Learning Objectives

- To understand the diagnostic criteria for bulimia nervosa and how to differentiate it from other eating disorders;
- To become aware of medical complications that occur in bulimia nervosa;
- To know and to understand the diagnostic criteria for Eating Disorder non specified (EDNOS);
- To know and to understand two subtypes of EDNOS: Binge Eating Disorder and Night Eating syndrome;
- To learn the treatment of bulimia nervosa and its features;
- To learn the treatment of Binge Eating Disorder;
- To learn the treatment of Night Eating syndrome.

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Key Messages

- Bulimia Nervosa is a psychiatric illness;
- The prognosis of Bulimia Nervosa is improved by early diagnosis and treatment;
- Eating disorders non specified (like Binge Eating Disorder, and Night Eating Disorder) are the most frequent eating disorders;
- Binge Eating Disorder is most common among the obese. Those who seek help do so for treatment of overweight rather than for binge eating;
- Night Eating Syndrome is uncommon in the general population, but it is very frequent in obese persons;
- All Eating Disorders are best by a multi disciplinary team with broad experience in this field. It is highly recommended that patients attend a unit specialising in eating disorders.

1. Introduction

Bulimia nervosa is understood to be a complex disorder with multiple factors contributing to its development. Researchers presently disagree about the relative influence of genetic factors, psychological patterns in the family of origin, and social trends. As with Anorexia Nervosa the main concern in Bulimia Nervosa is the morbid fear of becoming obese. Bulimia differs in being more frequent in people that have been obese in the past. Bulimia often appears after an
2. Diagnosis of Bulimia Nervosa

2.1 Clinical History

Bulimia nervosa is characterised by repeated episodes of binge eating. These episodes are followed by purging, usually in the form of self-induced vomiting or excessive laxative use, to avoid weight gain. (1) It typically occurs in adolescents and young women, the lifetime prevalence among women ranging from 1 to 4.2 % (2) In 1979, Russell proposed three diagnostic criteria for bulimia nervosa: 1) presence of binges, 2) induced vomiting or laxative abuse, 3) Excessive fear of becoming obese.

The onset of bulimia nervosa is most common in late adolescence or early adult life. Buts of dieting and bodily dissatisfaction often occur in the teenage years. For these reasons, it is often described as a developmental disorder. Although genetic researchers have identified specific genes linked to susceptibility to eating disorders, the primary factor in the development of bulimia nervosa is environmental stress related to the onset of puberty. Girls who have strongly negative feelings about their bodies in respose to puberty are at high risk of developing bulimia.

1. Binges

The Diagnostic and Statistical Manual of Mental Disorders (DSM, actual number version: IV, revised text: RT) (1) adds precision to Russell's criteria. DSM-IV-RT specifies the components of a binge and its minimum frequency. It describes one of the characteristics of a binge in this way: "to eat, in a discreet period (for example, two hours), an amount of food greater than the majority of people would eat in a similar period" (some authors describes 5000 calories) Another characteristic is the sensation of loss of control (the subject cannot control the kind or amount of food eaten). To fulfill the diagnosis binges must occur, at least, twice a week for three months.

The binge eating associated with bulimia begins most often after a period of strict dieting. Different dysphoric mood states appear before the binge. The binge reduces these unpleasant mood states, but, when it finishes, the patient suffers physical discomfort and an excessive fear of gaining weight. Emotions following a binge are usually negative with guilt, regret, disgust, and self loathing. Some nutrition experts have pointed to the easy availability of foods high in processed carbohydrates in developed countries as a social factor that contributes to the incidence of bulimia. It is suggest that bulimics who binge on ice cream, bread, cookies, pizza, and fast food items that are high in processed carbohydrates are simply manipulating their neurochemistry in a highly efficient manner. The incidence of bulimia may be lower in developing countries because diets that are high in vegetables and whole-grain products but low in processed carbohydrates do not affect serotonin levels in the brain as rapidly or as effectively.
2. **Vomiting and laxative abuse**  
The use of induced vomiting or laxative abuse are considered pathological behaviour in our society and they are not as well accepted methods of weight control as dieting or physical activity. The patients that present with these symptoms are different in psychopathological terms and in prognosis from those who do. 
Most people with bulimia develop purging behaviours as an antidote to bingeing. Vomiting is often induced to relieve an uncomfortable sensation of fullness in the stomach following a binge as well as to prevent absorption of the calories in the food. Vomiting is frequently induced by stimulating the gag reflex at the back of the throat with the fingers or a toothbrush, but a minority of patients use syrup of ipecac to induce vomiting. Vomiting is used by 80%-90% of patients diagnosed with bulimia. About a third of bulimics use laxatives after binge eating to empty the digestive tract, and a minority use diuretics or enemas. A small proportion of bulimics exercise excessively (strenuous exercise, in this case, means exercising for more than an hour just to keep from gaining weight after binge eating) or fast after a binge instead of purging. Until more data are available, the experts have classified bulimia into two subtypes: purgative and non-purgative.

3. **Excessive fear of gain weight**  
Even thought Russell emphasised this fear, the DSM shows it as an "exaggerated concern about the body shape and weight". ICD-10 also proposes morbid fear of obesity as a characteristic psychopathological feature in bulimia nervosa.

### 2.2 Laboratory and Image Findings

Bulimia may cause electrolyte disturbances and varying degrees of malnutrition, but the changes are less striking than those seen in Anorexia Nervosa. Useful basic investigations are similar to those in other eating disorders A- Blood chemistry studies:

1. serum electrolytes  
2. blood urea nitrogen  
3. serum creatinine (interpretations must incorporate assessments of weight since serum creatinine reflects muscle mass as well as renal function)  
4. thyroid-stimulating hormone and, if indicated, free T4, T3  
5. complete blood count including differential  
6. erythrocyte sedimentation rate  
7. aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase

B - Urinalysis

There are some nonroutine investigations, that should be carried out in specific cases:

- **Patients with suspected surreptitious vomiting:**  
  Serum Amylase: fractionated for salivary gland isoenzyme if available to rule out pancreatic involvement
- **Patients with suspected GI bleeding**  
  Stool for Guaiac
- **Patients with suspected laxative abuse**  
  Stool or Urine for Bisacodyl, Emodin, Aloe-emodin, Rhein
• Patients with suspected substance use:
  Toxicology screen
• Patients with significant cognitive deficits, other neurological soft signs, unremitting course, or other atypical features
  Brain Magnetic Resonance Imaging, Computed Tomography.

2.3 Instruments for the assessment of bulimia nervosa

Formal measurements are available for the assessment of eating disorders, including bulimia nervosa. We can use self-reported questionnaires and semistructured interviews. Clinical decisions about diagnosis cannot be made only on the basis of self-reported screening instruments: patients must be followed up in a second-stage more detailed assessment by trained clinical interviewers.

Some of these instruments are:

**EAT-40, Eating attitudes test**
Brief, standardised self-reported screening test of symptoms and concerns characteristic of eating disorders.

**EDI- Eating disorder inventory**
Standardised measurement of psychological traits and symptom clusters presumed to have relevance to the understanding and treatment of eating disorders.

**BITE- Bulimic Investigatory Test Edinburgh**
Brief questionnaire for the detection and description of binge-eating. This scale measures both symptoms and severity. All items in the DSM definition of bulimia and Russell's definition of bulimia nervosa are covered.

**BULIT-R Bulimia Test-Revised**
Brief measure designed to assess eating behaviours and attitudes related to bulimia.

2.4 Differential Diagnosis

The main differential diagnosis is:

• psychiatric disorders:
  o depression - loss of interest in food or delusions of worthlessness
  o conversion disorders - some patients may present with weight loss and vomiting because of a disgust for food in the absence of a desire for thinness
  o psychogenic vomiting
  o Anorexia Nervosa (see anorexia nervosa criteria, module 31.1)- binge eating occurs exclusively during the course of anorexia nervosa
  o Binge-Eating/Purging Type of Bulimia nervosa - Some authors have reported that BN patients are younger and have higher levels of depression than the Binge Eating Group. Women with Bulimia Nervosa have greater disturbances on the personality scales, although it seems that the main differences are between the Bulimia and the obese Binge Eating Disorder groups, whereas the bulimia nervosa and the non-obese group are quite similar on most measures. (see Binge Eating Disorder criteria). Persons with bulimia nervosa, however, usually purge, fast, or do strenuous exercise after they binge eat.
Binge eating in Borderline Personality Disorder - does not require inappropriate compensatory behaviours or over-concern with body shape and weight and includes characteristic features.

- physical disorders:
  - Vomiting or diarrhoea in general medical conditions or with excessive substance abuse is due to the direct physiological effects of the general medical condition or substance abuse
  - chronic wasting diseases - for instance, malignancy, tuberculosis
  - endocrine disorders - for example, Addison's disease, hyperthyroidism, anterior pituitary insufficiency
  - Kleine-Levin syndrome: this rare disorder is characterized by compulsive hyperphagia, hypersomnia and an abnormally uninhibited sexual drive
  - Pancreatitis: it is a life-threatening disease that may be difficult to diagnose in eating disorder patients because the symptoms may be similar. There is no evidence that pancreatitis is more common in eating disorder patients than in the general population. Certain medications and alcohol consumption increase the risk of pancreatitis. Because of the potential seriousness if the diagnosis is not made, acute pancreatitis should be considered in eating-disorder patients presenting with suggestive symptoms, particularly if one or more of the known predisposing factors are present. Patients suffering from either an eating disorder or acute pancreatitis may complain of abdominal pain or discomfort, nausea or vomiting. It is possible that a bulimic subject with these symptoms may not seek medical advice because of their familiarity. In such cases, measurement of serum amylase levels can be helpful. However, elevated amylase values may occur in subjects with bulimia nervosa (because of the binges and purges) without pancreatitis. Serum levels of enzymes that are specific for the pancreas, including serum lipase, trypsinogen, the pancreatic isoenzyme of amylase, can be measured to determine whether an elevated level serum amylase is the result of pancreatitis. In the same way, an abdominal computed tomography scan can help to confirm or exclude the diagnosis (3).

3. Medical complications of Bulimia nervosa

Knowledge of these changes may be of critical importance in avoiding misdiagnosis and successful therapy. Between bulimic episodes, the patients often show extreme variations in food intake with corresponding decreases or increases in body weight.

3.1 Gastrointestinal disturbances

Bulimia nervosa patients show delayed gastric emptying and diminished gastric relaxation. Diminished release of cholecystokinin and abnormalities in enteric autonomic function were also found in these patients. These factors may play a role in the perpetuation of the disease (4). Gastrointestinal (GI) complaints appear to be common among BN patients. Bloating, constipation, abdominal pain and nausea have been reported. However, GI complaints may pre-exist in some of these patients and may have an impact on the eating disorder symptoms (5). Some gastrointestinal pathology that is believed to occur as a complication of bulimia nervosa
(specially purging BN) has been described: Esophagitis, Esophageal rupture, Gastritis, Gastric reflux, Gastric dilation, Cathartic colon (non-specific inflammation, atrophy, ulceration and mononuclear cell infiltration) and Melanosis coli (pigmentation of the colonic mucosa associated with anthaquinone abuse).

3.2 Oropharyngeal symptoms

The most striking dental features are the intrinsic erosions due to the regular surreptitious vomiting. They occur in very typical locations within the dental arches and have been termed "perimolysis". In the same way, it is usual to find erythema of pharynx, palatal scratches and characteristic enlarged salivary glands.

3.3 Cardiovascular symptoms and signs

Some arrhythmias can appear: Hypokalaemia-associated depressed ST segment and QT prolongation. In severe cases, hypokalaemia causes widened QRS complexes, increased P-wave amplitude, increased PR interval, increased supraventricular and ventricular ectopic rhythms, torsade de pointes, and autonomic dysfunction on spectral analysis.

3.4 Central nervous system complications

Apathy, poor concentration, anxious, depressed and irritable mood. In severe cases, cognitive impairment and peripheral neuropathymay occur. It is possible to find cortical atrophy and ventricular enlargement on TC scan, abnormal cerebral blood flow and metabolism in PET (positron emission tomography) and f MRI (functional magnetic resonance imaging) and decreased grey and white matter in a usual MRI (magnetic resonance imaging).

3.5 Skin symptoms and signs

The most characteristic complication is scarring on the dorsum of hand (Russell's Sign). Other signs that can appear are petechia and conjunctival haemorrhages shortly after vomiting.

3.6 Metabolic symptoms and signs

- Weight fluctuation
- Proximal weakness
- Irritability, muscle cramping
- Poor skin turgor
- Pitting oedema
- Chvostek's and Trousseau's signs (rare)
- Urinalysis: dehydration (increased urine specific gravity, osmolality)
- Serum electrolyte abnormalities: hypokalaemia, hypochloraemia, alkalosis in vomiters; hypokalemic acidosis in purgative abuse. Hypomagnesemia and hypophosphatemia in vomiters and laxative abusers.

3.7 Muscular symptoms and signs
In ipecac abusers, weakness, palpitations. Muscle weakness, peripheral myopathy without specific abnormalities in electromyogram.

3.8 Reproductive symptoms and signs

- Fertility problems
- Spotty/scanty menstrual periods, oligomenorrhea or amenorrhea
- Serum gonadotropins variable: may be hypoestrogenic

3.9 Skeletal symptoms and signs

- Bone pain with exercise
- Point tenderness
- Short stature
- Arrested skeletal growth
- Possible pathological stress fractures
- Delayed bone age in some cases
- DEXA: possible osteopenia or osteoporosis specially in hip or lumbar spine

4. Therapy of Bulimia Nervosa

4.1 The aims of the treatment

The aims of treatment of patients with bulimia nervosa are to 1) reduce and, where possible, eliminate binge eating and purging, 2) treat physical complications of bulimia nervosa, 3) enhance patients' motivation to cooperate in the restoration of healthy eating patterns and participate in treatment, 4) provide education regarding healthy nutrition and eating patterns, 5) help patients reassess and change core dysfunctional thoughts, attitudes, motives, conflicts, and feelings related to the eating disorder, 6) treat associated psychiatric conditions, including deficits in mood and impulse regulation, self-esteem, and behaviour, 7) enlist family support and provide family counselling and therapy where appropriate and 8) prevent relapse.

4.2 Nutritional rehabilitation counselling

A primary focus for nutritional rehabilitation is to help the patient develop a structured meal plan as a means of reducing episodes of dietary restriction and the urges to binge and purge. Adequate nutritional intake can prevent craving and promote satiety. It is important to assess nutritional intake in all patients, even those with a normal body weight (or normal BMI), as normal weight does not necessarily indicate appropriate nutritional intake or normal body composition. Among patients of normal weight, nutritional counselling is a useful part of treatment and helps reduce food restriction, increase the variety of food restriction, increase the variety of foods eaten, and promote healthy but nor compulsive exercise patterns.

4.3 Therapeutic team

The biopsychosocial nature of bulimia nervosa dictates the need for a multidisciplinary approach. Professionals from several disciplines should collaborate in the patient's care. The specific role of each professional may vary with the organisational structure of the eating disorders program. The psychiatrist often assumes the leadership role but the therapeutic team
may include other physicians: specialists in internal medicine, paediatrics, and nutrition, psychologists and trained (in mental disorders and specially in eating disorders) nurses. Also registered dieticians with training in eating disorders often provide nutritional counselling. Other physician specialist and dentists may be consulted for the management of acute and ongoing medical complications. Dental professionals are often the first to discover and diagnose eating disorders by detecting perimolysis. The primary goal of dental care is to preserve the remaining teeth and to prevent further erosive loss of dental hard tissue. Dental restorative therapy must be part of a combined medical and dental treatment plan and should not be started before the eating disorder has been assessed and treated, and the patients are considered to have stable prognosis. In view of the young age of the patients, the large extension of the erosive lesions and in order to avoid endodontological treatment of mostly sound pulps, non-invasive restorative treatment using adhesive technology are preferred.

4.4 Evidence on efficacy for the treatment

Evidence for the efficacy of medication or behavioural treatment in BN is strong. Fluoxetine (60 mg/day) decreases the core symptoms of binge eating and purging and also the associated psychological features in the short term. Cognitive behavioural therapy reduces core behavioural and psychological features in the short and long term. Evidence for the efficacy of self-help is weak.

5. Diagnosis of Eating Disorder 'Not Otherwise Specified'

The 'not otherwise specified' category is a conceptually problematic one and comprises a clinically heterogeneous group of diagnoses. Two of these disorders are Binge Eating Disorder and Night Eating Syndrome.

5.1 Binge eating disorder

Binge eating disorder (BED) was included in the DSM IV as a proposed category for further study and as an example of an eating disorder 'not otherwise specified'. Community surveys have estimated the current prevalence of BED to be between 2 and 5 % (9,10). Binge eating disorder is more equal in gender ratio (65% female, 35% male) than bulimia nervosa in which only 10% of sufferers are male. Although BED is not limited to obese individuals, it is most common in this Group and those who seek help do so for treatment of overweight rather than for binge eating. The binging episodes of overweight individuals seem to differ in important ways from those described by patients with bulimia nervosa. BED subjects consume approximately half the calories of those with bulimia nervosa during binging and they also binge less frequently. In line with the data on bulimia nervosa subjects, obese binge eaters appear to increase their intake of fat rather than carbohydrates. There is no evidence that, in obese patients, binge eating develops as a result of therapeutic dietary restraint. Trigger factors for binging in BED patients have been variously reported as: negative emotional states, e.g. anger and frustration, depression and anxiety; a nonspecific feeling of tension in social situations; time of day and the type of meal.

5.1.1 Risk factors
Binge eating disorder appears to be associated with exposure to risk factors for psychiatric disorders (e.g. negative self-evaluation, parental depression, adverse childhood experiences including sexual and physical abuse and a range of parental problems, and pregnancy before onset) and with exposure to risk factors for obesity (e.g. childhood obesity, critical comments by family about shape, weight or eating): However, compared with bulimia nervosa the risk factors for BED are weaker. Even vulnerability to obesity seems to be more pronounced in bulimia nervosa.

5.1.2 Metabolic characteristics

There is no evidence that obese subjects with BED are more prone to the medical consequences of obesity than obese subjects without BED if one controls for weight. No significant differences were observed in blood pressure, resting metabolic rate, resting energy expenditure, body fat distribution (waist/hip ratio), percentage body fat, and blood serum measures such as glucose, insulin levels, lipid levels, and thyroid hormones. Others found no association between binge eating severity and glycemic control in obese patients with type 2 diabetes (11).

5.1.3 Differential Diagnosis

- The main differential diagnosis in cases suspected of BED is Bulimia Nervosa: Persons with bulimia nervosa, however, usually purge, fast and overexercise and whereas the prognosis of patients with bulimia nervosa is relatively poor, the great majority of people with binge eating disorder recover
- Another diagnosis that needs to be discussed in relation to BED is Night Eating Syndrome

5.2 Night Eating Syndrome

Night Eating Syndrome (NES) was introduced into the medical literature in 1955 by Stunkard, Grace and Wolff, based on their clinical observations of eating disturbances in a group of morbidly obese patients seeking treatment for weight control at a speciality clinic. Stunkard defined NES as involving morning anorexia, evening hyperphagia, and insomnia or sleeplessness. Morning anorexia was defined as negligible or no intake at the traditional breakfast time. Evening hyperphagia was defined as consuming at least 25% of the total daily calories after the evening meal. Insomnia or sleepless was required to occur three or more times a week.

In recent work, Stunkard (12) has emphasised that NES may represent a unique combination of eating disorder, mood disorder, and sleep disorder.

Night eating syndrome is uncommon in the general population (1.5 %), prevalence increases with increasing weight, from 8.9 % to 15 % in obesity clinics and from 10 % to 27 % and 42 % among obese persons undergoing assessment for bariatric surgery (13).

Even though, a long time has passed since its first description, little research has been focused on night eating. Research is now beginning to examine the possibility that, like binge eating, night eating may contribute to obesity(14).

Further research may accumulate the necessary evidence to determine whether NES should be included in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders The most pressing step towards clarifying the status of NES is to develop a uniform definition of this syndrome (15).
6. Therapy of Eating Disorder Non Specified

6.1 Binge eating disorder treatment

The literature regarding the efficacy of treatment in BED is variable. Future directions include the identification of interventions that help to sustain abstinence from binge eating and permanent weight loss. The strength of the evidence for drug and behavioural interventions is moderate, and for self-help and other interventions is weak(16). Binge eaters have received three types of treatment: psychotherapy, pharmachotherapy, and, notably, weight reduction programs that have ignored the issue of binge eating. Psychological treatment: Cognitive behaviour therapy is consistently associated with reductions in binge eating, ranging from 48 to 98% Interpersonal therapy has been associated with a clinically significant reduction (71 %) in binge eating and with maintenance of these effects (17). Pharmacotherapy has also reduced the frequency of binge eating. A significant reduction in bingeing compared to control groups has been found with antidepressant medication (desipramine, fluvoxamine, sertraline, fluoxetine). Therapeutic efficacy seems to be shown by the prompt recurrence of bingeing when medication is discontinued. Topiramate (100 to 1400 mg daily) may also be an effective and well-tolerated agent in the treatment of BED. A surprising finding is that traditional weight loss programs that ignore the issue of binge eating may be as effective in reducing bingeing as cognitive behaviour therapy and drugs. They are also even more successful than cognitive behavioural therapy and interpersonal therapy and most pharmacological interventions in producing weight loss.

6.2 Night Eating Syndrome treatment

The pharmacological treatment of NES is similar to that of BED (16). There are some studies (18) that show good clinical response to selective serotonin reuptake inhibitors (SSRI) and to antidepressants like sertraline. It has also been suggested that SSRI antidepressants improve NES independently of its effect on depression. Some authors (19) have suggested using topiramate in otherwise unresponsive cases of NES. Phototherapy is effective in seasonal affective and sleep disorders and has also been proposed as a treatment for NES. Progressive muscle relaxation therapy has also been shown to reduce symptoms associated with night eating syndrome. It is suggested that successful treatment of Night Eating Syndrome may contribute to control of the associated obesity.

7. Summary

Bulimia nervosa is a complex disorder characterised by repeated episodes of binge eating. These episodes are usually followed by purging, usually in the form of self-induced vomiting or excessive laxative use, to avoid weight gain. It typically occurs in adolescents and young women. The lifetime prevalence among women ranges from 1 to 4.2%. It can cause electrolytic disturbances and varying levels of malnutrition. Laboratory investigations are less abnormal than in Anorexia Nervosa. The aims of treatment are to reduce and, where possible, eliminate binge eating and purging, treat physical complications, enhance patients’ motivation, provide education regarding healthy nutrition and eating patterns, help patients reassess and change core dysfunctional thoughts and attitudes related to the eating disorder,
treat associated psychiatric conditions, enlist family support and provide family counselling and therapy (where appropriate) and prevent relapse.

The eating disorder category described as 'not otherwise specified' consists of a clinically heterogeneous group of diagnoses. Two eating disorders are proposed as syndromes found in the obese population: Binge Eating Disorder and Night Eating Syndrome.

There is no empirical evidence supporting a correlation between dieting and the development of the above eating disorders. Combined therapy by cognitive-behavioural / interpersonal therapy and psychopharmacologic treatment and weight reduction programs has achieved better results than isolated treatment of the 'not otherwise specified' group.

8. Clinical case

CASE HISTORY

A 27 years old female is admitted to our eating disorders unit.

She refers a family record of mother's dieting.

At puberty, she suffered strong negative feelings about her body and, when she was 16 years old, began to restrain food (restricting carbohydrates), to pursue a lower weight. She lost 8 kg during one year and suffered amenorrhea during 5 months. When amenorrhea turned up, she worried about her nutritional status and stopped dieting. Then, she began to suffer impulsive desires to eat, forcing her to binge. She tried to provoke self-vomiting in order to prevent weight gain, but she was unable to vomit. Therefore, she began running and going to the gym many hours a week. She never engaged in the misuse of laxatives, diuretics or enemas.

Last ten years, she has suffered recurrent episodes of binge eating (at least, three times a week, occasionally at night time) with compensatory behaviours like excessive exercising (for more than an hour after binge eating), just to keep her from gaining weight. She has not fasted, her self-evaluation is unduly influenced of body weight or shape, but she hasn't got disturbance in her body's shape perception.

Currently, her weight is 53,650 kg and her BMI 20, 95 kg/m². Laboratory findings are normal. She complains of gastric pain and uncomfortable feeling of fullness in her stomach after binging. She has lost appetite and satiety senses, and she feels very worried and unhappy about this recurrent sense of lack of control over eating. She has develop some co-morbid depression and anxious symptoms.

Q1: Which is the present diagnosis?
Q2: Which was the diagnosis when she was 16 years old?
Q3: Which are the aims of the treatment? 

9. Self-assessment test

10. References


