

Therapeutic apartments (assisted or not) constitute another public network resource that enables social reinsertion of patients suffering from different disorders. In our public network there are no apartments specifically for eating disorder patients.

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8.2. In eating disorders, what clinical criteria may be useful to assess referral amongst the health care resources available in the NHS?

8.2.1. Referral to mental health services

Criteria for referral from primary care to mental health services (CSMA and CSMIJ) are as follows:

- When there is an established diagnosis of eating disorder.
- Weight loss equal to or higher than 10%-25% of weight, without a cause to account for it.
- Presence of regular bulimic episodes, meaning over-eating and/or persistent purging behaviour (self-induced vomiting, laxative abuse and use of diuretics).
- Presence of associated psychopathological disturbances.
- Lack of disease awareness.
- If, despite following PC indications, weight and bulimic behaviour do not improve.

8.2.2. Referral to emergency hospitalisation

Criteria for referral from primary care to emergency hospitalisation (Emergency service of a general hospital) to receive emergency medical treatment are as follows:

- Weight loss >50% in the last 6 months (30% in the last 3 months).
- Consciousness disturbances.
- Convulsions.
- Dehydration.
- Severe liver or kidney disturbances.
- Pancreatitis.
- Decreased potassium <3 mEq/l or sodium (<130 o >145).
- Serious arrhythmia or conduction disorder.
- Bradycardia of <40 bpm.
- Other ECG disorders.
- Syncopes or hypotension with SBP <70 mm Hg.
- HDH: hematemesis, rectal bleeding.
- Acute gastric dilation.

8.2.3. Referral to emergency psychiatric assessment

Criteria for referral from primary care to emergency psychiatric assessment (at a hospital's Psychiatry Service) are as follows:

- Absolute refusal to eat or drink.
- Depressive symptomatology, with autolytic risk.
- Significant self-injurious behaviour.

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8.3. In eating disorders, what clinical criteria may be useful to assess inpatient care (complete hospitalisation) in one of the health care resources available in the NHS?

8.3.1. Inpatient care (complete hospitalisation) criteria

- Biological state that entails a risk of serious complications (no food intake, especially liquids, BMI<16, ionic disturbances, repeated self-induced vomiting, laxative abuse and use of diuretics, hemametesis and rectal bleeding).
- There are significant depressive symptoms with autolytic risk.
- Serious self-injurious behaviour.

These are not absolute criteria and, depending on their intensity, day care may be indicated (partial hospitalisation).

There are other psychopathological disorders that prevent outpatient treatment:

- Due to the psychopathology presented, the patient is unable to follow guidelines in the outpatient programme: frequency of visits, limitation of physical activity, recommended diet, etc.
- The patient's difficult behaviour at home, the existence of conflict in family relationships and/or family psychopathology are excessive and impossible to manage at an outpatient level.
- Weight evolution does not follow the rate indicated in the weight restoration programme.

In order to pursue complete hospitalisation, the judge's authorisation is required. In the case of minors, it is advisable though not indispensable to have the parents' authorisation as well as the judge's, and in the case of over-age patients, to have judicial authorisation (see chapter 12, "Legal Aspects").

Inpatient management (complete hospitalisation) can be carried out in a general hospital (or one specialised in eating disorders), the psychiatric hospital being the most recommended resource in special cases such as chronicity and severe mental disorders (delirium, repeated self-aggression, cognitive deterioration, etc.).

Specific eating disorder units are found in general hospitals and depend on the psychiatry service (although in some alienated cases they depend on the endocrinology service). Amongst other functions, they tackle especially resistant cases. These specific units are in contact with other hospital services such as Internal Medicine, Gynaecology, etc. and perform interventions on complications derived from eating disorders.

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8.3.2. Criteria for admission to day patient care (day hospital)

- From the adult or children MHC. If the patient does not meet emergency medical care or emergency psychiatric admission criteria and does meet any of the following criteria:
  - The patient is unable due to the psychopathology presented to follow the guidelines in the outpatient programme: frequency of visits, limitation of physical activity, recommended diet, etc.
  - There are serious behavioural problems at home, conflicts in family relationships and/or family psychopathology that cannot be modified at an outpatient level.
  - Weight evolution does not follow the rate indicated in the outpatient programme for weight restoration.
- From complete hospitalisation (once discharge criteria have been fulfilled). If additional eating and behaviour management is necessary, but can be carried out on an outpatient basis. Also in those cases in which psychopathological intensity requires psychological treatment to modify the patient's image, beliefs, assertiveness and other aspects related with eating disorders.

8.4. In eating disorders, what clinical criteria may be useful to assess discharge from health care resources available in the NHS?

8.4.1. Criteria for discharge from inpatient care (complete hospitalisation)

- Normalisation of biological disturbances that have led to complete hospitalisation.
- Weight restoration as is established in an individualised programme.
- Improved psychopathological state.
- Elimination of self-injurious behaviour.
- Remission of abnormal eating behaviour and compensatory behaviour.
- Improved family conflicts.
- Improved general functioning.

8.4.2. Criteria for discharge from day patient care (day hospital)

In order to refer to outpatient care (adult or children MHCs):

- Weight restoration and/or maintenance as established in the ITP.
- Completion of group programmes that had been initiated.
- Improved eating pattern and compensatory behaviour (if any).
- Improved family conflicts and general functioning that enable outpatient treatment.

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For hospital admission:

- Weight does not increase as is established in the weight restoration programme.
- Does not comply with rules established in the day hospital in terms of meals, physical activity restriction and general functioning.
- Presence of significant psychopathology.
- The patient's biological state entails a risk of serious complications (heart rate less than 45, potassium less than 3.5 mEq/l, hemametesis and rectal bleeding).
- At any moment during the patient's stay at the day hospital, referral to the Internal Medicine Service may be indicated.

8.4.3. Criteria for discharge from the outpatient treatment programme

Discharge criteria will depend on the ITP.

- Maintenance of non-altered eating behaviour (diets, over-eating and purging) throughout a period of one year.
- Maintenance of stable weight that is within normal values throughout a period of one year.
- Absence of menstrual irregularities secondary to eating disorders.
- Decreased recurrence risk.
- Absence of other psychopathological disturbances that require treatment.

8.4.4. Discharge criteria for eating disorders

The process will end when clinical improvement is evident and enables the patient to resume normal daily life, verifying that during a period of time greater than two years the following criteria are being fulfilled:

- Maintenance of weight and absence of nutritional imbalances.
- Absence of eating peculiarities that are harmful to health.
- Adequate relational life.

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Recommendations

D	8.1.	Individuals with eating disorders should be treated in the appropriate care level based on clinical criteria: outpatient, day hospital (day care) and general or psychiatric hospital (inpatient care). (Adapted from recommendation 6.5.8.1 of the NICE CPG).
D	8.2.	Health care professionals without specialist experience in eating disorders or who are faced with uncertain situations should seek the advice of a trained specialist when emergency inpatient care is deemed the most appropriate option for a patient with an eating disorder. (Adapted from recommendation 6.5.8.6 of the NICE CPG).
D	8.3.	The majority of patients with BN can be treated on an outpatient basis. Inpatient care is indicated when there is risk of suicide, self-injuries and serious physical complications. (Adapted from recommendations 6.5.8.1 and 6.5.8.4 of the NICE CPG).
D	8.4.	Health care professionals should assess patients with eating disorders and osteoporosis and advise them to refrain from performing physical activities that may significantly increase the risk of fracture. (Adopted from recommendation 6.4.5.3 of the NICE CPG).
D	8.5.	The paediatrician and the family physician must be in charge of the management of eating disorders in children and adolescents. Growth and development must be closely monitored. (Adapted from recommendation 6.4.5.4 of the NICE CPG).
D	8.6.	Primary care centres should offer monitoring and management of physical complications to patients with chronic AN and repeated therapeutic failures who do not wish to be treated by mental health services. (Adapted from recommendation 5.2.5.8 of the NICE CPG).
D	8.7.	Family members, especially siblings, should be included in the individualized treatment plan (ITP) of children and adolescents with eating disorders. The most common interventions involve sharing of information, advice on behavioural management of eating disorders and improving communication skills. The patient's motivation to change should be promoted by means of family intervention. (Adapted from recommendation 6.2.9.13 of the NICE CPG).
D	8.8.	Where inpatient care is required, it should be carried out within a reasonable distance to the patient's home to enable the involvement of relatives and carers in treatment, to enable the patient to maintain social and occupational links and to prevent difficulties between care levels. This is particularly important in the treatment of children and adolescents (Adopted from recommendation 6.5.8.4 of the NICE CPG).

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D	8.9.	Patients with AN whose disorder has not improved with outpatient treatment must be referred to day patient treatment or inpatient treatment. For those who present a high risk of suicide or serious self-injuries, inpatient management is indicated. (Adapted from recommendation 6.5.8.2 of the NICE CPG).
D	8.10.	Inpatient management should be considered for patients with AN whose disorder is associated with high or moderate risk due to common disease or physical complications of AN. (Adapted from recommendation 6.5.8.3 of the NICE CPG).
D	8.11.	Patients with AN who require inpatient treatment should be admitted to a centre that ensures adequate renutrition, avoiding the re-feeding syndrome, with close physical monitoring (especially in the first few days), along with the appropriate psychological intervention. (Adapted from recommendation 6.5.8.5 of the NICE CPG).
D	8.12.	The family physician and paediatrician should take charge of the assessment and initial intervention of patients with eating disorders who attend primary care. (Adapted from recommendation 5.2.5.1 of the NICE CPG).
D	8.13.	When management is shared between primary and specialized care, there should be close collaboration between health care professionals, patients and relatives and carers. (Adapted from recommendation 5.2.5.2 of the NICE CPG).
✓	8.14.	Patients with confirmed diagnosis or clear suspicion of an eating disorder will be referred to different health care resources based on clinical and age criteria.
✓	8.15.	Referral to adult and children MHCs by the family physician or paediatrician should consist of integrated management with shared responsibilities.
✓	8.16.	Cases referred to adult of children MHCs still require different levels to work together and short- and mid-term monitoring of patients, to avoid complications, recurrences and the onset of emotional disorders, and to detect changes in the patient's environment that could influence the disease.
✓	8.17.	The need to prescribe oestrogen treatment to prevent osteoporosis in girls and adolescents with AN should be carefully assessed, given that this medication can hide the presence of amenorrhoea.
✓	8.18.	In childhood, specific eating disorder treatment programmes designed for these ages will be required.

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9. Treatment of Eating Disorders

The treatment of eating disorders requires multidisciplinary collaboration and can be carried out at different levels of care and health care resources (See Chapter 8, "Interventions at the different levels of care").

The objectives of eating disorder treatments are:

1. To restore or normalise patient weight and nutritional state to healthy levels in the case of AN (See question 9.1. of this chapter).

Nutritional support can range from a base diet, if the patient presents normonutrition, or specific diet-therapy (oral diet) if there is an associated pathology, to the administration of artificial nutrition (enteral oral or parenteral intravenous) if there is severe energetic-protein malnutrition.

In the outpatient, standard refeeding includes following a normal diet. In day patient management, treatment is more intensive and prolonged than outpatient treatment. When outpatient treatment or day patient treatment is insufficient, or if the patient presents an acute problem, inpatient treatment is indicated.

2. To treat physical complications. This CPG does not include the specific treatment for all potential physical complications (See Chapter 4 "Definition and Classification of Eating Disorders").

3. To provide education on healthy, nutritious eating habits. Additionally, general recommendations on the principles of a balanced diet with a variety and frequency of foods, or more specific recommendations based on the type of eating disorder, can be delivered. The aim of nutritional education is to guide the patient and his/her family in terms of the behaviours that should be adopted and to increase the patient's motivation to cooperate and participate in treatment (See Question 9.2. of this chapter).

4. To modify/improve prior or acquired dysfunctions due to eating disorders (thoughts, attitudes, inadequate behaviour, etc. [197-199]), as well as to increase weight, reduce/eliminate binge-eating and purging, depression, amongst other relevant clinical variables. To this end there are several different treatments available:

– Psychological therapies (see questions 9.3.-9.8. of this chapter).

– Pharmacological treatments (see questions 9.9.-9.15. of this chapter).

– Combined interventions (see question 9.16. of this chapter).

The therapeutic contract includes ITP (see chapter 8, "Interventions at the different levels of care") and is signed by the patient and the multidisciplinary team of health care professionals involved in the care. This contract ensures that the patient has been informed and that he/she has accepted the ITP.

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5. To treat associated disorders (comorbidities), both psychiatric (including mood disturbances, low self-esteem, behaviour, etc.) and physical (diabetes mellitus, etc) (see questions 9.17 and 9.18 of this chapter).

6. To obtain family support for the patient and provide counselling and therapy when necessary (see chapter 7, "Diagnosis" and question 9.6. in the section "Family therapy").

7. To prevent relapse. This includes addressing and preventing situations that may favour recurrences and planning strategies to tackle them. Amongst the aspects that should be managed, body weight variations, patient requests for diets and drugs, management of proper eating habits, engagement in purging behaviour, performance of excessive exercise, management of appropriate ponderal-statural and psychomotor development, detection of any emotional imbalance or environmental pressure and family management[200, 201].

8. To treat chronic eating disorder cases, as well as the management of eating disorders in special situations such as pregnancy and delivery, which are described at the end of this chapter (see questions 9.19 and 9.20 of this chapter, respectively).

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MEDICAL MEASURES

Of all the different interventions or medical measures that can be indicated in the treatment of eating disorders, only evidence concerning certain types of renutrition (vitamin-mineral supplements, enteral oral artificial feeding and artificial feeding) and NC has been identified.

9.1. Renutrition

9.1.1. What is the efficacy of renutrition in patients with eating disorders?

Response is based on the NICE CPG (2004)[30]. Two high-quality (1++) SRSE, one elaborated by the AHRQ of the US (2006)[31] and an additional one by Bulik, *et al*. (2007)[202] have been considered. The updated search has identified a new RCT (Rigaud, 2007)[203].

Scientific Evidence

In an RCT (Birmingham, 1994; Canada)[204] a Zn supplement (14 mg/day) was compared to placebo in a sample of 54 females with AN aged over 15 years. This study yields preliminary evidence that Zn can increase the BMI.	**RCT 1+**

An RCT (Rigaud, 2007; France) [203] compared enteral feeding (cyclic enteral nutrition) (N=41, 97% women, mean age: 22.5 years) with a control group (N=40, 98% women, mean age: 24.2 years) in a sample of individuals with AN who had been treated for two months and with one-year follow-up. When treatment was completed, weight gain was 39% greater in the group treated with enteral feeding than in the control group ($p<0.01$); fat-free body mass increase was also greater in the group treated with enteral feeding ($p<0.01$); energy intake was greater in the group treated with enteral feeding ($p<0.05$); the number of binge-eating episodes was lower in the group treated with enteral feeding than in the control group ($p<0.01$). The majority of patients treated with enteral feeding improved in regards to their eating disorder. After treatment, the recurrence-free period was greater in the group treated with enteral feeding than in the control group ($p<0.05$). Treatment with enteral nutrition helps correct malnutrition in patients with AN, without interfering in the treatment of their eating disorder or increasing the recurrence-free period. **RCT 1+**

Summary of the evidence

CPG[30]	There is not sufficient significant clinical evidence that determines that treatment with Zn supplement in hospitalised adults with AN is effective at increasing weight when compared to placebo at the end of treatment (2 RCTs; N=68; Birmingham, 1994[204]; Katz, 1987[205]).
CPG[30]	There is not sufficient significant clinical evidence that determines that the Zn supplement *vs*. placebo in hospitalised children with AN is effective at increasing weight at the end of treatment (1 RCT; N=26; Lask, 1993)[206].
CPG[30]	There is limited evidence that indicates significant clinical differences in the treatment with nasogastric feeding *vs*. nasogastric feeding-standard treatments in the increase of weight in hospitalised adults with AN (2 RCTs; N=116; Arii, 1996[207]; Robb, 2002[208]).
CPG[30]	There is not sufficient evidence to indicate that there are significant clinical differences between total parenteral nutrition *vs*. standard treatment in weight gain in adults with AN who are under inpatient management (1 RCT; N=22; Pertschuk, 1981)[209].

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Recommendations

		(See also recommendations 9.GM.1 to 9.GM.6)
D	9.1.1.1.	Physical exploration and in some cases treatment with multi-vitamin/mineral supplements in oral form, both on an outpatient and inpatient basis, is recommended for patients with AN who are at the stage of body weight restoration. (Adopted from recommendation 6.4.5.2 of the NICE CPG).
D	9.1.1.2.	Total parenteral nutrition should not be used for patients with AN unless the patient refuses nasogastric feeding and/or presents gastrointestinal dysfunction. (Adopted from recommendation 6.4.11.1 of the NICE CPG).
✓	9.1.1.3.	Enteral or parenteral renutrition must be applied using strict medical criteria and its duration will depend on when the patient is able to resume oral feeding.

9.1.2. What is the safety of renutrition in patients with AN?

The answer is based on the evidence described for renutrition efficacy (question 9.1.1.).

Scientific Evidence

In an RCT no adverse effects were reported. In an RCT (Birmingham, 1994)[204] No adverse effects were reported. Dropout rate in the group treated with the Zn supplement was 39% and 32% in the placebo group.	**RCT 1+**

Summary of the Evidence

CPG[30]	There is not sufficient evidence to indicate significant clinical differences between treatment with zinc supplement *vs*. placebo in the number of adult inpatients with AN who drop out of treatment for any reason (2 RCTs; N= 68; Birmingham, 1994[204]; Katz, 1987[205]).
CPG[30]	There is not sufficient evidence to indicate significant clinical differences between treatment with nasogastric feeding-standard treatment, in the number of inpatients with AN who drop out of treatment for any reason (1 RCT; Arii, 1996[207]; N=16; RR: 2.33; 95% CI: 0.30 to 17.88).
CPG[30]	There is limited evidence regarding significant clinical differences between total parenteral nutrition *vs*. standard treatment in the number of patients with adverse effects (1 RCT; N=22; Pertschuk, 1981)[209].

Recommendations

(See recommendations 9.GM.1. to 9.GM.5.)

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9.2. Nutritional Counselling (NC)

9.2.1. Anorexia nervosa

9.2.1.1. What is the efficacy of NC in patients with AN?

The answer is based on the NICE CPG (2004)[30]. Two high-quality (1++) SRSE, one elaborated by the AHRQ of the US (2006)[31] and a more recent one conducted by Bulik, *et al*. (2007)[202]. No new evidence has been identified in the updated search.

Scientific Evidence

In a further RCT (Pike, 2003; USA)[210], CBT (N=18) was compared to NC (N=15) in women (18-45 years) with AN who had been under inpatient treatment for 12 months. After weight restoration, a lower percentage of failures, a higher percentage of positive results and a longer time period until recurrence were reported in the group that received CBT. However, one of the limitations found in this study was the fact that many patients had received concomitant antidepressant treatment. **RCT 1+**

Summary of the Evidence

CPG[30]	There is limited evidence to indicate that in inpatients with AN after weight restoration, CBT is superior to NC in the proportion of recoveries and relapses. (1 RCT; N=33; Pike, 2003)[210].
SRSE[31] 1++	There is little evidence that CBT is superior to NC in the proportion of patients who recover and in weight gain.

Recommendations

(See recommendations 9.GM.1. to 9.GM.5.)

9.2.1.2. What is the safety of NC in patients with AN?

The answer is based on the evidence described for NC efficacy (question 9.2.1.1.).

Scientific Evidence

AN RCT (Pike, 2003)[210] reported an overall dropout rate of 9% (CBT: 0% *vs.* NC: 20%). There was one case of depression and suicidal ideation. The design of this RCT is limited by the fact that many studied patients were also taking antidepressants, rendering its results questionable. **RCT 1+**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Summary of the Evidence

CPG[30]	There is limited evidence to indicate that CBT is more acceptable than NC in inpatients with AN after weight restoration (1 RCT; N=33; Pike, 2003)[210].

Recommendations

(See recommendations 9.GM.1. to 9.GM.5.)

9.2.2. Bulimia nervosa

9.2.2.1. What is the efficacy of NC in patients with BN?

The answer is based on the NICE CPG (2004)[30]. High-quality (1++) SRSE have also been taken into account, one elaborated by the AHRQ of the US (2006)[31] and a more recently published one by Shapiro, *et al*., . (2007)[211], which include the same RCT. The updated search has not yielded new evidence. Results are presented according to the variables studied in the RCT.

Variables: reduction/ remission of binge-eating and purging episodes

There is limited evidence to suggest that CBT-EN is more effective than NC in reducing the frequency of purging episodes by the end of treatment (SMD: -0.95; 95% CI: -1.70 to -0.20) and strong evidence for post-treatment follow-up (SMD: -1.34; 95% CI: -2.13 to -0.55) according to 1 RCT (N=31; Sundgot-Borgen, 2002)[212]. **RCT 1++**

There is no evidence or insufficient evidence to determine that CBT-exposure with response prevention (ERP) for binge-eating episodes differs from NC (nutritional therapy) in the remission of binge-eating and purging (2 RCTs; N=90; Hsu, 2001[213]; Jansen, 2002[214]; RR: 0.82; 95% CI: 0.65 a 1.04) and in the frequency of binge-eating and purging (1 RCT; N=49; Hsu, 2001[213]; SMD: 0.43; 95% CI: -0.14 to 0.99) by the end of treatment. **RCT 1 ++**

Variables: depression and/or interpersonal and psychosocial functioning and/or general psychiatric symptoms

There is no evidence or insufficient evidence to determine that CBT-ERP differs from NC (nutritional therapy) in the scores obtained from the instrument used to assess depression (1 RCT; N=49; Hsu, 2001[213]; SMD: 0.15; 95% CI: -0.41 to 0.72). **RCT 1 ++**

Summary of the Evidence

SRSE[31] 1++	If a cognitive component is added to NC (nutritional therapy) its effectiveness improves (Hsu, 2001)[213].

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Recommendations

(See recommendations 9.GM.6. to 9.GM.9.)

9.2.2.2. What is the safety of NC in patients with BN?

The answer is based on the evidence described for NC efficacy (question 9.2.2.1.).

Summary of the Evidence

1++	There is limited evidence to indicate significant differences between CBT-BN *vs*. NC in the number of dropouts by the end of treatment (1 RCT; N=31; Sundgot-Borgen, 2002[212]; SMD: -0.95; 95% CI: -0.70 to -0.20).
1++	There is not sufficient evidence of clinically significant differences between CBT-ERP and NC (nutritional therapy) in the number of dropouts for any reason by the end of treatment (2 RCTs; N=90; Hsu, 2001[213]; Jansen, 2002[214]; RR: 0.95; 95% CI: 0.16 to 5.51).

Recommendations

(See recommendations 9.GM.6. to 9.GM.9.)

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GENERAL RECOMMENDATIONS ON MEDICAL MEASURES FOR EATING DISORDERS (QUESTIONS 9.1-9.2)

Eating Disorders

Recommendations

✓	9.GM.01.	Nutritional support for patients with eating disorders will be selected based on the patient's degree of malnutrition and collaboration, and always with the psychiatrist's approval.
✓	9.GM.02.	Before initiating artificial nutrition the patient's degree of collaboration must be assessed and an attempt must always be made to convince him/her of the benefits of natural oral feeding.
✓	9.GM.03.	In day hospitals, nutritional support for low-weight patients, where an oral diet is insufficient, can be supplemented with artificial nutrition (oral enteral nutrition). To ensure its intake, it must be administered during the day hospital's hours, providing supplementary energy ranging from 300 to 1,000 kcal/day.
✓	9.GM.04.	Oral nutritional support in eating disorder inpatients is deemed adequate (favourable progress) when a ponderal gain greater than 0.5 kg per week is produced, with up to 1 kg increments being the usual during that period. Sometimes, when the patient with moderate malnutrition resists resuming normal feeding, the diet can be reduced by 500-700 kcal and be supplemented by complementary oral enteral nutrition in the same amount, which must be administered after meals and not instead of meals.
✓	9.GM.05.	In the case of severe malnutrition, extreme starvation, poor progress or lack of cooperation of the patient in terms of intake, artificial nutrition treatment is indicated. If possible, an oral diet with or without oral enteral nutrition is always the first step, followed by a 3 to 6 day period to assess the degree of collaboration and medical-nutritional evolution.
✓	9.GM.06.	Regarding estimated energetic requirements, it is recommended that caloric needs at the beginning always be below the usual, that real weight, as opposed to ideal weight, be used to make the estimation and that in cases of severe malnutrition energetic requirements be 25 to 30 kcal/kg real weight or total kcal not higher than 1,000/day.

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Anorexia nervosa

Recommendations

D	9.GM.1.	In feeding guidelines for children and adolescents with anorexia nervosa, carers should be included in any dietary information, education and meal planning. (Adopted from recommendation 6.5.8.9 of the NICE CPG).
D	9.GM.2.	Feeding against the will of the patient should be used as a last resort in the management of AN. (Adopted from recommendation 6.4.13.5 of the NICE CPG).
D	9.GM.3.	Feeding against the will of the patient is an intervention that must be performed by experts in the management of eating disorders and related clinical complications. (Adopted from recommendation 6.4.13.6 of the NICE CPG).
D	9.GM.4.	Legal requirements must be acknowledged and complied with when deciding whether to feed a patient against his/her will. (Adopted from recommendation 6.4.13.7 of the NICE CPG).
D	9.GM.5.	Health care professionals must be careful with the healthy weight restoration process in children and adolescents with AN, administering the nutrients and energy required by providing an adequate diet to promote normal growth and development. (Adopted from recommendation 6.5.8.8 of the NICE CPG).

Bulimia nervosa

Recommendations

D	9.GM.6.	Patients with BN who frequently vomit and abuse laxatives can develop abnormalities in electrolyte balance. (Adopted from recommendation 7.5.3.1 of the NICE CPG).
D	9.GM.7.	When electrolyte imbalance is detected, in most cases elimination of the behaviour that caused it is sufficient to correct the problem. In a small number of cases, oral administration of electrolytes whose plasmatic levels are insufficient is necessary to restore normal levels, except in cases involving gastrointestinal absorption. (Adopted from recommendation 7.5.3.2 of the NICE CPG).
D	9.GM.8.	In the case of laxative misuse, patients with BN must be advised on how to decrease and stop abuse. This process must be carried out gradually. Patients must also be informed that the use of laxatives does not decrease nutrient absorption.

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		(Adopted from recommendation 7.5.3.3 of the NICE CPG).
D	9.GM.9.	Patients who vomit habitually must have regular dental check-ups and be provided with dental hygiene advice. (Adapted from recommendations 7.5.3.4 and 7.5.3.5 of the NICE CPG).

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PSYCHOLOGICAL THERAPIES

In this section scientific evidence on the efficacy and safety of a series of psychological therapies studied in patients with eating disorders is described. Only randomised controlled trials (RCT) of sufficient quality have been included. The following therapies have been assessed: cognitive-behavioural therapy, self-help and guided self-help, interpersonal therapy, family therapy (systemic or unspecified), psychodynamic therapy and behavioural therapy.

9.3. Cognitive-Behavioural Therapy (CBT)

9.3.1. Anorexia nervosa

9.3.1.1. What is the efficacy of CBT in patients with AN?

The answer is based on the NICE CPG (2004)[30]. High-quality (1++) SRSE have been taken into account, one elaborated by the AHRQ of the US (2006[31]) and a further one recently published by Bulik, *et al*. (2007)[202] where the same RCTs are included. The updated search has not yielded any new evidence.

Scientific Evidence

In an RCT (Channon, 1989; UK[215]), CBT (N=8; mean age=21.6 years) was compared to behavioural therapy (BT) (N=8; mean age=24.1 years) and a control group (standard treatments) (N=8; mean age=25.8 years) in women with AN (mean age range: 21.6-25.8 years) treated on an outpatient basis for 12 months. At 6 months of treatment, CBT was more effective than BT in improving psychosexual functioning. In contrast, BT was more effective than CBT in improving the menstrual cycle. At one year of treatment, BT was more effective than the other two treatments in restoring patients' weight. **RCT 1 +**

In a further RCT (Pike, 2003; USA)[210], CBT (N=18) was compared to NC (N=15) in women (18-45 years) with AN who had been under inpatient treatment for 12 months. After weight restoration, a lower percentage of failures, a higher percentage of positive results and a longer period of time until recurrence was reported in the group that received CBT. However, one of the limitations of the study was the fact that many patients had received concomitant antidepressant treatment. **RCT 1 +**

In the third RCT (McInstosh, 2005; New Zealand)[216], CBT (N=19) was compared to IPT (N=21) and non-specific supportive clinical management (NSCM) (N=16), in women (17-40 years) with AN who were treated on an outpatient basis for 20 weeks. Non-specific supportive clinical management (NSCM) was more effective than IPT in improving patients' general behaviour and in dietary restraint at 20 weeks of treatment and was also more effective than CBT in improving general behaviour at 20 weeks. **RCT 1 +**

CBT was superior to IPT in improving dietary restraint at 20 weeks.

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Summary of the Evidence

	(See also the summary of the evidence for psychological treatment)
SRSE[31] 1++	There is little evidence to determine that CBT is more effective than NC in the number of patients with AN who recover and in weight gain.
SRSE[31] 1++	CBT did not prove to be more effective than BT or non-specific supportive clinical management (NSCM) in AN patients' weight restoration, general behaviour and attitudes regarding food. Based on the results, CBT does not exceed IPT.
SRSE[31] 1++	There is not enough evidence to determine that CBT is effective in the acute phase of AN.
CPG[30]	There is limited evidence to indicate that CBT is more acceptable than NC in AN inpatients after weight restoration (1 RCT; N=33; Pike, 2003)[210].
SRSE[31] 1++	CBT treatment can reduce recurrence risk in adults with AN after weight restoration.

Recommendations

(See recommendations 9.GP.1. to 9.GP.11.)

9.3.1.2. What is the safety of CBT in patients with AN?

The answer is based on the evidence pertaining to the question on CBT efficacy (question 9.3.1.1.): NICE's CPG (2004)[30] and 2 high-quality SRSE (1++) (2006)[31] and (2007)[202]. The updated search did not yield any new evidence. Question 9.3.1.1. briefly describes the studies.

Scientific Evidence

One of the RCT (Channon, 1989)[215] reported that 13% of patients dropped out of treatment. Dropouts per groups were: CBT: 0%, BT: 13%, control: 25%; and no adverse effects were reported in any group. **RCT 1 +**

In the second RCT (Pike, 2003)[210], 9% dropped out of treatment (CBT 0%, NC 20%) and depression and suicidal ideations was reported in one case of the CBT group and 3 of the NC group. **RCT 1 +**

The third RCT (McInstosh, 2005)[216] reported that 38% of studied patients dropped out of treatment. Dropouts per groups were: CBT 37%, IPT 43%, non-specific supportive clinical management (NSCM) 31%; however, no adverse effects were reported in any group. **RCT 1 +**

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Summary of the Evidence

	(See also the summary of the evidence for psychological treatment)
CPG[30]	There is limited evidence to determine that CBT is more acceptable than NC for AN inpatients after weight restoration (1 RCT; N=33; Pike, 2003)[210].
SRSE[31] 1++	Psychological interventions do not usually produce adverse effects in treated patients.

Recommendations

(See recommendations 9.GP.1. to 9.GP.11.)

9.3.2. Bulimia nervosa

9.3.2.1. What is the efficacy of CBT in patients with BN?

The source of evidence to answer this question has been NICE's CPG (2004)[30], which describes evidence based on outcome variables. It is also based on high-quality (1++) SRSE, one elaborated by the AHRQ of the US (2006)[31] and a further more recent publication by Shapiro, *et al*. (2007)[211], where six new quality (1++ and 1+) RCT were identified. The updated search subsequently yielded two more RCT.

Variable: reduction / remission of binge-eating and purging

There is strong evidence that points to a higher efficacy of CBT-BN *vs.* wait-list in terms of remission in adults with BN by the end of treatment (3 RCTs; N=136; Griffiths, 1994[217]; Lee, 1986[218];Treasure, 1999[219]; RR: 0,73; 95% CI: 0.61 to 0.88; NNT:4; 95% CI: 3 a 9). **RCT 1 ++**

There is strong evidence that indicated the efficacy of CBT-BN vs. wait-list in terms of the frequency of binge-eating (5 RCTs: N=185; Griffiths, 1994[217]; Lee, 1986[218]; Treasure, 1999[219]; Freeman, 1988[220]; Wolf, 1992[221]; SMD: -0.75 95% CI: -1.05 to -0.44) and the frequency of purging in adults by the end of treatment (6 RCTs; N=192; Griffiths, 1994[217]; Lee, 1986[218]; Freeman, 1988[220]; Wolf, 1992[221]; Agras, 1989[222]; Leitenberg, 1988[223]; SMD according to the random effects model: -1.00; 95% CI:-1.63 to -0.36). **RCT 1 ++**

It is not likely that CBT-BN is more effective than BT in reducing the frequency of binge-eating (SMD:-0.11; 95% CI:-0.45 to 0.24) and purging episodes by the end of treatment (SMD: 0.08; 95% CI: -0.27 to 0.42) (3 RCTs; N=131; Fairburn, 1991[224]; Freeman, 1988[220]; Wolf, 1992[221]). **RCT 1 ++**

There is not sufficient evidence that CBT-BN-ERP is superior to CBT-BN in terms of remission of binge-eating (2 RCTs; N=53; Cooper, 1995[225];Wilson, 1991[226]; RR: 0.97; **RCT 1 ++**

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95% CI: 0.58 to 1.63) and purging (3 RCTs; N=92; Cooper, 1995[225];Wilson, 1991[226];Agras, 1989[222]; RR: 0.88; 95% CI: 0.62 to 1.24) by the end of treatment.

There is sufficient evidence to determine that CBT-BN-ERP is superior to CBT-BN alone in the remission of binge-eating (RR: 1.20; 95% CI: 0.52 to 2.79) and purging (RR: 1.60; 95% CI: 0.76 to 3.36) at follow-up after the end of treatment (1 RCT; N=22; Cooper, 1995)[225]. **RCT 1 ++**

There is not sufficient evidence to suggest that CBT-BN-ERP treatment is superior to CBT-BN in reducing the frequency of binge-eating (1 RCT; N= 27; Cooper, 1995[225]; SMD: -0.25 95% CI: -1.01 to 0.50) and purging (3 RCTs: N=83; Agras 2000[227]; Cooper, 1995[225], Lenteinberg, 1988[223]; SMD: -0.17; 95% CI:-0.60 to 0.27) by the end of treatment. **RCT 1 +**

There is not sufficient evidence to suggest that CBT-BN-ERP treatment is superior to CBT-BN in reducing the frequency of purging at follow-up after the end of treatment (2 RCTs; N=48; Cooper, 1995[225]; Lenteinberg, 1988[223]; SMD= -0.47; 95% CI: -1.06 to 0.11). **RCT 1 ++**

There is limited evidence that indicates that CBT-BN-ERP treatment is superior to CBT-BN in reducing the frequency of purging episodes at follow-up after the end of treatment (1 RCT; N=25; Wilson, 1991[226]; SMD: -0.90; 95% CI: -1.73 to -0.07). **RCT 1 ++**

There is strong evidence that suggests that CBT-BN treatment is more effective than IPT-BN in the remission of binge-eating (2 RCTs; N=270; Agras, 2000[227]; Fairburn, 1986[228]; RR: 0.77; 95% CI: 0.67 to 0.87; NNT: 5; 95% CI: 4 a 20) and purging by the end of treatment (1 RCT; N=220; Cooper, 1995[225]; RR: 0.76; 95% CI: 0.67 to 0,86; NNT: 5; 95% CI: 4 a 8). **RCT 1 ++**

There is evidence that it is not likely that there are significant differences between CBT-BN and IPT-BN treatments in the remission of binge-eating episodes by the end of treatment and at follow-up (2 RCTs; N=270; Agras, 2000[227]; Fairburn, 1986[228]; RR: 0.93; 95% CI: 0.82 to 1.06). **RCT 1 ++**

It is not likely that CBT-BN is superior to IPT-BN in reducing the frequency of binge-eating (2 RCTs; N=262;Agras, 2000[227]; Fairburn, 1986[228]; SMD: -0.24; 95% CI: -0.48 to 0.01) and purging (2 RCTs; N=257; Agras, 2000[227]; Fairburn, 1986[228]; SMD: -0.04; 95% CI: -0.29 to 0.20) by the end of treatment. **RCT 1 ++**

There is sufficient evidence that there may be significant differences between CBT-BN vs. PDT in the frequency of binge-eating (1 RCT; N=46; Garner, 1993[229]; SMD: -0.19; 95% CI: -0.77 to 0.39) and purging (1 RCT; N=50; Garner, 1993[229]; SMD: -0.56; 95% CI: -1.13 to 0.01) by the end of treatment. **RCT 1 ++**

It is not likely that CBT-BN is superior to FSP (focal supportive psychotherapy) in reducing the frequency of binge-eating (3 RCTs; N=111; Fairburn, 1991[224]; Freeman, 1988[220]; Wolf, 1992[221]; SMD: 0.00; 95% CI: -0.37 to 0.38) and purging episodes (4 RCTs, N=144; Fairburn, 1991[224]; Freeman, 1988[220]; Wolf, 1992[224], Agras, 2000[227]; SMD:-0.13; 95% CI: -0.46 to 0.20) by the end of treatment. **RCT 1 ++**

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There is limited evidence to suggest that CBT-BN is superior to NC in reducing the frequency of purging episodes at the end of treatment (SMD: -0.95; 95% CI:-1.70 to -0.20) and strong evidence at post-treatment follow-up (SMD:-1.34; 95% CI: -2.13 to -0.55) (one RCT; N=31, Sundgot-Borgen, 2002)[212]. **RCT 1 ++**

There is not sufficient evidence that CBT-BN differs from GSH (guided self-help) in the remission of binge-eating and purging episodes at the end of treatment or at post-treatment follow-up (RR: 1.63; 95% CI: 0.42 to 6.36) (1 RCT; N=81, Bailer, 2004)[230]. **RCT 1 ++**

There is limited evidence that CBT-BN differs from GSH in reducing the frequency of binge-eating (SMD: 1.20; 95% CI: 0.63 to 1.78) and purging (SMD: 0.55; 95% CI: 0.01 to 1.08) by the end of treatment (1 RCT; N=56, Bailer, 2004)[230]. **RCT 1 ++**

There is not sufficient evidence that CBT-BN differs from GSH in reducing the frequency of binge-eating (SMD: 0.30; 95% CI: -0.24 to 0.83) and purging (SMD: 0.38; 95% CI: -0.15 to 0.92) at post-treatment follow-up (1 RCT; N=55, Bailer, 2004)[230]. **RCT 1 ++**

There is not sufficient evidence that CBT-BN treatment differs from SH (self-help) in the remission of binge-eating (RR: 0.94; 95% CI: 0.73 to 1.21) and purging (RR: 0.98; 95% CI: 0.82 to 6.36) by the end of treatment (1 RCT; N=83, Treasure, 1994)[231]. **RCT 1 ++**

There is evidence that treatment with CBT-BN differs from SH in reducing the frequency of binge-eating (SMD: 0.03; 95% CI: -0.43 to 0.49) by the end of treatment (1 RCT; N=80, Treasure, 1994)[231]. **RCT 1 ++**

There is not sufficient evidence that individual CBT differs from group CBT in the remission of binge-eating (RR: 0.83; 95% CI: 0.59 to 1.16) and purging (RR: 0.85; 95% CI: 0.57 to 1.27) by the end of treatment (1 RCT; N=60, Chen, 2003)[232]. **RCT 1 ++**

There is insufficient evidence that individual CBT differs from group CBT in the remission of binge-eating (RR: 0.95; 95% CI: 0.64 to 1) and purging (RR: 1.12; 95% CI: 0.74 to 1.69) at post-treatment follow-up (1 RCT; N=60, Chen, 2003)[232]. **RCT 1 ++**

There is not sufficient evidence that individual CBT differs from group CBT in reducing the frequency of binge-eating (SMD: -0.18; 95% CI: -0.68 to 0.33) and purging (SMD: -0.25; 95% CI: -0.76 to 0.26) by the end of treatment (1 RCT; N=60, Chen, 2003)[232]. **RCT 1 ++**

There is not sufficient evidence that individual CBT differs from group CBT in reducing the frequency of binge-eating (SMD: 0.06; 95% CI: -0.45 to 0.57) and purging (SMD: 0.08; 95% CI: -0.42 to 0.59) at post-treatment follow-up (1 RCT; N=60, Chen, 2003)[232]. **RCT 1 ++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Variables: depression and/or interpersonal and psychosocial functioning and/or general psychiatric symptoms

There is strong evidence that points to a higher efficacy of CBT-BN *vs*. wait-list in reducing depression in adults with BN by the end of treatment (3 RCTs; N=87; Agras, 1989[222]; Lee, 1986[218]; Leitenberg, 1988[223]; SMD according to random effects model: -1.19; 95% CI: -1.99 to -0.39). **RCT 1 ++**

There is not sufficient evidence to determine if CBT-BN differs from BT in reducing general psychiatric symptoms (SMD: -0.26; 95% CI: -0.89 to 0.37) and in interpersonal and psychosocial functioning (SMD: -0.21; 95% CI: -0.84 to 0.42) by the end of treatment (1 RCT; N=39, Fairburn, 1991)[224]. **RCT 1 ++**

There is not sufficient evidence that CBT-BN differs from BT in general psychiatric symptoms (SMD: -0.09; 95% CI: -0.79 to 0.61) and interpersonal and psychosocial functioning (SMD: 0.14; 95% CI: -0.56 to 0.85) at follow-up (1 RCT; N=32, Fairburn, 1991)[224]. **RCT 1 ++**

There is insufficient evidence that indicates that CBT-BN differs from PDT in interpersonal and psychosocial functioning (N=41, SMD: -0.39; 95% CI: -1.01 to 0.23) or in general psychiatric symptoms (N= 48, SMD: -0.60; 95% CI: -1.18 to -0.02) by the end of treatment (1 RCT, Garner, 1993)[229] **RCT 1 ++**

There is no evidence or insufficient evidence to determine that CBT-BN differs from FSP in interpersonal and psychosocial functioning (SMD: -0.47; 95% CI:-1.28 to 0.34) (1 RCT; N=24, Fairburn, 1986)[228] and in general psychiatric symptoms (2 RCTs; N=69; Fairburn, 1986[228]; Walsh, 1997[233]; SMD: -0.29; 95% CI: -0.77 to 0.19) by the end of treatment or at post-treatment follow-up. **RCT 1 ++**

There is insufficient evidence that CBT-BN treatment vs. GSH differs in terms of depression scores by the end of treatment (SMD: 0.55; 95% CI: 0.02 to 1.09) and at post-treatment follow-up (SMD: 0.38; 95% CI: -0.15 to0.92) (1 RCT; N=55, Bailer, 2004)[230]. **RCT 1 ++**

There is insufficient evidence that individual CBT differs from group CBT in terms of depression (SMD: 0.09; 95% CI: -0.41 to 0.60), general psychiatric symptoms (SMD: 0.07; 95% CI: -0.58 to 0.44) and psychosocial and interpersonal functioning (SMD: -0.16; 95% CI: -0.67 to 0.34) by the end of treatment (1 RCT; N=60, Chen, 2003)[232] **RCT 1 ++**

There is insufficient evidence that individual CBT differs from group CBT in terms of depression (SMD: 0.28; 95% CI: -0.23 to 0.179), general psychiatric symptoms (SMD: 0.14; 95% CI: -0.37 to 0.64) and psychosocial and interpersonal functioning (SMD: 0.46; 95% CI: -0.05 to 0.98) at post-treatment follow-up (1 RCT; N=60, Chen, 2003)[232]. **RCT 1 ++**

Other results

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

In an RCT (Wilson, 2002; USA)[234] CBT was compared to IPT in a sample of 20 adult women with BN under outpatient treatment for 6 weeks. In this study, CBT-BN was superior than IPT in reducing dietary restraint at 6 weeks of treatment, improving binge-eating and purging at 10 weeks of treatment, and reducing binge-eating after treatment. CBT was more effective at reducing the frequency of vomiting at 6 weeks after treatment than IPT. **RCT 1 +**

In an RCT (Wilfley, 1993; USA)[235] that compared group CBT with group IPT and wait-list, in 56 adult women with BN, CBT and IPT were superior to the control group (wait-list) in reducing frequency of binge-eating episodes, disinhibition and restraint at 16 weeks of treatment. There were no significant differences between both group therapies. **RCT 1 +**

A further RCT (Bulik, 1998; New Zealand)[236] performed on 111 adult women with BN under outpatient treatment compared CBT (8 weeks) followed by ERP for binge-eating (G1) with CBT followed by ERP for vomit-inducing signs (G2) and with CBT followed by relaxation training (G3). G3 was superior to G1 in reducing depressive behaviour and body dissatisfaction at the end of treatment and at 2-years follow-up. Relaxation was superior to G2 in reducing depressive behaviour and psychological eating disorder and behavioural attitudes at the end of treatment and at 3-years follow-up. **RCT 1 ++**

An additional RCT (Sundgot-Borgen, 2002; Norway)[212] compared exercise with CBT, NC, wait-list and healthy control subjects in a group of 74 adult women with BN under outpatient treatment. Exercise was superior to CBT in reducing binge-eating episodes, laxative abuse and the desire to be thin throughout 18 months of follow-up. **RCT 1 +**

An RCT (Bailer, 2004;Austria)[230] compared group CBT with group GSH in 81 adult women with BN under outpatient treatment. The GSH was administered by psychiatrists in training. Both treatments significantly reduced binge-eating, vomiting, laxative abuse, the EDI questionnaire score, the desire to be thin and body dissatisfaction. At one year follow-up, patients treat with GSH obtained greater reduction in purging and EDI questionnaire score. CBT was associated with greater reduction in the desire to be thin at the end of treatment and at follow-up. Both therapies significantly improved depression at the end of treatment. At follow-up, individuals with GSH obtained better results in decreasing depression scores. Within the group of patients who completed treatment, there was a significant difference in the group treated with GSH in the maintenance of remission for more than 2 weeks (74% vs. 44%). There were no significant changes in weight in patients treated with GSH. **RCT 1 +**

In an RCT (Thiels, 1998;Alemania)[237] CBT was compared to GSH for 16 weeks in a group of 62 adult women with BN under outpatient treatment. Treatment was administered by psychotherapists. In both treatments, a significant decrease in binge-eating, purging and BITE and EAT scores was reported. There were no significant differences among groups in terms of depression. **RCT 1 +**

In a recent RCT (Schmith, 2007; UK)[238] FT (N=31 BN, N=10 EDNOS) was compared to CBT-GSH (N=30 BN, N=14 EDNOS) in adolescents aged between 13 and 20 years. Treatment was administered for 6 months and follow-up was carried out throughout 12 months. CBT-GSH reduced binge-eating more than FT at 6 months (p=0.03) although at 12 months the difference between them disappeared. There were **RCT 1 ++**

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no differences between groups in terms of BMI, diet, eating fast and other behavioural attitudes related to eating behaviour. The direct cost of treatment was lower for CBT-GSH and no differences were observed in other types of costs.

In a recent RCT (Burton, 2007; U.S.)[239] 45 women (age range: 14-23 years) from different educational institutions were recruited. They belonged to different ethnic groups and social classes and presented depressive symptoms. The group treated with depression-aimed CBT was compared to a control group (on wait-list). CBT was administered for 4 weeks (weekly one-hour sessions) with follow-up at 1 and 6 months. A significant reduction of depressive symptoms was observed in the group treated with CBT, compared to the control ($p<0.001$), at one-month follow-up ($p<0.001$) and at 6-months follow-up (p=0,018). A significant reduction of BN symptoms was reported in the group treated with CBT compared to the control group (p=0.004) and at one-month follow-up (p=0.016). These effects were not significant at 6-months follow-up (p=0.384) There were no significant differences in terms of substance abuse among groups (p=0.548) or at 6-months follow-up (p=0.714) and one-year follow-up (p=0.647). CBT is effective at reducing depressive symptoms at 6-months follow-up, providing an opportunity to study long-term effects of depressive symptoms. CBT produces a short-term reduction of BN symptoms but not substance abuse. Future research with RCT that use risk factors for psychiatric disorders are necessary. **RCT 1 ++**

General summary of the evidence

	(See also summary of the evidence for psychological treatment)
SRSE[240] 1++	An RCT suggests that CBT reduces symptoms in less time than IPT. (Fairburn, 1991)[224].
SRSE[240] 1++	An RCT shows that group CBT is more economical and yields the same results as individual CBT (Chen, 2003)[241].
SRSE[240] 1++	CBT is more effective than BT due to the cognitive component that seems to be the most important aspect of this therapy (Cooper, 1995[225]; Fairburn, 1991[224]).
SRSE[240] 1++	The administration of ERP after CBT does not provide any additional advantage to CBT (Bulik, 1998[236]; Cooper, 1995[225]).
SRSE[240] 1++	CBT has better results than psychodynamically oriented supportive expressive therapy (Garner, 1993)[229].
SRSE[240] 1++	Adding a cognitive element to NC (nutritional therapy) improves its effectiveness (Hsu, 2001)[213].
SRSE[240] 1++	There are scarce conclusions regarding the efficacy of SH and GSH treatments for BN (Bailer, 2004[230]; Carter, 2003[242]; Thiels, 1998[237]).

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

SRSE[240] 1++	CBT treatment in patients with BN at 6 and 12 months follow-up produced a 90% decrease in binge-eating and purging and, at one-year follow-up, 36% of patients remitted in terms of binge-eating and purging.
SRSE[240] 1++	Individual and group CBT treatment reduces the main symptoms of BN (binge-eating and purging) and related short and long-term psychological effects.
CPG[30]	CBT-GSH is superior to FT by reducing binge-eating more rapidly, being less expensive and more acceptable for adolescents with BN and/or EDNOS. Further research is necessary to determine if different groups of adolescents respond differently to FT and CBT-GSH and which different ways of involving the family in treatment can be more or less beneficial (Schmidt, 2007)[238].

Recommendations

A	9.3.2.1.1.	CBT-BN is a specifically adapted form of CBT and it is recommended that 16 to 20 sessions be performed over 4 or 5 months of treatment. (Adopted from recommendation 7.2.7.3. of the NICE CPG). intervention can be incorporated. (Adopted from recommendation 7.2.7.4. of the NICE CPG).
B	9.3.2.1.2.	Patients with BN who do not respond to or refuse to receive CBT treatment may be offered alternative psychological recommendation (Adopted from recommendation 7.2.7.5. of the NICE CPG).
D	9.3.2.1.3.	Adolescents with BN can be treated with CBT adapted to the their age needs, level of development, and, if appropriate, the family's treatment. (Adopted from recommendation 7.2.7.4. of the NICE CPG).

9.3.2.2. What is the safety of CBT in patients with BN?

The answer is based on available evidence for CBT efficacy, where studies are briefly described (question 9.3.2.1.).

Scientific Evidence

There is evidence that indicates that CBT treatment dropout is not likely (9 RCTs; N=384;Agras, 1989[222]; Freeman, 1988[220]; Griffits, 1994[217]; Lee, 1986[218]; Leitenberg, 1988[223]; Wolf, 1992[221]; Treasure, 1994[231]; Mitchell, 1990[243]; Sundgot-Borgen, 2002[212]; RR: 1.14; 95% CI: 0.74 to 1.74). **RCT 1 ++**

There is insufficient evidence that there are significant differences between CBT-BN and BT in the number of dropouts by the end of treatment (3 RCT; N=142; Fairburn, 1991[224]; Freeman, 1988[220]; Wolf, 1992[221]; RR: 1.20; 95% CI: 0.61 to 2.37). **RCT 1 ++**

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There is insufficient evidence to determine if there are significant differences between CBT-BN and CBT-ERP in the number of dropouts by the end of treatment (4 RCTs; N=115;Agras, 1989[222]; Leitenberg, 1988[223]; Cooper, 1987[75];Wilson, 1991[226]; RR: 1.14; 95% CI: 0.48 to 2.68). **RCT 1 ++**

There is insufficient evidence to determine if there are significant differences between CBT-BN and IPT in the number of dropouts by the end of treatment (2 RCTs; N=70; Agras, 1989[222]; Fairburn, 1991[224]; RR: 1.24; 95% CI: 0.84 to 1.83). **RCT 1 ++**

There is insufficient evidence to determine if there are significant differences between CBT-BN and PDT in the number of dropouts by the end of treatment (1 RCT; N=50; Garner, 1993[229]; RR: 1.00; 95% CI: 0.33 to 3.03). **RCT 1 ++**

There is insufficient evidence to determine if there are significant differences between CBT-BN and FSP in the number of dropouts by the end of treatment (4 RCTs; N=155; Agras, 1989[222]; Fairburn, 1991[224]; Freeman, 1988[220]; Kirckley, 1985[244]; RR: 0.84; 95% CI: 0.49 to 1.45). **RCT 1 ++**

There is limited evidence to indicate significant differences between CBT-BN and NC in the number of dropouts by the end of treatment (1 RCT; N=31; Sundgot-Borgen, 2002[212]; SMD: -0.95; 95% CI: -1.70 to -0.20). **RCT 1 ++**

There is insufficient evidence to determine if there are significant differences between CBT-BN and GSH in the number of dropouts by the end of treatment (1 RCT; N=81; Bailer, 2004[230]; RR: 1.46; 95% CI: 0.75 to 2.86). **RCT 1 ++**

There is insufficient evidence to determine if there are significant differences between CBT-BN and SH in the number of dropouts by the end of treatment (1 RCT; N=83; Treasure, 1994[231]; RR: 0.98; 95% CI: 0.45 to 2.15). **RCT 1 ++**

There is insufficient evidence to determine if there are significant differences between group CBT and individual CBT in the number of dropouts by the end of treatment (1 RCT; N=60; Chen, 2003[232]; RR: 1.00; 95% CI: 0.43 to 2.31). **RCT 1 ++**

In an RCT (Agras, 2000)[227], the number of dropouts in the CBT group was 28% and 24% in the IPT group. There was one case of acute panic attack as an adverse effect in the CBT group. **RCT 1 ++**

In another RCT (Wilfley, 1993; USA)[235], where group CBT was compared to group IPT and wait-list, in adult females with BN (N=56), the number of dropouts in the CBT group was 14%, 11% in the IPT group and there were no dropouts in the control group (wait-list). No adverse effects were reported. **RCT 1 +**

In an RCT (Wilson, 2002)[234], the total dropout rate by the end of treatment was 30% and 41% at follow-up. No adverse effects were reported. **RCT 1 +**

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In an RCT (Bulik, 1998)[236] the overall dropout rate was 17%. Dropouts per groups were: CBT-ERP for binge-eating: 5%, CBT-ERP for vomit-inducing signs: 6%, relaxation training: 3%. No adverse effects were reported. **RCT 1 ++**

In an RCT (Sundgot-Borgen, 2002)[212], after exercise, 20% of dropouts; after CBT, 13%; after NC, no dropouts; after wait-list, 6%; after healthy control, 0%. No adverse effects were reported. **RCT 1 +**

In an RCT (Bailer, 2004)[230], after SH, the dropout rate was 25%; after CBT, dropout rate was 37%. No adverse effects were reported. **RCT 1+**

In an RCT (Thiels, 1998)[237], dropout rate in the CBT group was 13%; in GSH, it was 29%. No adverse effects were reported. **RCT 1+**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12. and 9.GP.13.)

9.3.3. Binge-eating disorder

9.3.3.1. What is the efficacy of CBT in patients with BED?

The evidence that answers this question in based on the NICE CPG (2004)[30] and on quality RCT (1++ and 1+) that had not been considered in the aforementioned guide but had been included in the high-quality SRSE, one elaborated by the AHRQ of the US (2006)[31] and a more recently published one conducted by Brownley, *et al*. (2007)[245]. Three more RCTs have been identified in the updated search.

Variable: BMI

There is evidence that indicates it is not likely that there is a clinically significant difference between CBT-BED and wait-list in body weight modification by the end of treatment (BMI when possible) (3 RCTs; N=176;Agras, 1995[246]; Gorin, 2001[247]; Telch, 1990[248]; SMD: -0.02; 95% CI: -0.33 to 0.30). **RCT 1++**

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There is evidence that indicates it is not likely that there is a clinically significant difference between CBT-BED and IPT-BED in body weight modification by the end of treatment (BMI when possible) (1 RCT; N=158; Wilfley, 2002[249]; SMD: 0.06; 95% CI: -0.26 to 0.37). **RCT 1++**

There is insufficient evidence to determine that there are significant differences between BCT-BED and behavioural weight control (BWC) in the variation of body weight (BMI when possible) by the end of treatment (SMD: 0.24; 95% CI: -0.41 to 0.90) and at follow-up (SMD: 0.05; 95% CI: -0.60 to 0.70) (1 RCT; N=37, Nauta, 2000)[250]. **RCT 1++**

Variables: reduction / remission of binge-eating and purging

There is strong evidence that CBT-BED is more effective than being on wait-list in terms of significant remission of binge-eating (4 RCT; N=226; Agras, 1995[246]; Gorin, 2001[247]; Telch, 1990[248]; Wilfley, 1993[235]; based on random effects model RR:0.64; 95% CI: 0.49 to 0.84; NNT: 3; 95% CI: 2 to 7) and reduction of binge-eating episodes (4 RCTs: N=214;Agras, 1995[246]; Gorin, 2001[247];Telch, 1990[248];Wilfley, 1993[235]; random effects model SMD: -1.30; 95% CI: -2.13 to -0.48) by the end of treatment. **RCT 1++**

There is evidence that indicates it is not likely that there is a significant difference in the reduction of binge-eating frequency between CBT-BED and IPT-BED, by the end of treatment (2 RCT: N=194; Wilfley, 1993[235] and 2002[249]; SMD:-0.07; 95% CI: -0.35 to 0.22) and at post-treatment follow-up (1 RCT; N=138; Wilfley, 2002[249]; SMD: 0.14; 95% CI: -0.19 to 0.48). **RCT 1++**

There is strong evidence that indicates a significant difference in binge-eating remission in favour of CBT-BED *vs*. BWC at post-treatment follow-up (1 RCT; N=37; Nauta, 2000[250]; RR: 0.25; 95% CI: 0.08 to 0.79; NNT: 3; 95% CI: 2 to 8). **RCT 1++**

Variables: depression and/or interpersonal and psychosocial functioning and/or general psychiatric symptoms

Evidence indicates that it is unlikely to be a significant difference between CBT-BED and the control group (wait-list) in depression scores by the end of treatment (4 RCTs; N=214; Agras, 1995[246]; Gorin, 2001[247]; Telch, 1990[248]; Wilfley, 1993[235]; SMD: -0.18; 95% CI: -0.46 to 0.10). **RCT 1++**

There is insufficient evidence to indicate that there are significant differences between CBT-BED and the control group (wait-list) in general psychiatric scores (1 RCT; N=42; Agras, 1995[246]; SMD: 0.00; 95% CI: -0.69 to 0.69) and in interpersonal and psychosocial functioning (2 RCTs; N=194; Agras, 1995[246]; Wilfley, 1993[235]; SMD: 0.22; 95% CI: -0.06 to 0.50) by the end of treatment. **RCT 1++**

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Evidence indicates that it is unlikely to be a significant difference between CBT-BED and IPT-BED in depression scores by the end of treatment (2 RCTs; N=194; Wilfley, 1993[235] y 2002[249]; DME; 0,22; 95% CI: -0,06 a 0,50) and at post-treatment follow-up (1 RCT; N=138; Wilfley, 2002[249]; SMD:0.10; 95% CI: -0.24 to 0.43). **RCT 1++**

Evidence indicates that it is unlikely to be a significant difference between CBT-BED and IPT-BED in psychosocial / interpersonal functioning by the end of treatment (2 RCTs: N=194; Wilfley, 1993[235] and 2002[249]; SMD: 0.06; 95% CI: -0.22 to 0.35). **RCT 1++**

Evidence indicates that it is unlikely to be a significant difference between CBT-BED and IPT-BED in depression scores at post-treatment follow-up (1 RCT, Wilfley, 2002[249]: N=138; SMD: 0.13; 95% CI:-0.20 to 0.47). **RCT 1++**

Evidence indicates that it is unlikely to be significant differences between CBT-BED and BWC in depression scores by the end of treatment (SMD:-0.31; 95% CI: -0.97 to 0.34) and at follow-up (SMD: 0.21; 95% CI: -0.45 to 0.86) (1 RCT; N=37, Nauta, 2000)[250]. **RCT 1++**

Other results

In an RCT (Gorin, 2003; USA)[251] group CBT was compared to CBT with partner involvement and wait-list, in a group of 94 adult women with BED under outpatient treatment over 12 months. Compared to the control subjects, both groups significantly reduced binge-eating and BMI, and improved psychological results such as disinhibition, depression and self-esteem. The partner's involvement in therapy did not produce significant improvement compared to normal CBT. Both group therapies have significantly reduced depression scores compared to the control group, but there are no differences between them. Average BMI decrease from baseline to follow-up was 0.11 for CBT and 0.77 for CBT-partner. This suggests that CBT alone does not produce a significant change in BMI. **RCT 1+**

In an RCT (Hilbert, 2004; Germany)[252] CBT-body exposure component was compared to CBT-cognitive interventions for image disturbance in 28 adult women with BED under outpatient treatment with at least one binge-eating episode per week for 4 months. Treatment duration was 5 months. Both groups decreased binge-eating and depression scores but there were no significant differences between them. **RCT 1+**

In a recent RCT (Schmith, 2007; UK)[238] FT was compared (N=31 BN, N=10 EDNOS) with CBT-GSH (N=30 BN, N=14 EDNOS) in adolescents between the ages of 13 and 20 years, with BN or EDNOS. Treatment was administered over 6 months with 12-month follow-up. CBT-GSH was more effective at reducing binge-eating at 6 months than FT (p=0.03) even though this difference disappeared at 12 months. There were no differences between groups in terms of BMI, diet, eating fast and other attitudinal eating disorder symptoms. The direct cost of treatment was lower for CBT-GSH and no **RCT 1++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

differences were observed in other types of costs. CBT-GSH is slightly superior to FT in reducing binge-eating more quickly, being less costly and presenting better acceptability for adolescents with BN and/or EDNOS.

An RCT (Munsch, 2007; Switzerland)[253] in 80 participants (88.85% women, age 18-70 years) diagnosed with BED compared CBT (N=44) to behavioural weight loss treatment (BWLT) (N=36) in 16 weekly sessions with 12-month follow-up. CBT was significantly more effective at reducing BMI ($p<0.001$) and in remission of binge-eating ($p=0.010$) than BWLT by the end of treatment. At 12-month follow-up, results in both treatments were comparable. CBT was more effective than BWLT in the treatment of BED. **RCT 1 +**

In an RCT (Shapiro, 2007; USA)[254] in 66 women (ages: 18-60 years) diagnosed with BED, where subclinical cases of BED (BMI>27 kg/m^2) and regular access to a computer were included, group CBT (N=22; 90-minute sessions in groups of 5 to 10 people) was compared to CBT-CD (N=22; CBT using CD-ROM for treatment of obesity and eating behaviour that affects health) and to waiting list (N=22). Treatment duration was 10 weeks.

Treatment acceptability was measured using debilitation of individuals who had been originally assigned to the waiting list group after changing from one treatment to another. The majority of patients on waiting list preferred to receive the CD-ROM than group CBT when their waiting time ended. There is a significant difference in both groups compared to the control group in reducing the number of daily binge-eating episodes. **RCT 1++**

Summary of the Evidence

SRSE[31] 1++	CBT is more effective at reducing the frequency of binge-eating episodes and increasing their remission. Both individual and group CBT, when compared to other interventions carried out in the control group, obtain better results in achieving abstinence and its maintenance over the 4 months following treatment (Hilbert, 2004[252]; Gorin, 2003[251]). CBT is not associated with weight loss.
SRSE[31] 1++	CBT also improved psychological aspects (disinhibition, etc.) (Hilbert, 2004[252]; Gorin, 2003[251]).
SRSE[31] 1++	The efficacy of CBT in reducing body weight in BED patients is not adequately documented (Hilbert, 2004[254]; Gorin, 2003[251]).

Recommendations

(See also recommendations 9.GP.14. and 9.GP.17.)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

A	9.3.3.1.	Adult patients with BED can be offered a specifically adapted form of CBT. (Adopted from recommendation 8.2.7.4. of the NICE CPG).

9.3.3.2. What is the safety of CBT in patients with BED?

The answer is based on the evidence described for CBT efficacy where studies are briefly described (question 9.3.3.1.): in the NICE CPG (2004)[30] and in two quality RCT (1++ and 1+) that were not considered in the aforementioned guide but were included in the high-quality SRSE (1++) published in the years 2006[31] and 2007[245]. The updated search has identified one more RCT. Question 9.3.3.1. briefly describes the studies.

Scientific Evidence

There is limited evidence to indicate that there are significant differences between CBT and waiting list in the number of dropouts for any reason by the end of treatment (4 RCTs; N=222;Agras, 1995[246]; Gorin, 2001[247],Telch, 1990[248],Wilfley, 1993[235]; RR:1.86; 95% CI: 1.10 to 3.15; NNT: 7; 95% CI: 4 to 34). **RCT 1+**

There is not sufficient evidence to indicate that there are significant differences between CBT and IPT in the number of dropouts for any reason by the end of treatment (2 RCTs; N=198; Wilfley, 1993[235] and 2002[249]; RR: 1.89; 95% CI: 0.89 to 4.02). **RCT 1+**

There is insufficient evidence to indicate that there are significant differences between CBT and BWC in the number of dropouts for any reason by the end of treatment (1 RCT; N=37: Nauta, 2000[250]; RR: 0.76; 95% CI: 0.18 to 3.29). **RCT 1+**

The RCT (Hilbert, 2004[252]; Gorin, 2003[251]; Peterson, 2001[255] and 1998[256]) did not report any adverse effects. The overall dropout rate in one of the RCT (Gorin, 2003)[251] was 34% and, in another RCT (Hilbert, 2004)[252], 14% in each treatment group. **RCT 1+**

In an RCT (Shapiro, 2007)[254] that compared group CBT to CBT-CDROM and wait-list, dropouts in each group were as follows: CD-ROM 32%; group CBT 41%; and wait-list 9%. The CBT-CD programme has better acceptability than group CBT in patients with BED. **RCT 1++**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12. and 9.GP.17.)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

9.4. Self-Help (SH) and Guided Self-Help (GSH)

9.4.1. Bulimia nervosa

9.4.1.1. What is the efficacy of SH and GSH in patients with BN?

The answer to this question is based on the NICE CPG (2004)[30] and on three high-quality SRSE (1++): the one elaborated by the AHRQ[31], one Cochrane review[257] and a more recent publication by Shapiro, *et al.* (2007)[211]. The updated search has not yielded any new evidence.

Variables: reduction / remission of binge-eating and purging

Evidence indicates that it is unlikely to be a clinically significant difference between SH and the control group (wait-list) in binge-eating (2 RCTs; N=139; Treasure, 1994[231]; Carter, 2003[242]; RR: 0.96; 95% CI: 0.85 to 1.09) and purging (2 RCTs; N=139;Treasure, 1994[231]; Carter, 2003[242]; RR: 0.97; 95% CI:0.87 to 1.07) remission by the end of treatment. **RCT 1++**

There is not sufficient evidence to indicate clinically significant differences between SH and the control group (wait-list) in the frequency of binge-eating episodes by the end of treatment (1 RCT; N=39; Mitchel, 2001[258]; SMD: 0.48; 95% CI: -0.16 to 1.12). **RCT 1++**

Variables: depression and/or interpersonal and psychosocial functioning and/or general psychiatric symptoms

There is not sufficient evidence to indicate clinically significant differences between SH and wait-list in depression scores by the end of treatment. (1 RCT, N=57; Carter, 2003[242]; SMD: 0.47; 95% CI: -0.06 to 1.00). **RCT 1++**

There is not sufficient evidence to indicate clinically significant differences between SH and wait-list in psychosocial interpersonal function by the end of treatment (1 RCT, N=57; Carter, 2003[242]; SMD: 0.15; 95% CI: -0.37 to 0.67). **RCT 1++**

Other results

In an RCT (Durand y King, 2003)[259] in a sample of 68 patients, GSH administered by a general practitioner was compared to clinical treatment administered by a specialist. Treatment duration was determined by the clinician. Both groups of patients reported decreased BITE and EDE scores. However, binge-eating and purging behaviours did not significantly decrease. Depression symptoms decreased in a similar fashion in both groups. No weight changes were observed in any of the groups. **RCT 1+**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

In an RCT (Carter, 2003; Canada)[242] the SH-based CBT manual specifically geared towards eating disorders was compared to SH with a self-affirmation manual (non-specific) and to wait-list in 85 adult women with BN under outpatient treatment. The individuals who provided guidance were non-specialised facilitators without clinical training. Both SH therapies significantly reduced binge-eating and purging behaviours when compared to waiting (wait-list). SH-based CBT was associated with a greater reduction of excessive exercise compared to the control group (on wait-list) and non-specific SH. **RCT 1+**

Summary of the Evidence

SRSE[31] 1++	Four RCTs provide evidence of the efficacy of SH in BN. There are no differences in efficacy between the different types of SH (Carter, 2003)[242]. There is preliminary evidence that indicates that general practitioners can carry out SH treatment. (Durand and King, 2003)[259].
SRSE[257] 1++	Due to the fact that SH and GSH treatments for BN, when compared to the control group (patients on wait-list or receiving standard treatment), produce a short-term reduction of eating disorder severity and other symptoms, these procedures seem useful as the first step of treatment. It is yet unclear if guidance is necessary, how much of it and who should provide it. Patient preference and availability of resources must be taken into account.
SRSE[257] 1++	No significant differences were found in any of the results when SH, GSH and formal psychological therapy administered by a therapist were compared, even though it is probable that these analyses do not have sufficient statistical power. However, it is suggested that SH treatments possibly be considered an alternative to treatments administered by a specialised therapist, taking both the patient's preference and availability of resources into consideration.

Recommendations

B	9.4.1.1.1.	A possible first step in BN treatment is encouraging patients to initiate a SH programme (guided or not). (Adapted from recommendation 7.2.7.1. of the NICE CPG).
B	9.4.1.1.2.	SH (guided or not) is sufficient treatment for a limited number of patients with BN. (Adapted from recommendation 7.2.7.2. of the NICE CPG).

9.4.1.2. What is the safety of SH and GSH in patients with BN?

The answer in based on the NICE CPG (2004)[30] and on high-quality SRSE (1++) published in 2006[31] and 2007[211]. The updated search has not yielded any new evidence. Studies are briefly described in question 9.4.1.1.

It has been 5 years since the publication of this Clinical Practice Guideline and is subject to updating.

Scientific Evidence

There is insufficient evidence to indicate clinically significant differences between SH and wait-list in the number of dropouts for any reason by the end of treatment (3 RCTs, N=183; Carter, 2003[242]; Mitchel, 2001[258]; Treasure, 1994[231]; RR: 0.68; 95% CI: 0.38 to 1.19). **RCT 1+**

There is insufficient evidence to indicate clinically significant differences between GSH and wait-list in the number of dropouts for any reason by the end of treatment (1 RCT; N=47;Walsh, 2004[328]; RR: 1.38; 95% CI: 0.98 to 1.96). **RCT 1+**

In an RCT (Durand and King, 2003[259]), the overall dropout rate was 21%. Dropouts per groups were: SH by family physician, 24%; specialist treatment, 18%. No adverse effects were reported. **RCT 1+**

In an RCT (Carter, 2003)[242] the dropout rate in the CBT manual group was 18%; non-specific treatment, 25%; wait-list, 28%. No adverse effects were reported. **RCT 1+**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12 to 9.GP.17)

9.4.2. Binge-Eating Disorder

9.4.2.1. What is the efficacy of SH and GSH in patients with BED?

The answer is based on the NICE CPG (2004)[30] and on three high-quality SRSE (1++): the one elaborated by the AHRQ[31], a Cochrane[257] review and a more recently published one by Brownley, *et al.*, 2007[245]. The updated search has not yielded any new evidence.

Variable: BMI

There is insufficient evidence to determine that there are clinically significant differences between SH and GSH in mean BMI by the end of treatment (2 RCTs, N=109; Loeb 2000[260]; Carter, 1998[262]; SMD: 0.08; 95% CI: -0.30 to 0.46) and at follow-up (1 RCT, N=69; Carter, 1998[262]; SMD: 0.19; 95% CI: -0.29 to 0.66). **RCT 1++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Variable: reduction / remission of binge-eating and purging

There is insufficient evidence to determine that there are clinically significant differences between SH and GSH in the remission of binge-eating by the end of treatment (1 RCT, N=40; Loeb, 2000[260]; RR: 0.71; 95% CI: 0.42 to1.21). **RCT 1++**

There is limited evidence to determine that GSH is superior to SH in reducing the frequency of binge-eating by the end of treatment (2 RCTs, N=109; Loeb, 2000[260]; Carter,1998[261]; SMD: -0.48; 95% CI: -0.86 to -0.09). **RCT 1++**

There is insufficient evidence to determine that there are clinically significant differences between SH and GSH in the frequency of binge-eating at post-treatment follow-up (1 RCT, N=69; Carter, 1998[261]; SMD: -0.24; 95% CI: -0.71 to 0.23). **RCT 1++**

Variables: depression and/or interpersonal and psychosocial functioning and/or general psychiatric symptoms

There is insufficient evidence to determine that there are clinically significant differences between SH and GSH in depression scores by the end of treatment (1 RCT, N=40; Loeb, 2000[260]; SMD: -0.22; 95% CI: -0.85 to 0.40). **RCT 1++**

There is insufficient evidence to determine that there are clinically significant differences between SH and GSH in general psychiatric symptoms by the end of treatment (2 RCTs, N=109; Loeb, 2000[260]; Carter, 1998[261]; SMD: -0.18; 95% CI: -0.55 to 0.20) and at follow-up (1 RCT, N=69; Carter, 1998[261]; SMD: -0.20; 95% CI: -0.68 to 0.27). **RCT 1++**

More results

In an RCT (Peterson, 1998; USA[256]) SH administered by a therapist (G1) was compared to partial SH (G2), structured SH (G3) and wait-list (G4) in 61 patients with BED. G1 used group discussion and the psychoeducational component; in G2, participants watched a 30-minute psychoeducational video and later engaged in a group discussion with the therapist, and in G3 participants watched the 30-minute video and discussed it amongst themselves. All treated groups obtained better results than the control group in remission and frequency of binge-eating and symptoms related with eating disorders. Abstinence proportion (complete remission) was 68% to 87% in the treated groups and 12.5% in the control group. There were no differences amongst groups in terms of depression and BMI. **RCT 1+**

In an RCT (Peterson, 2001; USA)[255] SH administered by a therapist (G1) was compared to partial SH (G2) and structured SH (G3) in 61 patients with BED. The three groups showed significant improvement in remission, binge-eating frequency and body dissatisfaction. The SH group obtained greater remission by the end of treatment, but not at follow-up. There were no differences amongst groups in terms of depression and BMI. **RCT 1+**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Summary of the Evidence

SRSE[31] 1++	SH and GSH are efficacious in reducing binge-eating remission and frequency and psychological traits associated with eating disorders. This therapy obtains similar values to those obtained in trials that use face-to-face psychotherapy in a short period of time. There are no changes in depression and BMI.
SRSE[257] 1++	Given that SH and GSH treatments for BED, when compared to controls (on wait-list or receiving standard treatment), produce a short-term reduction of eating disorder severity and other symptoms, these procedures seem useful as a first step of treatment. It remains unclear if guidance is necessary, how much of it and who should provide it. Patient preference and availability of resources should be taken into consideration.
SRSE[257] 1++	No significant differences were found in several results pertaining to SH and GSH interventions and formal psychological therapy administered by a therapist, even though it is probable that these analyses do not have sufficient statistical power. However, it is suggested that SH treatment possibly be considered an alternative to treatments administered by a specialised therapist, taking both patient preference and availability of resources into account.

Recommendations

(See recommendations 9.GP.12. to 9.GP.17.)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

9.4.2.2. What is the safety of SH and GSH in patients with BED?

The answer is based on the NICE CPG (2004)[30] and on the high-quality (1++) SRSE of the AHRQ and a more recent one conducted by Brownley, *et al*., 2007[211]. The updated search has not yielded any new evidence. Studies are briefly described in question 9.4.2.1.

Scientific Evidence

There is insufficient evidence to determine that there are significant differences between SH and GSH in the number of dropouts for any reason by the end of treatment (1 RCT, Loeb, 2000[260]; N=40; RR: 0.86; 95% CI; 0.35 to 2.10). **RCT 1++**

In an RCT (Peterson, 1998)[256], the overall dropout rate was 16%. Dropouts per groups were: G1: 13%, G2: 11%, G3: 27% and G4: 0%. No adverse effects were reported. **RCT 1++**

Another RCT (Peterson, 2001[255]), reported an overall dropout rate of 14%. No adverse effects were reported. **RCT 1+**

In yet another RCT (Carter, 1998[261]), the overall dropout rate was 12%. Dropouts per groups were: GSH: 24%, SH: 0% and wait-list: 4%. No adverse effects were reported. **RCT 1+**

Recommendations

(See recommendations 9.GP.12. to 9.GP.14.)

9.5. Interpersonal Therapy (IPT)

9.5.1. Anorexia nervosa

9.5.1.1. What is the efficacy of IPT in patients with AN?

The answer is based on a high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31]. The results of the previous SRSE are maintained in a further high-quality (1++) SRSE based on the same RCT published one year later (Bulik, *et al*., 2007)[202]. The updated search has not yielded any new RCTs.

Scientific Evidence

In an RCT (McInstosh, 2005; New Zealand)[216] CBT (N=19) was compared to IPT (N=21) and to non-specific supportive clinical management (NSCM) (N=16) in women (17-40 years) with AN and low weight who were treated on an outpatient basis over 20 weeks. NSCM was superior to IPT in improving the general functioning of patients and in dietary restraint at 20 weeks of treatment; NSCM was superior to CBT in improving general functioning at 20 weeks, and CBT was superior to IPT in improving dietary restraint at 20 weeks. **RCT 1+**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.1. to 9.GP.11.)

9.5.1.2. What is the safety of IPT in patients with AN?

The answer is based on two high-quality SRSE (1++) described in the question regarding IPT efficacy[31, 202].

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Scientific Evidence

In an RCT (McInstosh, 2005)[216], there was an overall dropout rate of 38% in the group treated with IPT. No adverse effects were reported.	**RCT 1+**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.1. to 9.GP.11.)

9.5.2. Bulimia nervosa

9.5.2.1. What is the efficacy of IPT in patients with BN?

The evidence for this question is based on the NICE CPG (2004)[30], where RCTs are described in terms of outcome variables, and on the high-quality (1++) SRSE elaborated by the AHRQ of the US (2006)[31] and a more recent high-quality (1++) one published by Shapiro, *et al*. (2007)[211]. The updated search has not yielded any new evidence.

Variable: reduction / remission of binge-eating and purging

There is strong evidence that suggests that CBT-BN treatment is more effective than IPT-BN in the remission of binge-eating (2 RCTs; N=270; Agras, 2000[227]; Fairburn, 1986[228]; RR: 0.77; 95% CI: 0.67 to 0.87; NNT: 5; 95% CI: 4 to 20) and purging by the end of treatment (1 RCT; N=220; Cooper, 1995[225]; RR: 0.76; 95% CI: 0.67 to 0.86; NNT: 5; 95% CI: 4 to 8).	**RCT 1 ++**
There is evidence that indicates that there are no significant differences between CBT-BN and IPT-BN in the remission of binge-eating at post-treatment follow-up (2 RCTs; N=270; Agras, 2000[227]; Fairburn, 1986[228]; RR: 0.93; 95% CI: 0.82 to 1.06).	**RCT 1 ++**
It is not likely that CBT-BN is superior to IPT-BN in reducing the frequency of binge-eating (2 RCTs; N=262;Agras, 2000[227]; Fairburn, 1986[228]; SMD: -0.24; 95% CI: -0.48 to 0.01) and purging (2 RCTs; N=257; Agras, 2000[227]; Fairburn, 1986[228]; SMD: -0.04; 95% CI: -0.29 to 0.20) by the end of treatment.	**RCT 1 ++**

Variables: depression and/or interpersonal and psychosocial functioning and/or general psychiatric symptoms

There is insufficient evidence to determine that there are significant differences between CBT and IPT in general psychiatric symptom scores by the end of treatment	**RCT 1 ++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

according to 2 RCTs (Agras, 2000[227]; Fairburn, 1991[224]) (SMD: 0.09; 95% CI: -0.54 to 0.7) and at follow-up according to 1 RCT (N=38; Fairburn, 1991)[224], (SMD: -0.03; 95% CI: -0.6 to 0.60).

There is insufficient evidence to determine that there are significant differences between CBT and IPT in interpersonal and psychosocial functioning by the end of treatment according to 2 RCTs (Agras, 2000[227]; Fairburn, 1991[224]; SMD: -0.26; 95% CI: -0.56 to 0.04) and at follow-up according to 1 RCT (N=39; Fairburn, 1991[224]; SMD: -0.04; 95% CI: -0.68 to 0.61). **RCT 1 ++**

More results

An RCT (Wilson, 2002; USA)[234] compared CBT to IPT in 220 adult women with BN under outpatient treatment with 6 weeks follow-up. CBT was superior to IPT in reducing dietary restraint at 6 weeks of treatment, in reducing binge-eating and purging frequency at 10 weeks of treatment, and in reducing binge-eating after treatment. CBT was more effective at achieving reduced frequency of vomiting at 6 weeks after treatment. **RCT 1 +**

In an RCT (Wilfley, 1993; USA)[235] that compared group CBT to group IPT and to wait-list in 56 adult women with BN, CBT and IPT decreased the frequency of binge-eating at one-year follow-up. Both therapies were superior to the control group (wait-list) in decreasing the frequency of binge-eating, disinhibition and limitations at 16 weeks of treatment. There were no significant differences between group therapies. **RCT 1 +**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendation

B	9.5.2.1.	IPT should be considered as an alternative to CBT even though patients should be informed it takes 8 to 12 months to obtain results comparable with CBT (Adopted from recommendation 7.2.7.6. of the NICE CPG).

9.5.2.2. What is the safety of IPT in patients with BN?

Evidence is derived from the NICE CPG (2004)[30] and high-quality SRSE (1++)[31, 211]. The updated search has not yielded any new evidence. Studies are briefly described in question 9.5.2.1.

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Scientific Evidence

There is insufficient evidence to determine if there are significant differences between CBT-BN and IPT in the number of dropouts by the end of treatment (2 RCTs; N=270; Agras, 1989[222]; Fairburn ,1991[224]; RR: 1.24; 95% CI: 0.84 to 1.83). **RCT 1 ++**

One RCT (Wilson, 2002)[234] claimed that the overall dropout rate by the end of treatment was 30% and 41% at follow-up. No adverse effects were reported **RCT 1 +**

One RCT (Wilfley, 1993; USA)[235] compared group CBT to group IPT and to wait-list in 56 adult women with BN. Overall dropout rate was 14% (CBT: 33%, IPT: 11%, and on wait-list: 0%). No adverse effects were reported. **RCT 1 ++**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12 to 9.GP.17)

9.5.3. Binge-Eating Disorder

9.5.3.1. What is the efficacy of IPT in patients with BED?

Evidence is derived from the NICE CPG (2004)[30] where outcome variables are described and from high-quality SRSE (1++) elaborated by AHRQ of the US (2006)[31] and by Brownley, *et al*. (2007)[245]. The updated search has not yielded any new evidence.

Variable: BMI

There is evidence that indicates is not likely that there is a clinically significant difference between CBT-BED and IPT-BED in body weight variation by the end of treatment (BMI when possible) (1 RCT, N=158; Wilfley, 2002[234]; SMD: 0.06; 95% CI: -0.26 to 0.37). **RCT 1 ++**

Variables: reduction / remission of binge-eating and purging

There is evidence that indicates it is not likely that there is a significant difference between CBT-BED and IPT-BED in the reduction of binge-eating frequency by the end of treatment (2 RCTs; N=194; Wilfley, 1993[235] and 2002[234]; SMD: -0.07; 95% CI: -0.35 **RCT 1 ++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

to -0.22) and at post-treatment follow-up (2 RCTs; N=138; Wilfley, 1993[235] and 2002[234]; SMD: 0.14; 95% CI: -0.19 to 0.48).

There is strong evidence that suggests there is a clinically significant difference between IPT-BED and wait-list in the remission of binge-eating (2 RCTs; N=38; Wilfley, 1993[235] and 2002[234]; RR: 0.56; 95% CI: 0.37 to 0.84; NNT: 3; 95% CI: 2 to 5) and in the reduction of binge-eating frequency by the end of treatment (2 RCTs; N=38; Wilfley, 1993[235] and 2002[234]; SMD: -1.44; 95% CI: -2.16 to -0.72). **RCT 1 ++**

Variables: depression and/or interpersonal and psychosocial functioning and/or general psychiatric symptoms

There is limited evidence that suggests a clinically significant difference between IPT-BED and wait-list in improving depression scores by the end of treatment (2 RCTs; N=38; Wilfley, 1993[235] and 2002[234]; SMD: -0.80; 95% CI:-1.46 to -0.13). **RCT 1 ++**

There is insufficient evidence to suggest that there is a clinically significant difference between IPT-BED and wait-list in improving interpersonal and psychosocial functioning by the end of treatment (2 RCTs; N=38; Wilfley, 1993[235] and 2002[234]; SMD: 0.00; 95% CI: -0.64 to -0.64). **RCT 1 ++**

There is evidence that indicates it is not likely that there is a significant difference between CBT-BED and IPT-BED in depression scores by the end of treatment (2 RCTs; N=194; Wilfley, 1993[235] and 2002[234]; SMD: 0.22; 95% CI: -0.06 to 0.50) and at post-treatment follow-up (1 RCT; N=138; Wilfley, 2002[234]; SMD:0.10; 95% CI: -0.24 to 0.43). **RCT 1 ++**

There is evidence that indicates it is not likely that there is a significant difference between CBT-BED and IPT-BED in psychosocial and interpersonal functioning by the end of treatment (2 RCTs, N=194; Wilfley, 1993[235] and 2002[234]; SMD: 0.06; 95% CI: -0.22 to 0.35). **RCT 1 ++**

There is evidence that indicates it is not likely that there is a significant difference between CBT-BED and IPT-BED in mean depression scores at post-treatment follow-up (1 RCT; Wilfley, 2002[234]; N=138; SMD: 0.13; 95% CI:-0.20 to 0.47). **RCT 1 ++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See also recommendations 9.GP.12. to 9.GP.17.)		
B	9.5.3.1.	IPT-BED can be offered to patients with persistent BED (adapted from recommendation 8.2.7.5. of the NICE CPG).

9.5.3.2. What is the safety of interpersonal therapy in patients with BED?

The evidence derives from the NICE CPG (2004)[30] and high-quality SRSE[31, 245]. The updated search has not yielded any new evidence. Studies are briefly described in question 9.5.3.1.

Scientific Evidence

There is insufficient evidence to determine that there are significant differences between CBT and IPT in the number of dropouts for any reason by the end of treatment (2 RCTs; N=198; Wilfley, 1993[235] and 2002[234]; RR: 1.89; 95% CI: 0.89 to 4.02).	**RCT 1 +**
In an RCT (Wilfley, 2002)[234], dropout rate for CBT was 20% and 16% for IPT. No adverse effects were reported.	**RCT 1 +**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12. to 9.GP.17.)

9.6. Family Therapy (FT) (systemic or unspecified)

9.6.1. Anorexia nervosa

9.6.1.1. What is the efficacy of FT (systemic) in patients with AN?

The answer is based on the NICE CPG (2004)[30] and on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and a more recently published SRSE (Bulik, *et al*., 2007)[202]. The updated search has not yielded any new evidence on FT.

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Scientific Evidence

In an RCT (Crisp, 1991; UK)[263] in adult women with AN the following treatments were compared: G1) in inpatients: individual therapy-FT-group therapy-NC-occupational therapy (N=30); G2) in outpatients: individual psychological therapy-FT-NC (N=20); G3) in outpatients: group FT-NC (N=20); G4) control group (N=20). A two-year follow-up was conducted. Individual therapy and FT in outpatients is more effective for weight restoration at one or two years of follow-up. FT in adults with AN is superior to standard treatments in increasing BMI, restoring menstruation and reducing bulimic symptoms. **RCT 1 +**

In an RCT (Dare, 2001; UK)[264] FT was compared to cognitive analytical therapy, focal supportive psychotherapy (FSP) and standard treatments in adult patients with AN (98% women) under outpatient treatment. At one-year follow-up, FT and FSP were associated with increased weight gain and a greater proportion of patients who had recovered or showed significant improvement. **RCT 1 +**

In two RCTs (Russel, 1987; UK[265]; Eisler, 1997[266]) FT was compared to individual therapy in 80 adolescent and adult females with AN under outpatient treatment. FT proved to be more effective in younger patients at earlier stages of the disease than in adults with chronic disease. **RCT 1 +**

In an RCT (Eisler, 2000; UK)[267] conjoint FT (family treated as a whole) was compared to FT in separate sessions for the parents and patients in 40 adolescent outpatients (98% females) with AN. Conjoint FT was more effective at reducing depression and obsessive behaviours, but not in weight restoration. In families where the mother's critical attitude was significant separate FT was more effective. **RCT 1 ++**

In an RCT (Geist, 2000; Canada)[268] FT was compared to group family psychoeducation in 25 adolescent females inpatients with AN. There were no differences in both groups at 16 weeks. **RCT 1 +**

In an RCT (Robin, 1994[269]; USA and 1995[270]) behavioural SFT (parents have control over the patients renutrition) was compared to ego-oriented individual therapy in 24 adolescent female inpatients and outpatients with AN. Behavioural SFT was more effective at increasing BMI and restoring menstruation. There were no differences between both therapies in terms of diet and the behaviour of patients. **RCT 1 +**

In an RCT (Lock, 2005; USA)[271] long-term FT (20 sessions over 12 months) was compared to short-term FT (10 sessions in 6 months) in adolescents (90% females) outpatients with AN. Long-term therapy presents better overall results in patients with destructured families and improves BMI in patients with severe diet-related obsessions. **RCT 1 ++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Summary of the Evidence

	(See summary of the evidence for psychological treatment)
CPG[30]	There is insufficient evidence to determine that conjoint or separate FT in children and adolescents is effective by the end of treatment and at post-treatment follow-up. (Eisler, 2000[267]; Robin, 1999[272]).
CPG[30]	There is limited evidence to determine that FT is superior to standard treatments in terms of achieving weight gain by the end of treatment and at post-treatment follow-up. (Crisp, 1991[263]; Dare, 2001[264]).
CPG[30]	In children and adolescents, there is insufficient evidence to determine that FT and body awareness therapy together are superior to FT alone (Wallin, 2000)[273].
CPG[30]	There is limited evidence to determine that individual supportive psychotherapy is superior to FT in terms of weight gain in adults with AN at one year post-treatment follow-up (Russell, 1987)[265].
CPG[30]	There is limited evidence to determine that FT is superior to supportive psychotherapy at one-year follow-up in terms of weight gain and number of recoveries when administered to patients with AN (evolution under 3 years and disorder onset age under 19 years) (Russell, 1987)[265].
CPG[30]	There is insufficient evidence to determine group education in the family is more effective than conjoint FT in terms of weight restoration in children and adolescents under inpatient treatment. (Geist, 2000)[268].
SRSE[31] 1++	FT (including family of origin) can be more effective in young people with a disease of shorter duration.
SRSE[31] 1++	There are no studies that explore FT in adults including the insertion family (spouse and children) and not only the family of origin.
SRSE[31] 1++	There is no evidence to determine that FT helps adults with AN in chronic stages.
SRSE[31]	Over time, different forms of FT produce good results in adolescents with AN.
SRSE[31] 1++	There is scarce evidence to determine that interventions involving the family are more effective in patients under the age of 15 than in older patients.

Recommendations

		(See also recommendations 9.GP.1. to 9.GP.11.)
B	9.6.1.1.1.	FT is indicated in children and adolescents with AN. (Adopted from recommendation 6.2.9.14. of the NICE guide).

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

D	9.6.1.1.2.	Family members of children with AN and siblings and family members of adolescents with AN can be included in treatment, taking part in improving communication, supporting behavioural treatment and sharing therapeutic information. (Adopted from recommendation 6.2.9.13. of the NICE guide).
D	9.6.1.1.3.	Children and adolescents with AN can be offered individual appointments with health care professionals, separate from those in which the family is involved. (Adopted from recommendation 6.2.9.15.of the NICE guide).
D	9.6.1.1.4.	The effects of AN on siblings and other family members justifies their involvement in treatment. (Adopted from recommendation 6.2.9.16. of the NICE guide).

9.6.1.2. What is the safety of FT (systemic) in patients with AN?

The answer in based on the NICE CPG (2004)[30] and on later SRSE[31, 202]. The updated search has not yielded any new evidence on the safety of FT in AN. Studies are briefly described in question 9.6.1.1.

Scientific Evidence

In an RCT (Crisp, 1991)[263], 19% of participants dropped out of treatment. Dropouts per groups were: G1: 40%; G2: 10%; G3: 15%; G4: 0%. No adverse effects were reported. **RCT 1 +**

In another RCT (Dare, 2001)[264], 36% of participants dropped out of treatment. Dropouts per groups were: FSP: 43%; FT: 27%; cognitive analytical therapy: 41%; and standard treatments: 32%). No adverse effects were reported. **RCT 1 +**

In two additional RCTs (Russell, 1987[265]; Eisler, 1997[266]), 35% of treated patients dropped out of treatment (FT, 37% and individual therapy, 33%). No adverse effects were reported. **RCT 1 +**

In a further RCT (Eisler, 2000)[267], 19% of patients dropped out of treatment. Dropouts per groups were: conjoint FT: 11% and separate FT: 10%. No adverse effects were reported. **RCT 1 ++**

In an RCT (Geist, 2000)[268], there were no dropouts. No adverse effects were reported either. **RCT 1 +**

In an RCT (Robin, 1994[269] y 1995[270]), 8% of participants dropped out of treatment in both groups. No adverse effects were reported. **RCT 1 +**

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In an RCT (Lock, 2005)[271], 20% of participants dropped out of treatment. 24% dropped out of long-term therapy and 18% dropped out of short-term therapy. Treatment dropout as a result of another psychological treatment was reported as an adverse effect. **RCT 1 ++**

Summary of the Evidence

	(See summary of the evidence for psychological treatment)
CPG[30]	In children and adolescents with AN there is insufficient evidence to determine that separate or conjoint FT has more or less acceptability. (2 RCTs: N=64; Eisler, 2000[267]; Robin, 1999[272]).
CPG[30]	In children and adolescents with AN there is insufficient evidence to determine that adding body awareness therapy to FT increases acceptability for patients more than FT alone (1 RCT; N=33;Wallin, 2000)[273].

Recommendations

(See recommendations 9.GP.1. to 9.GP.11.)

9.6.2. Bulimia nervosa

9.6.2.1. What is the efficacy of FT (systemic) in patients with BN?

There is no evidence to respond to this question in the NICE CPG (2004)[30] or in later SRSE[31, 211]. The updated search has identified two more RCTs.

Scientific Evidence

In a recent RCT (Schmith, 2007; UK)238 FT was compared (N=31 BN, N=10 EDNOS) with CBT-GSH (N=30 BN, N=14 EDNOS) in adolescents between the ages of 13 and 20 years, with BN or EDNOS. Treatment was administered over 6 months with 12-month follow-up. CBT-GSH was more effective at reducing binge-eating at 6 months than FT (p=0.03) even though this difference disappeared at 12 months. There were no differences between groups in terms of BMI, diet, eating fast and other attitudinal eating disorder symptoms. The direct cost of treatment was lower for CBT-GSH and no differences were observed in other types of costs. CBT-GSH is slightly superior to FT in reducing binge-eating more quickly, being less costly and presenting better acceptability for adolescents with BN and/or EDNOS. **RCT 1 ++**

In a recent RCT (Le Grange, et al., 2007; USA)274 FT (N=41) was compared to FSP (N=31). Patients receiving FT obtained significantly better results after treatment in terms of binge-eating and purging abstinence (p=0.049). At 6-months follow-up binge-eating and purging abstinence was statistically significant in favour of FT (12 patients, 29%) compared to SFT (4 patients, 10%; p=0.005). In the measurement of all other **RCT 1++**

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results related with eating disorders a significant difference in favour of FT (p=0.003 to p=0.03) was also reported. FT had a statistically significant clinical advantage over FSP after treatment and at 6-months follow-up. There was a reduction of BN symptoms in the group treated with FT.

9.6.2.2. What is the safety of FT (systemic) in patients with BN?

Safety of FT is not addressed in the NICE CPG (2004)[30] or in later high-quality SRSE (1++)[31, 211]. Only one RCT has been identified that provides results on this issue. Studies are briefly described in question 9.6.2.1.

Scientific Evidence

In an RCT (Schmith, 2007)[238], 28% of young people refused to participate in the study due to the family's mandatory involvement, which suggests these patients should be offered individual therapy. **RCT 1++**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12. to 9.GP.17.)

9.6.3. Binge-eating disorder

9.6.3.1. What is the efficacy of FT (systemic or unspecified) in patients with BED?

There is no evidence in the NICE CPG (2004)[30], or in later high-quality SRSE[31, 245]. The updated search has identified one new RCT.

Scientific Evidence

In an RCT conducted by Schmith, 2007[238] that compared FT (N=31 BN; N=10 EDNOS) and CBT-GSH (N=30 BN; N=14 EDNOS) in adolescents aged 13-20 years with BN or EDNOS over 6 months with a 12-month follow-up, CBT-GSH was more effective at reducing binge-eating at 6 months than FT (p=0.03) even though this difference disappeared at 12 months. There were no differences between groups in terms of BMI, diet, eating fast and other attitudinal eating disorder symptoms. The direct cost of treatment was lower for CBT-GSH and no differences were reported in other types of costs. **RCT 1++**

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Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12. to 9.GP.17.)

9.6.3.2. What is the safety of FT (systemic or unspecified) in patients with BED?

There is no evidence in the NICE CPG[30] or in later high-quality SRSE[31, 245] that address the safety of FT. The updated search has identified one RCT. Studies are briefly described in question 9.6.3.1.

In the RCT conducted by Schmith, 2007[238] 28% of young people refused to participate in the study due to the family's mandatory involvement, which suggests these patients should be offered individual therapy. **RCT 1++**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12. to 9.GP.17.)

9.7. Psychodynamic Therapy (PDT)

9.7.1. Anorexia nervosa

9.7.1.1. What is the efficacy and safety of PDT in patients with AN?

There is no evidence that supports or denies the efficacy and safety of PDT in patients with AN.

Recommendation

(See also recommendations 9.GP.1. to 9.GP.11.)

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9.7.2. Bulimia nervosa

9.7.2.1. What is the efficacy of PDT in patients with BN?

The answer to this question is based on the NICE CPG (2004)[30], where it is described in terms of several outcome variables. No evidence has been identified in later SRSE[31, 211]. The updated search has not identified any new evidence on PDT in BN.

Variables: reduction / remission of binge-eating and purging

There is insufficient evidence to determine that there are significant differences between CBT and PDT in the frequency of binge-eating (1 RCT; N=46; Garner, 1993[229]; SMD: -0.19; 95% CI: -0.77 to 0.39) and purging (1 RCT; N=50; Garner, 1993[229]; SMD: -0.56; 95% CI: -1.13 to 0.01) by the end of treatment.	**RCT 1++**

Variables: depression and/or interpersonal and psychosocial functioning and/or general psychiatric symptoms

There is no evidence or insufficient evidence to determine that CBT-BN differs from PDT in psychosocial and interpersonal functioning (1 RCT; N=41; Garner,1993[229]; SMD: -0.39; 95% CI: -1.01 to 0.23) and in general psychiatric symptoms (1 RCT; N=48; Garner, 1993[229]; SMD: -0.60; 95% CI: -1.18 to -0.02) by the end of treatment.	**RCT 1++**

Summary of the Evidence

	(See also summary of the evidence for psychological treatment)
CPG[30]	CBT does not offer any advantage over PDT.

Recommendations

(See recommendations 9.GP.12. to 9.GP.17.)

9.7.2.2. What is the safety of PDT in patients with BN?

The answer is based on the NICE CPG (2004)[30], where different outcome variables are described. No evidence has been identified in later SRSE [31, 211]. The updated search has not yielded any new evidence on PDT in BN.

Scientific Evidence

There is no significant evidence on the number of dropouts between CBT and PDT by the end of treatment (1 RCT; N=50; Garner, 1993[229]; RR: 1.00; 95% CI: 0.33 to 3.03).	**RCT 1++**

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Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12. to 9.GP.13.)

9.7.3. Binge-eating disorder

9.7.3.1. What is the efficacy and safety of PDT in patients with BED?

There is no evidence that supports or denies the efficacy and safety of PDT in patients with BED.

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendation

(See recommendations 9.GP.12. to 9.GP.17.)

9.8. Behavioural Therapy (BT)

9.8.1. Anorexia nervosa

9.8.1.1. What is the efficacy of BT in patients with AN?

The answer is based on the NICE CPG[30], where different outcome variables are described, on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and on a more recently published one (Bulik, *et al*., 2007)[202]. The updated search has not identified any new evidence on BT in AN.

Scientific Evidence

An RCT (Channon, 1989; UK)[215] compared CBT (N=8) to BT (N=8) and to FSP used as control treatment (N=8) in women with AN (mean age for CBT: 21.6 years; BT: 24.1; control: 25.8) that underwent outpatient treatment over 12 months with 6-month follow-up. At 6 months of treatment, CBT was more effective than BT in improving psychosexual functioning; however, BT was more effective than CBT in improving the **RCT 1+**

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menstrual cycle. At one year of treatment, BT was more effective at patient weight restoration.

Summary of the Evidence

	(See also summary of the evidence for psychological treatment)
SRSE[31] 1++	CBT did not prove to be more effective than BT and FSP in terms of patient weight restoration, general behaviour and attitude regarding food.

Recommendations

(See recommendations 9.GP.1. to 9.GP.11.)

9.8.1.2. What is the safety of BT in patients with AN?

The answer is based on high-quality SRSE[31, 202] (1++). The updated search has not yielded any new evidence on the safety of BT in AN. The study is briefly described in question 9.8.1.1.

Scientific Evidence

An RCT (Channon, 1989)[215] determined that 13% of patients dropped out of treatment. No adverse effects were reported. **RCT 1+**

Summary of the Evidence

	(See also summary of the evidence for psychological treatment)
SRSE[31] 1++	Behavioural psychological interventions do not usually have any harmful effects on patients.

Recommendations

(See recommendations 9.GP.1 to 9.GP.11)

9.8.2. Bulimia nervosa

9.8.2.1. What is the efficacy of BT in patients with BN?

The answer is based on the NICE CPG (2004)[30], on the high-quality SRSE (1++)

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elaborated by the AHRQ of the US (2006)[31] and on a more recently published one (Shapiro, *et al*., 2007)[211]. The updated search has not identified any new evidence on the safety of BT in BN.

Variables: reduction / remission of binge-eating and purging

It is not likely that CBT-BN is more effective than BT in reducing the frequency of binge-eating (SMD: -0.11; 95% CI: -0.45 to 0.24) and purging (SMD: 0.08; 95% CI: -0.27 to 0.42) by the end of treatment, according to 3 RCTs (N=131; Fairburn, 1991[224]; Freeman, 1988[220]; Wolf, 1992[221]). **RCT 1++**

Variables: depression and/or interpersonal and psychosocial functioning and/or general psychiatric symptoms

There is insufficient evidence to determine that CBT-BN differs from BT in terms of general psychiatric symptoms (1 RCT; N=33; Fairburn, 1991[224]; SMD: -0.09; 95% CI:-0.79 to 0.61) and interpersonal and psychosocial functioning (1 RCT; N=32; Fairburn, 1991[224]; SMD: 0.14; 95% CI: -0.56 to 0.85) at follow-up. **RCT 1++**

There is insufficient evidence to determine that CBT-BN differs from BT in the reduction of general psychiatric symptoms (SMD: -0.26; 95% CI: -0.89 to 0.37) and in interpersonal and psychosocial functioning (SMD: -0.21; 95% CI: -0.84 to 0.42) by the end of treatment according to 1 RCT (N=39; Fairburn, 1991)[224]. **RCT 1++**

There is insufficient evidence to determine that there are significant differences between BT and IPT in general psychiatric symptoms scores by the end of treatment (1 RCT; N=39; Fairburn, 1991[224]; SMD: 0.09; 95% CI: -0.54 to 0.7) and at follow-up (1 RCT; N=31; Fairburn, 1991[224]; SMD: 0.07; 95% CI: -0.65 to 0.78). **RCT 1++**

There is insufficient evidence to determine that there are significant differences between BT and IPT in interpersonal and psychosocial functioning by the end of treatment (1 RCT: N=39; Fairburn, 1991[224]; SMD: -0.06; 95% CI: -0.69 to 0.55) and at follow-up (1 RCT; N=31; Fairburn, 1991[224]; SMD: -0.19; 95% CI: -0.91 to 0.52). **RCT 1++**

In two RCTs (Fairburn, 1991; UK[224] and 1993[275]) in 75 adult women with BN under outpatient treatment, CBT was compared with BT and IPT. CBT was superior to BT at 18 months of treatment in reducing symptoms related with diet, psychopathology and body figure. CBT was more effective than IPT in reduction of vomiting. At 12-months follow-up, CBT was superior to BT in abstinence of symptoms. **RCT 1+**

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Summary of the Evidence

	(See also summary of the evidence for psychological treatment)
SRSE[31] 1++	CBT is more effective than BT alone due to the cognitive component it incorporates, which seems to be the most important aspect. (Fairburn, 1991[224] and 1993[275]).

Recommendations

(See recommendations 9.GP.12. to 9.GP.17.)

9.8.2.2. What is the safety of BT in patients with BN?

Information is based on An RCT included in high-quality SRSE[31, 211]. A brief description of this RCT is presented in the section on efficacy. No new RCTs on the safety of BT in BN have been identified.

Scientific Evidence

In 2 RCTs (Fairburn, 1991[224] and 1993[275]), 20% of treated patients dropped out of treatment. In the BT group one case of severe weight loss was reported.	**RCT 1+**

Summary of the Evidence

(See summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12. to 9.GP.17.)

9.8.3. Binge-eating disorder

9.8.3.1. What is the efficacy and safety of BT in patients with BED?

There is no evidence to support or deny the efficacy and safety of BT in patients with BED.

Summary of the Evidence

(See also summary of the evidence for psychological treatment)

Recommendations

(See recommendations 9.GP.12. to 9.GP.17.)

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SUMMARY OF THE EVIDENCE FOR PSYCHOLOGICAL THERAPY

(QUESTIONS 9.3. to 9.8.)

Anorexia nervosa

CPG[30]	There is insufficient evidence to determine that a certain psychological treatment (including CBT, IPT, SFT and BT) is more effective than another psychological treatment for adults with AN by the end of treatment and at post-treatment follow-up. (6 RCTs; N=297; Bachar, 1999[276]; Channon, 1989[215]; Crisp, 1991[263]; Dare, 2001[264]; McIntosh, 2005[216]; Treasure, 1995[277]).
CPG[30]	There is limited evidence to determine that the outcomes of a certain psychological treatment (including CBT, IPT, SFT, PDT and BT) are more effective at increasing body weight and that patients with AN should be referred to reference centres (tertiary) by the end of treatment and at post-treatment follow-up (more than 5 years) (5 RCTs; N=258; Channon, 1989[215]; Crisp, 1991[263]; Dare, 2001[264]; Hall, 1987[278]; Treasure, 1995[277]).
CPG[30]	There is insufficient evidence to determine that psychological treatment (including CBT, IPT, SFT, PDT and BT) has more or less acceptability for patients with AN when compared to standard treatments (3 RCTs; N=198; Channon, 1989[215]; Crisp, 1991[263]; Dare, 2001[264]).
CPG[30]	There is insufficient evidence to determine that psychological treatment (including CBT, IPT, SFT, PDT and BT) has more or less acceptability in adults with AN (6 RCTs; N=297; Bachar, 1999[276]; Channon, 1989[215]; Crisp, 1991[263]; Dare, 2001[264]; McIntosh, 2005[216]; Treasure, 1995[277]).
CPG[30]	There is insufficient evidence to determine that psychological treatments (CBT, SFT and PDT) on an outpatient basis for patients with AN have more or less acceptability in comparison with standard treatments (3 RCTs; N=198; Channon, 1989[215]; Crisp, 1991[263]; Dare, 2001[264]).
SRSE[31] 1++	Current evidence on the efficacy of psychological treatment for AN is weak, with the exception of evidence relating to psychological treatment for adolescents with AN, which is moderate.

Bulimia nervosa

SRSE[31] 1++	In BN, evidence is strong for behavioural interventions and poor for SH (guided or not).

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SRSE[31] 1++	The best psychological treatment of choice when individual and group CBT are not effective in patients with BN remains unknown.

Binge-eating disorder

SRSE[31] 1++	In BED, evidence is moderate for behavioural interventions and weak for SH (guided or not).

GENERAL RECOMMENDATIONS FOR PSYCHOLOGICAL THERAPY IN EATING DISORDERS

(QUESTIONS 9.3. to 9.8.)

Anorexia nervosa

D	9.GP.1.	The psychological therapies to be assessed for AN are: CBT, SFT, IPT, PDT and BT. (Adapted from recommendation 6.2.9.1. of the NICE guide).
D	9.GP.2.	In the case of patients who require special care, the selection of the psychological treatment model that will be offered is even more important. (Adopted from recommendation 6.2.9.2. of the NICE guide).
D	9.GP.3.	The objective of psychological treatment is to reduce risk, to encourage weight gain by means of a healthy diet, to reduce other symptoms related with eating disorders and to facilitate physical and psychological recovery (Adopted from recommendation 6.2.9.3. of the NICE guide).
D	9.GP.4.	Most psychological treatments for patients with AN can be performed on an outpatient basis (with physical monitoring) by professionals specialised in eating disorders. (Adopted from recommendation 6.2.9.4. of the NICE guide).
D	9.GP.5.	The duration of psychological treatment should be of at least 6 months when performed on an outpatient basis (with physical monitoring) and 12 months for inpatients (Adopted from recommendation 6.2.9.5. of the NICE guide).

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D	9.GP.6.	For patients with AN who have undergone outpatient psychological therapy but have not improved or have deteriorated, the indication of more intensive treatments (combined individual and family therapy, day or inpatient care) must be considered. (Adopted from recommendation 6.2.9.6. of the NICE guide).
D	9.GP.7.	For inpatients with AN, a treatment programme aimed at suppressing symptoms and achieving normal weight should be established. Adequate physical monitoring is important during renutrition. (Recommendation 6.2.9.8. of the NICE guide is adopted).
D	9.GP.8.	Psychological treatments must be aimed at modifying behavioural attitudes, attitudes related to weight and body shape and the fear of gaining weight. (Adopted from recommendation 6.2.9.9. of the NICE guide).
D	9.GP.9.	The use of excessively rigid behaviour modification programmes is not recommended for inpatients with AN. (Adopted from recommendation 6.2.9.10. of the NICE guide).
D	9.GP.10.	Following hospital discharge, patients with AN should be offered outpatient care that includes monitoring of normal weight restoration and psychological intervention that focuses on eating behaviour, attitudes to weight and shape and the fear of social response regarding weight gain, along with regular physical and psychological follow-up. Follow-up duration must be of at least 12 months. (Recommendations 6.2.9.11. and 6.2.9.12. of the NICE guide are adopted).
D	9.GP.11.	In children and adolescents with AN who require inpatient treatment and urgent weight restoration, age-related educational and social needs should be taken into account. (Recommendation 6.2.9.17. of the NICE guide is adopted).

Binge-Eating Disorder

A	9.GP.12.	Patients must be informed that all psychological treatments have a limited effect on body weight. (Adopted from recommendation 8.2.7.6. of the NICE guide).
B	9.GP.13.	A possible first step in the treatment of patients with BED is to encourage them to follow a SH programme (guided or not). (Adapted from recommendation 8.2.7.2. of the NICE guide).

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B	9.GP.14.	Health care professionals can consider providing BED patients with SH programmes (guided or not) that may yield positive results. However, this treatment is only effective in a limited number of patients with BED. (Adapted from recommendation 8.2.7.3. of the NICE guide).
D	9.GP.15.	If there is a lack of evidence to guide the care of patients with EDNOS or BED, health care professionals are recommended to follow the eating disorder treatment that most resembles the eating disorder the patient presents. (Adopted from recommendation 8.2.7.1. of the NICE guide).
D	9.GP.16.	When psychological treatments are performed on patients with BED, it may be necessary in some cases to treat comorbid obesity. (Adopted from recommendation 8.2.7.7. of the NICE guide).
D	9.GP.17.	Adolescents with BED must be provided with psychological treatments adapted to their developmental stage. (Adopted from recommendation 8.2.7.8. of the NICE guide).

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PHARMACOLOGICAL TREATMENT

In this section, scientific evidence on the efficacy and safety of several drugs studied for the treatment of eating disorder patients is described. Only randomised controlled trials (RCT) of sufficient quality have been included. The drugs assessed are included within the following groups: antidepressants, antipsychotics, appetite stimulants, opioid antagonists, anticonvulsants, psychostimulants and antiemetics.

Although benzodiazepines such as alprazolam and lorazepam are indicated as anti-anxiety drugs no RCTs that address their use for eating disorders have been identified. A similar situation has been encountered with lithium.

9.9. Antidepressants

Evidence relating to eating disorders has been identified for the following antidepressants, according to the group they belong to:

– Selective serotonin reuptake inhibitors (SSRIs): fluoxetine, citalopram, fluvoxamine.

– Other antidepressants: sibutramine and trazodone.

– Tricyclic antidepressants: amitriptyline, clomipramine and imipramine.

9.9.1. Anorexia nervosa

9.9.1.1. What is the efficacy of antidepressants in patients with AN?

The answer is based on the NICE CPG (2004)[30] where the RCT results are described, on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and on a more recently published one conducted by Bulik, *et al*. (2007)[202]. The update search has not yielded any new evidence.

Scientific Evidence

There is limited evidence to indicate that there are significant differences between fluoxetine and placebo in body weight restoration in inpatients treated over one year (1 RCT; N=35; Kaye, 2001[279]; RR: 0.45; 95% CI: 0.23 to 0.86)	**RCT 1++**
In an RCT (Attia, 1998; USA)[280] fluoxetine (60 mg/day) was compared to placebo in 31 female inpatients with AN (age: 16-45 years). Both groups experienced decreased clinical symptoms related with eating disorders, obsessive-compulsive disorders, depressive behaviours and pre-occupation with food and its rituals. In both groups an increase in the percentage of BMI was reported. There were no significant differences between fluoxetine and placebo in any of the results.	**RCT 1++**

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In an RCT (Kaye, 2001; USA)[279] fluoxetine was compared to placebo in 39 adult female inpatients and outpatients with AN. Patients, before being discharged, received 20mg/day of fluoxetine, which was adjusted to a maximum dose of 60mg/day at 52 weeks. The group treated with fluoxetine presented significant increases in BMI and decreases in depression, anxiety, obsessive-compulsive disorders and symptoms related with eating disorders. **RCT 1+**

In an RCT (Biederman, 1985; USA)[281] amitriptyline (175 mg/day) was compared to placebo in 25 patients and outpatients (age: 11-17 years) with AN. There were no significant differences in symptoms related with eating disorders, behaviour or weight when compared to placebo. **RCT 1+**

In an RCT (Halmi, 1986; USA)[282] amitriptyline (160 mg/day) was compared to cyproheptadine (appetite stimulant) (32 mg/day) and to placebo in 72 female inpatients (age: 13-36 years). In the group treated with cyproheptadine, daily calorie intake was significantly greater than in the placebo group. In the groups treated with amitriptyline and cyproheptadine, weight restoration increased in less days than the placebo group. Patients with nonbulimic AN obtained better results with cyproheptadine than with amitriptyline or placebo. **RCT 1+**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Summary of the Evidence

SRSE[31] 1++	The literature that exists on pharmacological treatment of AN is scarce and inconclusive. Most RCTs are based on small samples and very few of them have statistical value to support their conclusions. Many studies include patients who are receiving other treatment, whether it is psychological or pharmacological. As far as cases studied, they include inpatients that later receive outpatient treatment, which prevents generalisation of results.
CPG[30]	There is evidence that indicates significant differences in weight increase by the end of multimode treatment with antidepressants when compared to placebo (4 RCT; N=146;Attia, 1998[280] [fluoxetine]; Biederman, 1985[281] [amitriptyline]; Halmi, 1986[282] [amitriptyline]; Lacey, 1980[283] [clomipramine]).
SRSE[31] 1++	There is insufficient evidence to determine that there are significant differences between antidepressants (citalopram) and wait-list in weight gain by the end of outpatient treatment (1 RCT; N=26; Fassino, 2002)[284].
SRSE[31] 1++	Tricyclic antidepressants can be associated with improvement of disorders secondary to eating disorders. However, these results are no associated with weight increase.

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

9.9.1.2. What is the safety of antidepressants in patients with AN?

The answer is based on the NICE guide (2004)[30], which describes RCT results based on variables of interest, the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and on a more recently published one conducted by Bulik, *et al*. (2007)[202]. The updated search has not identified any new evidence. The studies are briefly described in question 9.9.1.1.

Scientific Evidence

There is insufficient evidence to determine that there are significant differences between antidepressants and placebo that lead AN patients to drop out of treatment for any reason, by the end of treatment (2 RCTs; N=47;Attia, 1998[280] [fluoxetine]; Lacey, 1980[283] [clomipramine]; RR: 1.26; 95% CI: 0.44 to 3.56). **RCT 1++**

In an RCT (Attia, 1998)[280], one case of insomnia and agitation and one of blurred vision were reported in the fluoxetine group. 3% of participants dropped out of treatment (in total). **RCT 1++**

In another RCT (Kaye, 2001)[279] no adverse effects were reported. Dropouts: 47% in the fluoxetine group and 85% in the placebo group. **RCT 1++**

In an RCT (Biederman, 1985)[281], the amitriptyline group presented diaphoresis (2 cases), drowsiness (6 cases), dry mouth (4 cases), blurred vision (1 case), urinary retention (1 case), hypotension (2 cases) and leukopenia (1 case). Dry mouth (2 cases), palpitations (1 case) and dizziness (2 cases) were reported in the placebo group. There were no dropouts. **RCT 1+**

In an RCT (Halmi, 1986)[282] the amitriptyline group presented drowsiness, excitement, confusion, increased motor activity, tachycardia, dry mouth and constipation. The cyproheptadine group did not yield conclusive results. In the placebo group: excitement increased motor activity and drowsiness. Dropouts: 30% amitriptyline, 25% cyproheptadina and 20% placebo. **RCT 1+**

Summary of the Evidence

CPG[30]	There is insufficient evidence to determine that there are significant differences between antidepressants (citalopram) and placebo that cause AN patients to dropout of outpatient treatment due to adverse effects by the end of treatment (1 RCT; N=26; Fassino, 2002)[284].
CPG[30]	The dropout of patients who receive pharmacological treatment is important, especially in RCTs performed on outpatient cases.

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

SRSE[31] 1++	Due to the small-sized samples of both RCTs (Kaye, 2001[279] and Attia, 1998[280]) that use fluoxetine, no conclusions can be drawn regarding whether harmful effects associated with treatment in low-weight individuals differ from the harmful effects in normal weight individuals or those with psychiatric disorders.
SRSE[31] 1++	In an RCT (Kaye, 2001)[279], no adverse effects were reported in the fluoxetine group. In another RCT (Attia, 1998)[280], one case of insomnia and agitation and one case of blurred vision were reported in the fluoxetine group. In regard to tricyclic antidepressants, another RCT (Halmi, 1986)[282] with amitriptyline reported sporadic cases of associated drowsiness, excitement, confusion, increased motor activity, tachycardia, dry mouth and constipation; however, the proportion of adverse effects was similar between the experimental group and the control group (placebo).

Recommendations

(See recommendations 9.GPH.1 to 9.GPH.6)

9.9.2. Bulimia nervosa

9.9.2.1. What is the efficacy of antidepressants in patients with BN?

The answer is based on the NICE CPG (2004)[30], which describes the RCT results based on variables of interest, on the SRSE of sufficient quality (1++) elaborated by the AHRQ of the US (2006)[31] and on a more recently published one by Shapiro, *et al*. (2007)[211]. The updated search has not identified any new evidence.

Variable: BMI

There is not sufficient evidence to indicate clinically significant differences between SSRI antidepressants (fluoxetine) and placebo in relation to body weight by the end of treatment (1 RCT; N=46; Kanerva, 1994[285]; SMD: -0.30; 95% CI: -0.88 to 0.28).

Variables: reduction / remission of binge-eating and purging

There is limited evidence to determine that antidepressant treatment (fluoxetine, desipramine, phenelzine [withdrawn from the Spanish market], trazodone, bupropion) is superior to placebo in the remission of binge-eating and purging by the end of treatment (6 RCTs; N=697; McCann, 1990262; Walsh, 1991286; Goldstein, 1995287; Walsh, 1987288; Horne, 1988289; Pope, 1989290; RR: 0.88; 95% CI: 0.83 to 0.94; NNT: 9; 95% CI: 6 to 15).	**RCT 1++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

There is strong evidence that antidepressant treatment (fluoxetine, desipramine, phenelzine [withdrawn from the Spanish market], trazodone, bupropion) is superior to placebo in clinical improvement (defined as reducing binge-eating by at least 50%) (6 RCTs; N=855; Mc Cann, 1990[262]; Walsh, 1991[286]; Goldstein, 1995[287]; Walsh, 1987[288]; Horne, 1988[289]; Pope, 1989[290]; RR: 0.68; 95% CI:0.60 to 0.78; NNT: 5; 95% CI: 4 to 8). **RCT 1++**

There is insufficient evidence to determine if there is a clinically significant difference between antidepressants (fluoxetine, desipramine, phenelzine [withdrawn from the Spanish market], trazodone, bupropion) and placebo in reducing the frequency of binge-eating (6 RCTs; N=290; McCann, 1990[262]; Walsh, 1991[286]; Walsh, 1987[288]; Pope, 1989[290]; Mitchell, 2001[258]; Carruba, 2001[291]; random effects model SMD: -0.33; 95% CI: -1.13 to 0.47) and purging by the end of treatment (3 RCTs; N=198; Walsh, 1991[286]; Pope 1989[290]; Mitchell, 2001[258]; SMD: -0.19; 95% CI: -0.66 to 0.28). **RCT 1++**

There is limited evidence to determine that MAOI treatment (phenelzine, [withdrawn from the Spanish market]) *is* superior to placebo in the remission of binge-eating and purging by the end of treatment (1 RCT; N=62; Walsh, 1987[288]; RR: 0.77; 95% CI: 0.62 to 0.95; NNT: 5; 95% CI: 3 to 17). **RCT 1++**

There is insufficient evidence to determine if there is a clinically significant difference between SSRI antidepressants (fluoxetine) and placebo in reducing the frequency of binge-eating (SMD: -0.30; 95% CI: -0.91 to 0.31) and purging (SMD: -0.56; 95% CI: -1.17 to 0.06) by the end of treatment according to 1 RCT (N=43; Kanerva, 2001)[285]. **RCT 1++**

There is insufficient evidence to determine if there is a clinically significant difference between tricyclic antidepressants (desipramine) and placebo in reducing the frequency of purging (1 RCT; N=78; Walsh, 1991[286]; SMD: -0.34; 95% CI: -0.79 to 0.11) by the end of treatment. **RCT 1++**

There is strong evidence to determine that SSRI antidepressant treatment (fluoxetine) is superior to placebo in reducing binge-eating (3 RCTs; N= 706; Goldstein, 1995[287]; Kanerva, 1994[285], Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992[292]; RR: 0.73; 95% CI: 0.62 to 0.84; NNT: 6; 95% CI: 5 to 12) and purging by at least 50% by the end of treatment (2 RCTs; N=656; Goldstein, 1995[287], Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992[292]; RR: 0.66; 95% CI: 0.57 to 0.76; NNT: 5; 95% CI: 4 to 7). **RCT 1++**

There is strong evidence to determine that tricyclic antidepressant treatment (imipramine) is superior to placebo in terms of clinical improvement (defined as reducing binge-eating by at least 50%) by the end of treatment (1 RCT; N=22; Pope, 1983[293]; RR: 0.30; 95% CI: 0.11 to 0.80; NNT: 2; 95% CI: 2 to 4). **RCT 1++**

There is strong evidence to determine that tricyclic antidepressants (imipramine, desipramine) *vs.* placebo decrease the frequency of binge-eating by the end of treatment (3 RCTs; N=120; Pope, 1983[293]; McCann, 1990[262]; Walsh, 1991[286]; SMD:-0.82; 95% CI: -1.20 to -0.45). **RCT 1++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Variables: depression and/or interpersonal and psychosocial functioning and/or general psychiatric symptoms

There is limited evidence to determine that antidepressant treatment (fluoxetine, desipramine, phenelzine [withdrawn from the Spanish market], trazodone, bupropion) *vs.* placebo is superior at improving depression scores by the end of treatment (6 RCTs; N=293; McCann, 1990[262]; Walsh, 1991[286], Goldstein, 1995[287]; Walsh, 1987[288]; Horne, 1988[289]; Pope, 1989[290]; SMD: -0.28; 95% CI: -0.51 to -0.05). **RCT 1++**

There is insufficient evidence to determine that there are clinically significant differences between SSRI antidepressants (fluoxetine) and placebo in depression scores by the end of treatment (1 RCT; N=46; Kanerva, 2001[285]; SMD: -0.44; 95% CI: -1.03 to 0.14). **RCT 1++**

There is limited evidence to determine that tricyclic antidepressant treatment (imipramine, desipramine) is superior to placebo at improving depression scores by the end of treatment (3 RCT; N=120; Pope, 1983[293]; McCann, 1990[262]; Walsh, 1991[286]; SMD: -0.47; 95% CI: -0.83 to -0.10). **RCT 1++**

Other results

In an RCT (Beumont, 1997;Australia)[294] fluoxetine was compared with placebo in 72 adult female outpatients with BN. The fluoxetine group obtained better results in the reduction of binge-eating and vomiting, weight restoration, pre-occupation with food, EDI's BN scores, obsession regarding slimness and body dissatisfaction. There were no differences in depression scores. **RCT 1+**

In an RCT (Fichter, 1991; Germany)[295] fluoxetine was compared with placebo in 39 adult female inpatients with BN. There were no differences in any of the outcomes measured. **RCT 1++**

In an RCT (Romano, 2002; USA)[296] fluoxetine was compared with placebo in 150 patients (98% women) with BN under outpatient treatment. The fluoxetine group obtained better results than the placebo group in the reduction of vomiting, binge-eating, disordered behaviour rituals, pre-occupation and other severe symptoms. **RCT 1+**

In two RCTs (Fichter, 1996; Germany[297]; Fichter, 1997[298]) fluvoxamine was compared with placebo in 72 adult female outpatients with BN. The fluvoxamine group obtained better results in the reduction of vomiting, purging and binge-eating frequencies when compared with the placebo group. Both groups gained weight, with no significant differences between them. **RCT 1++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Summary of the Evidence

SRSE[31] 1++	The fluoxetine dose (60 mg/day) administered over 6 to 18 weeks has managed, in all moderate (1+) and high quality (1++) RCT, to reduce binge-eating and purging as well as psychological symptoms related with eating disorders in a short period of time.
SRSE[31] 1++	Fluoxetine at 60mg/day obtains better results than a 20mg/day dose (Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992)[292]; this dose is also associated with relapse prevention in a study with a considerable amount of discontinuation.
SRSE[31] 1++	There is considerable evidence of the short-term effect of fluoxetine treatment (60mg/day) in patients with BN. There is no evidence of the effectiveness of long-term treatment or the optimal duration of treatment.
SRSE[31] 1++	Some studies have reported remissions in a small number of patients treated exclusively with pharmacological treatment, which leads us to borne in mind that although symptoms improve, they persist in time and do not disappear.
SRSE[31] 1++	In an RCT (Walsh, 1991)[286] it was reported that patients with greater body mass and weight and longer disease length responded better to desipramine treatment.
SRSE[31] 1++	In the study Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992[292], patients with greater weight responded better in all treatment groups.

Recommendations

(See also recommendations 9.GPH.1. to 9.GPH.6.)		
B	9.9.2.1.1.	Patients should be informed that antidepressant treatment can reduce the frequency of binge-eating and purging episodes but effects are not immediate. (Adopted from recommendation 7.3.6.2. of the NICE guide).
B	9.9.2.1.2.	In the treatment of BN pharmacological treatments other than antidepressants are not recommended. (Adopted from recommendation 7.3.6.4. of the NICE guide).
D	9.9.2.1.3.	The dose of fluoxetine used in patients with BN is greater than the dose used for treating depression (60 mg/day). (Adopted from recommendation 7.7.6.5. of the NICE guide).

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

D	9.9.2.1.4.	Amongst SSRI antidepressants, fluoxetine is the first-choice drug for treatment of BN, in terms of acceptability, tolerability and symptom reduction. (Adopted from recommendation 7.3.6.3. of the NICE guide).

9.9.2.2. What is the safety of antidepressants in patients with BN?

The answer is based on the NICE CPG (2004)[30], which describes the results obtained from RCTs, on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and on a more recently published one by Shapiro, *et al*. (2007)[211]. The updated search has not identified any new evidence. The studies are briefly described in question 9.9.2.1.

Scientific Evidence

There is limited evidence to suggest that antidepressant treatment (fluoxetine, desipramine, imipramine, phenelzine [withdrawn from the Spanish market], trazodone, mianserine, moclobemide) is superior to placebo in treatment discontinuation due to adverse effects (9 RCTs; N=1.078; Pope, 1983[293], 1989[290]; Mitchell, 1990[243]; Walsh, 1991[286]; 1987[288]; Kanerva, 1994[285]; Goldstein, 1995[287]; Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992[292]; Carruba, 2001[291]; RR: 1.90; 95% CI: 1.20 to 2.99; NNH: 20; 95% CI: 13 to 50). **RCT 1++**

There is insufficient evidence to suggest which types of antidepressants (fluoxetine, desipramine, imipramine, phenelzine [withdrawn from the Spanish market], trazodone, mianserine, moclobemide, brupopion) are well or poorly tolerated by patients with BN (13 RCTs; Pope, 1983[293], 1989[290]; Mitchell, 1990[243], 2001[258]; Walsh, 1991[286], 1987[288], 2004[225]; Kanerva, 1994[285]; Goldstein, 1995[287]; Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992[292]; Carruba, 2001[292]; McCann, 1990[262]; Sabine, 1983[299]; Horne, 1988[289]; N=1.336; random effects model RR: 0.91; 95% CI:0.72 to 1.16). **RCT 1++**

There is limited evidence suggesting that treatment with antidepressants (fluoxetine, desipramine) *vs*. placebo favours treatment acceptability (5 RCTs Mitchell, 2001[258]; Walsh, 1991[286] 2004[233]; Kanerva, 1994[285]; Goldstein, 1995[287], Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992[292]; N=803; RR:0.79; 95% CI: 0.67 to 0.95). **RCT 1++**

There is insufficient evidence suggesting that there are significant differences between SSRI antidepressants (fluoxetine) and placebo in the number of patients who discontinue treatment due to adverse effects (3 RCTs Kanerva, 1994[285]; Goldstein, 1995[287], Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992[292]; N=706; RR: 1.59; 95% CI: 0.88 to 2.88). **RCT 1++**

There is strong evidence to determine that the dropout rate is greater in the placebo group than in the group receiving tricyclic antidepressants (imipramine, desipramine) (4 RCTs; N=217; Pope, 1983[293], Mitchell, 1990[243]; Walsh, 1991[286]; McCann, 1990[262]; RR: 2.03; 95% CI: 1.18 to 3.49; NNT: 7; 95% CI: 4 to 20). **RCT 1++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

There is insufficient evidence suggesting that there are significant differences between tricyclic antidepressants (imipramine, desipramine) and placebo in the number of patients who discontinue treatment due to adverse effects (3 RCTs; N=187; Pope, 1983[293]; Mitchell, 1990[243]; Walsh, 1991[286]; RR: 3.17; 95% CI: 0.84 to 11.90). **RCT 1++**

There is insufficient evidence to determine that there is a significant difference between MAOI antidepressants (phenelzide [withdrawn from the Spanish market] and moclobemide) and placebo in the number of patients who discontinue treatment due to adverse effects (random effects model RR: 2.55; 95% CI: 0.20 to 33.22) or any other reason (random effects model RR: 0.94; 95% CI: 0.50 to 1.77; based on 2 RCTs; N=139; Walsh, 1987[288]; Carruba, 2001[291]). **RCT 1++**

In an RCT (Beumont, 1997)[294] fluoxetine was compared with placebo. Adverse effects in the fluoxetine group included: insomnia, nausea and instability, the latter being significantly more common. Depression was the most common adverse effect in the placebo group. No dropouts were reported. **RCT 1+**

In an RCT (Fichter, 1991)[295], that compared fluoxetine *vs*. placebo, the experimental group showed greater tremor (significant differences). No dropouts were reported. **RCT 1+**

In an RCT (Romano, 2002)[296] fluoxetine was compared with placebo. This study explored the efficacy of fluoxetine (60mg/day) in the number of relapses over 52 weeks. Relapses were less frequent in the placebo group (33% *vs.* 51% in the fluoxetine group). Treatment discontinuation occurred more frequently within the first 3 months of the 52 weeks of complete treatment. The same study reported a dropout rate of 83% in the fluoxetine group and 92% in the placebo group. Rhinitis was reported to be the adverse effect. (fluoxetine, 24 cases; placebo, 12 cases; p<0.04). **RCT 1+**

Fichter, 1996[297] and Fichter, 1997[298] compared fluvoxamine *vs*. placebo. The fluvoxamine group was associated with less relapses. However, dropouts were greater in this group than in the placebo group (51% fluvoxamine *vs.* 14% placebo). There were 8 dropouts in the fluvoxamine group due to adverse effects. (The most significant were: nausea, drowsiness and instability.) **RCT 1++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Summary of the Evidence

SRSE[31] 1++	The adverse effects produced by second-generation antidepressants (MAOIs) in patients with BN are similar to those that occur in other disorders (Kennedy, 1993)[300].

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

9.9.3. Binge-Eating Disorder

9.9.3.1. What is the efficacy of antidepressants in patients with BED?

The answer is based on the NICE CPG[30], which describes RCT results based on variables of interest, on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and on a more recently published one conducted by Brownley, *et al*. (2007)[245]. The updated search has not identified any new evidence.

Variable: BMI

There is evidence suggesting that there are no clinically significant differences between antidepressants (fluoxetine, imipramine, sertraline) and placebo in terms of body weight (BMI when possible) by the end of treatment (3 RCTs; N=95; Arnold,2002[301]; McElroy, 2000[302]; Laederach, 1999[303]; SMD: 0.00; 95% CI: -0.42 to 0.42). **RCT 1++**

Variable: reduction / remission of binge-eating and purging

There is limited evidence to determine that there are clinically significant differences between antidepressants (fluoxetine, fluvoxamine, sertraline, imipramine and citalopram) and placebo in favour of antidepressants in terms of binge-eating remission (4 RCTs; N=217; Arnold, 2002[301]; McElroy, 2000[302]; 2003[304]; Hudson, 1998[305]; RR: 0.75; 95% CI: 0.62 to 0.90; NNT: 5; 95% CI: 4 to 13) and reduction of binge-eating frequency by the end of treatment (4 RCTs; N=122; Arnold, 2002[301]; McElroy, 2000[302], 2003[304]; Laederach, 1999[303]; SMD: -0.59; 95% CI: -0.96 to -0.22). **RCT 1++**

There is limited evidence to suggest that there are clinically significant differences between tricyclic antidepressants (imipramine) and placebo in favour of tricyclic antidepressants in terms of reducing the frequency of binge-eating by the end of treatment (1 RCT; N=29; Laederach, 1999[303]; SMD: -0.77; 95% CI: -1.53 to -0.01). **RCT 1++**

Variable: depression

There is insufficient evidence to determine that tricyclic antidepressants (imipramine) are superior to placebo in terms of reducing depression scores (1 RCT; N=29; Laederach, 1999[303]; SMD: -0.73; 95% CI: -1.49 to 0.02). **RCT 1++**

Other results

An RCT (Pearlstein, 2003; USA)[306] compared fluvoxamine (average dose 239 mg/day) *vs.* placebo over 12 weeks in 20 adult outpatients (88% women) with BED. There were no differences between both groups in terms of frequency of binge-eating, but both groups showed a decrease in frequency of binge-eating, loss of body weight and size and reduced self-reported depression. **RCT 1++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

An RCT (Appolinario, 2003; Brazil)[307] compared sibutramine *vs.* placebo in outpatients with BED (88% women; mean age 35.2 to 36.6). Sibutramine is associated with decreased depressive behaviour and is superior to placebo in reducing the frequency and severity of binge-eating episodes. In the placebo group weight gain was observed during and by the end of treatment. In the group treated with the drug, weight loss by the end of treatment was reported. **RCT 1++**

Summary of the Evidence

SRSE[31] 1++	In short-term RCTs with SSRI antidepressants, the group treated with these drugs presents better results in the reduction of symptoms related with eating disorders, in behaviour, weight and severity of the disease. The clinical impact of these conclusions cannot be judged due to the lack of data on total BED remission and on follow-up.
SRSE[31] 1++	Low doses of imipramine as a complementary strategy to nutritional counselling and psychological therapy are associated with reduced binge-eating and weight loss that is sustained after drug discontinuation.

Recommendations

(See also recommendation 9.GPH.6.)		
B	9.9.3.1.1.	SSRI antidepressant treatment can be offered to a patient with BED, regardless of whether he/she follows a guided SH programme or not. (Adopted from recommendation 8.3.5.1. of the NICE guide).
B	9.9.3.1.2.	Patients must be informed that SSRI antidepressant treatment can reduce the frequency of binge-eating, but the duration of long-term effects is unknown. Antidepressant treatment may be beneficial for a small number of patients. (Adopted from recommendation 8.3.5.2. of the NICE guide).

9.9.3.2. What is the safety of antidepressants in patients with BED?

The answer is based on the NICE CPG (2004)[30], which describes the results of RCTs, on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and a more recently published one carried out by Brownley, *et al.* (2007)[245]. The updated search has not identified any new evidence. Studies are briefly described in question 9.9.3.1.

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Scientific Evidence

There is insufficient evidence to determine if there is a clinically significant difference between antidepressants (fluoxetine, fluvoxamine, sertraline, imipramine and citalopram) and placebo in treatment discontinuation for any reason (5 RCTs; N=248; Arnold, 2002[301]; McElroy, 2000[302], 2003[304]; Hudson, 1998[305]; Laederach, 1999[303]; random effects model RR: 1.02; 95% CI: 0.43 to 2.42). **RCT 1++**

There is insufficient evidence to determine if there is a clinically significant difference between antidepressants (fluoxetine, fluvoxamine, sertraline) *vs.* placebo in treatment discontinuation due to adverse effects (3 RCTs; N=176; Arnold, 2002[301]; McElroy, 2000[302]; Hudson, 1998[305]; RR: 2.48; 95% CI: 0.74 to 8.39). **RCT 1++**

There is insufficient evidence to determine if there is a clinically significant difference between antidepressants (fluoxetine, fluvoxamine, sertraline and citalopram) and placebo in treatment discontinuation for any reason (4 RCTs; N=217; Arnold, 2002[301]; McElroy, 2000[302], 2003[304]; Hudson, 1998[305]; random effects model RR: 1.02; 95% CI: 0.39 to 2.68) or as a result of adverse effects by the end of treatment (3 RCTs; N=183; Arnold, 2002[301]; McElroy, 2003[304]; Hudson, 1998[305]; RR: 3.03; 95% CI: 0.84 to 10.86). **RCT 1++**

There is insufficient evidence to determine if there is a clinically significant difference between tricyclic antidepressants (imipramine) and placebo in treatment discontinuation due to adverse effects or any other reason (1 RCT; N=31; Laederach, 1999[303]; RR: 1.07; 95% CI: 0.07 to 15.57). **RCT 1++**

In an RCT (Pearlstein, 2003)[306] that compared fluovoxamine with placebo, 20% of participants dropped out of treatment. **RCT 1++**

In an RCT (Appolinario, 2003)[307], 23% of participants in the sibutramine group and 17% of participants in the placebo group dropped out of treatment. The adverse effects of sibutramine were: dry mouth (22 cases); headache (6); constipation (7). In the placebo group: dry mouth (3); headache (14); constipation (0) ($p<0.01$). Other adverse effects do not differ significantly between groups (nausea, insomnia, sweating, lower back pain, depressive behaviour, distress, etc). **RCT 1++**

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

9.10. Antipsychotics

Evidence in AN has been identified for the following Antipsychotics:
sulpiride, pimozide and olanzapine.

9.10.1. Anorexia nervosa

9.10.1.1. What is the efficacy of Antipsychotics in patients with AN?

The answer is based on the NICE CPG (2004)[30] and on a high-quality SRSE (1++) (Dunican,2007)[308] . The updated search has not yielded any new evidence.

Scientific Evidence

There is insufficient evidence to determine that ant psychotics (pimozide and sulpiride) have an impact on body weight *vs*. placebo during multimode treatment of inpatients with AN (2 crossed studies; N=32; pimozide [Vandereycken, 1982][309] and sulpiride [Vandereycken, 1984][310]).	**RCT 1++**
There is insufficient evidence to determine that there is any significant difference between antipsychotics and antidepressants in terms of weight gain in cases of AN.	**RCT 1++**

Summary of the Evidence

SRSE[308] 1++	In some of the case series and in most of the RCTs, patients with AN gained weight while receiving treatment with olanzapine, achieving healthy weight in many cases. Reported cases and RCTs identified additional benefits of olanzapine, including the reduction of delusional beliefs, improvement of body self-image, decrease of agitation and anxiety prior to meals, improvement of sleep, depressive symptoms and treatment adherence and other symptoms related with eating disorders.
SRSE[308] 1++	Preliminary evidence supports the use of olanzapine for the treatment of AN, demonstrating that a 2.5 to 15 daily mg dose promotes weight gain and has positive effects on related psychological symptoms. The limitations of the data analysed are: small sample, high dropout rate in the RCT and the open design of the RCT.

Summary of the Evidence

(See also recommendations 9.GPH.1. to 9.GPH.6.)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

9.10.1.2. What is the safety of Antipsychotics in patients with AN?

The answer is based on the NICE CPG (2004)[30]. The updated search has not yielded any new evidence.

Scientific Evidence

There is insufficient evidence, stemming from 2 small studies, to determine if there is a clinically significant difference between antipsychotics (pimozide and sulpiride) and placebo in terms of dropout rate due to adverse effects or any other reason by the end of treatment (2 crossed studies; N=32; pimozide [Vandereycken, 1982][309] and sulpiride [Vandereycken, 1984][310]).	**RCT 1++**

Recommendations

(See recommendations 9.GF.1.to 9.GPH.6.)

9.11. Appetite stimulants (orexigens)

Of the group of appetite stimulants, evidence of cyproheptadine in AN has been identified. No evidence has been identified on the use of pizotifen (also an appetite stimulant) in AN.

9.11.1. What is the efficacy of cyproheptadine in patients with AN?

The answer is based on en la NICE CPG (2004)[30]. The updated search has not yielded any new evidence.

Scientific Evidence

There is insufficient evidence to determine that Antipsychotics or antihistamines (cyproheptadine) have any impact on body weight when compared to placebo in patients with AN who are under inpatient management and under multimode treatment when compared to placebo (2 RCTs; N=177; Goldberg, 1980[311]; Halmi, 1986[282]).	**RCT 1++**

Recommendations

(See recommendations 9.GPH.1 to 9.GF.6)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

9.11.2. What is the safety of cyproheptadine in patients with AN?

The answer is based on the NICE CPG (2004)[30]. The updated search has not yielded any new evidence.

Scientific Evidence

There is insufficient evidence to determine that antidepressants, antihistamines or antipsychotics are more or less acceptable to patients with AN when compared to placebo or wait-list.	**RCT 1++**
There is insufficient evidence to determine that antidepressants, antihistamines or antipsychotics produce more or less adverse effects in patients with AN when compared with placebo or wait-list.	**RCT 1++**

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

9.12. Opioid antagonists

Evidence has been found for the following opioid antagonist in the treatment of BN: naltrexone. No evidence on the use of naloxone (also an opioid antagonist) in BNT/BED treatment has been found.

9.12.1. Bulimia nervosa

9.12.1.1. What is the efficacy of naltrexone in patients with BN?

The answer is based on the NICE CPG (2004)[30]. The updated search has not yielded any new evidence.

Scientific Evidence

There is insufficient evidence to determine that opioid antagonists (naltrexone) are more or less effective when compared to placebo in women with mean age 25 years diagnosed with BN (2 RCT; N=28; Huseman, 1990[312]; Mitchell, 1989[313]).	**RCT 1++**

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

9.12.1.2. What is the safety of opioid antagonists in patients with BN?

The answer is based on the NICE CPG (2004)[30]. The updated search has not yielded any new evidence.

Scientific Evidence

There is insufficient evidence to determine that opioid antagonists (naltrexone) are more or less acceptable to patients when compared to placebo in women with mean age 25 years who have been diagnosed with BN (2 RCTs; N=28; Huseman, 1990[312]; Mitchell, 1989[313]).	**RCT 1++**

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

9.13. Topiramate

9.13.1. Bulimia nervosa

9.13.1.1. What is the efficacy of topiramate in patients with BN?

Evidence is based on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and also on a more recently published high-quality SRSE (1++) (Shapiro, *et al*., 2007)[211]. The updated search has not identified any new evidence.

Scientific Evidence

2 RCTs (Hoopes, 2003; USA[314]; Hedges, 2003; USA[315]) compared topiramate *vs.* placebo in 68 adult patients (97% women) with BN who were being treated on an outpatient basis. The proportion of abstinence from binge-eating and purging was 22.6% in the topiramate group and 6% in the placebo group, a non-significant difference. The topiramate group was associated with significant reduction of anxiety but not depression, and with a greater tendency towards weight loss. The control group had a tendency towards weight gain.	**RCT 1+**

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

9.13.1.2. What is the safety of topiramate in patients with BN?

Evidence is based on two RCTs included in the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and also on a more recently published high-quality SRSE (1++) conducted by Shapiro, *et al*. (2007)[211]. The updated search has not identified any new evidence. The studies are briefly described in question 9.13.1.1.

Scientific Evidence

In the two previous RCTs (Hoopes, 2003[314]; Hedges, 2003[315]) one case of adverse effect was reported in the form of facial rash and irritability in the topiramate group. The overall dropout rate was higher (47%) in the placebo group than in the topiramate group (34%).	**RCT 1+**

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

9.13.2. Binge-Eating Disorder

9.13.2.1. What is the efficacy of topiramate in patients with BED?

The answer is based on the NICE CPG (2004)[30], on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and on a more recently published high-quality SRSE (1++) conducted by Brownley, *et al*. (2007)[245]. The updated search has no identified any new evidence.

Scientific Evidence

In an RCT (McElroy, 2003; USA)[304] topiramate (average dose 212 mg/day) was compared to placebo in 61 patients with eating disorders over 14 weeks on an outpatient basis. Patients who received topiramate presented remission of binge-eating and significant reduction of its frequency. There were significant differences in weight loss between the group treated with topiramate (5.9 kg) and the placebo group (1.2 kg). There were no differences between groups in terms of depression scores [31].	**RCT 1+**
There is limited evidence to suggest that there may be a clinically significant difference between topiramate and placebo in the remission of binge-eating by the end of treatment (1 RCT; N=61; McElroy, 2003[304]; RR: 0.56; 95% CI: 0.34 to 0.92; NNT: 4; 95% CI: 2 to 15).	**RCT 1++**

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

9.13.2.2. What is the safety of topiramate in patients with BED?

The answer is based on the NICE CPG[30], where the results of RCTs are described based on variables of interest, on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and on a more recently published high-quality SRSE (1++) conducted by Brownley, *et al*. (2007)[245]. The updated search has no identified any new evidence. The studies are briefly described in question 9.13.2.1.

Scientific Evidence

There is insufficient evidence to determine that there are clinically significant differences between topiramate (anticonvulsant) and placebo in the number of patients who drop out of treatment for any reason (RR: 1.21; 95% CI: 0.67 to 2.16) or due to adverse effects (RR: 2.07; 95% CI: 0.57 to 7.52) based on 1 RCT (1 RCT; N=61; McEnroy, 2003)[304]. **RCT 1++**

According to An RCT (McElroy, 2003)[304], the following adverse effects were reported: headache, paresthesia and amenorrhoea in the topiramate group. In the placebo group: cramps, sedation and testicular pain. The overall dropout rate was 47% in the topiramate group and 39% in the placebo group. **RCT 1+**

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6)

9.14. Atomoxetine

9.14.1. What is the efficacy of atomoxetine in patients with eating disorders?

No evidence has been found in the NICE CPG (2004)[30] or in the high-quality SRSE [31, 202, 211, 245]. The updated search has identified one RCT.

Scientific Evidence

One RCT (McElroy, 2007)[316] compared atomoxetine (40-120 mg/day doses; N=20) *vs.* control group (N=19) over 10 weeks in a group of patients with BED (age: 18 to 65 years). The group treated with atomoxetine significantly reduced the weekly frequency of binge-eating (p=0.018), the daily frequency of binge-eating (p=0.003) and the disease scale (p=0.015). No significant differences were found in other results. 30% of patients in the treated group and 45% in the placebo group did not complete the study. **RCT 1+**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

9.14.2. What is the safety of atomoxetine in patients with eating disorders?

The answer is based on the RCT described in the question regarding efficacy.

Scientific Evidence

An RCT (McElroy, 2007)[316] compared atomoxetine (40 a 120 mg/day doses; N=20) *vs.* control group (N=19) over 10 weeks in a group of patients with BED (age: 18 to 65 years). There were 3 dropouts due to adverse effects in the treated group (depressive symptoms, constipation and agitation) and 1 in the placebo group. Atomoxetine is effective and well tolerated in the short-term treatment of BED. **RCT 1+**

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

9.15. Antiemetics

9.15.1. Bulimia nervosa

9.15.1.1. What is the efficacy of ondansetron in patients with BN?

The answer is based on the NICE CPG (2004)[30], on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and on a more recently published high-quality SRSE (1++) conducted by Shapiro, *et al*. (2007) [211]. The updated search has no identified any new evidence.

Scientific Evidence

An RCT (Faris, 2000; USA)[317] compared ondansetron treatment to placebo in adult females with BN (N=26) who were being treated on an outpatient basis over 4 weeks. In the ondansetron group frequency of binge-eating and purging decreased significantly and the restoration of a well-balanced diet improved. There are no measures of depression or anxiety. There are no differences in patients' weight changes. **RCT 1++**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Summary of the Evidence

CPG[30]	There is insufficient evidence to determine that antiemetics (ondansetron) are more or less effective when compared to placebo (2 RCTs; N=37; Faris, 1998[317], 2000[317]).

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

9.15.1.2. What is the safety of ondansetron in patients with BN?

The answer is based on the NICE CPG (2004)[30], on the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and on a more recently published high-quality SRSE (1++) conducted by Shapiro, *et al*. (2007) [211]. The updated search has no identified any new evidence. The study is briefly described in question 9.15.1.1.

Scientific Evidence

In an RCT (Faris, 2000)[317], one participant dropped out of treatment in the ondansetron group and there were no dropouts in the placebo group. No adverse effects were reported.	**RCT 1++**

Summary of the Evidence

CPG[30]	There is insufficient evidence to determine that antiemetics (ondansetron) are more or less acceptable *vs*. placebo (2 RCTs; N=37; Faris, 1998[317], 2000[317]).

Recommendations

(See recommendations 9.GPH.1. to 9.GPH.6.)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

SUMMARY OF THE EVIDENCE FOR PHARMACOLOGICAL TREATMENT

(QUESTIONS 9.9. to 9.15.)

SRSE[31] 1++	Dropout rates in the RCT assessing pharmacological treatments ranged from 0% to 51% of treated cases. No drug in particular showed a greater relapse rate than others.

Anorexia nervosa

SRSE[31] 1++	The literature relating to medication in AN is scarce and inconclusive.

Binge-Eating Disorder

SRSE[31] 1++	There is moderate evidence that supports the role of drugs in the treatment of BED

GENERAL RECOMMENDATIONS FOR THE PHARMACOLOGICAL TREATMENT OF EATING DISORDERS

(QUESTIONS 9.9. to 9.15.)

Anorexia nervosa

D	9.GPH.1.	Pharmacological treatment is not recommended as the only primary treatment in patients with AN. (Adopted from recommendation 6.3.6.1. of the NICE guide).
D	9.GPH.2.	Caution should be exercised when prescribing pharmacological treatment for patients with AN who have associated comorbidities such as obsessive-compulsive disorder (OCD) or depression. (Adopted from recommendation 6.3.6.2. of the NICE guide).
D	9.GPH.3.	Given the risk of heart complications presented by patients with AN, prescription of drugs whose side effects may affect cardiac function must be avoided. (Adopted from recommendation 6.3.6.4. of the NICE guide).

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D	9.GPH.4.	If drugs with adverse cardiovascular effects are administered, ECG monitoring of patients should be carried out. (Adopted from recommendation 6.3.6.3. of the NICE guide).
D	9.GPH.5.	All patients with AN must be warned of the side effects of pharmacological treatments. (Adopted from recommendation 6.3.6.5. of the NICE guide).

Binge-Eating Disorder

D	9.GPH.6	In the absence of evidence to guide the management of BED, it is recommended that the clinician treat the patient based on the eating problem that most closely resembles the patient's eating disorder according to BN or AN guides. (Adopted from recommendation 8.2.7.1. of the NICE guide).

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COMBINED INTERVENTIONS

This section describes the evidence for the efficacy and safety of combined interventions, whether they are psychological or pharmacological, in the treatment of eating disorders. Occasionally, the experimental and control groups receive one or more treatments.

9.16. What is the efficacy and safety of combined interventions in patients with eating disorders?

9.16.1. Anorexia nervosa

9.16.1.1. What is the efficacy and safety of combined interventions in patients with AN?

The answer is based on the NICE CPG (2004)[30], which describes the results from the RCT according to variables of interest. There are no results regarding combined interventions neither in the quality systematic review of scientific evidence (1++) elaborated by the AHRQ of the US (2006)[31] nor in the more recent one conducted by Bulik, *et al*. (2007)[202]. The updated search has identified two new 2 RCTs.

The revised literature does not inform on the adverse effects declared over the course of the RCT that studied combined interventions on AN.

Scientific evidence

In patients whose clinical picture is not as severe as to require emergency treatment, there is insufficient evidence on whether to administer an outpatient psychological treatment or to proceed to a complete hospitalisation for administration of such treatment (group therapy-FT-NC *vs.* individual therapy-NSFT-NC) according to 1 RCT (N=90; Crisp, 1991)[263]. **RCT 1++**

An RCT (Brambilla, 2007, Italy)[318] comprised of 30 women with AN receiving outpatient treatment compared CBT-olanzapine (daily doses 2.5 mg during the first month and 5 mg the two following months) N=15 (8 restrictive type AN [AN-R] and 7 bulimic-purging type AN [AN-BP]) *vs.* CBT-placebo, N=15 (10 AN-R and 5 AN-BP). There were no significant differences between the two groups in BMI or in the EDI questionnaire score. However, AN-BP treated with CBT-olanzapine improved the BMI score significantly (p=0.01) as compared with other groups. Both groups showed significant improvement in *The Yale Brown Cornell for Eating Disorder Rating Scale* (p=0.08) score. Differences were not significant upon stratification of the groups. Both groups obtained a significant in the measurement of aggressiveness (CBT-olanzapine: p=0.006 *vs.* CBT-placebo: p=0.05) and depression (CBT-olanzapine: p=0.01 *vs.* CBT-placebo: p=0.01). Upon stratification of the groups, AN-BP treated with CBT-olanzapine obtained better results in aggressiveness (p=0.05) than AN-R. There were no differences between these groups in terms of depression. **RCT 1++**

It has been 3 years since the publication of this Clinical Practice Guideline and this is subject to updating.

The CBT-olanzapine group obtained better results in the TCI questionnaire (*The Temperament and Character Inventory*) (p=0.007) as compared with placebo. Upon stratification of AN-R treated with CBT-olanzapine, this type of AN obtained better results than the other groups (p=0.04). The concentration of hemovallinic acid increased significantly in the group treated with CBT-olanzapine. There was no correlation between the concentration of hemovallinic acid and psychopathological parameters. Pharmacological treatment may improve significantly specific aspects of AN.

An RCT (Brambilla, 2007; Italy)319 comprised of 20 women with AN receiving outpatient treatment (mean age: 23 years) compared CBT-nutritional rehabilitation programme-olanzapine (N=10) vs. CBT-nutritional rehabilitation programme-placebo (N=10) administered over 3 months (olanzapine: 2.5 mg 1 month and 5 mg 2 months). BMI increased significantly in both groups but without differences between them. Leptine and ghreline concentrations did not change throughout the treatment course and no correlation was observed between plasma levels and BMI. Data suggest that the increase in BMI of subjects treated with olanzapine cannot be attributed to the administration of this drug. Leptine and ghreline secretion was not accountable for such changes. **RCT 1++**

An RCT (Walsh, 2006; USA)[320], comprised of a group of patients with AN (N=93) with a minimum BMI of 19.0 in a day hospital, compared CBT-fluoxetine (N=49) *vs.* CBT-placebo (N=44) over one full year. A similar percentage of patients from both groups (fluoxetine: 26.5% *vs.* placebo: 31.5%, p=0,57) maintained the BMI score (≥18.5) and continued in the study for a further 52 weeks. There were no significant differences in relapse time between both groups. (p=0.64). This study failed to show that treatment with fluoxetine benefits patients with AN after recovery of body weight. **RCT 1++**

9.16.2. Bulimia nervosa

9.16.2.1. What is the efficacy of combined interventions in patients with BN?

The answer is based on the NICE CPG (2004)[30], which describes the results yielded by RCT according to variables of interest and in the high-quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and in a more recent publication (Shapiro, *et al*. 2007)[211]. The updated search has not identified any new evidence.

In the case of BN, there are some RCTs that compare treatment with antidepressants against a combination of antidepressants and psychological therapy. Other RCTs also compare single psychological therapy against a combination of single psychological therapy and antidepressants.

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Antidepressants *vs*. antidepressants-psychological therapy

Variables: reduction / remission of binge-eating and purging

There is insufficient evidence to show that antidepressants (desipramine, fluoxetine, imipramine) differ from the combination of CBT-antidepressants with regard to remission of binge eating episodes (5 RCTs; N=291; Leitenberg, 1994[321]; Jacobi, 2002[322]; Goldbloom, 1997[323]; Agras, 1992[324]; Walsh, 1997[233]; randomised effects model RR: 1.29; 95% CI: 0.99 to 1.69) and remission of purging episodes (5 RCTs; N=199; Leitenberg, 1994[321]; Jacobi, 2002[322]; Goldbloom, 1997[323]; Agras, 1992[324]; Walsh, 1997[233]; randomised effects model RR: 1.23; 95% CI: 0.93 to 1.64) at treatment completion. **RCT 1++**

There is strong evidence to determine that the combination antidepressants-CBT *vs.* antidepressants (desipramine, fluoxetine) is suitable to reduce the frequency of binge-eating episodes (4 RCTs; N=133; Jacobi, 2002[322]; Goldbloom, 1997[323]; Agras, 1992[324]; Walsh, 1997[233]; SMD: 0.55; 95% CI: 0.21 to 0.90) but limited to reduce the frequency of purging episodes at treatment completion (5 RCTs; N=141; Leitengerg, 1994[321]; Jacobi, 2002[322]; Goldbloom, 1997[323]; Agras, 1992[324]; Walsh, 1997[233]; SMD: 0.49; 95% CI: 0.15 to 0.83). **RCT 1++**

There is insufficient evidence to show that antidepressants (desipramine, fluoxetine) differ from the combination of CBT-antidepressants with regard to remission and frequency of binge eating episodes (2 RCTs; N=81; Jacobi, 2002[322]; Agras, 1992[324]; RR: 1.12; 95% CI: 0.88 to 1.42), (1 RCT; N=21; Agras, 1992[324]; SMD: 0,29; 95% CI: -0.58 to 1.15), respectively, and in the remission and frequency of purging episodes (3 RCTs; N=95; Leitenberg, 1994[321]; Jacobi, 2002[322]; Agras, 1992[324]; RR: 1.10; 95% CI: 0.87 to 1.39), (2 RCTs; N=29; Leitenberg, 1994[321]; Agras, 1992[324]; SMD: 0.46; 95% CI: -0.31 to 1.23) at post-treatment follow up, respectively. **RCT 1++**

There is insufficient evidence to show that antidepressants (fluoxetine) differ from the combination of SH-antidepressants with regard to remission and frequency of binge-eating episodes (1 RCT; N=47; Mitchell, 2001[258]; RR: 1.11; 95% CI:0.83 to 1.48), (1 RCT; N=34; Mitchell, 2001[258]; SMD: 0.37; 95% CI: -0.24 to 0.97) and remission and frequency of purging episodes (1 RCT; N=47; Mitchell, 2001[258]; RR:1.11; 95% CI: 0.83 to 1.48), (1 RCT; N=34; Mitchell, 2001[258]; SMD: 0.31; 95% CI:-0.29 to 0.91) respectively, at treatment completion. **RCT 1++**

There is insufficient evidence to show that antidepressants (desipramine) differ from the combination de SFP-antidepressants with regard to remission and frequency of binge eating episodes (RR: 0.95; 95% CI: 0.75 to 1.21), (SMD: -0,29; CI 95%: -0.85 to 0.27) and remission and frequency of purging episodes (RR: 0.95; 95% CI:0.75 to 1.21 and SMD: 0.26; 95% CI: -0.30 to 0.83) respectively at treatment completion according to 1 RCT (N=50; Walsh, 1987)[288]. **RCT 1++**

Variable: depression

RCT 1++

There is insufficient evidence to show that the combination of CBT-antidepressants

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(desipramine, fluoxetine) differs from antidepressants alone in depression scores (4 RCTs; N=125; Jacobi, 2002[322]; Goldbloom, 1997[323];Agras, 1992[324]; Walsh, 1997[233]; SMD: 0.31; 95% CI: -0.05 to 0.66) and in generalized psychiatric symptoms (2 RCTs; N=85; Jacobi, 2002[322];Walsh, 1997[233]; SMD: 0.10; 95% CI: -0.33 to 0.53) at treatment completion.

There is insufficient evidence to show that treatment with antidepressants (desipramine) differs from SFP-antidepressants with regard to the score obtained on depression (SMD: 0.26; 95% CI: -0.30 to 0.83) and in generalized psychiatric symptoms (SMD: -0.22; 95% CI: -0.78 to 0.34) at treatment completion according to 1 RCT (N=50; Walsh, 1997)[233]. **RCT 1++**

Psychological Therapy vs. Antidepressants-Psychological therapy

Variables: reduction / remission of binge-eating and purging

There is insufficient evidence to show that CBT differs from combination of CBT-antidepressants (desipramine, fluoxetine, imipramine) with regard to remission and frequency of binge-eating (5 RCTs; N=225; Jacobi, 2002[322]; Agras, 1992[324]; Walsh, 1997[233]; Fichter, 1991[295]; Goldbloom, 1997[323]; randomised effects model RR:1.09; 95% CI: 0.83 to 1.42), (5 RCTs; N=185; Jacobi, 2002[322]; Agras, 1992[324]; Walsh, 1997[233]; Fichter, 1991[295]; Goldbloom, 1997[323]; SMD: 0.38; 95% CI: 0.09 to 0.68) and remission of purging episodes (5 RCTs; N=199; Jacobi, 2002[323]; Agras, 1992[324]; Walsh,1997[233]; Leitenberg, 1994[321]; Goldbloom, 1997[323]; RR: 0.94; 95% CI: 0.69 to 1.28) at treatment completion. **RCT 1++**

There is insufficient evidence to show that CBT differs from the combination CBT-antidepressants (desipramine, fluoxetine) with regard to remission and frequency of binge eating episodes (2 RCTs; N=84; Jacobi, 2002[322]; Agras, 1992[324]; RR: 0,86; 95% CI:0.64 to 1.14), (1 RCT; N=32; Agras, 1992[324]; SMD: -0.19; 95% CI: -0.94 to 0.56) and remission of purging episodes (3 RCTs; N=98; Jacobi, 2002[322]; Agras, 1992[324]; Leitenberg,1994[321]; RR: 0.87; 95% CI: 0.68 to 1.13), (2 RCTs; N=43; Leitenberg, 1994[321]; Agras 1992[324]; SMD: -0.27; 95% CI: -0.90 to 0.37) at post-treatment follow up. **RCT 1++**

There is insufficient evidence to show any differences between SH *vs.* SH-antidepressants (fluoxetine) with regard to remission and frequency of binge eating episodes (1 RCT; N=43; Mitchell, 2001[258]; RR: 1.01; 95% CI: 0.73 to 1.41), (1 RCT; N=40; Mitchell, 2001[258]; SMD: 0.20; 95% CI: -0.43 to 0.82) and remission and frequency of purging episodes (1 RCT; N=43; Mitchell 2001[258]; RR: 1.01; 95% CI: 0.73 to 1.41), (1 RCT; N=40; Mitchell, 2001[258]; SMD: 0.36; 95% CI: -0.27 to 0.98) at treatment completion. **RCT 1++**

There is insufficient evidence to show that SFT-antidepressants (desipramine) differs from SFT alone with regard to remission and frequency of binge-eating episodes (RR: 1.00; 95% CI: 0.73 to 1.38; SMD: -0.07; 95% CI: -0.66 to 0.52) and in remission and frequency of purging episodes (RR: 1.05; 95% CI: 0.85 to 1.30; SMD: 0.25; 95% CI: -0.35 to 0.84) at treatment completion according to 1 RCT (N=44; Walsh, 1997)[233]. **RCT 1++**

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There is insufficient evidence to show that NC differs from its combination with antidepressants (fluoxetine) with regard to remission and frequency of binge-eating episodes (1 RCT; N=67; Beumont, 1997[294]; RR: 0.97; 95% CI: 0.62 to 1.54), (1 RCT; N=65; Beumont, 1997[294]; SMD: -0.17; 95% CI: -0.66 to 0.31) and in remission and frequency of purging episodes (1 RCT; N=77; Beumont, 1997[294]; RR: 0.97; 95% CI: 0.62 to 1.54), (1 RCT; N=65; Beumont, 1997[294]; SMD: 0.34; 95% CI: -0.15 to 0.84) at treatment completion. **RCT 1++**

There is insufficient evidence to show that NC differs from its combination with antidepressants (fluoxetine) with regard to remission and frequency of binge-eating episodes (1 RCT; N=67; Beumont, 1997[294]; RR: 0.70; 95% CI: 0.50 to 0.97; NNT: 4; 95% CI: 3 to 25), (1 RCT; N=40; Beumont, 1997[294]; SMD: -0.08; 95% CI: -0.71 to 0.54) and in remission and frequency of purging episodes (1 RCT; N=67; Beumont, 1997[294]; RR: 0.70; CI 95%: 0.50 to 0.97; NNT: 4; 95% CI: 3 a 25), (1 EC; N=40; Beumont, 1997[294]; SMD:-0.05; 95% CI: -0.68 to 0.58) at treatment completion. **RCT 1++**

Variable: depression

There is insufficient evidence to show that CBT-antidepressants (desipramine, fluoxetine) differ from CBT alone with regard to depression (5 RCTs; N=179; Fichter,1991[295]; Jacobi, 2002[322], Goldblomm, 1997[323],Agras, 1992[324],Walsh, 1997[233]; SMD: 0.18; 95% CI: -0.12 to 0.48) and generalised psychiatric symptoms (2 RCTs; N=85; Jacobi,2002[322]; Walsh, 1997[233]; SMD: -0.09; 95% CI: -0.52 to 0.34) at treatment completion. **RCT 1++**

There is insufficient evidence to show SFT-antidepressants (desipramine) differs from SFT alone with regard to depression (SMD: 0.37; 95% CI: -0.22 to 0.97) and generalised psychiatric symptoms (SMD: 0.00; 95% CI: -0.59 to 0.59) at treatment completion, according to 1 RCT (N=44; Walsh, 1997)[233]. **RCT 1++**

There is insufficient evidence to show that NC differs from its combination with antidepressants (fluoxetine) in depression at treatment completion (1 RCT; N=67; Beumont, 1997[294]; SMD: 0.25; 95% CI: -0.23 to 0.73) and in the follow up after treatment (1 RCT; N=40; Beumont, 1997[294]; SMD: 0.30; 95% CI: -0.34 to 0.93). **RCT 1++**

Other results

An RCT (Goldbloom, 1997; Canada)[323] compared fluoxetine (60 mg/day) *vs.* CBT *vs.* fluoxetine (60 mg/day)-CBT in 60 adult women with BN receiving outpatient treatment for 12 weeks. At 12 weeks no differences were found among the groups in the measurements relating to feeding. The 3 interventions improved the main bulimic symptoms. The combination (fluoxetine 60mg/day-CBT) and CBT alone achieved better results than fluoxetine alone to reduce binge-eating and purging episodes, both subjective and objectively, as well as thoughts relating to abstinence or remission.

The scores achieved in the depression scale and the weight did not show any differences among the 3 groups.

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

An RCT (Walsh, 2004; USA)[328] compared fluoxetine *vs.* placebo *vs.* GSH *vs.* fluoxetine-GSH in 91 adult women diagnosed with BN undergoing outpatient BN. Fluoxetine alone and in combination with GSH achieved better results to reduce purging, binge-eating, food restriction and depression than those observed in other groups. **RCT 1++**

An RCT (Mitchell, 2001; USA)[258] compared fluoxetine *vs.* placebo *vs.* SH *vs.* fluoxetine-SH in 91 adult women with BN receiving outpatient treatment. Fluoxetine was associated with a higher decrease in the number of purging episodes as compared to placebo, but not so in the number of binge-eating episodes. There are no significant differences in depression, and neither in remissions nor abstinence. **RCT 1+**

In 2 RCTs (Agras, 1992; USA324 and Agras, 1994325) compared desipramine (for 16 weeks) vs. desipramine (24 weeks) vs. desipramine-CBT (16 weeks) vs. desipramine-CBT (24 weeks) vs. CBT alone (24 weeks) in 71 adult women with BN undergoing outpatient treatment. The combined treatment of 16 and 24 weeks was more effective than desipramine alone to decrease binge-eating and purging episodes. The combined treatment of 24 weeks was more effective in the patient's concerns over the diet and the feeling of hungriness. There were no differences in remission or abstinence, neither in the weight. Depression was not reported. At one year follow up, the 24 week combination and CBT alone were better than the 16 week combination and desipramine alone to reduce the number of binge-eating and purging episodes. Of the patients who presented remission or abstinence at treatment completion, between 78% and 100% maintained the effect at the 1 year follow up. This was only observed in cases in which treatment had been combined. **RCT 1+**

2 RCTs (Walsh, 1997[233]; Wilson, 1999[326]) compared CBT-placebo *vs.* CBT-desipramine alone or desipramine and afterwards fluoxetine *vs.* support therapy-placebo *vs.* support therapy-medication (desipramine alone or desipramine followed by fluoxetine) *vs.* medication alone (desipramine alone or desipramine followed by fluoxetine) administered over 16 weeks to 120 adult women with BN undergoing outpatient treatment. In analysing all the groups that included CBT *vs.* all the groups that included support therapy, it was observed that CBT was better to reduce the frequency of binge-eating and purging episodes. The combinations of CBT + medication were superior to CBT alone to reduce the frequency of binge-eating episodes, EAT scores, depression, weight and to increase the number of remissions. The combinations of CBT and medication were superior to medication alone to reduce the frequency of binge-eating episodes, EAT scores, body image and to increase the number of remissions. Medication alone was superior to CBT alone to reduce BMI and weight. Medication alone was superior to IPT-medication to reduce the frequency of binge-eating and purging episodes. Medication was superior to lessen depression. CBT was not associated with a greater probability for remission. **RCT 1++**

An RCT (Mitchell, 2002)[327] compared IPT *vs.* fluoxetine (16 weeks) *vs.* fluoxetine (8 weeks)-desipramine (8 weeks) in 62 adult women diagnosed with BN receiving outpatient care. No differences were noted in any of the dietary or psychological aspects. **RCT 1+**

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Summary of the Evidence

SRSE[31] 1++	The above mentioned RCTs show preliminary evidence of the most suitable combinations between psycho-drugs and behavioural psychological therapies. The studies have different designs and there is no repetition of their results. Hence, more studies are needed in order to be able to evaluate and ascertain their efficacy.
SRSE[31] 1++	CBT and the combination CBT-fluoxetine afford better results than treatment with fluoxetine alone to reduce binge-eating and purging at treatment completion (Goldbloom, 1997)[323].
SRSE[31] 1++	Within the context of special programmes for EDs, both fluoxetine as well as SH therapies (guided or not) are better to reduce purging. Nonetheless, the combination of both therapies does not increase treatment efficacy (Mitchell, 2001)[258].
SRSE[31] 1++	The treatment of affected individuals who do not respond to behavioural psychological therapies continues to pose a limitation to the current evidence. (Mitchell, 2002)[327].

9.16.2.2. What is the safety of combined interventions in patients with BN?

The answer is based on the NICE CPG (2004)[30] and on the high-quality SRSEs (1++), one elaborated by the AHRQ of the US (2006)[31] and the more recent one elaborated by Shapiro, *et al.* (2007)[211]. The updated search has not identified any new evidence. Question 9.16.1.1. describes the studies briefly.

Scientific Evidence

There is insufficient evidence to determine if there are any significant clinical differences in the number of withdrawals for any given reason between treatment with antidepressants *vs.* CBT-antidepressants (desipramine, fluoxetine, imipramine) (4 RCTs; N=206; Mitchell, 1990[243]; Jacobi, 2002[322]; Goldbloom, 1997[323]; Leitenberg, 1994[321]; RR: 1.17; 95% CI: 0.81 to 1.68). **RCT 1++**

There is insufficient evidence to determine if there are any significant clinical differences in the number of withdrawals due to adverse effects between treatment with antidepressants (imipramine) *vs.* CBT-antidepressants (1 RCT; Mitchell, 1990[243], N=106; RR: 0.64; CI 95%: 0.11 to 3.69). **RCT 1++**

There is insufficient evidence to determine if there is a clinically significant difference between treatment with CBT *vs.* CBT-antidepressants (desipramine, fluoxetine, imipramine) in the number of withdrawals for any given reason (5 RCTs; N=230; Fichter, 1991[295]; Jacobi, 2002[322]; Goldbloom, 1997[323]; Mitchell, 1990[243]; Leitenberg, 1994[321]; RR: 0.70; 95% CI: 0.45 to 1.08). **RCT 1++**

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There is insufficient evidence to determine if there is a clinically significant difference between the treatment with CBT and the combination of CBT-antidepressants (imipramine) in the number of withdrawals due to adverse effects (1 RCT; N=86; Mitchell, 1990[243]; RR: 0.51; 95% CI: 0.06 to 4.70). **RCT 1++**

There is insufficient evidence to determine if there is a clinically significant difference in the treatment with antidepressants (fluoxetine) *vs.* fluoxetine-SH regarding the number of withdrawals for any given reason at treatment completion (2 RCTs; N=91; Mitchell, 2001[258]; Walsh 2004[328]; RR: 1.15; 95% CI: 0.72 to 1.84). **RCT 1++**

There is insufficient evidence to determine whether treatment with SH *vs.* SH-antidepressants (imipramine) differs in the number of withdrawals for any given reason (1 RCT; N=43; Mitchell, 1990[243]; RR: 0.48; 95% CI: 0.05 to 4.88). **RCT 1++**

There is insufficient evidence to determine whether treatment with NC differs from treatment with NC-antidepressants (fluoxetine) in the number of withdrawals for any given reason at treatment completion (RR=0.66; 95% CI: 0.29 to 1.49) or due to adverse effects (RR: 0.11; 95% CI: 0.01 to 2.04) according to 1 RCT (N=67; Beumont, 1987)[294]. **RCT 1++**

An RCT (Goldbloom, 1997)[323] compared fluoxetine *vs.* fluoxetine-CBT. There were 2 withdrawals due adverse effects in the fluoxetine group and four in the combination fluoxetine-CBT. **RCT 1+**

In an RCT (Walsh, 2004)[328], there was a 54% withdrawal rate in the group treated with fluoxetine-GSH *vs.* 88% in the group treated with placebo-GSH *vs.* 70% in the group treated with fluoxetine *vs.* 64% in the placebo group. Adverse effects were not reported. **RCT 1++**

In an RCT (Mitchell, 2001)[258], the number of withdrawals was low: placebo (5%), fluoxetine (0%), placebo-SH (0%), fluoxetine-SH (5%). No adverse effects were reported. **RCT 1+**

In 2 RCTs (Agras, 1992[324]; Agras, 1994[325]), no information was reported regarding adverse effects. The mean withdrawal rate was 25%. **RCT 1+**

In 2 RCTs (Walsh, 1997[233];Wilson, 1999[326]), the mean withdrawal rate was 34%, and no information was provided on adverse effects. **RCT 1++**

In an RCT (Mitchell, 2002)[327], no information was provided on adverse effects. The percentage of withdrawals was 32% in the IPT group and 48% in the antidepressants group. **RCT 1+**

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Summary of the Evidence

CPG [30]	When these therapies were implemented in primary care, the number of withdrawals in the group treated wit fluoxetine was 70%, higher than the 54% observed in the group treated with fluoxetine-GSH (Walsh, 2004)[328].

9.16.3. Binge-eating disorder

9.16.3.1. What is the efficacy of combined interventions in patients with BED?

The answer is based on NICE CPG (2004)[30], which describes the results yielded by RCTs, and on the high-quality SRSEs (1++), one elaborated by the AHRQ of the US (2006)[31] and the more recently published one conducted by Brownley, *et al*. (2007)[245]. The updated search has identified one new RCT.

Scientific Evidence

An RCT (Grilo, 2005; USA)[329] compared fluoxetine (60 mg/day) *vs.* placebo *vs.* CBT *vs.* CBT-fluoxetine in 108 adult patients (78% women) diagnosed with BED and treated on an outpatient basis for 16 weeks. The groups that received CBT (alone or in combination with fluoxetine) achieved better results than the placebo and the fluoxetine alone groups to reduce the frequency of binge-eating episodes, ED-related symptoms, depression, and greater frequency of remission. There were no differences with regard to weight. **RCT 1++**

An RCT (Agras, 1994; USA)[325] compared CBT (3 months) – weight loss therapy (6 months) –weight loss therapy (9 months) *vs.* CBT (3 months) – weight loss therapy (6 months) - desipramine (300 mg/day) in 109 adult women with BED on an outpatient basis. The groups that received CBT experienced a significant in the binge-eating episodes after 12 weeks of treatment, even though the effect was not maintained at the 1 year follow up. There were no differences regarding depression scores among the groups. At treatment initiation, the weight loss was greater in the group receiving weight loss therapy. At the 3-month follow up, the weight loss was greater in the CBT-desipramine group (mean weight loss: 4.8 Kg). **RCT 1+**

An RCT (Grilo, 2005; USA)[330] compared CBT-orlistat (120 mg 3/day) *vs.* CBT-placebo over12 weeks of treatment in 50 adult patients (88% women) (BMI>30) with BED undergoing outpatient treatment. The group on orlistat had a greater remission rate at treatment completion, but not so at the 2-month follow up. This group also experienced weight loss. **RCT 1++**

An RCT (Devlin, 2007; USA)[331] comprised of 116 people suffering from BED (90 females and 26 males) (age range: 18-70 years) compared individual CBT- fluoxetine *vs.* individual CBT over 5 months with a follow up at +2 years. Both groups showed an improvement in the frequency of the binge-eating episodes and their remission after 2 months of treatment. The *odds ratio* (*odds* at 24months/ post-treatment) for remission of the binge-eating episodes at 2 years after treatment was 1.373 times the *odds* at completion of 5 months of treatment. Subjects who received individual CBT experienced a greater reduction in the frequency of binge-eating episodes and greater remission odds ratio of such episodes as compared to the subjects who had not received this therapy at 2 years. There were no differences regarding weight in neither of the groups. The group that received fluoxetine achieved better results in the reduction of depressive symptoms at 2 years. A short-term treatment may show benefits in the long term and not all treatments are equivalent in terms of the benefits they provide. **RCT 1++**

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RSEC[31] 1++	The combination of pharmacological treatment with CBT can reduce the occurrence of binge-eating episodes and improve weight loss. However, no definition has yet been provided as to what the best medication is to maintain the weight loss. Moreover, there are no empiric data on what the ideal treatment duration might be to maintain BED remission and weight loss.

9.16.3.2. What is the safety of combined interventions in patients with BED?

The answer is based on NICE CPG (2004)[30], on the quality SRSE (1++) elaborated by the AHRQ of the US (2006)[31] and on other more recently published publication conducted by Brownley, *et al*. (2007)[245]. Question 9.16.3.1. describes the studies briefly.

Scientific Evidence

In an RCT (Grilo, 2005; USA)[329] that compared fluoxetine *vs.* placebo *vs.* CBT-placebo *vs.* CBT-fluoxetine, the percentage of withdrawals was as follows: placebo (15%), fluoxetine (22%), CBT-placebo (21%), CBT-fluoxetine (23%). No information was provided on adverse effects. **RCT 1++**

In an RCT (Agras, 1994)[325], the withdrawal rate from weight loss therapy was 27%, from CBT-weight loss therapy it was 17%, and from CBT- weight loss therapy-desipramine it was 23%. Eight subjects stopped taking desipramine due to adverse effects. **RCT 1+**

An RCT (Grilo, 2005)[330] compared CBT-orlistat (120 mg 3/day) *vs.* CBT-placebo for 12 weeks of treatment in 50 adult patients (88% women) (BMI>30) with BED receiving treatment on an outpatient treatment basis. The number of withdrawals was as follows: orlistat-CBT (24%); placebo-CBT (20%). The group treated with orlistat-CBT had more gastrointestinal adverse effects. **RCT 1++**

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TREATMENT OF EATING DISORDERS THAT OCCUR WITH COMORBIDITIES

9.17. What is the treatment for eating disorders that occur with mental disorders?

The mental disorders most frequently associated with eating disorders are: substance-related disorders, anxiety disorders, OCD, personality disorders, mood disorders and impulse control disorders.

Information on treatment in the presence of these associations has been extracted from the NICE guide (2004)[30], although it only establishes recommendations for certain associations.

Scientific Evidence

There are special programmes (that have not been sufficiently assessed) for patients with BN associated with substance abuse, self-inflicted injuries and/or BPD. **CPG[30]**

The presence of impulse control disorder or personality disorder (type B) in patients with BN is associated with poor treatment outcomes. **CPG[30]**

Care must be exercised when using medication to treat comorbid conditions such as depression and OCD compulsive symptomatology in patients with AN when these clinical pictures could be resolved through weight gain. **CPG[30]**

9.18. What is the treatment for eating disorders that occur with organic disorders?

The organic disorders that are most frequently associated with eating disorders are: diabetes mellitus (DM), obesity, malabsorbtion syndromes and thyroid diseases.

Information on treatment in the presence of these combinations has been extracted from the NICE guide[30], although it only formulates recommendations for certain associations.

Scientific Evidence

The presence of type 1 DM requires changes in the psychological treatment of an eating disorder. In CBT-BN, control over eating is very important and can clash with NC aimed at diabetics (Peveler, 1993[332]). There are special guidelines that adapt CBT-BN to type 1 DM. **CPG[30]**

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Recommendations

D	9.18.1.	Treatment in clinical and subclinical cases of eating disorders in patients with DM is essential due to the increased risk presented by this group. (Adopted from recommendation 7.5.8.1. of the NICE guide).
D	9.18.2.	Patients with Type 1 DM and an eating disorder must be monitored due to the high risk of developing retinopathy and other complications. (Adopted from recommendation 7.5.8.2. of the NICE guide).
D	9.18.3.	Young people with Type 1 DM and poor adherence to antidiabetic treatment should be assessed for the probable presence of an eating disorder. (Adopted from recommendation 5.2.5.5. of the NICE guide).

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TREATMENT OF CHRONIC EATING DISORDERS

9.19. How are chronic eating disorders treated?

Tertiary prevention aims to resolve the most serious symptoms, palliate the most severe complications and avoid vital risk in chronic cases, which have generally been refractory to treatment and in which expectations of remission are low or inexistent.

In chronic eating disorders, tertiary prevention includes management of the organic (cachexia, electrolyte imbalance, immune deficiency, etc.) and mental (loss of impulse control, risk of self-aggression, suicide prevention, etc.) state, maintenance pharmacological treatment, behavioural pact within the family (acceptance of certain behaviours and reaching agreement on others, indications for the family to identify relapse in maladaptive symptoms, etc.) and social reinsertion measures in which family, health care resources and support associations for patients with eating disorders and their family members play a very important role.

There is no evidence on the effect of different interventions aimed at chronic eating disorder cases.

Tertiary prevention of eating disorders was not addressed in the NICE CPG (2004)[30]. Four of the documents[10, 16, 19, 147] elaborated in our setting formulate recommendations on this aspect and based on them the working group has formulated the recommendations for its approach.

Recommendations

✓	9.19.1.	The health care professional in charge of the care of chronic eating disorder cases should inform the patient on the possibility of recovery and advise him/her to see the specialist regularly regardless of the number of years elapsed and previous therapeutic failures.
✓	9.19.2.	It is necessary to have access to health care resources that are able to provide long-term treatments and follow-up on the evolution of chronic eating disorder cases, as well as to have social support to decrease future disability.

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TREATMENT OF EATING DISORDERS IN SPECIAL CASES

9.20. What is the treatment for eating disorders in special situations such as pregnancy and delivery?

Evidence on the considerations that should be acknowledged when the patient is pregnant or giving birth has been extracted from the NICE guide (2004)[30].

Summary of the Evidence

CPG[30]	Pregnancy with purging practices or pregnancy with overweight or obesity in an eating disorder is specific risk situations. Delivery can also be considered a risk situation that can lead to the onset of an eating disorder (Crow, 2008[333]; Franko, 1993[334]; Fairburn, 1990[335]).
CPG[30]	In a follow-up study of patients with AN (N=140), fertility was reduced by a third of what was expected, prematurity doubled and perinatal mortality was six times greater than in people without an eating disorder (Brinch, 1988)[336].
CPG[30]	In another follow-up study of 66 pregnant women with AN, these patients presented a higher percentage of C-sections and prematurity than the group without eating disorders (Bulik, 2008)[337].
CPG[30]	In a study conducted on pregnant women with AN, monitoring of the foetus' weight was performed. The foetus's growth was slow, especially in the last trimester (Treasure, 1988)[338].
CPG[30]	Pregnant women with AN have high risk of presenting premature deliveries. There are case-series that suggest the difficulties encountered by women with AN to feed their infants (Russell, 1998[339]; WezelMeijle, 1989[340]), while other studies report abnormal growth of these children (Hodes, 1997)[341].

Recommendations

D	9.20.1.	Pregnant patients with AN, whether it is the first episode or a relapse, require intensive prenatal care with adequate nutrition and follow-up of foetal development. (Adopted from recommendation 6.4.8.1. of the NICE guide).
D	9.20.2.	Pregnant women with eating disorders require careful follow-up throughout pregnancy and the postpartum period. (Adopted from recommendation 7.5.10.1. of the NICE guide).

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10. Assessment of Eating Disorders

Key Questions:

10.1. What instruments are useful to assess eating disorder symptoms and behaviour?
10.2. What instruments are useful for the psychopathological assessment of eating disorders?

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10.1. What instruments are useful to assess eating disorder symptoms and behaviour?

In the past few years several instruments have been designed for the assessment of symptoms and behaviours presented by patients with eating disorders. Self-report questionnaires and semi-structured interviews (see chapter 2, "Diagnosis") are the two main assessment instruments. There are also other measures related to preoccupation with body image, dietary consumption, amongst others, as well measures related to comorbidity (depression, anxiety, etc.).

Self-report questionnaires measure symptoms and risk behaviour of eating disorders, and do not provide a specific diagnosis (see chapter 2, "Diagnosis"). In contrast to semistructured interviews that require experience and individualised administration and, hence, entail increased cost and time, self-report questionnaire are relatively economical, require less time and can be applied to big groups of people, however, there is greater difficulty as far as definition and interpretation of concepts, which mandates caution in the generalisation of results[342].

When applying a questionnaire, it is important to borne in mind its psychometric properties: validity and reliability, sensitivity and specificity, amongst other characteristics, with the aim of selecting the most convenient questionnaire in accordance with assessment objectives, throughout the initial diagnostic phase and also throughout the treatment phase.

This chapter presents a review of different self-report questionnaires that are most frequently used in the assessment of eating disorders. Additionally, some instruments that are used to assess characteristics related with eating disorders but that are not per se instruments to detect/diagnose/assess eating disorders are mentioned. Instruments employed in the psychopathological assessment of eating disorders are also described.

10.1.1. Specific instruments for the assessment of eating disorders:

EAT-40, EAT-26 and ChEAT

Adapted Spanish versions that have been validated in our setting are available.
(See chapter 6, "Detection" and Annexes 2.2. to 2.4.)

EDI

Eating Disorder Inventory. Garner, *et al.*, 1983

The EDI (or EDI-I) is a self-report instrument designed to assess different cognitive and behavioural dimensions of AN and BN. It consists of 64 items grouped in 8 subscales that are positively correlated. The first three subscales measure behaviour and attitudes toward food, weight and body image (drive for thinness, bulimic symptomatology, self body image dissatisfaction), disturbances expressed in these areas are not specific of AN, given that similar responses appear in groups of people who are concerned about their diet. The other five subscales (ineffectiveness and low self-esteem, perfectionism, interpersonal distrust, interoceptive awareness or identification and maturity fears) assess general psychological characteristics associated with eating disorders, which are fundamental aspects of AN[343].

Each item is scored using a 6-point Likert scale. All subscales can be added to obtain an overall score or use each scale separately; clinically speaking, the quantitative value of each of the eight subscales is more relevant than the overall score. The maximum total score of this questionnaire is 192, the cut-off point is 42 points or less in the eight original subscales to diagnose an eating disorder. Some authors claim that the specificity of EDI is low, since it cannot properly differentiate individuals with eating disorders from those who present other psychological disorders. In a study carried out on women, it was reported that the subscales that best differentiate AN from BN are the ones addressing bulimic symptomatology and body image dissatisfaction, although the latter is high in both eating disorders. On the other hand, subscales such as low self-esteem, interpersonal fears, maturity fears, interoceptive identification and motivation to lose weight, which are usually high in both pathologies, distinguish patients with an eating disorder from control subjects. Subscales such as body image dissatisfaction, motivation to lose weight and dieting are usually high both in patients with eating disorders and in the general population[345].

There are several opinions on the utility of the EDI. Several authors suggest that it can be used as a screening test in non-clinical samples, but the fact that its ability to distinguish different types of eating disorders is questionable must be taken into account. However, it is suggested that the EDI is able to discriminate individuals with eating disorders and individuals without any psychiatric disturbances. This questionnaire has proven to be sensitive to changes registered in individuals when treatment is applied.

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Spanish version of the EDI

The Spanish adaptation of the EDI was developed by Guimerá and Torrubia, 1987, in a clinical sample (hospital) of 24 patients with AN and 24 control subjects sharing similar ages and sociodemographic characteristics[346].

EDI-2 (Version 2 of the EDI-1)

Later (1991), Garner, *et al*. developed the EDI-2, comprised of 91 items (64 items of the EDI-1 and an additional 27 items). The items are grouped in 11 scales, 8 of them main scales (obsession about thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness and maturity fears) and 3 provisional scales (not a part of the EDI-1) (ascetism, impulse regulation and social insecurity). A 5th grade educational level is required to answer this self-report measure[347].

Spanish version of the EDI-2

The Spanish version was adapted by the TEA publishing house 1998[348]

There is also a Spanish version validated in the Mexican population. When the cut-off point is 80 for the overall score, sensitivity is 91% and specificity is 80%; when it is 105 points, sensitivity is 82% and specificity is 89%[349].

BULIT

Bulimia Test. Smith and Thelen, 1984

There is an adapted Spanish version validated in our setting. (see chapter 6, "Detection" and Annex 2.5.)

BITE

Bulimia Investigatory Test Edinburgh. Henderson and Freeman, 1987

There is an adapted Spanish version validated in our setting. (see chapter 6, "Detection" and Annex 2.6.)

EDE-Q

Eating Disorders Examination-questionnaire. Fairburn and Beglin, 1993

The EDE-Q is a self-report questionnaire (it can be completed in less than 15 minutes) derived from the EDE semistructured interview developed by Fairburn and Beglin, 1993, and which contains its three main subscales (restraint, weight concern and shape concern)[350]. Results indicate a consistent positive correlation, albeit moderate, between the EDE and the EDE-Q. Correlation was higher in characteristics that do not present definition difficulties (for example, frequency of self-induced vomiting or average –days per week- laxative abuse); the greatest discrepancy indexes were found in the days-per-week assessment in which binge-eating episodes took place. These values were consistently higher in the EDE-Q. There is no information concerning the adaptation and validation of the EDE-Q in the Spanish population. However, there is an adapted Spanish version in a Colombian population sample (S-EDE-Q)[351].

SCOFF

Survey *Sick, Control, One, Fat, Food questionnaire*. Morgan, *et al*., 1999

There is an adapted Spanish version validated in our setting. (see chapter 6, "Detection" and Annex 2.1.)

ACTA

(Attitude Regarding Change in Eating Disorders) Beato and Rodríguez, 2003

The ACTA questionnaire was developed by L Beato and T Rodríguez, 2003[352], with the objective

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of assessing attitude regarding change in eating disorders. It consists of 59 items distributed in 6 subscales: precontemplation, contemplation, decision, action, maintenance and relapse. The ACTA is an easily administered instrument with adequate reliability and validity, and its use within the motivational approach can contribute useful information relating to therapeutic process knowledge.

ABOS

Anorectic Behaviour Observation Scale for parents/spouse.
Vandereycken, 1992.

The ABOS is a self-report questionnaire developed to obtain information from family members (parents) on their children's behaviours and attitudes that may be symptomatic of AN or BN. Using a cut-off point of 19 points, the ABOS obtained 90% sensitivity and 89.6% specificity in a sample of female students, values that were reliable based on subsequent clinical assessment. It can be a complementary instrument to eating disorder screening tools[353].

Spanish version of the ABOS

There is no information regarding the availability of an adapted version of the ABOS scale validated in our setting.

10.1.2. Instruments with measures related with eating disorders

One of the most important features of eating disorders is the perception of body image. There are several tools available for its assessment, including: BSQ, BIA, BAT and BES. It is also important to evaluate eating disorders in terms of internalisation of cultural influences on the aesthetic body model (culture of thinness). To this end, the CIMEC and its revised version can be used. Another aspect that needs to be assessed is eating restraint, diet, weight, physical activity, etc. To assess these aspects and others it is important to select the most reliable and valid questionnaire, one that is also brief and specific to what it intends to measure.

BSQ

Body Shape Questionnaire. Cooper, *et al*.,1987

The BSQ is a self-report questionnaire that measures body dissatisfaction, the fear of becoming fat, self-devaluation due to physical appearance, the desire to lose weight and avoidance of situations in which physical appearance might draw others' attention.

It can also be a useful tool in the exploratory study of individuals who are at risk of developing an eating disorder, acknowledging that a body scheme disorder is only one of the symptoms of this pathology. It consists of 34 items and it is scored on a 1-to-6 Likert type scale, 34-204 being the range of scores. An overall score (the sum of direct item scores) can be obtained and 4 subscales can be derived: body dissatisfaction, fear of becoming fat, low self-esteem due to appearance and the desire to lose weight. The cut-off point for the overall score has been determined to be 105[354].

Spanish version of the BSQ

The BSQ has been adapted and validated in our population by Raich, *et al.,* 1996[355]

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BIA

Body Image Assessment. Collins, *et al.,*1991

The BIA is a visual scale in which 7 figural stimuli of children and 7 figural stimuli of young adolescents, both male and female, appear separately, representing the standard percentile curves for BMI in children, ranging from the representation of a very thin figure to an overweight figure, with a score range of 1 (thinness) to 7 (obesity), with 0.5 point increments. The BIA can obtain an indicative discrepancy index between different "me's" by subtracting the scores obtained from the real me (for example, real-ideal)[356].

Spanish version of the BIA

Spanish version developed by Sánchez, 2005

BAT

Body Attitude Test. Probst, 1995

The BAT is a questionnaire that assesses a subjective dimension of body image, specifically a disturbance in the attitude towards one's own body. This test is endorsed by good psychometric results. Although it was initially conceptualised to assess body experience and attitude towards one's own body in patients with eating disorders, it has also been used in the non-pathological population. The 20 items it is comprised of are grouped into three main factors: negative appreciation of body size, loss of familiarity with one's own body and general body dissatisfaction[357].

Spanish version of the BAT

There is a Spanish version adapted and validated in our setting by Gila, *et al*., 1999, in a sample of 165 patients with eating disorders (79 AN and 86 BN) and 220 girls from the general population. Its results indicate adequate validity and reliability[358].

BES

Body-Esteem Scale. Mendelson and White, 1982

The BES is a self-report instrument comprised of 24 items that assess body self-esteem in children over the age of 7 with acceptable reading competence. It is a one-dimensional scale with yes/no answers that gathers information regarding feelings and self-evaluation of one's appearance and evaluations attributed to others about one's appearance. The BES has acceptable reliability and validity[359].

Spanish version of the BES

There is a Spanish version adapted and validated in our setting by Sperber, *et al*, 2004[360]

CIMEC

Questionnaire of Influences of the Aesthetic Body Model

Toro, *et al*., 1994

The CIMEC was designed to measure relevant cultural influences that contribute to triggering, facilitating or justifying weight loss, especially for aesthetic or social reasons. It is comprised of 40 direct items than assess body image related anxiety, influence of social models and influence of social situations. Answers are assessed on a scale of 1 to 3 points. A higher score reflects greater influence of social models. The highest possible score is 80 and the lowest is 0, with a

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cut-off point of 23/24 points[89]. It was elaborated and validated in a sample of young Spanish females (59 anorectics and 59 control subjects), matched by age and social class. The questionnaire showed satisfactory internal consistency, as well as appropriate sensitivity (81.4%) and specificity (55.9%), making it a possibly useful screening instrument given the relationship between AN and sociocultural influences.

CIMEC-26 (abbreviated version of the CIMEC)

When the CIMEC-40 was assessed in both a clinical group and a control group, 26 items that presented statistically significant differences were alienated. These questions were used to create the CIMEC-26, from which 5 dimensions are derived: body image distress, mass media influence, influence of verbal messages, influence of social models and influence of social situations.

Results indicate that the CIMEC (CIMEC-40 and CIMEC-26) is a valid and reliable instrument to assess the influence of aesthetic body models in the Spanish population by means of certain specific sources (mass media, social models and social situations).

CIMEC-12 (CIMEC version for prepubertal children)

Spanish version developed by T Saucedo, 2000[361]

Recommendations

D	10.1.1.	Assessment of patients with eating disorders should be comprehensive and include physical, psychological and social aspects, as well as a complete assessment of risk to self. (Adopted from recommendation 2.8.1.1. of the NICE guide).
D	10.1.2.	The therapeutic process modifies the level of risk for the mental and physical health of patients with eating disorders, and thus should be monitored throughout treatment. (Adopted from recommendation 2.8.1.2. of the NICE guide).
D	10.1.3.	Throughout treatment, health care professionals who evaluate children and adolescents with eating disorders should be alert to possible indicators of abuse (emotional, physical and sexual) to ensure an early response to this problem. (Adapted from recommendation 2.8.1.3. of the NICE guide).
D	10.1.4.	Health care professionals who work with children and adolescents with eating disorders should familiarise themselves with national CPGs and current legislation regarding confidentiality. (Adapted from recommendation 2.8.1.5. of the NICE guide).

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✓	10.1.5.	The use of questionnaires adapted and validated in the Spanish population is recommended for assessment of eating disorders. At present, the following specific instruments for eating disorders are recommended: EAT, EDI, BULIT, BITE, SCOFF, ACTA and ABOS (version selection based on the patient's age and other application criteria). To assess aspects related with eating disorders, the following questionnaires are recommended: BSQ, BIA, BAT, BES and CIMEC (the selection of the version should be based on age and other application criteria).

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10.2. What instruments are useful for the psychopathological assessment of eating disorders?

(See also chapter 7, "Diagnosis")

10.2.1. Impulsiveness

BIS-11

Barratt Impulsiveness Scale, version 11. Patton, *et al.*, 1995

The BIS-11 is a self-report survey used to assess impulsiveness. It consists of 30 items grouped into four subscales: cognitive impulsiveness, motor impulsiveness, non-planned impulsiveness and total impulsiveness. These scales are scored using a 4-point Likert scale. The total score of the BIS-11 is a valid and reliable measure of impulsiveness. There are no cut-off points, although a distribution median has been proposed[362].

Spanish version of the BIS-11

There is a Spanish version adapted and validated in our setting by M Oquendo, *et al.,* 2001[363].

10.2.2. Anxiety

STAI

State-Trait Anxiety Inventory. Spielberger, *et al.*, 1970

The STAI is a self-report assessment of anxiety as a transitory state (anxiety/state; A/S) and as a latent trait (anxiety/trait; A/T). It can be applied to adolescents (aged 13 and over) and adults. The instrument consists of two parts comprised of 20 questions each. The first section (A/S) assesses a transitory emotional state, characterised by subjective, consciously perceived feelings of tension and apprehension and by hyperactivity of the autonomous nervous system. The second section (A/T) denotes relatively stable anxiety proneness and characterises individuals with a tendency to perceive situations as threatening. It has adequate validity and reliability[364].

Spanish version of the STAI
There is a Spanish version adapted to our setting by TEA Publishing House, 1982[365]

Children version of the STAI (STAI-C)
The STAI-C can be administered to children aged 9 to 15 years and takes 15 to 20 minutes. Its objective is the assessment of anxiety-state and anxiety-trait in this age group [366].

Spanish version of the STAI -C
There is a Spanish version adapted to our setting by TEA Publishing House, 1990[367]

HARS
Hamilton Anxiety Rating Scale. Hamilton, 1959
The HARS is a hetero-administered scale used to assess anxiety by evaluating anxiety, tension, neurovegetative and somatic symptoms. The scale consists of 14 items with values ranging from 0 to 4 and determines the intensity of the symptoms it describes in the past month[368].

Spanish version of the HARS
There is a Spanish version that has been adapted to our setting by A Lobo and L Chamorro, 2002[369].

CETA - Assessment of Anxiety Disorders in Children and Adolescents
Ezpeleta, *et al*.

Spanish original version developed by L Ezpeleta[370]

10.2.3. Depression

BDI or Beck
Beck Depression Inventory. Beck, *et al*.,1961
The BDI o Beck Depression Inventory is a self-report questionnaire used to assess the existence or severity of depressive symptoms. Given its proven validity and reliability both in clinical and non-clinical populations it is one of the most widely used tests. It is also useful in the screening of the general population and somatic patients. It is comprised of 21 items and its objectives are to identify typical symptoms of severe depression and estimate depression severity. The overall score is used to estimate depression severity. The score obtained ranges from 0 to 63 points and the cut-off points are as follows: 0-9 (normal), 10-18 (mild depression), 19-29 (moderate depression) and 30-63 (severe depression). It can be used in patients aged 16 and over [371].

Spanish version of the BDI or Beck depression
There is an adapted version that has been validated for the Spanish population by C Conde and E Useros, 1975[372]. J Sanz, *et al*., 2003, later adapted the Beck-II (BDI-II)[373].

HAM-D
Hamilton Depression Rating Scale. Hamilton, 1959
The HAM-D is a hetero-administered questionnaire that assesses the severity of depressive symptoms, such as insomnia, agitation, anxiety and weight loss. Since its initial publication is

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has been widely used. It consists of 21 multiple choice items. The first 17 questions contribute to the total score, while questions 18 to 21 provide more information on depression such as, for example, the presence of paranoid symptoms in the patient[368, 374].

Spanish version of the HAM-D

There is a Spanish version adapted to our setting by A Lobo and L Chamorro, 2002[369].

CDI

Children Depression Inventory. Kovacs, 1991

The CDI is the most widely used questionnaire and is highly endorsed by experts in child depression, since it has proven to be very solid from a psychometric point of view and very useful for clinical purposes. It can be applied both in the general and clinical populations, in the first case, it is used for screening and in the second it constitutes the first element of diagnosis. The general depression score obtained is comprised of two scales: dysphoria and negative self-esteem. It is a self-report scale that contains 27 items. Each item has three possible answers that are quantified on a range of 0 to 2, based on the absence or severity of symptoms. This questionnaire can be administered to a population aged 8 to 15 years, with a completion time of 10 to 25minutes[375].

Spanish version of the CDI

There is a Spanish version, adapted by TEA Publishing House[376]

10.2.4. Personality

MCMI-III – Millon Clinical Multiaxial Inventory

Millon, 1990

This tool enables the assessment and identification of patients with emotional and personal difficulties who may require a more comprehensive assessment or professional management. It consists of 175 items that asses the following scales: reliability and validity; basic personality aspects, pathological personality, moderate clinical syndromes and severe clinical syndromes. Easy to use, the interpretative procedures are computerised and the user can obtain them on the spot in the case of outpatients in mental health centres, general hospitals or private clinics for an expert report. There are cut-off points in the scales to aid decision-making when faced with behavioural disorders or clinical syndromes. Its application can be individual and collective, with completion time ranging from 20 to 25 minutes (individuals 18 years and older)[377].

Spanish version of the MCMI-III

There is a version adapted by TEA publishing house[378]

MACI (adolescent version of the MCMI-III)

The MACI (*Millon Adolescent Clinical Inventory*) has been designed to assess personality characteristics and clinical syndromes in adolescents aged 13-19 years. It is applied on an individual basis. Its specific design for adolescents contrasts with other questionnaires aimed at the adult population. The whole theoretical system it is based on and the confluence of diagnostic suggestions and elements shared with the current DSM-IV, as well as a complete validation study, make it a valuable and relevant instrument. It is especially useful for the

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assessment and confirmation of diagnostic hypotheses, for treatment planning and for measuring progress in the different phases of treatment. It is comprised of 160 items that are grouped in 27 scales that encompass three main dimensions: personality characteristics, expressed concerns and clinical syndromes. Cut-off points are provided to aid decision-making in the case of disorders or clinical syndromes and validity and control indexes (original version of the MACI).

Spanish version of the MACI

There is a version adapted by TEA publishing house[379]

TCI-R

Revised version of the *Temperament and Character Inventory*. Cloninger, *et al*., 1994

The TCI-R is a self-report instrument that quantifies seven personality dimensions and 25 secondary traits. It contains 240 items on a 5-point Likert scale. This questionnaire has been used in students, general population and clinical population. Its psychometric properties, as well as the empirical work conducted with the TCI-R, are described in the development group's manual [380].

Spanish version of the TCI-R

There is a version that has been adapted and validated in our setting by JA Gutiérrez-Zotes, *et al*., 2004, in a sample of 400 volunteers (18-65 years) from geographic areas of Madrid, Tarragona and Barcelona[381]. This group had already developed the Spanish version of the original TCI[382].

IPDE

International Personality Disorder Examination. Loranger, 1979

The IPDE is a diagnostic tool, based on a semistructured clinical interview that is compatible with ICD-10 and DSM-IV assessment criteria. Its results enable the measure of other major personality disorder categories that until now had been omitted, providing a uniform reliable diagnosis that can be internationally accepted[383].

Spanish version of the IPDE

There is a Spanish version that was developed by López-Ibor, *et al.,* 1996[384]

10.2.5. Obsessiveness

Y-BOCS

Yale-Brown Obsessive-Compulsive Scale. Goodman *et al.,* 1989

In 1989 Goodman, *et al*. designed the Y-BOCS scale for OCD defined in terms of DSM-III-R criteria. The scale measures the intensity of OCD without analysing symptom content; it assesses obsessions and compulsions separately; it is sensitive and selective in changes of symptom severity. It is not a diagnostic tool, it is quickly and easily applied, and does not confound trait and state variables. Hence, the Y-BOCS has been designed to be used in patients with an OCD diagnosis, constituting an adequate method to measure severity of symptoms and their variation due to treatment. Several comparison and validation studies have been conducted to compare instruments designed to measure obsessive-compulsive symptomatology and Y-BOCS stands

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out as the most adequate, given its higher reliability, internal consistency and sensitivity to change [385].

Spanish version of the Y-BOCS

There is a version adapted and validated in our setting by Sal, *et al.,* 2002[386]

CY-BOCS (children and adolescent version of the Y-BOCS)

Spanish version of the CY-BOCS

There is a version that has been adapted and validated in our setting by Ulloa, *et al.,* 2004[387]

Recommendation

✓	10.2.	It is recommended to use questionnaires that have been adapted and validated in the Spanish population for the psychopathological assessment of eating disorders. At present, the following instruments are recommended to carry out the psychopathological assessment of eating disorders (version selection based on age and other application conditions): - Impulsiveness: BIS-11 - Anxiety: STAI, HARS, CETA - Depression: BDI, HAM-D, CDI - Personality: MCMI-III, MACI, TCI-R, IPDE - Obsessive ness: Y-BOCS.

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11. Prognosis of Eating Disorders

Key Questions:

11.1. What is the prognosis of eating disorders?

11.2. Are there prognostic factors for eating disorders?

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11.1. What is the prognosis of eating disorders?

Anorexia nervosa

There are contradictory data on the prognosis of AN despite its long historical trajectory, motivated by several factors: follow-up studies with great temporal variability, different results depending on when these studies are performed, lack of randomised intervention studies with sufficient outcome assessment, etc.

Acknowledging the previous limitations, results derived from the review of the main studies and SRSE of AN prognosis indicate that: mean gross mortality rate is 5% with a high standard deviation (5.7) and a 0 to 22 interval[388, 389]. Gross mortality rates are generally superior to those of the general population and increase significantly with the duration of follow-up. In contrast to this data, it seems that the overall outcome in survivors improves with duration of follow-up (with the resulting therapeutic implications).

50% of AN cases resolve in complete remission[390-392]. Partial remission is observed in 20%-30% of AN cases[388, 391]. Between 10% and 20% of cases result in chronicity[389, 391, 393].

Bulimia nervosa

The natural course of BN at 5 years in patients who live in a community is as follows: each year, 33% result in remission and 33% in relapse. This information indicates a relatively poor prognosis for non-treated individuals[74].

In 6-year follow-up studies of treated individuals, 60% were determined to have good prognosis, 30% were considered partially recovered and 10% were determined to have poor prognosis[394].

Risk factors of diagnosis modification (AN to BN or vice versa)

There is a lack of prospective studies focused on the evolution of AN based on the survival analysis model. However, the following risk factors for diagnosis modification from AN to BN or from BN to AN are: low self-sufficiency or autonomy, high paternal criticism, alcohol

abuse/dependency, low sensation seeking level[395].

Binge-Eating Disorder

In follow-up studies at 5 years, 10% of cases diagnosed with BED maintained the diagnosis, 18% to 20% presented partial remission and 70% were determined to have good prognosis. The presence of binge-eating predicts weight gain. The prevalence of obesity was duplicated in the group of patients who presented disorder maintenance by the end of the study [74].

11.2. Are there prognostic factors in eating disorders?

Anorexia nervosa

– Good prognostic factors: histrionic personality traits[388], onset in adolescence[393, 396]. In the latter case, it would not exactly be a good prognostic factor but rather a better prognostic factor than onset of AN in adulthood.

– Poor prognostic factors: comorbidity with other psychiatric disorders (mood disorders, anxiety disorders, substance abuse), presence of self-induced vomiting, binge-eating or laxative abuse, obsessive-compulsive personality traits, social adjustment and disease duration[388, 395, 397].

Bulimia nervosa

– Good prognostic factors: the earlier treatment has been initiated, the better prognosis. This factor is deemed to be the best indicator of good prognosis, even more than the type or duration of given treatment[398]; it also correlates with good prognosis at the beginning of the disease in adolescence when compared to onset in adulthood[399].

– Poor prognostic factors: history of substance abuse or laxative abuse predict suicide attempts[400]; comorbidity with OCD is associated with longer disease duration[401]; history of eating conflicts and/or refusal at early stages of childhood[402]; poor psychosocial functioning and greater disturbance of body image are the factors that most influence BN relapse[403].

Binge-Eating Disorder

– Poor prognostic factors: the presence of binge-eating episodes predicts weight gain. The prevalence of obesity was duplicated in the group of patients who sustained BED by the end of the study[74]. The presence of Cluster B personality traits predicts greater frequency of binge-eating episodes[404].

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12. Legal Aspects Concerning Patients with Eating Disorders in Spain

Key Questions:

12.1. What legal procedure must be followed when a patient with an eating disorder refuses to receive treatment?

12.2. Is the informed consent of a minor with an eating disorder legally valid?

12.3. In the case of a minor with an eating disorder, what is the legal solution to the dilemma stemming from the responsibility of confidentiality, respect of autonomy and obligations towards the minor's parents or legal guardians?

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12.1. What legal procedure must be followed when a patient with an eating disorder refuses to receive treatment?

To receive a medical treatment an informed consent is needed after the patient is informed of all the pertaining information in relation to the treatment, in virtue of Article 10 of the Ethics Code [405] and Articles 4, 8 and 9 of Law 41/2002[406].

Legally, the informed consent form is a non-transferable, non-delegable personal act. It is not a formal legal act and because of this it is not regulated by the civil code. Hence, the personal informed consent of the patients is a right that must be respected at all times, save for those exceptions explicitly established by the law.

One exception to the above is the lack of capacity of the patient. Spanish law allows the physician to carry out any clinical interventions that are essential even without the consent of the affected individual (article 9.2 Law 41/2002)[406] in cases where there is severe immediate risk to the individual's physical or mental integrity, prior consultation with family members or people related to the affected individual.

The application of Law 41/2002[406] sets forth some exceptional scenarios to the valid consent of a patient aged 16-17 years, since the legal age to make health-related decisions is 16 years in the application scope of this guide.

Any treatment that might require admission to hospital (article 9.4 Ethics Code)[405] must be regulated by the organic law and provide sufficient warranties, as it represents an exception to

the freedom of articles 17 of the Spanish Constitution[407] and 5 of the Rome Convention of the 4th of November, for the protection of human rights and fundamental freedoms [408].

In cases where total hospitalisation is deemed essential without having obtained the informed consent, article 763 of Law 1/2000 of 7th of January of the Civil Procedures Rules[409] establishes the occurrence of two situations: for the condition to be a psychological/psychiatric disorder and for the affected individual to be unable to make his or her own decisions.

Two situations are foreseen:

a) To apply for a Court authorisation for voluntary admission to hospital.

b) To admit the patient first and to report the admission to the Court with jurisdiction over the case within 24 hours.

In either of these two scenarios, it is a non-contradictory procedure before a Court, being the affected individual able to express his or her agreement or disagreement by themselves and if the individual so requires by means of representation and defence. The Court will issue either an order of authorisation or denial that will be open to appeal. The Court will state in the order the obligation on the part of the physicians to inform such Court of the need to maintain the measure every six months, being possible to establish a shorter period depending upon the type of disorder.

It is the physician's ultimate responsibility to decide when to discharge the hospitalised individual and the physician shall inform the Court immediately of such decision.

Recommendation

According to current legislation	12.1	The use of legal (judicial) channels is recommended in cases where the health professional deems it appropriate to safeguard the health of the patient, observing in all circumstances the patient's right to be heard and to be conveniently informed of the process and the medical and legal measures that will be applied. The well-informed procedure not only respects the right to information but also encourages the patient's cooperation and motivation and that of his or her closer relatives in the total hospitalisation procedure.

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Current legislation

– Article 10 of the Ethics Code and Medical Deontology, 1999. OMC, *Revista del Consejo General de Colegios Médicos de España*, 1999 (66): 2.124 y 2.931[405].

– Articles 4, 8 and 9 of Law 41/2002, of 14th November, a basic regulatory law that governs the patient's autonomy and embraces rights and obligations concerning clinical information and documentation (State's Official Gazette no. 274, dated 15-11-2002, pp. 40.126-40.132)[406].

– Article 9.2 of Law 41/2002, of 14th November, a basic regulatory law that governs the patient's autonomy and embraces rights and obligations concerning clinical information and documentation (State's Official Gazette no. 274, dated 15-11-2002, pp. 40.126-40.132)[406].

– Article 9.4 of the Ethics Code and Medical Deontology, 1999. OMC, *Revista del Consejo General de Colegios Médicos de España,* 1999 (66): 2.124 y 2.931, concerning observance of the patient's freedom and the possibility to request judicial intervention, if and when necessary[405].

– Article 17.1 of the Spanish Constitution of 6th of December 1978, concerning the right to freedom and the prohibition against deprivation of freedom, save for those cases expressly stipulated by the Law, as approved by the General Courts in plenary sessions of the Congress of Deputies and the Senate held on the 31st of October 1978 and ratified by the Spanish people via referendum on the 6th of December 1978 and sanctioned by his Royal Majesty the King before the General Courts on the 27th of December 1978[407].

– Article 5 of the European Convention for the Protection of Human Rights and Fundamental Freedoms of 4th November, ratified by Spain with date 26th of September 1979 and published in the Spanish Official Gazette on the 10th of October 1979. Revised in conformity with Protocol number 11 (coming into effect date 1 November 1998), based upon which the causes for non-voluntary admission to hospital may only be regulated by law[408].

– Article 763 of Law 1/2000, of 7th of January of the Civil Procedures Rules. Non-voluntary admission to hospital due to a psychological/psychiatric disorder, which regulates two types: non-voluntary admission to hospital and emergency admission to hospital [409].

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12.2. Is the informed consent of an under-age individual suffering from ED legally valid?

Legislation accepts the legal validity of the minor's autonomy contingent upon the minor's level of maturity in accordance with Organic Law for the Protection of Minors 1/1996, of 15th of January, which regards the minor as a titleholder of rights and not just as a mere object of such rights [410].

The legal treatment (article 9.3.c. of Law 41/2002) of an informed consent issued by a minor takes into account objective considerations (age) as well as subjective considerations (the patient's capacity to discern correctly)[406].

In general, in lieu of the Spanish legislation, the physician is obliged to listen to the minor (article 10.6 of the Ethics Code[405] and article 9.3.c. of Law 41/2002[406]) and make the will of the family prevail in those cases in which the minor is unable to decide neither emotionally nor intellectually, by means of the informed consent form, under the following legal limitations:

- It is the obligation of the physician to always listen to minors aged 12 and over and to take into account their opinion contingent upon their level of mental growth and personal attitude, making them participate in as much as possible in the making of decisions throughout the health process.

- Over the age of 16, the Law understand that the consent must be rendered directly by the patients in all cases, save for situations that pose severe immediate risk to health, in which the physician shall only inform and listen to the parent's views and opinions.

Recommendation

According to current legislation	12.2	One characteristic symptom of EDs and specifically of AN is the absence of awareness of the disease among sufferers. The disease itself often causes a lack of sufficient judgement to issue a valid and unbiased consent concerning the acceptance and choice of treatment. Hence, in the assumptions of severe risk to the health of a minor afflicted with AN who refuses treatment, established legal and judicial channels must specially be followed.

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Current legislation

- Organic Law 1/1996, of 15th of January, for Legal Protection of Minors, partial amendment of the Civil Code and the Civil Procedures Rules Law. Spanish Official Gazette, 17th January 1996 (number 0015)[410].

- Article 9.3.c. of Law 41/2002, of 14th November, a basic regulatory law governing the patient's autonomy and the rights and obligations with regard to clinical information and documentation (Official Gazette no. 274, de 15-11-2002, pp. 40.126-40.132)[406].

- Article 10.6 of the Ethics Code and Medical Deontology, 1999. OMC, *Revista del Consejo General de Colegios Médicos de España*, 1999 (66): 2.124 y 2.931, concerning observance of the patient's freedom and possibility to apply for the intervention of the Court, in cases where deemed necessary[405].

12.3. In the case of a minor diagnosed with ED, how is the dilemma: duty of confidentiality, respect to autonomy, and obligations with regard to the parents or legal guardians of the minor solved?

The controversy between the knowledge and authorisation from the parents or legal guardians and the right to privacy and confidentiality of the minor (article 7 of Law 41/2002)[406], is present in all norms and regulations and there is not one single norm that serves as a guide to the physician to generalise cases. In general, regulation tends to be inspired on some general principles concerning respect to privacy and professional secret, and to establish certain limitations and exceptions to the main general principle.

The physician may –according to article 16.1.d) of the Ethics Code[405]– disclose the secret prior authorisation from the patient or without such authorisation in cases in which the physician's silence may cause damage to the actual patient or to third parties.

Likewise, in an extended reading of the Civil Code on how the minor must be regarded, it is important for the physician to take into account the minor's age and maturity level.

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Recommendation

According to current legislation	12.3	The balance among the different rights in conflict makes it mandatory for the physician to observe and to interpret the best solution to each case. Nonetheless, it is always of outmost importance to inform and to listen attentively to both sides so that they understand the relationship between safeguarding health and the decision taken by the physician.

Current legislation

- Article 7 of Law 41/2002, of 14th November, a basic regulatory law governing the patient's autonomy and the rights and obligations with regard to clinical information and documentation (Official Gazette no. 274, de 15-11-2002, pp. 40.126-40.132)[406].

- Article 16 of the Ethics Code and Medical Deontology, 1999. OMC, *Revista del Consejo General de Colegios Médicos de España*, 1999 (66): 2.124 y 2.931, concerning exceptions to the physician's professional secret[405].

- Law 41/2002, of Law 41/2002, of 14th November, a basic regulatory law that governs the patient's autonomy and embraces rights and obligations concerning clinical information and documentation (State's Official Gazette no. 274, dated 15-11-2002, pp. 40.126-40.132)[406].

- Instrument of ratification for the Convention for the Protection of Human Rights and the Dignity of Human Beings, with regard to the applications of biology and medicine (Convention relating to Human Rights and Biomedicine) signed in Oviedo on the 4th of April 1997. Official Gazette number 251 of 20th October 1999[411].

- European Convention for the Protection of Human Rights and Fundamental Freedoms of 4th November, ratified by Spain with date 26th of September 1979 and published in the Spanish Official Gazette on the 10th of October 1979. Revised in conformity with Protocol number 11 (coming into effect date 1 November 1998)[408].

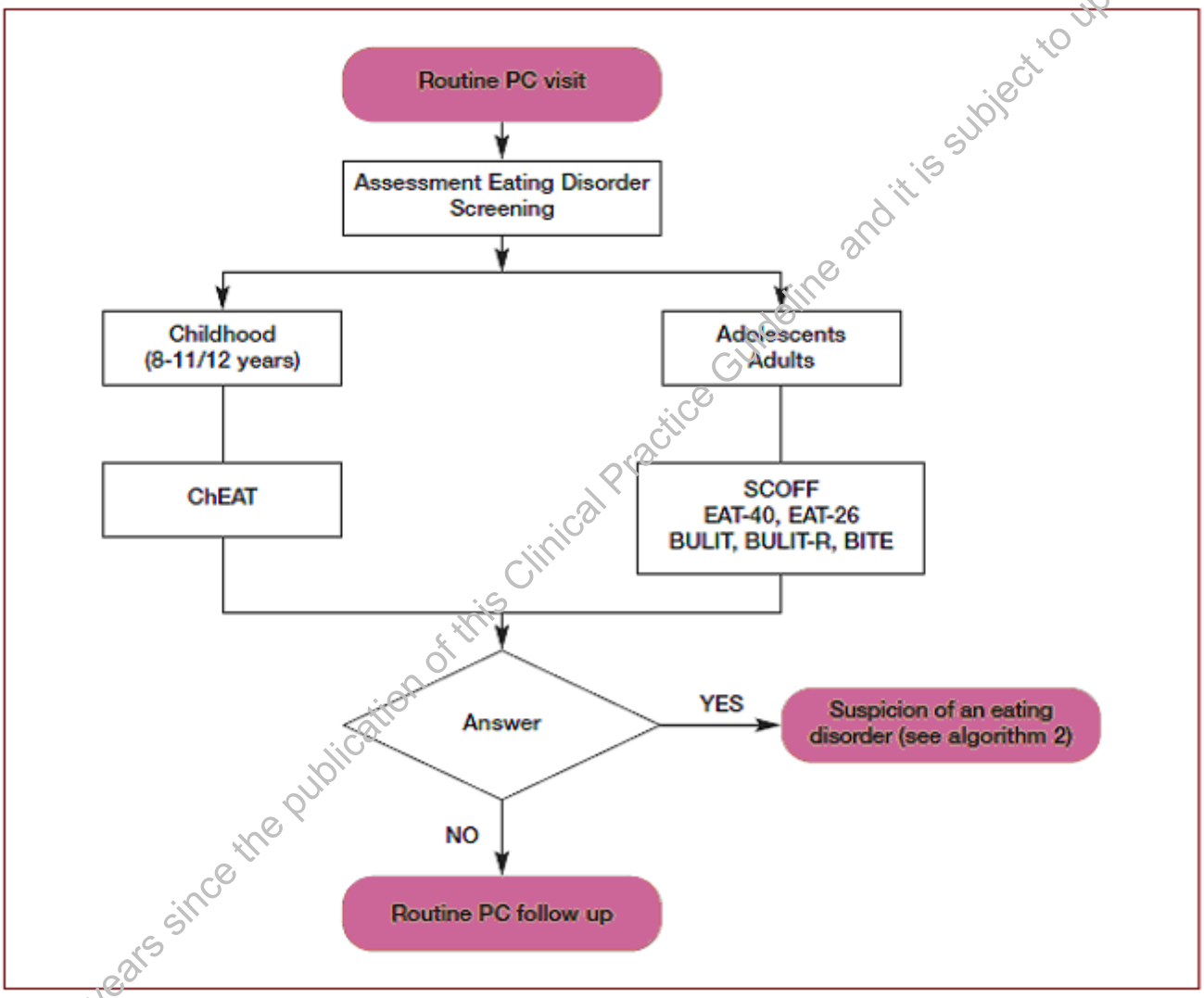
- Law 1/2000, of 7th of January, of Civil Procedures Rules (Official Gazette. 7, of 8th January 2000, pp. 575-728. Error corrections that appeared in the Official Gazette no. 90, de 14-04-2000, p. 15.278 and Official Gazette no. 180, dated 28-07-2001, p. 27.746)[409].

- Order 129/99 issued by the Constitutional Court concerning circumstances for non-voluntary admission to hospital.[412]

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

13. Detection, diagnosis and treatment strategies for eating disorders

Algorithm 1. Detection of potential cases of eating disorders



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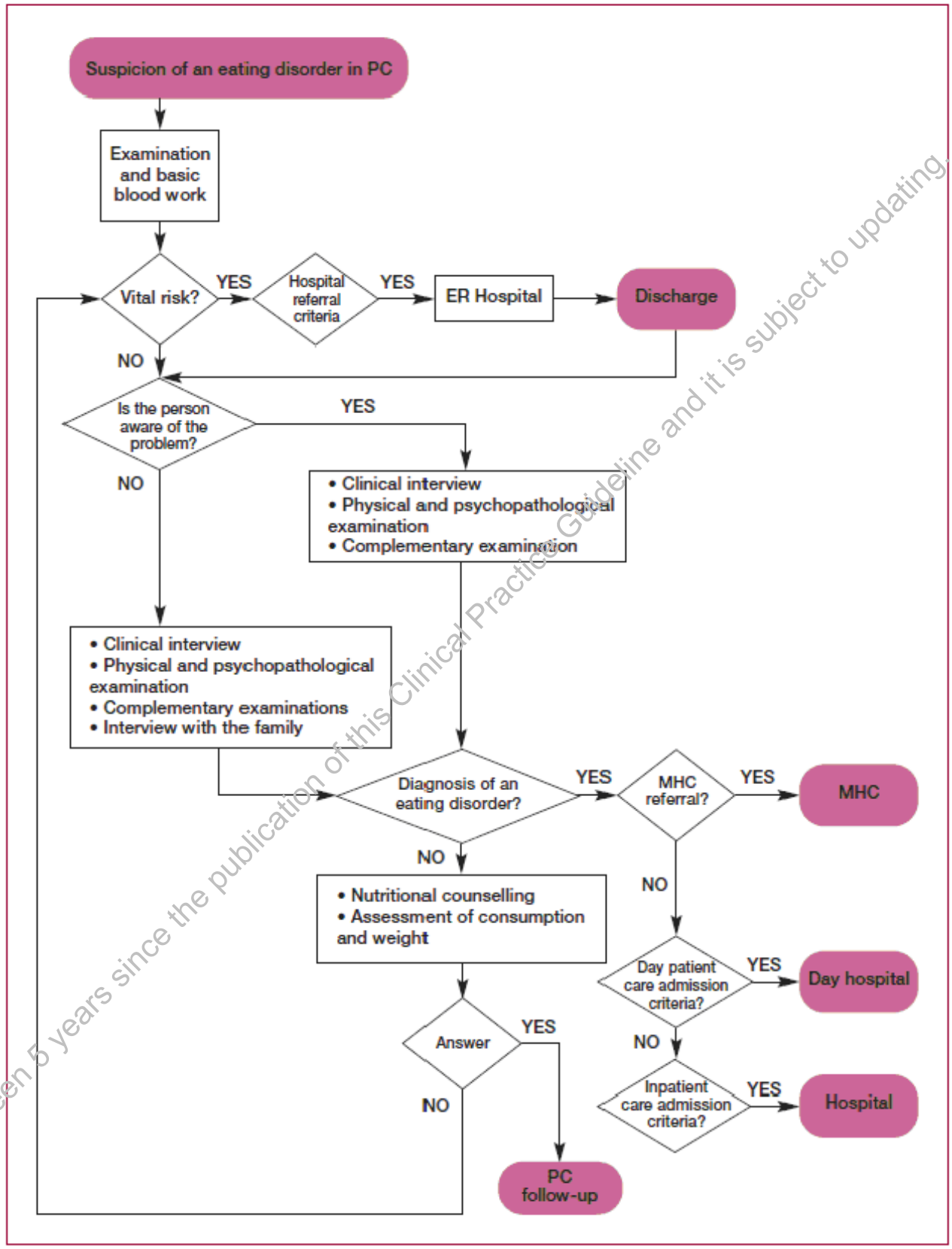
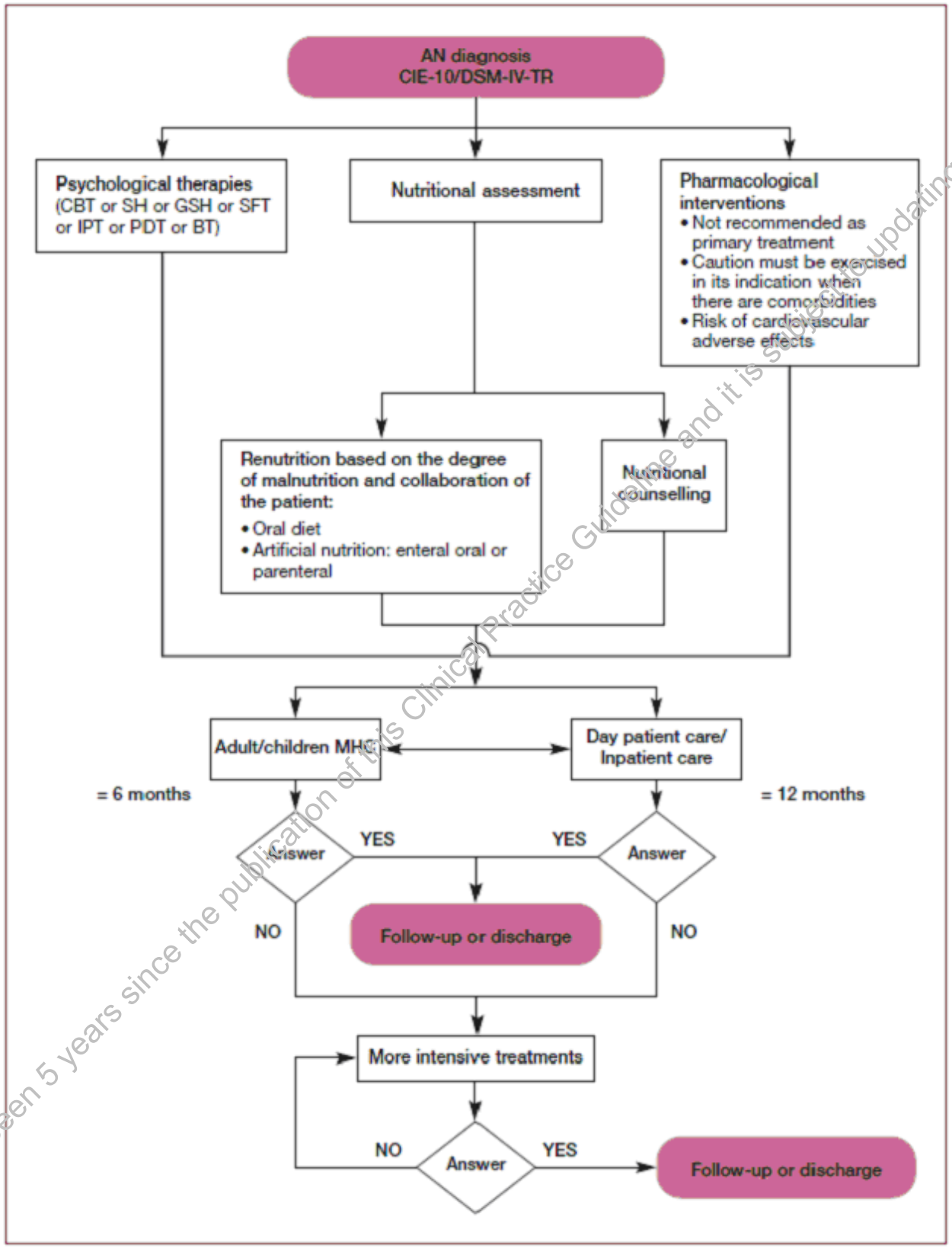
Algorithm 2. Intervention if there is suspicion of an eating disorder



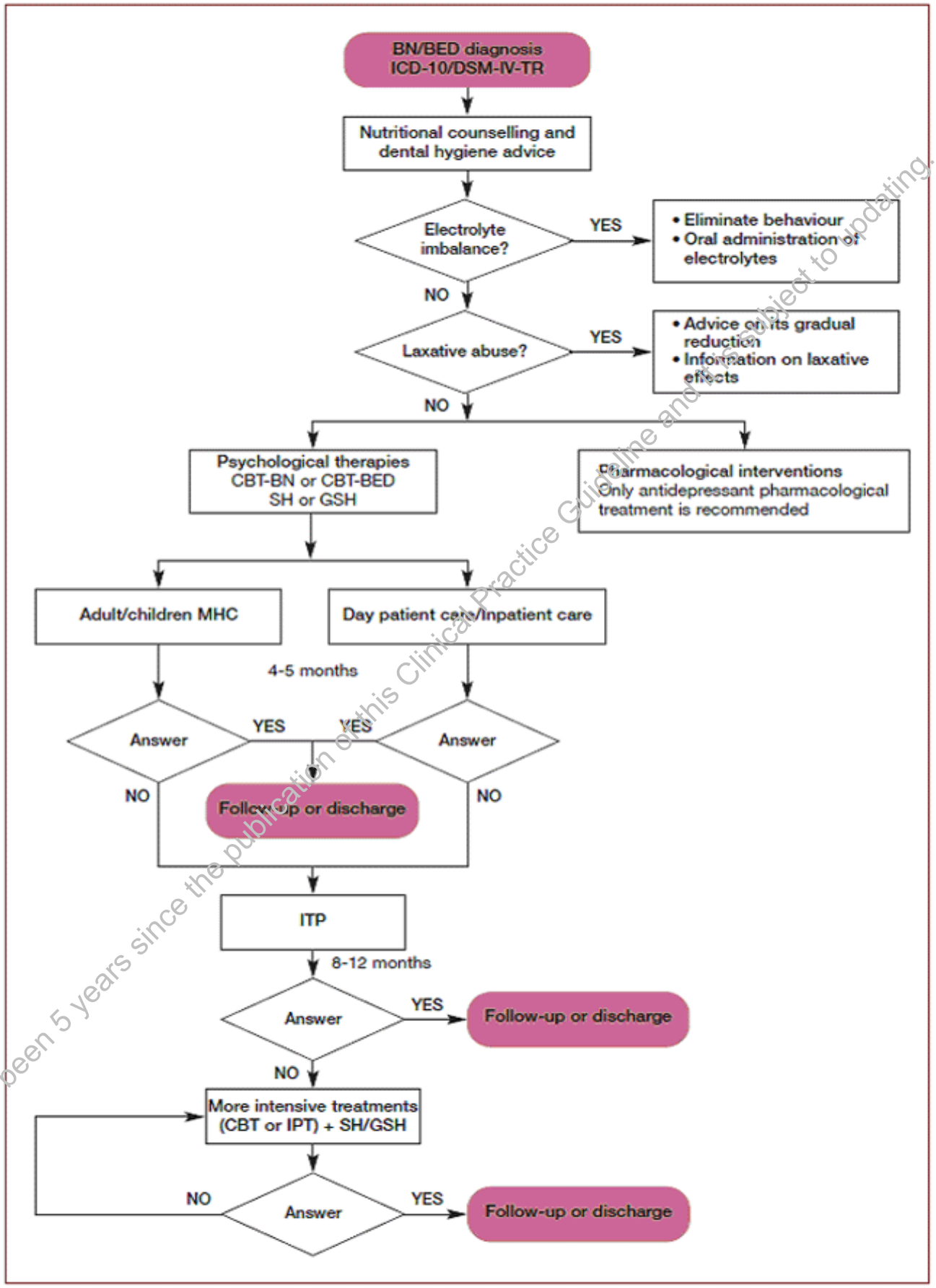
It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

Algorithm 3. Treatment of AN



It has been 5 years since the publication of this Clinical Practice Guideline and it is slightly outdating.

Algorithm 4. Treatment of BN and BED



It has been 5 years since the publication of this Clinical Practice Guideline and is subject to updating.

Algorithm Notes

Algorithm 1. Detection of potential cases of eating disorders

1) Visits of healthy children/adolescents and visits prior to participating in sports, for example, would be a good opportunity to carry out integrated prevention (primary) and screening of different disorders, including eating disorders.

2) Screening instruments are useful for a quick, first assessment aimed at ruling out the existence of suspicious symptoms in the first phase of the two-phase screening process, in which individuals who obtain high scores are re-assessed to determine if they fulfil formal diagnostic criteria.

3) Screening instruments are inefficient at establishing the diagnosis of an eating disorder.

4) In order to identify potential cases of eating disorders, several different screening self-reported questionnaires have been designed that enable systematic assessment of eating behaviour.

5) It is recommended to use questionnaires that have been adapted and validated in the pulation to detect cases (screening) of eating disorders. The following instruments are recommended (✓Good clinical practice). Recommendation 6.5.:

   – Eating disorders in general: SCOFF (for individuals aged 11 years and over).
   – AN: EAT-40, EAT-26 and ChEAT (the latter for individuals aged between 8 and 12 years).
   – BN: BULIT, BULIT-R y BITE (the three for individuals aged 12-13 years and over).

Algorithm 1 abbreviations

PC = Primary Care
BITE = Bulimic Investigatory Test, Edinburgh
BULIT = Bulimia Test
BULIT-R = revised version of the BULIT
ChEAT = Children's version of the EAT-26
EAT-40 = Eating Attitudes Test
EAT-26 = Abbreviated version of the EAT-40
SCOFF = *Sick, Control, One, Fat, Food questionnaire*
EDs = Eating Disorders

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Algorithm 2. Intervention if there is suspicion of an eating disorder

1) If a person attends the primary care practice presenting symptoms of a suspected eating disorder, the clinician must assess whether he/she is in a borderline biological or mental situation after performing basic examinations and blood work.

2) If the person seeking help is aware of the problem, the standard diagnostic procedure must be carried out, including anamnesis, physical and psychopathological examinations and complementary examinations (✓Good clinical practice). Recommendation 7.2.4.

3) Although communication must always be established with the family, the health care practitioner must study the patient's family environment and generate a climate of trust and confidentiality. When it is the family that seeks help, it becomes a key piece in the physician-patient relationship.

4) It is recommended to follow the WHO's (ICD-10) and the APA's (DSM-IV or DSM-IV-TR) diagnostic criteria. (✓Good clinical practice). Recommendation 7.1.

5) NC is provided with the aim of modifying what the patient eats, as well as maladaptive eating habits and attitudes. The advice given includes the description of a healthy diet, the benefits of maintaining a regular eating schedule, eating three meals a day, eating normal rations according to age, eating with the family, in a relaxed environment without distractions, without being the one to prepare the meal or staying in the kitchen and resting after meals, amongst others. Weight restoration requires a normocaloric and healthy diet except in cases where it is contraindicated due to the patient's condition.

6) Criteria for referral from PC to the hospital (emergency service of a general hospital) depend on whether the emergency is medical or psychiatric.

Criteria for referral from PC to emergency hospitalisation (emergency service of a general hospital) to receive emergency medical treatment are as follows:

– Weigh loss >50% in the last 6 months (30% in the last 3 months)
– Consciousness disturbances
– Convulsions
– Dehydration
– Severe liver or kidney disturbances
– Pancreatitis
– Decreased potassium <3 mEq/l or sodium (<130 or >145)
– Serious arrhythmia or conduction disorder
– Bradycardia <40 bpm
– Other ECG disorders
– Syncopes or hypotension with SBP <70 mmHg
– HDH: hematemesis, rectal bleeding
– Acute gastric dilation

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Criteria for referral from PC to emergency psychiatric assessment (psychiatric service of a hospital) are as follows:

– Absolute refusal to eat or drink.
– Depressive symptomatology, with autolytic risk.
– Significant self-inflicted injury behaviour.

7) Criteria for referral from PC to adult or child/adolescent mental health centres/units are as follows:

– When there is an established diagnosis of eating disorder.
– Weight loss equal to or higher than 10%-25% of weight, without a justifiable cause.
– Presence of regular bulimic episodes, meaning binge-eating behaviour and/or persistent purging practices (self-induced vomiting, laxative abuse and use of diuretics).
– Presence of associated psychopathological disturbances.
– Lack of disease awareness.
– If neither weight nor bulimic behaviour improve after following PC guidelines.

8) Complete hospitalisation (inpatient care) criteria:

– The biological state entails serious risk of complications (no consumption of food or liquids, BMI <16, ionic alterations, repeated self-induced vomiting, laxative abuse, use of diuretics, hematemesis and rectal bleeding).
– There are significant depressive symptoms and autolytic risk.
– Significant self-inflicted injury behaviour.

These are not absolute criteria and, depending on their intensity, partial hospitalisation (day patient care) may be indicated.

There are other psychopathological disorders that prevent outpatient treatment:

– The patient is unable due to her psychopathological state to follow the guidelines of the outpatient treatment programme: frequency of visits, limitation of physical activity, recommended diet, etc.
– The patient's problematic behaviour at home, the existence of family conflicts and/or family psychopathology is excessive and impossible to manage on an outpatient basis.
– Weight progress does not follow the rate established in the weight restoration programme.

If the patient refuses to undergo inpatient treatment, the parents' and judge's authorisation in the case of minors, and judicial authorisation in the case of over-age patients, will be required.

Inpatient treatment can be performed in a general hospital (or one specialised in eating disorders), the psychiatric hospital being the most recommended resource in special cases such as those involving chronicity and serious mental disorders (delirium, repeated self-aggression, cognitive deterioration, etc.)

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

9) Day care admission criteria:

– From the adult or children's mental health centre. If the patient does not meet emergency medical care or emergency inpatient treatment in a psychiatric hospital criteria and does fulfil one of the following criteria:
  - The patient is unable due to her psychopathological state to follow the guidelines of the outpatient treatment programme: frequency of visits, limitation of physical activity, recommended diet, etc.
  - There are serious behavioural problems in the patient's home, family conflicts and/or family psychopathology that are not modified on an outpatient basis.
  - Weight progress does not follow the rate established in the outpatient weight restoration programme.

– From inpatient care (complete hospitalisation) (once discharge criteria have been fulfilled). More control must be exercised on eating and diet and disordered behaviour and though it can also be performed on an outpatient basis, it is not indicated if there are serious behavioural problems in the patient's home, family conflicts and/or history of family psychopathology that cannot be modified by outpatient management.

Algorithm 2 abbreviations

PC = Primary care
MHC = Mental health centre
NC=Nutritional counselling
DH = Day hospital
NC = Nutrional counselling
EDs = Eatind Disorders

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Algorithm 3. Treatment of AN

1) It is recommended to follow the WHO's (ICD-10) and the APA's (DSM-IV or DSM-IV-TR) **diagnostic criteria** to establish the diagnosis of AN. (✓Good clinical practice). Recommendation 7.1.

2) **Nutritional or dietary counselling**. This type of intervention aims mainly to modify what the patient eats, as well as maladaptive eating habits and attitudes, providing a model to follow (description of a healthy diet, the benefits of maintaining a regular eating schedule, eating three meals a day, eating normal rations according to age, eating with the family, in a relaxed environment without distractions, without being the one to prepare the meal or and resting after meals, amongst others.). A normocaloric and healthy diet is required for weight restoration, except in cases where it is contraindicated due to the patient's condition.

– In feeding guidelines for children and adolescents with anorexia nervosa, carers should be included in any dietary information, education and meal planning. (Grade D). Recommendation 9.GM.1.

3) **Renutrition**

– Performance of standard treatments to resolve the situation acknowledging individual needs, especially in the case of children and adolescents. (Grade D). Recommendation 9.GM.1.

– A physical exploration and in some cases oral multivitamin and/or mineral supplements are recommended, both in outpatient and inpatient care, for patients with AN who are in the stage of body weight restoration. (Grade D). Recommendation 9.1.1.1.

– Feeding against the will of the patient should be used as a last resort in the management of AN. It is an intervention that must be performed by experts in the management of eating disorders and related clinical complications. Legal requirements must be taken into account and complied with when deciding whether to feed a patient against his/her will. (Grade D). Recommendations 9.GM.2, 9.GM.3 and 9.GM.4.

Total parenteral nutrition should not be used in patients with AN unless the patient refuses nasogastric feeding and/or when there is gastrointestinal dysfunction. (Grade D). Recommendation 9.1.1.2.

– Nutritional support for patients with eating disorders will be selected based on the patient's degree of malnutrition and collaboration, and always with the psychiatrist's approval. (✓Good clinical practice). Recommendation 9.GM.01.

– Before initiating artificial nutrition the patient's degree of collaboration must be assessed and an attempt must always be made to convince him/her of the benefits of natural feeding. (✓Good clinical practice). Recommendation 9.GM.02.

– In day hospitals, nutritional support for low-weight patients, where an oral diet is insufficient, can be supplemented with artificial nutrition (oral enteral nutrition). To ensure its intake, it must be administered during the day hospital's hours, providing supplementary energy ranging from 300 to 1,000 kcal/day. (✓Good clinical practice). Recommendation 9.GM.03.

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- Oral nutritional support in eating disorder inpatients is deemed adequate (favourable progress) when a ponderal gain greater than 0.5 kg per week is produced, with up to 1 kg increments being the usual during that period. Sometimes, when the patient with moderate malnutrition resists resuming normal feeding, the diet can be reduced by 500-700 kcal and be supplemented by complementary oral enteral nutrition in the same amount, which must be administered after meals and not instead of meals (✓Good clinical practice). Recommendation 9.GM.04.
- In the case of severe malnutrition, extreme starvation, poor progress or lack of cooperation of the patient in terms of eating, artificial nutrition treatment is indicated. If possible, an oral diet with or without oral enteral nutrition is always the first step, followed by a 3 to 6 day period to assess the degree of collaboration and medical-nutritional evolution. (✓Good clinical practice). Recommendation 9.GM.05.
- Regarding estimated energetic requirements, it is recommended that caloric needs at the beginning always be below the usual, that real weight, as opposed to ideal weight, is used to make the estimation and that in cases of severe malnutrition energetic requirements be 25 to 30 kcal/kg real weight or total kcal not higher than 1,000/day. (✓Good clinical practice). Recommendation 9.GM.06.

4) **Pharmacological interventions**

- Pharmacological treatment is not recommended as the only primary treatment of AN. (Grade D). Recommendation 9.GPH.1.
- Caution should be exercised when prescribing pharmacological treatment for patients with AN who have associated comorbidities such as obsessive-compulsive disorder (OCD) or depression. (Grade D). Recommendation 9.GPH.2.
- If drugs with adverse cardiovascular effects are administered, ECG monitoring of patients should be carried out. All patients with AN must be warned of the side effects of pharmacological treatments. (Grade D). Recommendations 9.GPH.4 and 9. GPH.5.

5) **Psychological therapies**

The objective of psychological treatment is to reduce risk, to encourage weight gain by means of a healthy diet, to reduce other symptoms related with eating disorders and to facilitate physical and psychological recovery. (Grade D). Recommendation 9.GP.3.

The psychological therapies that should be considered for AN are: CBT, SFT, IPT, PDT and BT. (Grade D). Recommendation 9.GP.1.

- The duration of psychological treatment should be of at least 6 months when performed on an outpatient basis (with physical monitoring) and 12 months for individuals who have been under inpatient management. (Grade D). Recommendation 9.GP.5.
- For patients with AN who have undergone outpatient psychological therapy but have not improved or have deteriorated, the indication of more intensive treatments (combined individual and family therapy, day or inpatient care) must be considered. (Grade D). Recommendation 9.GP.6.

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– Following hospital discharge, patients with AN should be offered outpatient care that includes monitoring of normal weight restoration and psychological intervention that focuses on eating behaviour, attitudes to weight and shape and the fear of social response regarding weight gain, along with regular physical and psychological follow-up. Follow-up duration must be of at least 12 months. (Grade D). Recommendation 9.GP.10.

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Algorithm 4. Treatment of BN and BED

1) It is recommended to follow the WHO's (ICD-10) and the APA's (DSM-IV or DSM-IV-TR) **diagnostic criteria** to establish the diagnosis of BN and BED. (✓Good clinical practice). Recommendation 7.1.

2) **Nutritional counselling and dental hygiene advice**

This type of intervention aims mainly to modify what the patient eats, as well as maladaptive eating habits and attitudes, providing a model to follow (healthy diet, maintenance of a fixed eating schedule, eating three meals a day, eating normal rations according to age, eating with the family in a relaxed environment without distractions, without being the one to prepare the meal and resting after eating).

In the case of laxative misuse, patients with BN must be advised on how to decrease and stop abuse. This process must be carried out gradually. Patients must also be informed that the use of laxatives does not decrease nutrient absorption. (Grade D). Recommendation 9.GM.8.

When electrolyte imbalance is detected, in most cases elimination of the behaviour that caused it is sufficient to correct the problem. In a small number of cases, oral administration of electrolytes whose plasmatic levels are insufficient is necessary to restore normal levels, except in cases involving gastrointestinal absorption. (Grade D). Recommendation 9.GM.7.

Patients who vomit habitually must have regular dental check-ups and be provided with dental hygiene advice. (Grade D). Recommendation 9.GM.9.

3) **Psychological therapies**

– CBT-BN is a specifically adapted form of CBT and it is recommended that 16 to 20 sessions are performed over 4 or 5 months of treatment. (Grade A). Recommendation 9.3.2.1.1.

Patients with BN who do not respond to or refuse to receive CBT treatment may be offered alternative psychological treatment. (Grade B). Recommendation 9.3.2.1.2.

Adolescents with BN can be treated with CBT adapted to their age, level of development, and, if appropriate, the family's intervention can be incorporated. (Grade D). Recommendation 9.3.2.1.3.

Adult patients with BED can be offered a specifically adapted form of CBT. (Grade A). Recommendation 9.3.3.1.

– A possible first step in the treatment of BED is to encourage patients to follow a SH programme (guided or not). (Grade B). Recommendation 9.GP.13.

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Health care professionals can consider providing BED patients with SH programmes (guided or not) that may yield positive results. However, this treatment is only effective in a limited number of patients with BED (Grade B). Recommendation 9.GP.14.

- If there is a lack of evidence to guide the care of patients with EDNOS or BED, health care professionals are recommended to follow the eating disorder treatment that most resembles the eating disorder the patient presents. (Grade D). Recommendation 9.GP.15.

- IPT should be considered an alternative to CBT although patients should be informed that it requires 8 to 12 months to achieve results similar to those obtained with CBT. (Grade B). Recommendation 9.5.2.1.

IPT-BED can be offered to patients with persistent BED. (Grade B). Recommendation 9.5.3.1.

4) **Pharmacological interventions**

- In the treatment of BN pharmacological treatments other than antidepressants are not recommended (Grade B). Recommendation 9.9.2.1.2.

Patients should be informed that antidepressant treatment can reduce the frequency of binge-eating and purging but effects are not immediate. (Grade B) Recommendation 9.9.2.1.1.

The dose of fluoxetine used in patients with BN is greater than the dose used for treating depression (60 mg/day). (Grade D). Recommendation 9.9.2.1.3.

Amongst SSRI antidepressants, fluoxetine is the first-choice drug for treatment of BN, in terms of acceptability, tolerability and symptom reduction. (Grade D). Recommendation 9.9.2.1.4.

- SSRI antidepressant treatment can be offered to a patient with BED, regardless of whether he/she follows a guided SH programme or not. (Grade B). Recommendation 9.9.3.1.1.

Patients must be informed that SSRI antidepressant treatment can reduce the frequency of binge-eating, but the duration of long-term effects is unknown. Antidepressant treatment may be beneficial for a small number of patients. (Grade B). Recommendation 9.9.3.1.2.

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14.Dissemination and implementation

In order for this CPG to reach health care professionals in the NHS, its dissemination will be carried out by means of the GuíaSalud Catalogue (www.guiasalud.es) and CAHTA's website (www.aatrm.net).

Once the national dissemination Plan within the general framework of GuíaSalud has been completed, the guide's working group and CAHTA will perform further dissemination activities that are deemed appropriate.

The CPG consists of three versions for health care professionals: the full version, the summary and the quick version. The first two include information for patients (Annex 3). The CPG is edited electronically and is available on the GuíaSalud and CAHTA websites. The summarised and quick versions are also in book and leaflet form, respectively. The book contains the CD-ROM of all versions.

The measure of adherence or implementation of the CPG's recommendations by means of monitoring and/or auditing can improve its use. The manual of the AGREE instrument addresses the importance of elaborating indicators, item 21 of the "applicability" dimension tackling this issue[45]. Hence, a CPG must offer a list of clear quantifiable criteria or quality indicators that derive from the key recommendations in the guide. The most well known and widely used indicator classification system in this guide is the one pertaining to Donabedian[413], which classifies indicators into: structure, process and outcome. In order to determine and assess the performance of the most important recommendations, the assessment of certain intervening process variables and the most relevant clinical outcomes is suggested.

In the clinical assessment of eating disorders it is recommended to measure key aspects related with quality for which certain indicators are initially proposed due to their validity, reliability and feasibility at different levels of care (primary care and specialised care).

Table 1 describes the 11 proposed indicators according to clinical area, type of indicator, the dimension of quality they address and the care level where they may be applied. It is important to borne in mind that, in practice, available indicators are not perfect and constitute an approximation of a real situation. Their objective is to provide useful information to facilitate decision-making. They are quantitative measures, which, if obtained periodically, enable analysis of their evolution over time (monitoring) [1].

Some of the indicators included in the Mental Health Strategy of the NHS (Quality Plan) [414] are common to eating disorders. Therefore, some of the indicators proposed are common to those included in the above-mentioned plan. Others have been adopted from the Contract Programme of the Regional Ministry of Andalusia/Andalusian Health Service[415] and the NICE CPG[30]. Additionally, the working group has proposed others.

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Table 1. Proposed indicators

Area	Type of indicator	Name of the indicator (standard)	Quality dimension	Health care level susceptible to application		
				1	2	3
Referral	Process	Proportion of patients with eating disorders who have been adequately referred to a specialised care level (See Annex 2.10.)	Adequacy	x		
Referral	Process	Proportion of patients with eating disorders who have been adequately referred with emergency criteria (See Annex 2.10.)	Adequacy	x	x	
Referral	Process	Average time elapsed between hospital discharge and the first outpatient (adult or children MHC) or day care follow-up visit (standard <15 days)	Continuity of care	x	x	
Diagnosis	Process	Proportion of patients who receive diagnostic confirmation within two months of the first consultation. (see Annex 2.10.)	Adequacy / Resolution	x	x	x
Treatment	Process	Proportion of patients diagnosed with an eating disorder who remain in treatment 6 or more months (standard >70%)	Adherence to treatment	x	x	x
Follow-up	Process	Proportion of patients diagnosed with an eating disorder during outpatient monitoring in day care or in the adult or children MHC who are discharged based on the number of months of follow-up (standard >60%)	Effectiveness/ Resolution	x	x	
Follow-up	Process	Number of interviews with family members in relation to visits for patients diagnosed with an eating disorder (standard 1/month)	Integral care	x	x	x
Follow-up	Outcome	Percentage of readmissions to the general hospital at 3, 6, 9 and 12 months. (See Annex 2.10.)	Effectiveness/ Continuity of care		x	x
Diagnosis	Outcome	Proportion of patients seen with an eating disorder diagnosis in PC, adult or children MHC, day hospital and general hospital. (see Annex 2.10.)	Disease management	x	x	x

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Integral care	Outcome	Satisfaction index of patients and family members by the end of treatment (see Annex 2.10.)	Satisfaction	x	x	x
Integral care	Outcome	Number of complaints (standard <5%)	Satisfaction	x	x	x

In Annex 2.10. some of the indicators proposed by this guide's working group are described. The authors did not intend to design a comprehensive and detailed assessment that entails the use of all proposed indicators. Quite the opposite, the aim is to provide clinicians and managers who may be interested with a tool that can be useful in the specific design of care assessment.

Those in charge of assessing the impact of the CPG and management of patients with eating disorders will have to choose the most appropriate sources of information, as well as the most convenient time frame that each indicator refers to (in some, however, both aspects are established). The standard use is provided for some, but in others it is yet to be determined.

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15. Future research recommendations

The following studies are required:

Studies that clarify the benefits, *versus* potential risks, of primary prevention programmes in schools and in mass media.

Studies that describe the value of preventive interventions (screening and risk factors) in childhood and adolescence, considered the highest risk group for developing eating disorders.

Studies that improve evidence regarding the choice of the treatment prescribed, the selection of other specific instruments and the expected duration and intensity of treatment in order to achieve the best results (immediate and long-term), based on clearly defined clinical indicators and on a more precise description of the stages of these disorders.

Studies that determine predisposing and precipitating biological and genetic risk factors for eating disorders. Also determine environmental, neuropsychological and personality risk factors that serve as precursors-protectors of eating disorders.

Studies that address proper methods for the treatment of osteopenia, osteoporosis and other "diseases-sequelae" of AN.

In the case of AN, studies that assess specific treatments for your patients, who are probably more sensitive to treatment and should be differentiated from treatments designed for older patients and patients with more chronic diseases, since that the characteristics and response to treatment, in other diseases, varies between these groups. Large multi-centre studies with adequate strength and required given that it is hard to recruit and retain patients with AN in controlled treatment studies due to the high dropout rate.

In the case of BN, studies that help to determine predictive factors of therapeutic success-failure and early predictors of change. Better studies are also required to address the treatment of complex cases with multiple comorbidities that are so common in health care practice.

In the case of BED, combined with obesity, studies on the best sequence of treatments (for example, if BED treatment precedes, or not, weight management treatment) and on the long-term benefits of treatment in terms of eating disorder symptoms and weight.

Studies that analyse in depth the clinical-phenomenological intrinsic characteristics of EDNOS (clinical and personality) and the efficacy of specific therapeutic approaches.

Studies that analyse the clinical-phenomenological characteristics of less prevalent eating disorder groups (late onset, males, etc.) and the efficacy of specific treatments.

Studies on the development and validation of SH therapeutic programmes not only in a printed format, but also and especially using the latest technological instruments (computerised

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manuals) and remote help (online, telephone support, etc.).

Studies that analyse endophenotypes associated with eating disorders (purging cases, impulsive subgroups, etc.) and therapeutic efficacy.

Studies that analyse cognitive styles and neuropsychological processes involved in the onset and maintenance of eating disorders.

Studies that analyse the clinical impact of comorbidity (Axis I and II) on eating disorders and specific therapeutic efficacy. Additionally, it would be necessary to design and analyse specific therapeutic approaches in these patient groups.

Studies that provide an in-depth analysis of diagnosis modification and factors involved in eating disorders.

Studies on the efficacy-efficiency of health care resources: structural and management aspects.

Studies that provide an in-depth analysis of the utility of new technologies (telemedicine, Internet, short mobile text messages [sms], videogames), as an additional therapeutic tool and in specialist education and training procedures.

Studies on neuroimaging, to facilitate an understanding of the morphological and functional involvement of certain brain areas in eating disorders (biological, environmental and personality vulnerability, nutritional changes associated with eating disorders, areas that regulate fullness-hunger sensations and therapeutic efficacy).

Animal and human studies on the mechanisms that regulate eating, versus energy expenditure.

Studies on the links between physiological and psychological processes of puberty and the onset of eating disorders.

Studies on the effects of exercise, including the role of extreme exercise and eating restraint, on the onset and development of eating disorders; and the opposite, the possible protective effect of some healthy popular athletes on boys' and girls' attitudes towards exercise, diet, weight and shape.

Studies on the impact of several comorbidities (including mood disorders, anxiety disorders, disorders related with substance abuse, personality disorders and others) frequently associated with the development of eating disorders and response to treatment.

Sociological studies on social values and eating disorders.

Family studies on factors associated with the onset and maintenance of eating disorders and the impact of these disorders on other family members.

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Annex 1. Levels of Evidence and Grades of Recommendation

SIGN Levels of Evidence and Grades of Recommendation

Levels of Evidence

1++	High quality meta-analysis, systematic reviews of clinical trials or high-quality clinical trials with low risk of bias.
1+	Well conducted meta-analysis, systematic reviews of clinical trials, or well conducted clinical trials with low risk of bias.
1-	Meta-analysis, systematic reviews of clinical trials, or clinical trials with high risk of bias.
2++	High quality systematic reviews of cohort or case-control studies Cohort or case-control studies with very low risk of bias and high probability of establishing a causal relationship.
2+	Well conducted cohort or case-control studies with low risk of bias and moderate probability of establishing a causal relationship.
2-	Cohort or case-control studies with high risk of bias and significant risk of non-causal relationship.
3	Non-analytic studies such as case reports, case series or descriptive studies.
4	Expert opinion.

Grades of Recommendation

A	At least one meta-analysis, systematic review or clinical trial classified as 1++ and directly applicable to the guide's target population, or a body of evidence composed of studies classified as 1+ with high consistency amongst them.
B	Body of evidence composed of studies classified 2++, directly applicable to the guide's target population and that have been shown to have high consistency amongst them, or evidence extrapolated from studies classified as 1++ or 1+.

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C	Body of evidence composed of studies classified as 2+ directly applicable to the guide's target population and that have shown to have high consistency amongst them; or evidence extrapolated from studies classified as 2++.
D	Level 3 or 4 evidence or evidence extrapolated from studies classified as 2+.

Good clinical practice

✓ *	Recommended practice based on clinical experience and the consensus of the drafting team.

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* Occasionally the working group becomes aware that there is an important practical aspect it wishes to emphasise and for which there probably is no supporting evidence available. These cases are generally related with a certain aspect of the treatment that is considered good clinical practice and which would rarely be questioned. These aspects are considered good clinical practice points. These messages are not an alternative to evidence-based recommendations, and hence should be considered only when there is no other way to highlight the aspect in question. They are represented with the √ symbol.

Annex 2. Clinical chapters

Annex 2.1. Spanish version of the SCOFF version

Reproduced with the authors' permission (J García-Campayo, 2004)[162]

Name: _____

Sex: _____

Age: _____

1. Do you make yourself Sick because you feel uncomfortably full?

YES NO

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

YES NO

3. Have you recently lost more than 6 kg in a 3 month period?

YES NO

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

YES NO

5. Would you say that Food dominates your life?

YES NO

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Annex 2.2. Spanish version of the EAT-40

Reproduced with the authors' permission (J Castro, *et al.,* 1991)[174]

1. I like to eat with other people.
2. I prepare meals for others, but I don't eat them.
3. I get nervous when meal time approaches.
4. I am very scared of weighing too much.
5. I try not to eat even if I am hungry.
6. I am very preoccupied with food.
7. Sometimes I have engaged in bingeing, feeling I was unable to stop eating.
8. I cut my food in small pieces.
9. I take the calories of the food I eat into account.
10. I especially avoid eating foods high in carbohydrates (for example, bread, rice, potatoes).
11. I feel full after meals.
12. I feel others would like me to eat more.
13. I vomit after eating.
14. I feel very guilty after eating.
15. I am preoccupied with the desire to be thinner.
16. I exercise a lot to burn calories.
17. I weigh myself several times a day.
18. I like wearing tight-fitting clothes.
19. I like eating meat.
20. I wake up early in the morning.
21. I eat the same foods every day.
22. I think about burning calories when I exercise.
23. I have regular menstruation.
24. Others think I am too thin.
25. I am preoccupied with the idea of having body fat.
26. I take longer to eat than other people.
27. I like eating at restaurants.
28. I take laxatives (purgatives).
29. I try to eat only sugar-free food.

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30. I eat diet food.
31. I feel food controls my life
32. I exert self-control during meals.
33. I notice others pressure me to eat.
34. I spend too much time thinking and worrying about food.
35. I have constipation.
36. I feel uncomfortable after eating sweets.
37. I commit to dieting.
38. I like to feel an empty stomach.
39. I enjoy trying new and tasty food.
40. I feel like vomiting after meals.

Answer categories:

Never / Rarely / Sometimes / Often / Most of the time / Always.

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Annex 2.3. Spanish version of the EAT-26

Reproduced with the authors' permission (A Gandarillas, *et al*., 2003)[170, 171].

1. I feel anxious about the idea of being too fat.
2. I try not to eat when I am hungry.
3. Food is a habitual preoccupation for me.
4. I have experienced bingeing episodes in which I felt I could not stop eating.
5. I cut my food in small pieces.
6. I know the amount of calories contained in the foods I eat.
7. I try not to eat foods that are high in carbohydrates (bread, rice, potatoes, etc.).
8. I feel others would like to see me eat more.
9. I vomit after eating.
10. I feel very guilty after eating.
11. I am obsessed with the desire to be thinner.
12. When I play sports I think mainly about burning calories.
13. Others think I am too thin.
14. I am worried about having fat body areas and/or cellulite.
15. I take longer than others to eat.
16. I try to eat only sugar-free foods.
17. I eat diet food.
18. I feel my life revolves around food.
19. I have good self-control over food.
20. I feel others pressure me to eat more.
21. I spend too much time thinking about food.
22. I do not feel good after eating sweets.
23. I am on a diet.
24. I like to have an empty stomach.
25. I like to try new food, tasty food that is high in calories.
26. After meals I feel compelled to vomit.

Answer categories:

Always / Very often / Often / Sometimes / Rarely / Never.

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Annex 2.4. Spanish version of the ChEAT

Reproduced with the authors' permission (M De Gracia, 2008)[173].

1. I am very scared of being overweight.
2. I try not to eat even if I am hungry.
3. I think about food constantly.
4. I have sometimes engaged in bingeing, and felt I was unable to stop.
5. I cut food in small pieces.
6. I know how many calories are in the foods I eat.
7. I avoid eating foods such as bread, potatoes or rice.
8. I notice others would like me to eat more.
9. I vomit after eating.
10. I feel very guilty after eating.
11. I am preoccupied with the desire to be thinner.
12. I think about losing calories when I exercise.
13. Others think I am too thin.
14. I am worried about having body fat.
15. I take longer than others to eat.
16. I try to eat only sugar-free food.
17. I notice others pressure me to eat.
18. I spend too much time thinking and worrying about food.
19. I feel uncomfortable after eating sweets.
20. I commit to dieting.
21. I like to feel an empty stomach.
22. I enjoy trying new and tasty foods.
23. I feel like vomiting after meals.
24. I eat diet food.
25. I feel food controls my life.
26. I exert self-control during meals.

Answer categories:

Always / Most of the time / Often / Sometimes / Rarely / Never.

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Annex 2.5. Spanish version of the BULIT

Reproduced with the authors' permission (AJ Vázquez, *et al.,* 2007)[176].

Name: _____Date:

Age: _____ Current Weight: _____ Height:

Answer each of the following questions indicating the correct option with a cross. Please, respond truthfully and remember that all information will be strictly confidential.

1. Have you ever eaten uncontrollably to the point where you felt bloated?
    1. Once a month or less (or never)
    2. Two or three times a month
    3. Once or twice a week
    4. Three to six times a week
    5. Once a day or more

2. I am satisfied with my eating behaviour
    1. Agree
    2. Neutral
    3. Slightly disagree
    4. Disagree
    5. Completely disagree

3. Have you ever continued eating until you felt you were going to explode?
    1. Practically every time I eat
    2. Very frequently
    3. Often
    4. Sometimes
    5. Rarely or never

4. Would you currently call yourself a big eater?
    1. Yes, completely
    2. Yes
    3. Yes, probably
    4. Yes, possible
    5. No, probably not

5. I prefer eating
    1. At home alone
    2. At home with others
    3. At a public restaurant
    4. At friends' houses
    5. It does not matter

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6. Do you feel you have control over the food you eat?
    1. Most of the time or all the time
    2. Often
    3. Occasionally
    4. Rarely
    5. Never

7. I use suppositories or laxatives for weight management
    1. Once a day or more
    2. Three to six times a week
    3. Once or twice a week
    4. Two or three times a month
    5. Once a month or less

8. I eat until I feel too tired to continue
    1. At least once a day
    2. Three to six times a week
    3. Once or twice a week
    4. Two or three times a month
    5. Once a month or less (or never)

9. How frequently do you like to eat ice-cream, smoothies during a binging episode?
    1. Always
    2. Frequently
    3. Sometimes
    4. Rarely or never
    5. I do not overeat

10. How concerned are you about bingeing?
    1. I do not binge
    2. I am not too concerned
    3. Moderate concern
    4. Intense concern
    5. It is probably the biggest concern of my life

11. Most people I know would be surprised if they know how much food I eat every time I sit down
    1. Absolutely
    2. Very probably
    3. Probably
    4. Possibly
    5. No

12. Have you ever eaten until you felt sick?
    1. Very frequently
    2. Frequently
    3. Often
    4. Occasionally
    5. Rarely or never

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13. I am afraid of eating because I am afraid I will not be able to stop
    1. Always
    2. Most of the time
    3. Frequently
    4. Sometimes
    5. Rarely or never

14. I do not feel good about myself after eating too much
    1. Always
    2. Frequently
    3. Sometimes
    4. Rarely or never
    5. I do not eat too much

15. How frequently do you vomit intentionally after eating?
    1. Two or more times a week
    2. Once a week
    3. Two or three times a week
    4. Once a month
    5. Less than once a month (or never)

16. How do you feel after bingeing?
    1. I do not binge
    2. I feel good
    3. I feel moderately disgusted with myself
    4. I feel pretty disgusted with myself
    5. I hate myself

17. I eat a lot even when I am not hungry
    1. Very frequently
    2. Frequently
    3. Occasionally
    4. Sometimes
    5. Rarely or never

18. My way of eating is different from the way of eating of most people
    1. Always
    2. Most of the time
    3. Frequently
    4. Sometimes
    5. Rarely or never

19. I have tried to lose weight by fasting or intense dieting
    1. Not in the past year
    2. Once in the past year
    3. Two or three times in the past year
    4. Four or five times in the past year
    5. More than five times in the past year

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20. I feel sad after eating more than I intended to eat
    1. Always
    2. Most of the time
    3. Frequently
    4. Sometimes
    5. Rarely, never or it is not the case

21. When I binge I tend to eat foods high in carbohydrates (sugars, starch)
    1. Always
    2. Most of the time
    3. Frequently
    4. Sometimes
    5. Rarely, or I do not overeat

22. Compared to most people, my ability to control my eating behaviour seems to be:
    1. Greater than others'
    2. More or less the same
    3. Lesser
    4. Much lesser
    5. I have no control whatsoever

23. One of your best friends suddenly suggests that you both have dinner at a new restaurant that evening. Although you had planned to eat lightly at home, you go out for dinner, eat quite a lot and feel uncomfortably full.
    How would you feel about yourself on the way home?
    1. Good, happy about trying a new restaurant
    2. Slightly bad about eating so much
    3. Somewhat frustrated with yourself
    4. Disgusted with yourself
    5. Totally disgusted with yourself

24. At present I could consider myself a "compulsive eater" (who has episodes of uncontrolled eating)
    1. Absolutely
    2. Yes
    3. Yes, probably
    4. Yes, possible
    5. No, probably not

25. What is the most weight you have lost in a month?
    1. More than 20 kg
    2. 12-20 kg
    3. 8-11 kg
    4. 4-7 kg
    5. Less than 4 kg

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26. If I eat too much at night, I feel depressed the following morning
    1. Always
    2. Frequently
    3. Sometimes
    4. Rarely or never
    5. I do not eat too much at night.

27. Do you think it is easier for you to vomit than for most people?
    1. Yes, I do it effortlessly
    2. Yes, it is easy for me
    3. Yes, it is a bit easier for me
    4. More or less the same
    5. No, it is less easy for me

28. I feel food controls my life
    1. Always
    2. Most of the time
    3. Frequently
    4. Sometimes
    5. Rarely or never

29. I feel depressed right after eating too much
    1. Always
    2. Frequently
    3. Sometimes
    4. Rarely or never
    5. I do not eat too much

30. How frequently do you vomit after eating trying to lose weight?
    1. Less than once a month (or never)
    2. Once a month
    3. Two-three times a month
    4. Once a week
    5. Twice or more times a week

31. When you eat a large amount of food, how fast do you usually it eat?
    1. Faster than anyone ever has
    2. Much faster than most people
    3. A bit faster than most people
    4. Like most people
    5. Slower than most people (or not applicable)

32. What is the most weight you have gained in a month?
    1. More than 20 kg
    2. Between 12-20 kg
    3. Between 8-11 kg
    4. Between 4-7 kg
    5. Less than 4 kg

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33. APPLICABLE ONLY TO WOMEN. My last period was
    1. One month ago
    2. Two months ago
    3. Four months ago
    4. Six months ago
    5. Over six months ago

34. I use diuretics to help manage my weight
    1. Once a day or more
    2. Three to six times a week
    3. Once or twice a week
    4. Two or three times a month
    5. Once a month or less (or never)

35. What do you think of your appetite compared to that of most people you know?
    1. Often bigger than most
    2. Much bigger
    3. A bit bigger
    4. More or less the same
    5. Smaller than most

36. APPLICABLE ONLY TO WOMEN. I have my period once a month
    1. Always
    2. Generally
    3. Sometimes
    4. Rarely
    5. Never

PC	1	10	20	30	40	50	60	70	80	90	99

TOTAL

Exc_____

Sen _____

Von_____

Foo _____

Wei _____

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Annex 2.6. Spanish version of the BITE

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1.	Do you have a regular daily eating pattern?	YES	NO
2.	Are you a strict dieter?	YES	NO
3.	Do you experience feelings of failure when you break a diet, even if you only do so once?	YES	NO
4.	Do you count the calories in everything you eat, even if you are not on a diet?	YES	NO
5.	Have you ever fasted for one whole day?	YES	NO
6.	... if you have responded «YES» to question 5, how frequently do you do so?		
	❑ Alternate days	5	
	❑ 2-3 times a week	4	
	❑ Once a week	3	
	❑ Occasionally	2	
		1	

7. Do you use any of the following methods to lose weight? (Draw a circle around each answer based on the frequency, in accordance with the table below).

WEIGHT-LOSS PILLS	0	2	3	4	5	6	7
USE OF DIURETICS	0	2	3	4	5	6	7
USE OF LAXATIVES	0	2	3	4	5	6	7
SELF-INDUCED VOMITING	0	2	3	4	5	6	7

-Never	0
-Occasionally	2
-Once a week	3
-2 or 3 times a week	4
- Daily	5
- 2 or 3 times per day	6
- 5 or more times a day 7	7

8.	Do your eating habits severely disrupt your life?	YES	NO
9.	Do you feel that food controls your life?	YES	NO
10.	Do you ever eat and eat until you are stopped by physical discomfort?	YES	NO
11.	Are there times when all you can do is think about food?	YES	NO

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12. Do you eat reasonable amounts of food in front of others and then binge in secret? YES NO

13. Are you able to stop eating when you wish? YES NO

14. Do you ever experience an OVERWHELMING desire to eat and eat? YES NO

15. Do you tend to eat a lot when you feel anxious? YES NO

16. Are you TERRIFIED of the idea of becoming obese? YES NO

17. Do you ever eat large amounts of food (not only during meals)? YES NO

18. Do you feel ashamed of your eating habits? YES NO

19. Do you worry that you have lost control over how much you eat? YES NO

20. Do you turn to food for comfort? YES NO

21. Are you able to leave food on your plate at the end of a meal? YES NO

22. Do you deceive other people about how much you eat? YES NO

23. Do the amounts you eat directly depend on the hunger you are experiencing? YES NO

24. Do you always binge? YES NO

25. ... if you have answered «YES», do you despise yourself when you binge? YES NO

26. If you do binge, do you only do it when you are alone? YES NO

27. If you do binge, how often do you do so? YES NO

2-3 times a day	6
Daily	5
2-3 times a week	4
Once a week	3
Once a month	2
Rarely	1

28. Would you do anything to satisfy the overwhelming desire to binge? YES NO

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29. If you binge eat, do feel very guilty? YES NO

30. Do you ever eat in secret? YES NO

31. Do you think your eating habits are normal? YES NO

32. Do you consider yourself a compulsive glutton? YES NO

33. Does your weight change more than 2.5 kg per week? YES NO

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Annex 2.7. Diagnostic Criteria for Eating Disorders

ICD-10 Diagnostic Criteria for Eating Disorders

In 1992 the WHO published the ICD (*International Statistical Classification of Diseases and Related Health Problems).* The ICD is used worldwide for statistics on morbidity and mortality, reimbursement systems and automatic decision support in medicine. This system is designed to promote international comparison of the recollection, processing, classification and presentation of these statistics.

At this moment, the classification currently in vigour is the tenth edition (ICD-10) and the WHO continues to work on it, publishing minor annual updates and bigger updates every three years.

Anorexia nervosa (F50.0)

Disorder characterised by the presence of deliberate weight loss, induced or sustained by the patient. The disorder occurs most frequently in adolescent girls and young women, although on rare occasions it may affect adolescent boys and young men, as well as prepubertal children or older women up to the menopause. AN constitutes an independent syndrome, in the following sense:

a) The syndrome's clinical features are easily recognized, resulting in a reliable diagnosis with a high degree of agreement between clinicians.

b) Follow-up studies have demonstrated that, amongst patients who do not recover, a considerable number continue to show the main characteristics of AN in a chronic form.

Despite the fact that the main causes of AN remain elusive, there is growing evidence suggesting that there are a series of sociocultural and biological factors that interact and contribute to the onset of AN, in which less specific psychological mechanisms and personality vulnerability also take part. The disorder is accompanied by malnutrition of varying severity, leading to endocrine and metabolic disturbances and other functional disorders. It remains uncertain whether the characteristic endocrine disorder is entirely due to malnutrition and the direct effect of the behaviours that have caused it (for example, restricted dietary choice, excessive physical exercise with metabolic balance disturbances, self-induced vomiting and laxative abuse, with the resulting electrolyte imbalances) or if other yet unknown factors are involved.

Diagnostic Guidelines:
All the following disturbances must be present:

a) Significant weight loss (BMI<17.5 kg/cm^2). Prepubertal patients may not present the expected weight gain corresponding to the period of growth.

b) The weight loss is self-induced by: 1) avoidance of "fattening foods", and by one or more of the following symptoms: 2) self-induced vomiting, 3) self-induced intestinal purging, 4) excessive exercise and 5) use of appetite suppressants or diuretics.

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c) Body image distortion that consists of a specific psychopathology characterised by a dread of fatness and flaccidity of body areas that persists as an intrusive, overvalued idea, leading the patient to impose a low weight threshold on himself or herself.

d) A widespread endocrine disorder that affects the hypothalamic-pituitary-gonadal axis manifesting in women as amenorrhoea and in men as loss of sexual drive and potency (An apparent exception is the persistence of vaginal bleeds in anorexic women who are receiving replacement hormonal therapy, most commonly taken as a birth control pill). There may also be elevated levels of growth hormone and cortisol, changes in the peripheral metabolism of the thyroid hormone, and insulin secretion abnormalities.

e) If onset occurs before puberty, the sequence of pubertal events is delayed or even stunted (growth ceases; in girls the breasts do not develop and there is a primary amenorrhoea; in boys the genitals remain juvenile). If recovery takes place, puberty is often completed normally, but the menarche is late.

Excludes:
Anorexia, loss of appetite (R63.0).
Psychogenic anorexia (F50.8).

Atypical Anorexia Nervosa (F50.1)

This term must be used in cases where one or more key features of AN (F50.0), such as amenorrhoea or significant weight loss, are missing, but that otherwise present a rather characteristic clinical picture. These types of patients are more frequent in consultative and liaison psychiatry and in primary care. Patients who present all key symptoms of AN in a mild degree an also be included in this group. This term must not be used for eating disorders that resemble AN but that are actually a result of a known physical ethiology.

Diagnostic Criteria for Bulimia Nervosa (F50.2)

Syndrome characterised by repeated episodes of binge-eating and excessive preoccupation with the control of body weight, leading the patient to use extreme measures to mitigate the weight gain caused by ingested food. This term should be limited to the forms of the disorder that are related to AN by virtue of sharing the same psychopathology.

The age and sex distribution is similar to that of AN, but the age of presentation tends to be slightly later. The disorder may be considered a sequel to persistent AN (although the reverse sequence may also occur). At first glance, a previously anorexic patient may appear to improve as a result of weight gain and menstruation may even return in the case of a female, but a harmful pattern of behaviour characterised by overeating and self-induced vomiting then becomes established. Repeated self-induced vomiting is likely to give rise to disturbances of electrolyte balance, physical complications (litany, epileptic seizures, cardiac arrhythmias, muscular weakness), and greater loss of weight.

Diagnostic Guidelines:
All the disturbances listed below must be present, thus constituting strict diagnostic guidelines.

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Within each guideline some variations can be accepted, as is indicated:

a) There is a persistent preoccupation with eating, and an irresistible craving for food; the patient succumbs to episodes of overeating in which large amounts of food are consumed in short periods of time.

b) The patient attempts to counteract the weight gain caused by ingested food by one or more of the following methods: self-induced vomiting; laxative abuse, alternating periods of fasting; use of drugs such as appetite suppressants, thyroid preparations or diuretics. When bulimia occurs in diabetic patients they may choose to neglect their insulin treatment.

c) The psychopathology consists of a morbid dread of fatness and the patient sets herself or himself a sharply defined weight threshold, well below the premorbid weight that constitutes the optimum or healthy weight. There is often, but not always, a history of an earlier episode of AN, the interval between the two disorders ranging from a few months to several years. This earlier episode may have been fully expressed, or may have assumed a minor cryptic form with a moderate loss of weight and/or a transient phase of amenorrhoea.

Include:
Bulimia NOS (not otherwise specified).
Hyperorexia nervosa.

Diagnostic criteria for atypical bulimia nervosa (F50.3)

This term must be used in cases where one or more key features of BN (F50.2) are missing but that otherwise present a rather typical clinical picture. Patients frequently have normal weight or are even slightly overweight, but they present repeated bouts of overeating followed by self-induced vomiting or purging. Partial syndromes accompanied by depressive symptoms are not uncommon (if these symptoms fulfil the guidelines of a depressive disorder a double diagnosis must be made).

Includes:
Bulimia with normalweight.

Diagnostic Criteria for Other Eating Disorders (F50.8) and Unspecified Eating Disorders (F50.9)

Other eating disorders must be coded using the ICD-10 F50.8 code (other eating disorders).

Unspecified eating disorders must be coded using the F50.9 code (Unspecified eating disorders).

DSM-IV-TR Diagnostic Criteria for Eating Disorders

The DSM-IV (*Diagnostic and Statistical Manual of Mental Disorders IV*) of the APA (American Psychiatric Association) is a classification of mental disorders that was elaborated with the objective of providing clear descriptions of diagnostic categories, to enable clinicians and researchers to diagnose, study and Exchange information and address different mental

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disorders. In 1994, the fourth version, the DSM-IV, was published. The latest version, the DSM-IV-TR, which is currently in use, was published in 2000 and includes some revisions. At present the APA is developing the upcoming DSM-V, the publication of which is expected to be in 2011.

The DSM is an instrument based on empirical data and presenting in the form of descriptive methodology with the aim of improving communication between clinicians pertaining to different orientations and between clinicians in general with different researchers. Thus, it does not try to explain different pathologies, or to propose pharmacological or psychotherapeutic treatment guidelines or specific branches within psychology or psychiatry. It is important to clarify that professionals with clinical experience must always use it given that it is used as a guide that must be accompanied with the necessary clinical judgement, professional knowledge and ethical criteria.

The DSM-IV is a diagnostic tool that presents a description of the current functioning of the patient by means of five "axes", with the aim of providing a general view of different functional areas.

Anorexia nervosa (307.1)

Diagnostic Criteria:

a) Refusal to maintain body weight at or above a minimally normal weight for age and height: (for example, weight loss leading to maintenance of body weight <85% of that expected or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).

b) Intense fear of gaining weight or becoming obese, even though under weight.

c) Disturbance in the way one's body weight or shape are experienced, undue influence of body weight or shape on self evaluation, or denial of the seriousness of the current low body weight.

d) Amenorrhoea in postmenarchal girls and women. For example, absence of three consecutive menstrual cycles. (Amenorrhoea is defined as periods occurring only following hormone (e.g., oestrogen) administration).

Specify type:

– Restricting type: During the current episode of AN, the person has not regularly engaged in binge-eating or purging behaviour (self-induced vomiting, laxative abuse, use of diuretics, or enemas).

– Binge-eating–purging type: During the current episode of AN, the person has regularly engaged in binge-eating or purging behaviour (self-induced vomiting, laxative abuse and use of diuretics or enemas).

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Bulimia nervosa (307.51)

Diagnostic Criteria:
a) Recurrent episodes of binge eating. An episode of binge eating is characterized by:

1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.

2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

b) Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.

c) The binge eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for 3 months.

d) Self-evaluation is unduly influenced by body shape and weight.

e) The disturbance does not occur exclusively during episodes of AN.

Specify type:

- Purging Type: during the current episode of BN, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.
- Non-purging Type: during the current episode of BN, the person has used other inappropriate compensatory behaviours, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas

EDNOS (307.50)

The EDNOS category refers to eating disorders that do not satisfy criteria for any specific eating disorders. For example:

- In women, all diagnostic criteria for AN are met, but menstruation is regular (EDNOS 1).

- All diagnostic criteria for AN are met but despite substantial weight loss, the patient's weight is in the normal range (EDNOS 2).

- All diagnostic criteria for BN are met except binges and inappropriate compensatory behaviours occur at a frequency of less than twice a week or for a duration of less than 3 months. (EDNOS 3).

- All diagnostic criteria for BN are met, except an individual of normal body weight engages in the regular use of inappropriate compensatory behaviours after eating small amounts of

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food (for example, self-induced vomiting after eating two cookies) (EDNOS 4).

- Chew and spit out, but not swallow, large amounts of food.
- Compulsive disorder: characterised by repeated episodes of binge eating in the absence of inappropriate compensatory behaviours typical of BN.

Though the DSM-IV includes research criteria for later studies, BED remains classified in eating disorders not otherwise specified.

Table 1. **Comparison of diagnostic criteria: DSM-IV, ICD-9, ICD-10**

Diagnosis	Diagnostic Classifications		
	DSM-IV-TR	ICD-9	ICD-10
Anorexia nervosa	307.1	307.1	F50.0
Bulimia nervosa	307.51	307.51	F50.2
Unspecified eating disorders	307.50	307.50	F50.9

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Annex 2.8. Spanish version of the EDE-12 semistructured interview (R M Raich)[191]

The twelfth edition of Fairburn and Cooper's, 1993[190] semistructured interview (EDE-12) is the result of the gradual improvement of the interview originally designed by Cooper and Fairburn in 1987. The current version can now be used for the diagnosis of eating disorders based on DSM-IV criteria, to assess the frequency of serious behaviours present in eating disorders and the severity of other important aspects of the psychopathological characteristics of eating disorders. The EDE-12 consists of 62 items that assess 4 subscales: restraint, preoccupation with food and preoccupation with shape. Each of the patient's answers is evaluated by the interviewer on a scale of 0 to 6 points that determine intensity or frequency. Scores can be obtained for items, subscales and also an overall severity score. Although it was designed to assess the symptoms of eating pathology in the last four weeks and focuses on them, it was later modified so it could be used to assess the presence of these symptoms in the last three months [192, 193].

Annex 2.9. Incorrect ideas about weight and health[10]

– Any kind of fat in food is bad.

– It is healthy to be thin.

– Fat can be lost quickly and without posing a risk to health.

– In women there is a special kind of fat called cellulite.

– Cellulite is caused by toxins.

– The fat in a certain body area can be reduced (hips and thighs).

– Certain foods, or combination of foods, can activate metabolism and accelerate weight loss.

– Obese women under the age of 50 are at risk for heart disease.

– Dieting is a healthy activity.

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Annex 2.10. Description of proposed indicators

Proportion of patients with eating disorders who have been adequately referred to a specialised level of care

Criteria for referral from PC to mental health services (MHCFA and MHSFCA)	– When there is an established diagnosis of eating disorder. – Weight loss equal to or higher than 10%-25% of weight, without a justifiable cause. – Presence of regular bulimic episodes, meaning binge-eating behaviour and/or persistent purging practices (self-induced vomiting, laxative abuse and use of diuretics). – Presence of associated psychopathological disturbances. – Lack of disease awareness. – If neither weight nor bulimic behaviour improve after following PC guidelines.
	Preferential cases (1-3 days) and other cases (max. 1 month).
Formula:	Patients with eating disorders who have been adequately referred to a specialised level of care / patients with eating disorders x 100.
Standard:	To be determined.

Proportion of patients with eating disorders adequately referred with emergency criteria

Criteria for referral from PC to emergency hospitalisation:	– Weight loss >50% in the last 6 months (30% in the last 3 months). – Consciousness disturbances. – Convulsions. – Dehydration. – Severe liver or kidney disturbances. – Pancreatitis. – Decreased potassium <3 mEq/l or sodium (<130 o >145). – Serious arrhythmia or conduction disorder. – Bradycardia of <40 bpm. – Other ECG alterations. – Syncopes or hypotension with SBP <70 mm Hg. – HDH: hematemesis, rectal bleeding. – Acute gastric dilation.
	No delay.

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Formula:	Patients with eating disorders adequately referred with emergency diagnosis / patients with eating disorders referred with emergency diagnosis x 100.
Standard:	To be determined.

Proportion of patients who receive diagnostic confirmation within two months of the first consultation

Appropriate time interval:	Except in high risk situations, all patients with eating disorder shall have the diagnosis and individual therapeutic plan established within two months of the first consultation.
Formula:	Patients who have been diagnosed in the appropriate time frame /patients diagnosed with an eating disorder x 100.
Standard:	To be determined.

Percentage of general hospital readmissions at 3, 6, 9 and 12 months

Formula:	Number of patients who are discharged from a general hospital due to eating disorders, at 3 months-total number of discharges in that time frame / total number of discharges to a general hospital in that same time frame due to mental disorder x 100.
Definitions:	All discharges classified in the ICD-10 will be included (codes F50.0, F50.1, F50.2, F50.3; F50.8 and F50.9 of the 10th version).
Levels of disaggregation:	Per autonomous community.
Sources of information:	Registry of hospital discharges (MBDS)-MSC (Ministry of Health and Consumer Affairs).
Periodicity:	3, 6, 9, 12 months (to be determined).
Clarifications:	Discharges from general hospitals will be counted. The numerator is currently biased by not being able to identify patients who have been discharged from different hospitals until all databases of the individual health care card of autonomous communities of the NHS are integrated.

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Proportion of patients seen with the diagnosis of eating disorder in PC, adult and children's MHC (MHCFA –MHCFCA), day hospital and general hospital

Formula:	Number of patients diagnosed with an eating disorder/total population x 100.
Definitions:	Prior agreement of the autonomous communities on register and coding criteria of cases managed on an outpatient level by mental health specialised services is required. All admissions classified in the ICD-10 will be included (codes F50.0, F50.1, F50.2, F50.3; F50.8 and F50.9 of the 10th version).
Levels of disaggregation:	According to type of eating disorder, level of care (PC, MHC, day hospital and general hospital), autonomous community, age and sex.
Sources of information:	Death statistics and population projections. NSI.
Periodicity:	Annual.
Observations:	At present there are no data for the entire NHS. It requires the previous agreement of normalised registry criteria and subsequent sharing of information by the MSC.

Satisfaction index of patients and family members by the end of treatment

Formula:	Number of surveyed individuals who deemed all aspects to be adequate / total number of surveyed individuals x 100.
Definition:	All patients treated in primary and specialised care for eating disorders will be included at the end of treatment.
Levels of disaggregation:	According to type of eating disorder, autonomous community and age and sex groups.
Periodicity:	Annual.
Clarifications:	Only those individuals who refuse to complete the questionnaire will be excluded. Returned questionnaires and the characteristics of those who answered and those who did not will have to be reported. Expected response rate is 50%.

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Annex 3. Information for patients with eating disorders and their families

Annex 3.1. Patient Information

Eating Disorders. What should you know?

This information will help you gain deeper knowledge on eating disorders and understand the importance of becoming involved in treatment and in the recovery process. The aspects addressed are the definition of these disorders, their symptoms and course and recommended treatments based on available research results.

Eating Disorders

What are eating disorders?

People with eating disorders are characterised by adopting maladaptive eating behaviours as a response to personal body image dissatisfaction. They frequently present distorted thoughts on weight (they feel inferior to others, and believe they are negatively judged due to their physical appearance) and health (fat in food is harmful, it is healthy to be thin, weight can be lost quickly without posing any risks to health, dieting is a healthy activity, etc). People with these disorders suffer nutritional, physical, psychological and social consequences. If left untreated, these disorders can be life-threatening.

These eating problems can affect all people, regardless of their socio-economic or cultural situation. Eating disorders are more common in females (90-95%), but lately there has been a growing incidence in men. These disorders can occur at any age, but most commonly develop in adolescence due to the vital changes that take place during this time and the search for self-identity it entails. In the past few years earlier and earlier ages of onset of eating disorders have been observed. It is a process that can initiate very subtly and remain initially undetected.

In general, people with anorexia nervosa (AN) and bulimia nervosa (BN) share an excessive preoccupation with losing weight, even if they have normal weight or even if their weight is well below the healthy standard. Food becomes the main focus of their concern and other aspects of life are gradually neglected.

AN and BN differ in the way people react to food and the different physical consequences that result. If adequate treatment is not followed, one disorder may lead to another.

What is anorexia nervosa?

AN is characterised by excessive dread of becoming fat and by a distortion of body image that leads patients to feel and view themselves as fat when in fact they are not. They refuse to maintain their weight within a normal range and to this end they impose dietary restrictions on themselves to lose weight. Their self-evaluation is determined by how they see their body and

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shape. Perfectionism and low self-esteem are common. Often depression and obsessive thoughts are also part of the disorder. Individuals with AN rarely seek help and hide their symptoms for as long as they can.

What is bulimia nervosa?

In the case of BN, individuals engage in bouts of eating large amounts of food (bingeing) and subsequently carry out activities to maintain their weight. This behaviour can take over their daily life and hamper social relations and interactions. Individuals with BN usually hide their behaviour and rarely seek help. They may have a normal weight, or, paradoxically, become overweight as a result of their inadequate eating behaviours. The fear of gaining weight and sense of lack of control over eating determine their mood and mental state, which can result in depressive processes.

What are atypical eating disorders?

These eating disorders are called atypical because they do not entirely fit into the clinical definition of AN or BN due to the absence of one of the key symptoms of these disorders. Individuals with these disorders may present a combination of AN and BN symptoms that may vary over time. Many people with atypical eating disorders have suffered AN or BN in the past or will develop in the future.

The most well known atypical eating disorder is binge-eating disorder. In this disorder, patients engage in bingeing episodes, but do not try to control their weight by means of purging practices. They may feel anxious, tense and depressed, with the resulting impairment of their social life and relationships.

Atypical disorders, included binge-eating disorder, account for more than half the cases of eating disorders.

What is the origin of eating disorders?

There are many predisposing factors in the development of eating disorders: biological predisposition which includes genes, society (pressure to be thin as a symbol of beauty and success), family environment (tense, distant, cold, overprotective, uncommunicative climate, high family expectations, parents' excessive preoccupation with weight and diet,, obesity in the family) and personal character (maturity fears, perfectionism and self-control or low self-esteem and personal dissatisfaction).

Experiencing certain situations can trigger the disorder. For example, puberty, stressful situations (physical or psychological abuse, loss of a family member, separation or divorce, relocation, adaptation difficulties, exams, etc.), comments and pressure to lose weight, low-calorie diets, succumbing to the cultural stereotype and adherence to mass media messages, including Internet, which promote an abnormal image of thinness, excessive exercise, etc.

Once the disorder has been triggered, some of its consequences serve its maintenance and lead to further deterioration (for example, the biological consequences of malnutrition, social alienation and mood problems).

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Alert Signs

What signs can alert us to a possible eating disorder?

There are several changes that help detect the presence of the disorder, of which the following should be highlighted:

In relation to eating
Feeling of guilt for eating or not eating; preferring to eat alone, eating reduced amounts of food, presence of bingeing in the last few weeks, the feeling of not being able to stop eating, continuous avoidance of certain foods (for example, sweets), consumption of low-calorie and high-fibre foods, use of diuretics and laxative abuse, self-induced vomiting and dietary restraint or fasting.

In relation to body image
Excessive preoccupation with body or shape, the belief that physical appearance is very valuable as a conduit to gain success in any aspect of life, excessive adherence to cultural stereotypes and depending on certain magazines for their advice on dieting, weight or figure.

In relation with physical exercise
Excessive physical exercise and restlessness if it is not performed, use of exercise to lose weight.

In relation with behaviour
Constant personal dissatisfaction, depressive and irritable state, frequent mood changes; decreased social relations with increasing alienation, apparent increase of study hours, difficulty concentrating and regular visits to websites, blogs or chats that preach thinness and give out advice on losing weight or purging.

What are the consequences of inadequate eating behaviour?

The adoption of inadequate eating behaviours lead to physical disturbances in the patient, which are different in the case of AN versus BN:

In anorexia nervosa
Unexplainable weight loss or stunted weight (in children), delayed menarche or loss of menstruation, paleness, hair loss, feeling cold, blue fingers, appearance of lanugo, low blood pressure, arrhythmia, weakness and dizziness.

In bulimia nervosa
Irregular periods, muscle pain and fatigue, chronic throat irritation or loss of teeth due to repeated self-induced vomiting and inflammation of salivary glands.

Where can we seek professional help?

If you think you may be suffering from an eating disorder, it is very important that you seek help. Management of this type of disease begins in your primary care centre, where the general practitioner or paediatrician will advise you and assess your case. In certain circumstances, the

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team of psychiatrists, psychologists, etc. of the mental health centre/unit in your area will undertake your management to resolve the problem. There are also hospitalisation units (reference) to manage those patients who, due to the severity of their situation, require day care or inpatient care. Do not worry about confidentiality. All specific details that you share with health care professionals are personal and private information and will not be disclosed.

Treatment

The family's collaboration is essential in the treatment of these diseases. Treatment must be carried out by multidisciplinary teams of specialised professionals who can manage the medical, psychological, social and family complications that may arise. Medical care is aimed at eliminating physical complications, restoring normal weight and learning about following a healthy diet.

Once the necessary physical balance has been achieved, psychic problems linked to the disease are addressed. Psychologists or psychiatrists will be in charge of these therapies. Thus, proper treatment of these disorders should take the following aspects into consideration:

Psychological treatment: aimed at modifying distorted thoughts and negative feelings regarding weight, shape and body size in the patient's systems of values to help construct or recover an identity that aids the person in coping with life's challenges and difficulties.

This treatment can be delivered individually or in group sessions with other patients who are experiencing the same problems and, in some cases, with family members (parents, siblings, spouse, etc.).

Nutritional treatment: its main objective is to provide counselling on healthy eating habits and to provide information on the dangers of following unnecessary diets that, although justifiable, are not prescribed or managed by a competent health care professionals.

The social dimension of food, understood in terms of how eating provides a pleasant opportunity for family interaction, is emphasised more than food's nutritional value. It is important to receive nutritional counselling from the beginning of treatment to prevent relapses.

Pharmacological treatment: aimed at patients who require drugs in combination with psychological treatment, especially in cases of BN and BED, or to resolve associated symptomatology such as depression, anxiety or impulsiveness.

Not all patients are the same or require the same treatments. That is why it is important for you to follow your health practitioner's advice and to get involved in your own treatment.

Eating disorders have varying degrees of severity. Treatment can be performed on an outpatient basis, although in very severe cases temporary hospitalisation (inpatient or day care) may be necessary.

National associations of individuals with eating disorders

Aside from the resources available in the NHS to address these diseases, patients and family

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members can seek support and guidance in associations (share experiences, listen to other testimonials, obtain support and professional counselling thanks to experts who collaborate with these associations, etc). Some of these associations are:

- Association in Defence of Anorexia Nervosa and Bulimia Management [Asociación en Defensa de la Atención de la Anorexia Nerviosa y Bulimia (ADANER)] http://www.adaner.org

- Spanish Federation of Support Associations for Anorexia and Bulimia (FEACAB) http://www.feacab.org

Annex 3.2. Support associations for patients with eating disorders and their family members

Association in Defence of Anorexia Nervosa and Bulimia Management (ADANER)]

ADANER is an association of patients with eating disorders and their families. Its activity is carried out all over the country by its delegations and support groups. Its objectives are based on improving the care and quality of life of patients and their family members, disseminating information and making society aware of all aspects related to these diseases, and preventing and supporting research in this field.

e-mail: info@adaner.org web: http://www.adaner.org

Telephone Number: 915 77 02 61

Spanish Federation of Support Associations for Anorexia and Bulimia (FEACAB)

FEACAB is composed of associations from different Spanish provinces or autonomous communities and more than 20,000 people with AN and BN and their families. Its objectives are based on promoting the existence of sociosanitary and educational solutions that are adequate and sufficient for the prevention and treatment of these diseases, promoting good practice standards, for both associations and other groups –mass media, commerce, etc.- and adding prevention efforts that are already being carried out by each association.

e-mail: arbada@arbada.org web: http://www.feacab.org

Telephone number: 976 38 95 75

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Annex 4. Glossary

Counselling

Performance of several personal interviews with patients and family members which serve to inform and educate on the disease and its main sanitary, family and social consequences, and to provide insight on the current situation of health care, legal, economic and social resources with the aim of reassuring and assisting the patient and/or family.

Alprazolam

Benzodiazepine that produces its effect by binding stereospecific receptors located in the NCS. The following therapeutic indications have been approved in Spain: generalised anxiety and depression-related anxiety, and anxiety disorders with or without agoraphobia.

Benzodiazepines are only indicated in the treatment of an intense disorder that limits the patient's activity or subjects him/her to significant stress.

Amitriptyline

Tricyclic antidepressant. The following therapeutic indications have been approved in Spain: treatment of depression and chronic neuropathic pain.

Atomoxetine

Psychostimulant drug. The following therapeutic indications have been approved in Spain: treatment of attention deficit disorder with hyperactivity (ADDH) in children >6 years and adolescents as part of a complete treatment programme.

"Pure" Self-Help (SH)

Intervention that employs a clear model and treatment structure and includes all necessary instructions on how the user can improve his/her skills to assess and manage difficulties. Material can be found in any format, including books, CD-ROM, Internet packages, etc.; it can be individual or group-based; psychoeducational material can be included depending on its objectives, that is, to alleviate symptoms *vs.* improving knowledge.

Guided Self-Help (GSH)

It is the previously defined self-help (SH) and contact with a "therapist" who may be a mental health professional or a non-specialised individual. This intervention does not include: exclusively educational prevention material; pure support groups (since it does not consider improved clinical outcome a direct objective); groups that do not focus their sessions on one recognisable tool, for example: book, CD-ROM, video; sessions where the emphasis lies on the

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therapist who guides the sessions and is present in most of the "action". The number of hours of guidance can vary.

Brofamine

Antidepressant belonging to the group of monoaminooxidase inhibitors (MAOI). It does not appear in the Vademecum or in the AGEMED (Spanish Drug Agency). It has been used in the control group of several RCTs

Cyproheptadine

It belongs to the group of antihistamines (which block histamine H1 receptors). Aside from blocking the action of histamine H1 receptors in the brain, alleviating allergic reaction symptoms, cyproheptadine can also block serotonin receptors in the brain, thus stimulating appetite. The following therapeutic indications have been approved in Spain: anorexia, prevention and symptomatic treatment of nutrient-deficient states in convalescent periods or inadequate dietary intake. Appetite and development stimulant.

Citalopram

Antidepressant belonging to the group of SSRIs. The following therapeutic indications have been approved in Spain: major depressive episodes, prevention of depression relapse/recurrence, anxiety disorder with or without agoraphobia and OCD.

Clomipramine

Tricyclic antidepressant. The following therapeutic indications have been approved in Spain: any kind of depression, symptomatology and severity, obsessive syndromes, phobias, panic attacks, narcoleptic syndromes with cataplexy crises, nocturnal enuresis (only after 5 years of age and after ruling out organic causes).

Nutritional Counselling
Dietary or nutritional counselling or nutritional therapy

Type of treatment in which the main objective is to modify what the patient eats, and his/her habits and attitudes towards food. It is not a clearly defined intervention and it is applied in several modalities. It consists of providing a model which involves following a series of indications such as eating a healthy diet, maintaining fixed eating schedules, eating 3 meals a day, eating normal rations depending on age, eating while sitting down with the family, in a relaxed environment without distractions, without preparing the meal and resting after eating. Weight restoration requires a normocaloric, healthy diet, except in cases in which it is contraindicated due to the patient's condition.

Media literacy

Primary prevention interventions based on cognitive behaviour and inoculation theories that aim to reduce risk factors of eating disorders by means of learning activities that enable the acquisition

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of skills to fend off social persuasion. The approach aims to prepare participants to adopt a critical assessment of media content so they can identify, analyse, challenge and propose alternatives to cultural ideals presented in the mass media. Videos are played and then discussed to broaden knowledge and stimulate critical analysis of the group participants.

Desipramine

Tricyclic antidepressant. It is not included in the Vademecum or in the AGEMED (Spanish Drug Agency). It has been identified as treatment in the control group of some RCTs

Phenelzine

Antidepressant belonging to the MAOI group, which has been withdrawn from the Spanish market.

Fluoxetine

Antidepressant belonging to the group of SSRIs. The following therapeutic indications have been approved in Spain: major depression, OCD and BN.

Fluvoxamine

Antidepressant belonging to the group of SSRIs. The following therapeutic indications have been approved in Spain: depression and OCD.

Mutual help groups (MHGs)

Groups of people who meet voluntarily with the aim of helping each other. They are generally comprised of individuals who share the same problem or who find themselves in a similar difficult situation. The MHG emphasises personal interaction and each member's capacity to assume responsibilities. It tends to provide emotional help and promote values that help members strengthen their own sense of self. These groups provide assistance and emotional support to families and patients, facilitating the success of the corresponding therapy. Groups are guided by facilitators (people who have experienced the same problem or situation as the participants) and are periodically aided by a professional who supervises the intervention and provides instruments to improve group dynamics.

Imipramine

Tricyclic antidepressant. The following therapeutic indications have been approved in Spain: depression and nocturnal enuresis in children.

Interventions aimed at eliminating risk factors of the disorder (in this case, eating disorders)

Primary prevention interventions that can be specific or non-specific depending on the issue/aspect that is being addressed, providing participants with certain skills to cope with problems (management skills). Specific interventions are focused on: age of onset, child obesity,

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negative attitudes towards weight and shape, irrational beliefs on the benefits of being on a diet, social pressure to manage weight, dieting, amongst others. Non-specific interventions address: risk exposure, psychopathology of parents, lack of parental bond, physical and/or psychological abuse by family members, amongst others. Discussions/debates are carried out in classrooms, programmed sessions, etc.

Interventions aimed at making the patient stronger to tackle the disease (in this case, eating disorders)

Primary prevention interventions. Protection is achieved by providing the patient (host) with different skills (life skills, skills to deal with social pressure, to solve problems, etc.) and performing activities to develop coping skills. Discussions/debates are carried out in classrooms, programmed sessions, etc.

Psychoeducational interventions

Primary prevention interventions that consist of communicating information on a certain matter (in this case, on eating disorders), which will later be the central topic of debate/discussion between group participants.

Lithium

Mood stabilising psychoactive drug. The following therapeutic indications have been approved in Spain: prophylaxis and treatment of manic-depressive psychosis, recurrent unipolar depression, endogenous depression resistant to standard treatment, neutropenia following treatment with cytostatics, medullary aplasia and Felty's syndrome.

Lorazepam

Benzodiazepine that binds with specific receptors in several locations of the NCS, boosting the effects of synaptic or presynaptic inhibition mediated by GABA or directly affecting the potential action of generation mechanisms. The following therapeutic indications have been approved in Spain: short-term treatment of all states of anxiety or tension, associated or not to functional or organic disorders, psychic disturbances, psychosomatic diseases, organic diseases, sleep disorders, insomnia, hyperemotivity, neurosis. Benzodiazepines are only indicated in the treatment of an intense disorder that limits the patient's activity or subjects him/her to significant stress.

Mianserine

Antidepressant that blocks peripheral alpha-adrenoreceptors increasing the exchange of noradrenaline in the brain. It has slightly sedative, H1 antihistaminic and scarcely anticolinergic activity. The following therapeutic indications have been approved in Spain: improvement of depressive symptoms in depression cases where pharmacological treatment is indicated. It is one of the interventions in the control group of the RCTs studied in this CPG.

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Moclobemide

MAOI antidepressant, mainly of the A subtype; decreases metabolism of noradrenaline, dopamine and serotonin, leading to greater extracellular concentrations of these transmitters. Its approved therapeutic indication in Spain is major depression.

Advocacy

In a social context, this technique is based on the influence on a person's or group's attitudes towards a specific matter.

Naloxone

Antagonist of exogenously administered opioids. The following therapeutic indications have been approved in Spain: to totally or partially counteract respiratory depression caused by overdose of narcotics and dextropropoxifen or pentazocine, and also respiratory depression in the newborn caused by administration of opioids to the mother during delivery. Diagnosis when there is suspicion of acute narcotic intoxication.

Naltrexone

Antagonist of exogenously administered opioids. The following therapeutic indications have been approved in Spain: alcoholism, opioid withdrawal, accompanied by other therapeutic measures.

Olanzapine

Antipsychotic, antimanic and mood stabiliser with an affinity for serotonin, dopamine, cholinergic muscarinic, alpha-1-adrenergic and histamine receptors. The following therapeutic indications have been approved in Spain: schizophrenia, moderate or severe manic episode, relapse prevention of bipolar disorder whose manic episode has responded to olanzapine treatment, intramuscular administration for immediate management of agitation and disordered behaviours in schizophrenia or manic episode, when oral administration is not recommended.

Ondasentron

Potent antiemetic antagonist that is highly selective of 5-HT3 receptors located in peripheral neurones and within the NCS. The following therapeutic indications have been approved in Spain: management of nausea and vomiting caused by chemotherapy and cytotoxic radiation therapy and post-operative nausea and vomiting.

Orlistat

Peripherally acting drug designed to treat obesity.

Pimozide

Antipsychotic drug that selectively blocks dopaminergic central receptors and has neuroleptic properties. The following therapeutic indications have been approved in Spain: acute and chronic psychosis and anxiety disorders.

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Pizotifen

This drug belongs to the group of antihistamines (whose effect is to block histamine H1 receptors). Aside from blocking the action of histamine H1 receptors in the brain, alleviating allergic reaction symptoms, pizotifen is capable of blocking serotonin receptors in the brain, stimulating appetite. The following therapeutic indications have been approved in Spain: anorexia, thinness conditioned by psychic or "nervous" factors, weight deficit or loss, in puberty and senile patients.

Sibutramine

Antidepressant. The following therapeutic indications have been approved in Spain: complementary therapy within a comprehensive weight management programme for patients with obesity (whose BMI is 30 kg/m^2 or more) and patients with overweight (whose BMI is 27 kg/m^2 or more), who present other risk factors associated with obesity such as type 2 diabetes or dyslipidemia. It will be prescribed only to those patients who have not responded satisfactorily to an appropriate regimen that aims exclusively to tackle ponderal loss; that is, for patients who present difficulties in achieving or maintaining weight loss >5% in 3 months.

Refeeding syndrome

Metabolic disturbances and liquid and electrolyte imbalance with hypophosphatemia, cardiac insufficiency, muscular weakness, immune and haematological dysfunction, digestive and nervous system disorders. It occurs when sudden refeeding is administered, with normal or excessive caloric content, consisting mainly of carbohydrates. The best treatment for this syndrome is to be aware of its existence and keep it in mind when refeeding a patient with an eating disorder. To tackle this syndrome, appropriate treatment will be based on the apparatus or system involved, modifying the route or load of deficitary micronutrients that are being administered (oral to parenteral, etc.).

Sulpiride

Specific dopamine D2 and D3 receptor antagonist anntipsychotic. The following therapeutic indications have been approved in Spain: diverse psychopathological pictures: neurosis, depression, neurotic somatizations, functional psychological disorders, psychosomatic syndromes, psychoasthenia, psychic involution of old age, gastrointestinal somatizations, vertigo. In psychiatry: acute psychotic, confusional, hallucinatory and delirious state, depressive state, schizophrenia, chronic delirium, autism, serious behavioural disorders, neurotic state with inhibition and depression and different cenestopathies.

Induction-Dissonance Techniques

Primary prevention psychosocial interventions that consist of performing exercises where each participant targets one of his/her strongly-held belief and argues against this belief, to find discrepancies between both attitudes and promote effective change towards a new, better standpoint.

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Cognitive-Behavioural Therapy (CBT)

Psychological therapy designed to enable people to establish links between thoughts, feelings or actions and current or past actions and reassess their perceptions, beliefs or reasoning on them. The interventions should include at least one of the following aspects: 1) control of thoughts, feelings or actions in relation to behaviour, 2) help in using alternative ways of addressing behaviour, 3) reduce stress. A specific form of CBT has been developed for BN (CBT-BN) which, in general, involves 16-20 hours of individual sessions over four or five months. Its aim is not just to help patients modify eating habits, but also to cope with their patterns of thought (especially regarding the excessive value bestowed upon body shape and weight). CBT for patients with BED (CBT-BED) has been derived from CBT-BN.

Behavioural Therapy (BT)

Therapy that emphasises behavioural modification especially and not cognitive aspects as much.

Systemic Family Therapy (SFT)

FT is a form of psychotherapy that focuses on improving relationships and behavioural patterns within the family as a whole, as well as between individual members and groups or subsystems in the family. Systemic FT emphasises present relationships within the family and reassesses the role of the designated patient, of the symbolic value and the secondary gain of the symptom for the family system. Another key feature of this approach is the contextualisation in the family of any event, action or judgement, where most things or events do not have an intrinsic value, but rather are granted one depending on the function if fulfils within the system.

Interpersonal Therapy (IPT)

IPT was originally developed by Klerman, 1984, with the aim of being applied as maintenance treatment after depression, although it was later used as independent treatment and, at present, its use has been extended to several different disorders. IPT mainly addresses current interpersonal relationships and is focused on the patient's immediate social contest: grief, interpersonal disputes, role transition and interpersonal deficits. It has been adapted to BN treatment (IPT-BN; Fairburn, 1997). IPT-BN does not attempt to directly modify eating habits. Instead, it waits for them to change by improving interpersonal behaviour. In the case of BED IPT-BN is adapted to patients with BED (IPT-BED).

Psychodynamic Therapy (PDT)

Type of psychotherapy based on psychoanalytical principles that consists of performing regular individual therapy sessions with a trained psychotherapist, or under his/her supervision. A wide range of strategies is used, such as interventions aimed at understanding, support interventions or directive activity.

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Topiramate

Anticonvulsant psychoactive drug that blocks state-dependent sodium channels in neurones and boosts the potency of GABA activity. The following therapeutic indications have been approved in Spain: epilepsy and migraine prophylaxis.

Trazodone

Antidepressant. The following therapeutic indications have been approved in Spain: organic, psychogenic, symptomatic and involutive, combined states of depression and anxiety, atypical or masked depressions (psychosomatic disorders), shakes, dyskinesia, mood/affective and behaviour disorders (irritability, aggressiveness, emotional lability, apathy, tendency toward alienation, decreased sleep duration), preanesthetic, preendoscopic and postoperative medication.

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Annex 5. Abbreviations

ABOS Anorectic Behaviour Observation Scale for parents/spouse

ACTA Attitude Towards Change in Eating Disorders (acronym in Spanish)

ADANER Association in Defence of Anorexia Nervosa and Bulimia Management (acronym in Spanish)

ADDH Attention Deficit Disorder with Hyperactivity

AGEMED Spanish Drug Agency (acronym in Spanish)

AHRQ *Agency for Healthcare Research and Quality*

AN Anorexia nervosa

APA American Psychiatric Association

AR Assessment Report

BAT Body Attitude Test

BED *Binge eating disorder*

BDI or Beck Depression Inventory

BES Body-Esteem Scale

BET Branched Eating Disorders Test

BIA Body Image Assessment

BIS-11 Barratt Impulsiveness Scale

BITE Bulimia Investigatory Test Edinburgh

BMI Body Mass Index

BN Bulimia nervosa

BPD Borderline Personality Disorder

BSQ Body Shape Questionnaire

BT Behavioural Therapy

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

BULIT Bulimia Test

BULIT-R Revised version of the BULIT

BWC Behavioural Weight Control

BWLT Behavioural Weight Loss Treatment

CAHTA Catalan Agency for Health Technology Assessment

CBT Cognitive-behavioural therapy

CBT-BN Cognitive-behavioural therapy in bulimia nervosa

CBT-BED Cognitive-behavioural therapy in binge-eating disorder

CDI Children Depression Inventory

CETA Assessment of Anxiety Disorders in Children and Adolescents (acronym in Spanish)

ChEAT Children Eating Attitude Test

ChEDE-12 Children's version of the EDE-12

CI Confidence Interval

CIMEC Questionnaire on Influences of the Aesthetic Body Model (acronym in Spanish)

CPG Clinical Practice Guidelines

CY-BOCS Children and Adolescent version of the Y-BOCS

DH Department of Health, Day Hospital

DM Diabetes mellitus

DSM-IV-TR Diagnostic and Statistical Manual of Mental Disorders, fourth Edition, revised text

EAT (EAT-40) Eating Attitudes Test

EAT-26 Short version of the EAT-40

ECG Electrocardiogram

ED Eating Disorder

EDDS Eating Disorder Diagnostic Scale

EDE-12 Semistructured interview, 12th edition *(*Eating Disorders Examination-*12)*

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

EDE-Q Eating Disorders Examination-questionnaire

EDI Eating Disorder Inventory

EDNOS Eating Disorder Not Otherwise Specified (un-specific ED)

ERP Exposure with response prevention

ES Effect size

EU European Union

FEACAB Spanish Federation of Support Associations for Anorexia and Bulimia (from its acronym in Spanish for Federación Española de Asociaciones de Ayuda y Lucha contra la Anorexia y la Bulimia)

FT Non-specific Family Therapy

GDG Guidelines Development Group

GM General recommendations for medical measures

GP General recommendations for psychological therapies

GPH General recommendations for pharmacological treatment

GSH Guided Self-Help

HAM-D Hamilton Depression Rating Scale

HARS Hamilton Anxiety Rating Scale

HIV Human Immunodeficiency Virus

HRF Health Care Research Fund (the acronym in Spanish is FIS)

IAS Health Care Institute (from its acronym in Catalan for Institut d'Assistència Sanitària)

ICD-9 International Statistical Classification of Diseases and Related Health Problems, 9th edition

ICD-10 International Statistical Classification of Diseases and Related Health Problems, 10th edition

INSALUD National Institute of Health (acronym in Spanish)

IPDE *International Personality Disorder Examination*

IPT-BN Interpersonal Therapy for bulimia nervosa

IPT Interpersonal Therapy

It has been 5 years since the publication of this Clinical Practice Guideline and it is subject to updating.

ISCIII Carlos III Health Institute

ITP Individualised Treatment Plan

kcal Kilocalories

kg Kilograms

MA Metaanalysis or quantitive review

MACI Millon Adolescent Clinical Inventory

MCMI-III Millon Multiaxial Clinical Inventory-III

MHC Mental Health Centre

MHCFA Mental Health Centre for Adults

MHCFCA Mental Health Centre for Children and Adolescents

MHGs Mutual Help Groups

MSC Ministry of Health and Consumer Affairs

NC Nutritional counselling

NCCMH *National Collaborating Centre for Mental Health*

NHS National Health System

NICE *National Institute for Clinical Excellence*

NNT Number needed to treat

NPV Negative Predictive Value

NSCM Non-specific Supportive Clinical Management

OCD Obsessive-Compulsive Disorder

PC Primary Care

PDT Psychodynamic Therapy

PPV Positive Predictive Value

Q-RCT Quasi-experimental RCT

RCT Randomised controlled trial

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r correlation coefficient r
RR Relative Risk

SCOFF *Sick, Control, One, Fat, Food questionnaire*

SD Standard Deviation

SED *Survey for Eating Disorders*

semFYC Spanish Society of Family and Community Medicine (from its acronym in Spanish for Sociedad Española de Medicina de Familia y Comunitaria)

SFP Supportive Focal Psychotherapy

SFT Systemic Family Therapy

SH Self-Help

SMD Standard Mean Difference

SSRI Selective Serotonin Reuptake Inhibibitors

SRSE Systematic Review Scientific Evidence

STAI *State-Trait Anxiety Inventory*

STAIC STAI's version for children

TCI-R Temperament and Character Inventory revised

UB University of Barcelona

UK United Kingdom

USA United States of America

vs. Versus

Y-BOCS *Y*ale-Brown Obsessive-Compulsive Scale for OCD

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Annex 6. Others

Annex 6.1. Protocols, recommendations, therapeutic orientations and guides on eating disorders

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Annex 6.2. Results of the search, selection and quality assessment of the evidence based on the stages performed

1) 115 documents were identified: guidelines, reviews and ARs. After applying selection criteria (to the title and/or abstract), 73 were excluded. The complete document of all 42 retrieved documents (14 were potential guidelines) was requested. After review, 28 more were excluded (11 of them for not being considered guidelines[421-431]) and 15 were included (3 guidelines and 12 reviews). After assessing the quality with specific instruments, 2 CPGs[14, 18] and 11 reviews were excluded. Only two documents met quality criteria: one NICE CPG[30] and one AHRQ SRSE[31]. The evidence tables in the different chapters state the quality of each SRSE. That is not the case in the only quality CPG, which is described solely as CPG.

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2) We identified 20 documents elaborated in our setting relating to protocols, recommendations, therapeutic orientations and guidelines on eating disorders developed by institutions of the health administration, scientific societies, hospitals and other organizations in our setting. None of these documents met the quality criteria established by the working group and were thus excluded[10-13, 15-17, 19, 20, 146-151, 416-420,] .

3) Of the documents retrieved, 12 RCTs [140, 145, 203, 238, 253, 254, 274, 316, 318, 319, 331, 432] were included, their quality being 1++ (9) and 1+ (3). The rest were excluded due to topic, language or methodology. One quality 1++ RCT was manually identified[320].

4) Of the documents identified in the CPG/SRSE/AR, 14 SRSE were included based on title and/or summary. After reviewing the entire document, 7 [137, 202, 211, 245, 308, 397, 433] were included and the other 7[434-440] were excluded for not being considered SRSE. The quality of all 7 SRSE included is 1++[137, 202, 211, 245, 308, 397, 433]. No guidelines were identified.

5) Of the documents identified on primary prevention, 14 SRSE, 103 RCTs and 1 guideline, which was excluded due to its low quality, were retrieved. 8 SRSE[134-136, 441-445] were preliminarily selected based on their title and/or summary. Excluded SRSE included one that had been identified and included in the previous search[137]. After reviewing the entire document, four[441-443, 445] were excluded (one due to its date and three due to low quality), 3 were included (quality 1++)[134-136] and the original version[444] of an already included SRSE[137] was excluded. Of the identified RCT, 51 RCTs were excluded (language or topic) and 52 were preliminarily included, of which 38 are included in one of the quality SRSE included[137] and two were repetitions. The entire document of the remaining 12 RCTs and 2 manually identified ones was reviewed, resulting in the inclusion of 8 RCTs[138-145].

6) Different reviews and articles on the topic have been identified, on which the prognosis chapter is based.

7) In the consultation of these different sources, one SRSE of the Cochrane Collaboration on SH and GSH in eating disorders[257] was identified. Another Cochrane Collaboration was excluded due to its date[446].

Annex 6.3. Description of the NICE CPG (January, 2004)[30]

Title: Eating disorders. Core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related eating disorders.

Developed by: National Collaborating Centre for Mental Health (NCCMH). The development group of the CPG (GDG) was selected by the e NCCMH. The GDG is composed by: psychiatrists, clinical psychologists, nurses, social workers, general physicians, an eating disorder association representative and a patient. The GDG received technical support from staff members of the NCCMH in the search and summarising of the scientific evidence. NICE staff advised the GDG on the development process of the CPG.

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Commissioned and Published by: National Institute for Clinical Excellence (NICE) and The British Psychological Society and The Royal College of Psychiatrists, respectively, January 2004.

Target population: Eating disorders (AN, BN and BED).

Scope: aimed at all patients with AN, BN and related eating disorders, who are aged 8 years and older. The CPG does not include all mental health or physical pathologies in which an eating disorder is a symptom.

Potential users: scientific associations, health care professionals, researchers, planners, family members and educators.

Funding: NICE.

Methodological Aspects:

1) Search for evidence

SRSE in English published or updated after 1995. The search was completed with RCT and other designs. The quality of the SRSE was determined by parallel independent assessment. The selection and assessment of quality was tested in a representative group of documents. The guide includes the general and specific search filters developed by the reviewer group. Electronic searches were carried out in the main bibliographic databases: MEDLINE, EMBASE, CINAHL and PSYCINFO. Also in the Cochrane Database of Systematic Reviews, the NHS R&D Health Technology Assessment database, Evidence-Based Mental Health and Clinical Evidence (Issue 5). In order to include evidence published after search dates, evidence identified by experts was reviewed, including reference lists and recent summaries of selected journals. When no SRSE were found, new searches were performed in compliance with the GDG's decision.

2) Quality and Strength of the Evidence

Intervention studies were classified in accordance with the accepted hierarchy of evidence: Level of evidence (I, IIa, IIb, III and IV) and grade of recommendation (A, B and C) depending on the type and level of respectively associated evidence.

The levels of evidence of the NICE guide scale correspond to the following levels of the SIGN scale: I=1++; IIa=1+; IIb=1-; III=2 (-, +, ++) and IV=3, 4. A and B grades of recommendation coincide in both the NICE scales and the SIGN scale used in this CPG. The C grade of the NICE scale could be extrapolated to the D grade of the SIGN scale.

When statistically significant results were obtained after controlling heterogeneity, the GDG assessed whether these findings were clinically significant (for example, if benefit for patients was probable) taking the RCT population, the nature of the outcome and effect size (ES) into account. On this basis, effect size was considered clinically significant or not. The next step was to assess the strength of the evidence, by examining the confidence intervals of the effect

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estimator, establishing the following categories: S1= strong evidence (clinically significant ES and CI with a range of clinical relevance (level of evidence I); S2= limited evidence (not a level of evidence I or situations where one of the CI extremes is not clinically significant); S3= unlikely that it is clinically significant, if it is not a level of evidence I or if ES is statistically but not clinically significant, and the CI excludes clinically important values; S4= when the ES is not statistically significant: if the CI is narrow and excludes a clinically significant ES, the difference is not considered to be clinically significant; S5= but if the CI is wide, it is considered insufficient evidence to determine if there was a clinically significant difference or not. S6= if the CI includes clinically important values, it is considered insufficient to determine clinical signification.

3) Method to formulate recommendations

The importance of acknowledging that the grade of recommendations does not always reflect their clinical significance or relevance is emphasised.

Annex 6.4. Description of the AHRQ'S SRSE (April, 2006)[31]

Title: Management of Eating Disorders.

Developed by: RTI-UNC Evidence-Based Practice Centre, Research Triangle Park, NC (centre ascribed to the AHRQ). The GDG of this guide was advised by and received feedback from a technical experts panel (TEP) comprised of 11 people, mainly psychiatrics from different settings and one family representative. 23 external reviewers, some from the TEP, also participated.

Commissioned and Published By: Agency for Healthcare Research and Quality (AHRQ), USA. AHRQ Publication Nº 06-E010, April 2006.

Target Population: AN, BN and EDNOS.

Scope: treatment of eating disorders (efficacy, adverse effects, factors associated with treatment efficacy, sex-, age-, race-, ethnic group- or cultural group-related differences in treatment efficacy, factors associated with outcomes and sex-, age-, race-, ethnic group- or cultural group-related outcome differences).

Results of Interest: eating-related behaviours, psychiatric or psychological outcomes and biomarkers.

Potential Users: scientific associations, health care professionals, researchers and planners.

Funding: Office of Research on Women's Health (NIH).

Methodological Aspects:

1) Search for Evidence

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Consulted bibliographic databases: MEDLINE, Cumulative Index to Nursing and Applied Health (CINAHL), PSYCHINFO, Educational Resources Information Centre (ERIC), National Agricultural Library (AGRICOLA) and Cochrane Collaboration Libraries. The guide includes the search performed. Study inclusion criteria: published in all languages, between 19080 and September 2005, in humans aged 10 years and older, of both sexes, diagnosed mainly with AN, BN or BED and informing on at least one of the relevant results.

2) Selection of the Evidence

A reviewer carried out the application of selection criteria to titles and/or summaries. Excluded summaries were assessed by another expert reviewer, and reasons for exclusion were classified.

3) Quality Assessment and Synthesis of the Evidence

Evidence tables were elaborated; an external reviewer extracted data on treatments and results; another reviewer confirmed that they were exact, complete and consistent; both reconciled disagreements. Each reviewer assessed the quality of the studies. The analysis was focused on studies qualified as moderate (1+) or high (1++) quality.

4) Quality and Strength of the Evidence according to West, *et al*. (2002)[447]

2 forms were developed to assess the quality of the studies about treatment (25 items) and results (17 items) that enabled the classification of quality as low, moderate or high. The body of evidence was graded for each key question based on three dimensions: study quality, results and consistency of findings. After consensus of three expert researchers, the following levels of evidence were defined: I=strong, II=moderate, III=weak and IV=no evidence.

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