Clinical Assessment of the Patient with Overweight or Obesity

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James D. Crane and Barbara M. McGowan

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Abstract

There is a global pandemic of obesity. Now widely recognized as a disease in its own right, obesity is capable of adversely affecting the function of all organ systems and worsening the course of many coexisting morbidities. While the ability to carry out a basic clinical assessment of obesity is increasingly necessary for healthcare professionals in all fields, the comprehensive assessment of obesity requires multidisciplinary expertise, including that of obesity physicians, dieticians, psychologists and/or psychiatrists, and bariatric/metabolic surgeons. This assessment must consider potential drivers of obesity, the effect of obesity on an individual's physical and mental health, physical function, and the impact of obesity on other aspects of their wellbeing including their family, social, and economic lives.

Keywords

Obesity · Clinical assessment · Multidisciplinary team working · Comorbidities · Severity scores

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Introduction

The epidemic of overweight and obesity has spread from advanced economies into the developed world, and in 2014, there were an estimated 1.9 billion adults globally who were overweight, of whom 600 million were obese (WHO 2016). Obesity is an established risk factor for, or is associated with, a multitude of other conditions including hypertension, coronary vascular disease, type 2 diabetes, stroke, and cancers to name but a few of the most important. It has been estimated that in 2010, overweight and obesity caused 3.4 million deaths and was responsible for loss of around 100 million disability-adjusted life years globally (Lim et al. 2012). The costs of treating obesity and its sequelae are huge. For the UK, the Foresight Report, commissioned in 2007, estimated that by 2050 the total direct and indirect economic cost of obesity could reach £50 billion per annum in 2007 pounds (Foresight 2007). People with obesity use primary care and acute care services more frequently than those of normal weight (Guallar-Castillón et al. 2002); healthcare workers of all types will encounter obesity with growing frequency over the coming decades. The clinical assessment of obesity will become a routine and increasingly important healthcare activity and should be directed toward both an understanding of the cause for a person's obesity and a search for any present or impending sequelae. This chapter will provide an overview of the comprehensive obesity assessment, some of which is possible in primary care with the remainder usually requiring a referral to a specialist multidisciplinary team.

The Clinical Assessment of Obesity

Overview of the Clinical Assessment of Obesity

While, on a practical level, obesity is usually rather crudely defined by a person's body mass index (BMI) (BMI = weight (kg) ÷ height (m^2)), most of the negative health effects of obesity can be attributed to an elevation of fat mass (the most common cause for an increased BMI). The extent to which increased fat causes or influences the numerous and varied diseases associated with obesity is dependent on many things: the underlying cause for its increase, its anatomical location, its distribution between the different types of adipose tissue, its overspill into ectopic (nonadipose) sites, and its interaction with other aspects of the patient's prevailing medical and social circumstances. Obesity may be "simple," i.e., the result of normal adaptive or maladaptive physiological processes faced with a sustained surfeit of energy, or it may be secondary to a pathological process. The location of excess fat at different body sites and in different adipose types is influenced heavily by genes, both through their determination of the intrinsic properties of adipose cells and of the cell signaling molecules that regulate adipose tissue. This is most obviously evident in the marked differences in male and female fat distribution. Fat depots vary markedly in their association with metabolic disease, with upper-body (principally abdominal) fat being more "unhealthy" than lower body (gluteal or femoral) fat

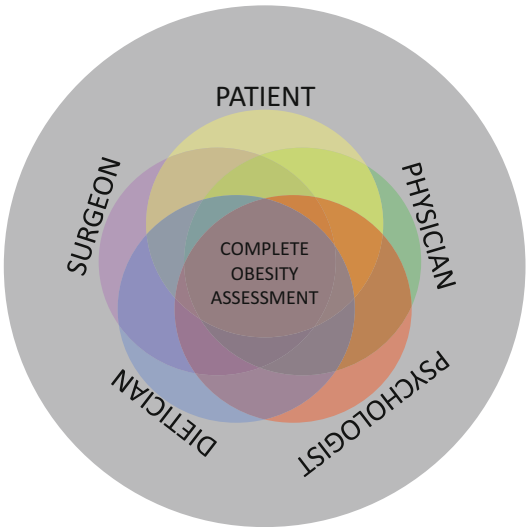

Fig. 1 The multidisciplinary assessment of the patient with obesity. The *gray circle* represents the context in which the assessment is made: the patient's health, economic, social, and family circumstances. The *colored circles* represent the unique expertise of each member of the multidisciplinary team, with the complete obesity assessment requiring the input of all of these

(Lee et al. 2013). Recent research has suggested that individuals may have a genetically determined upper limit to their capacity to store fat in "healthy" sites, and as such have different thresholds above which excess fat is stored in metabolically unhealthy or ectopic sites (Lotta et al. 2017). Once present in ectopic sites, there may also be a person-specific threshold for fat to cause tissue or organ dysfunction (Taylor and Holman 2015). Increased body weight can impact on health independent of the pathophysiology of adiposity, for example by exacerbating pain from joint disease or physically compressing the airways to cause obstructive sleep apnea. In addition, individuals with obesity continue to be subject to discrimination in various forms in many modern societies with a knock-on effect on quality of life and psychological or psychiatric ill health. Thus, a complete assessment of obesity must investigate all these aspects to provide a proper description of its severity and requires the expertise of multiple healthcare disciplines including physicians, surgeons, dieticians, and psychologists. Importantly, it also requires self-reflection and honest testament from the patient themselves (Fig. 1).

History, Examination, and Screening Investigations

Clues to Etiology

Taking a careful weight history can reveal many clues as to the cause of an individual's weight gain. While the exact causes of the current obesity epidemic continue to be elucidated and debated, it is widely accepted to be driven largely by a surfeit of readily available, high energy food for a population leading ever more sedentary lives yet who remain evolutionarily adapted to survive in an environment of relative food scarcity. Notwithstanding this, under ordinary circumstances food intake is regulated by long-term neurohumoral homeostatic mechanisms, principally

involving signaling of overall energy stores from leptin and insulin, that over time equilibrate energy intake with energy expenditure to maintain a body weight set point. In some individuals, these homeostatic mechanisms result in a stable bodyweight above the healthy weight range, while in others they fail to hold body weight and progressive weight gain ensues (Vistisen et al. 2014). This may be the result of specific impairments: for example, obesity has been linked to resistance to leptin (Frederich et al. 1995), exemplified by the relative lack of potency of leptin as a pharmacotherapy to successfully reduce weight in patients with obesity (other than those with leptin deficiency) (Heymsfield et al. 1999). Superimposed on the homeostatic control of food intake are the so-called "hedonic" influences involving higher cortical and limbic pathways (concerned with the reward value of eating certain foods), and short-term neurohumoral signaling patterns controlling meal onset and conclusion (satiety). It is hypothesized that, at times, hedonic drivers overcome the metabolic homeostatic mechanisms and drive excessive energy intake (Yu et al. 2015). Exploring the circumstances surrounding periods of accelerated weight gain, or indeed weight loss (outside concerted efforts to lose weight), can often elucidate important contributory factors. The activity of hedonic pathways is influenced by, among other things, emotional stress. Indeed, it is commonplace for obese patients to describe periods of accelerated weight gain coinciding with stressful life events or as part of coping mechanisms for psychological or psychiatric illnesses, e.g., depression, in which case addressing the underlying cause is essential to the successful treatment of their obesity. Energy intake may be driven by other factors external to these control mechanisms. For example, alcoholic drinks are rarely consumed for their nutritional value, yet often contain a considerable amount of energy and may be drunk due to dependency or social imperatives leading to periods of significant excesses of energy intake. Another frequently encountered scenario is weight gain following cessation of high levels of physical activity, for example in an ex-elite athlete or when someone loses a very physical job. In such cases, habits around the timing, frequency, or size of meals may alter the neurobiological control of initiation of feeding, meal cessation (satiety), or interval between meals (Asher and Sassone-Corsi 2015), which subsequently fails to revert to normal as energy expenditure falls.

Obesity resulting from secondary pathology may also give rise to abrupt weight increase. Although almost always accompanied by other discernible symptoms or signs, the high-profile of body image and negative connotations of obesity often lead to weight gain being the presenting complaint. Thyroid hormone receptor activity is a key modulator of resting energy expenditure through its regulation of futile substrate cycles and uncoupling of oxidative respiration from ATP synthesis (Vaitkus et al. 2015). Hypothyroidism may induce weight gain from a stable baseline or accelerate the course of ongoing weight increase. Cushing's syndrome (glucocorticoid excess) has a complex effect on adipose tissue (Fardet and Fève 2014), initiating first the liberation of free fatty acids from adipose stores, followed by a regionally specific hypertrophic and hyperplastic effect on central adipose tissue. In combination with its catabolic effect on muscle mass and appetite stimulating effects in the hypothalamus (Spencer and Tilbrook 2011), chronic overexposure to

glucocorticoids causes a characteristic central adiposity with wasting of limb tissues. A similar pattern may also be seen with partial lipodystrophy syndromes. Lesions of the hypothalamus leading to dysregulation of energy balance and obesity may result from head injury, vascular, or neoplastic conditions and lead to abrupt increase in weight coincident with the onset of the lesion.

Work from the Genetics of Obesity Study at Cambridge University (www.goos.org.uk), and elsewhere, has identified specific monogenic causes of obesity sharing the characteristic of very early onset of excessive weight gain. With increasing prevalence of obesity in children, the discriminatory power of the presence of an early-onset of obesity will diminish. However, when combined with a careful family history revealing a pattern suggestive of Mendelian inheritance, a monogenic cause should be considered. In the case of leptin deficiency, hyperphagia and obesity can be completely ameliorated by administration of replacement hormone (Farooqi et al. 2002), transforming prognosis for these individuals.

Obesity is a prominent feature of other genetic syndromes, most prominently Bardet-Biedl and Prader-Willi syndromes. Bardet-Biedl, characterized by obesity, retinitis pigmentosa, polydactyly, renal anomalies, urogenital tract defects, and learning difficulties, is an autosomal recessive condition of cilia dysfunction and may cause obesity due to defective trafficking of the leptin receptor (Guo and Rahmouni 2011). Prader-Willi is an autosomal dominant syndrome usually due to sporadic chromosomal events leading to deletion or loss of function of a region on the long arm of chromosome 15, 15q11-13. Sufferers are typically short in stature with learning difficulties, behavioral problems, hypotonia, hypogonadism, and abnormalities of sleep. Feeding difficulties related to hypotonia result in initial failure-to-thrive, but this is followed by pronounced hyperphagia and obesity. The underlying molecular mechanisms linking Prader-Willi to obesity remain unclear but are likely to be polyfactorial (Khan et al. 2016). Other genetic syndromes linked to obesity have been characterized but are extremely rare. Nevertheless, genetic investigations are warranted when obesity is of early onset and accompanied by other congenital or developmental anomalies.

Summary

General factors to consider in the obesity history:

- Age of onset of excess weight
- Where onset as a young child, the presence of other traits suggestive of genetic syndromes
- Family history of obesity and its pattern, especially if severe obesity is dichotomously present with normal weight
- Pattern of weight gain, noting periods of acceleration or weight loss and their relation to health or life events
- Intake of alcohol or other highly calorific liquids
- Success and failure of previous attempts at losing weight

Identification of Sequelae and Important Comorbidities

Cardiovascular disease. The prevalence of hypertension (usually defined as systolic BP >140 mmHg and/or diastolic BP >90 mmHg) may be up to 75% in obese people, compared to approximately 25% of the population as a whole. Diagnosis in obese patients may be confounded by a lack of availability of appropriately sized sphygmomanometry cuffs and the absence of a prior diagnosis should not be relied upon as an indicator of normotension. Both due to the high prevalence of hypertension and its role as a prominent risk factor for cardiovascular death, improvement in blood pressure is a key health goal for patients with concomitant obesity and hypertension; its identification is an important priority in the obesity clinic. It is widely accepted that 24 h ambulatory blood pressure monitoring outperforms clinic measurements in predicting cardiovascular events and this is the preferred diagnostic test in most guidelines.

Obesity is strongly associated with atherosclerotic disease, both independently and through sequelae such as dyslipidemia and diabetes. Some patients with obesity attribute even quite marked exercise-induced chest or limb pain as being simply due to their weight and may not volunteer the presence of these symptoms without direct questioning. The presence of significant coronary arterial disease may have important implications for both short-term health and potential treatment options for obesity, in particular bariatric surgery and anesthetic risk, and should be characterized as fully as possible.

Even in the absence of significant ischemic heart disease, obesity is associated with a specific cardiomyopathy. The etiology is multifactorial and due to the effects of volume overload from a hyperdynamic circulation, the metabolic effects of excess fat deposition in the heart ("lipotoxicity") and the bioenergetic effects of insulin resistance and a switch to exclusive fatty acid metabolism (Abel et al. 2008). Comorbidities, including diabetes, hypertension, and obstructive sleep apnea, have additional deleterious effects on the myocardium. Hypertrophy, which may be eccentric or concentric, and fibrosis result in ventricular dysfunction that is often predominantly diastolic in the early stages. Breathlessness on exertion, orthopnea, and paroxysmal nocturnal dyspnea are all commonly encountered symptoms that raise suspicion of cardiomyopathy. Technical limitations of readily available investigations often lead to challenges in accurately characterizing the heart's function. While fluid retention and the presence of subcutaneous edema of the lower limbs may be due to heart failure, it may also be due to other factors such as the activation of the renin-angiotensin-aldosterone system associated with obesity that leads to salt and water retention, the increase in hydrostatic pressure of the lower limb venous system due to compression of the abdomino-pelvic vessels, or through dysfunction of the lymphatic system (linked to obesity through unknown mechanisms). A raised jugular venous pulsation/pressure may be difficult to see but when present usually indicates pulmonary arterial hypertension that may be due to congestive cardiac failure or cor pulmonale due to the chronic hypoxia of obstructive sleep apnea and obesity hypoventilation. Valvular disease, somewhat counterintuitively, may be *less* frequent in obese people (Singh et al. 1999).

A quantified estimate of the 10-year risk for cardiovascular and cerebrovascular events can be made using risk engines, such as QRISK-2 (Hippisley-Cox et al. 2008), developed and validated against datasets of the general primary care population in the UK and which, in contrast to other risk engines such as those derived from the Framingham and UKPDS datasets, includes BMI as a variable.

Summary
Basic cardiovascular assessment should include:

- Enquiry after personal history of exercise induced pain of the chest or limb
- Family history of hypertension or vascular disease
- Smoking and alcohol history
- History of exertional breathlessness, orthopnea, or paroxysmal nocturnal dyspnoea
- Examination of the cardiovascular system
- Electrocardiography
- Echocardiography, nuclear medicine perfusion scan or other cardiac imaging if symptoms are present
- FBC, creatinine, electrolytes, B-type natriuretic peptide (be aware that false negatives are more frequent in obesity), urinalysis for protein

Metabolic disease. The obesity epidemic advances hand-in-hand with that of type 2 diabetes, with insulin resistance increasing directly as a function of BMI. However, the degree of insulin resistance for a given level of adiposity is subject to much inter-individual variability. Fat is stored in several adipose tissue depots which are functionally distinct (Fig. 2) and site of fat deposition is highly influential. Central fat accumulation in the "android" pattern results in greater insulin resistance than peripheral fat accumulation in the "gynecoid" pattern (these common terms are used advisedly as body fat distribution does not always split neatly along gender lines). These patterns are easily identifiable visually and are evaluated by the waist-to-hip circumference ratio (as defined by the World Health Organization: the waist is taken to be the point midway between the apex of the iliac crest and the lower limit of the ribcage, and the hip is taken to be the maximum circumference around the buttocks with the tape parallel to the floor). More important, but invisible on examination, are interindividual differences in liver and pancreatic ectopic fat accumulation and their impact on insulin sensitivity and beta cell function, respectively. Gross steatosis of the liver may result in detectable hepatomegaly, with or without tenderness, if significant inflammation (steatohepatitis) is present. Diabetes is often present without symptoms (or without symptoms having been recognized) for a prolonged period of time prior to diagnosis. Symptoms should be enquired after, including those of hyperosmolarity (polyuria, nocturia, polydipsia, intermittent visual acuity deficit), frequent bacterial

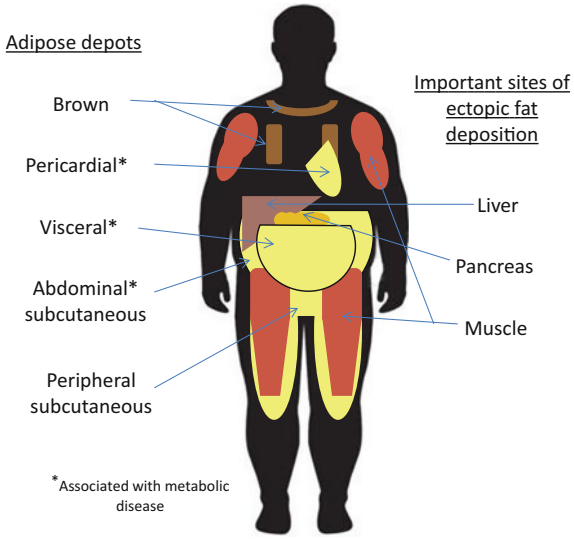

Fig. 2 Location of fat depots and sites of ectopic fat deposition. White adipose tissue exists in functionally distinct depots. Abdominal subcutaneous, visceral (omental, mesenteric, retroperitoneal, and perinephric) and pericardial fat are associated with an increased risk of metabolic and vascular disease. Brown fat tissue is found around the neck and mediastinum. Lipid may also deposit ectopically in nonadipose tissue with adverse biochemical and metabolic effects. Important sites of ectopic fat deposition include skeletal muscle and liver (causing insulin resistance) and the pancreas (causing islet dysfunction)

or fungal infections of the genitourinary tract or skin, or weight loss that is significant or out of keeping with a patient's efforts. Glycated hemoglobin (HbA1c) remains the best available measure of diabetes control, with the risk of vascular complications increasing in line with HbA1c (Stratton et al. 2000). With standardization of laboratory measurement of HbA1c, diagnosis of diabetes (and "prediabetes") is now approved without the need for formal oral glucose tolerance testing (WHO 2011). An initial assessment of the "severity" of diabetes in essence equates to the measurement of glycemia (using HbA1c) and identification of microvascular complications (retinal photography, estimation of glomerular filtration rate, screening for albuminuria, and foot examination) and macrovascular complications (identification of cardiovascular or stroke disease through history and examination and electrocardiography). More sophisticated investigations of diabetes complications can be guided by initial findings. In those with poor glycemic control and retinopathy for whom aggressive weight loss interventions are being considered, sequential retinal photographs should be planned during and after intervention due to the likelihood of rapid correction of HbA1c and the possibility that this may lead to worsening of retinopathy (Thomas et al. 2014). When established neuropathy is present, podiatry

surveillance may be necessary as increased mobility and altered biomechanics following large weight loss may increase the risk of new pressure ulcers or Charcot foot (Murchison et al. 2014).

Obesity, insulin resistance, and hyperlipidemia/dyslipidemia are intimately linked. Energy excess leads to obesity and adiposity, adiposity results in saturation and dysfunction of adipose depots, impaired lipid storage leads to elevated circulating free fatty acids and triglycerides resulting in ectopic lipid deposition, ectopic fat in the liver and pancreas impairs insulin activity, reduced hepatic insulin signaling leads to altered hepatic lipoprotein synthesis and impairs clearance of atherogenic apolipoprotein B100-containing particles (VLDL, IDL, LDL), strongly increasing cardiovascular risk. Hyperlipidemia itself is largely asymptomatic and detectable signs on examination relatively uncommon; corneal arcus may be present. A basic lipid profile including total cholesterol, HDL-cholesterol, and triglyceride concentrations enables a QRISK-2 calculation and assessment of pancreatitis risk (triglyceride >1000 mg/dl (11.2 mmol/L) (Berglund et al. 2012).

Summary

Basic metabolic assessment should include:

- Waist circumference and waist-to-hip ratio
- In the absence of previous diabetes diagnosis:
  - Enquiry after symptoms of hyperglycemia
  - Fasting glucose and HbA1c
- In the presence of a previous diabetes diagnosis:
  - Family history of type 2 diabetes
  - HbA1c and review of home blood glucose monitoring (if available)
  - Foot examination
  - Urinalysis for protein and urine sample for albumin:creatinine ratio
  - Enquiry after adherence to retinal screening, outcomes from this and fundoscopy if not-adherent
  - Screening questions for contraindications to metformin, SGLT-2 inhibitors and GLP-1 agonists (since these agents may be preferred for the purposes of facilitating weight loss)
- Examination for stigmata of familial hyperlipidemia syndromes
- Lipid profile (total and HDL-cholesterol and triglyceride as a minimum)

Reproductive Health. Obesity is associated with impairments of both male and female reproductive health. In men, aromatase, present in adipose tissue, is responsible for the aromatization of the A-ring of testosterone, converting it to estradiol. Since estradiol is equally as able to suppress the hypothalamo-pituitary-gonadal axis through negative feedback as testosterone, leutinizing hormone (LH) is not

upregulated to restore testosterone levels resulting in symptoms of hypogonadism which include erectile dysfunction, loss of libido, gynecomastia, testicular and penile atrophy, reduced muscle bulk, neurocognitive symptoms, and infertility. Hypogonadism may be found in up to 60% of men presenting for bariatric surgery (Calderón et al. 2014). Erectile dysfunction is commonly the trigger for seeking medical attention for hypogonadism, but may also be caused by many of the other consequences of obesity, for example vascular disease, medications, or depression. Medical practitioners may be misled by low *total* testosterone levels in obese patients and diagnose hypogonadism, missing alternative etiologies. Two-thirds of testosterone circulates tightly bound to sex hormone binding globulin (SHBG) (and much of the rest weakly bound to albumin) rendering it unavailable for biological activity but nonetheless detectable on a total testosterone assay. Reductions in SHBG thus reduce the total testosterone concentration but have little impact on free testosterone (or bioavailable testosterone – free + albumin-bound testosterone). As such, total testosterone is unreliable in obese patients for whom free (or bioavailable) testosterone should be measured instead. Obesity also adversely impacts spermatogenesis in men resulting in both reduced sperm count and motility (Hammoud et al. 2008) and an assessment of male fertility is also warranted.

In women, the predominant impact of obesity on reproductive health is through its association with polycystic ovarian syndrome (PCOS). Although the precise pathophysiology of PCOS is incompletely understood, hyperinsulinism driven by adiposity and insulin resistance is thought to be of primary importance. Hyperinsulinism is associated with increased pituitary LH, and the combination of the two results in increased androgen production by the ovary. This is exacerbated by suppressed hepatic SHBG and results in the clinical features of PCOS that may be present in up to three quarters of obese women of child-bearing age. These include: oligomenorrhea (menstrual cycle of >35 days), anovulatory amenorrhea, infertility, acne, male-pattern alopecia, and hirsutism. Diagnosis is confirmed by the Rotterdam Criteria (The Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group 2004): presence of 2 out of 3 of oligo/anovulation, hyperandrogenism (clinical or biochemical), and polycystic ovaries (on ultrasound), with exclusion of other etiologies (for example Cushing's syndrome, congenital adrenal hyperplasia and androgen-secreting tumors). Elevated LH, while not formally part of the diagnostic criteria, is usually present.

Overall, obesity is associated with reduced fecundity even in women without ovulatory disruption (Gesink-Law et al. 2007) and also with reduced success of assisted conception (Shah et al. 2011). As a result, access to assisted conception is often restricted for obese women. For example, in the United Kingdom, although national guidelines fall short of excluding obese women from in-vitro fertilization, funding bodies typically insist on a BMI <30 kg/m^2. Due to the central importance placed on fertility by many patients, the identification of fertility as a motivation for

weight loss and knowledge of local rules for access to fertility interventions is an important part of the clinical assessment of obese female patients and will affect treatment decisions and timescales.

Summary
Basic reproductive health assessment should include:

- In men:
  - Enquiry after symptoms of low libido and erectile dysfunction
  - Enquiry after desire for fertility
  - Systemic examination for signs of hypogonadism (loss of body hair, gynecomastia, loss of muscle bulk)
  - Genital examination (pubic hair, phallus length, testicular size, and consistency)
  - Testosterone, LH/FSH, SHBG, LFT, blood to be taken in early morning (at of before 9 am)
  - Semen analysis if evidence of hypogonadism and fertility is desired
- In women:
  - Obstetric history
  - Menstrual history
  - Examination for signs of androgen excess (facial/body hair, male pattern balding, acne)
  - If PCOS/androgen excess suspected: testosterone, LH/FSH, SHBG, transvaginal ultrasound

Gastrointestinal disease. Gastroesophageal reflux disease has a clear association with obesity (Jacobson et al. 2006). Increased abdominal pressure predisposes to hiatus hernia and lower-esophageal sphincter dysfunction, which may be further impaired by metabolic changes including elevation of estrogen. Obesity is also associated with the development of Barrett's esophagus and esophageal adenocarcinoma (Singh et al. 2013). Presence of upper gastrointestinal (UGI) pathology may influence choice of intervention for obesity: history of inflammatory disease is a contraindication to intragastric balloon insertion and endoluminal liners (e.g., Endobarrier™). Bariatric surgery has been variously reported to improve or worsen symptoms of reflux, which may influence surgical decisions on the choice of, or contraindication to, the surgical procedure (Mion and Dargent 2014). Symptoms of UGI pathology include retrosternal burning pain (particularly associated with food), dysphagia, odynophagia, acid brash, nocturnal wheeze, laryngitis, and pharyngitis, particularly in the morning. Dysphagia may be due to obesity-associated dysmotility or strictures of the esophagus, either inflammatory or neoplastic.

Obesity, in particular central obesity, is a potent risk factor for cholesterol gallstone disease. Gallstone cholecystitis is the likely reason for obesity being linked to an increased risk of gallbladder cancer. Cholecystitis can be detected through the presence of symptoms, principally right-upper quadrant pain that may radiate to the right shoulder tip and is exacerbated by fat intake, which may be accompanied by a positive Murphy's sign on examination. Symptoms may be acute and accompanied by vomiting and fever, or milder and chronic, but are absent altogether in the majority of cases. Choledocholithiasis may result in a history of intermittent jaundice or of gallstone pancreatitis. Rapid weight loss, for example through very low calorie diets or bariatric surgery, predisposes to gallstone formation lending importance to the detection of gallstone disease prior to such interventions. Cholesterol gallstones are radiolucent and rarely seen on plain x-ray, while computed tomography is only 80% sensitive. Ultrasound is highly sensitive and is the investigation of choice.

The presence of nonalcoholic fatty liver disease (NAFLD) is almost universal in patients with morbid obesity and is instrumental in driving insulin resistance. In a proportion of cases it is accompanied by steatohepatitis (NASH), probably driven by the accumulation of toxic lipid intermediates in the hepatocyte (Caligiuri et al. 2016), which is associated with progression to cirrhosis in up to a third of cases. In one meta-analysis, NASH was found in over one-third of patients undergoing bariatric surgery (Machado et al. 2006). NAFLD and NASH may be detectable by clinically apparent hepatomegaly which may or may not be tender. If cirrhosis is present, the signs of portal hypertension may be found including ascites, splenomegaly, and distended abdominal veins. Other signs of liver failure include spider naevi, Dupuytren's contracture, palmar erythema, and jaundice. NASH-related cirrhosis is becoming an increasingly common indication for liver transplant. Although the impact of obesity on graft and patient survival is variable across studies (Perez-Protto et al. 2013; La Mattina et al. 2012) and may not actually be that deleterious, obesity often features as a relative contraindication in transplant criteria (Varma et al. 2011) and obese patients can face a longer wait on the transplant list (Segev et al. 2008). Significant weight loss, for example after bariatric surgery (Lasailly et al. 2015), is a highly effective treatment for NAFLD and NASH. The early detection of NASH is thus of great importance. Transaminases are often normal, although NAFLD is characteristically associated with elevation of alanine aminotransferase (ALT) before aspartate aminotransferase (AST) with an AST:ALT ratio of <0.8 (in contrast to alcoholic liver disease). Gamma glutamyl transferase (GGT) is often also raised. Noninvasive attempts to differentiate steatohepatitis from simple steatosis have been sought to avoid the need for liver biopsy. At the most basic level, further AST rise resulting in an AST:ALT ratio of >0.8 is associated with the presence of cirrhotic scarring (fibrosis). More complex scoring systems, such as the FIB-4 index (Sterling et al. 2006), may be helpful. Conventional imaging is generally unable to differentiate the two. Ultrasonographic transient elastography scores correlate with the presence of fibrosis, but the technique is subject to technical

challenges in obese patients. Biopsy is usually necessary if confirmation of cirrhosis is required.

Summary
Basic gastrointestinal assessment should include:

- Alcohol history
- Enquiry after symptoms of reflux, inflammation, ulceration, or strictures of the UGI tract
- Enquiry after right-upper quadrant symptoms associated with gallbladder pathology or hepatitis
- Examination of gastrointestinal system
- FBC, ALT, AST, gamma GT, ALP, bilirubin, electrolytes
- UGI endoscopy if symptoms present
- Liver ultrasound

Respiratory disease. Respiratory symptoms, such as exertional dyspnoea, are highly prevalent in obese people and are often due to the impact of obesity on normal cardiorespiratory physiology rather than any underlying respiratory disease. The increased oxygen requirement of a high body mass, reduced chest wall compliance, and an increased work of breathing lead to higher respiratory muscle oxygen demand. However, a number of respiratory pathologies are specifically linked to obesity, most notably obstructive sleep apnea and obesity hypoventilation syndrome.

Obstructive sleep apnea (OSA) refers to the repetitive occlusion of the upper airways due to loss of dilatory muscle tone during normal rapid eye movement (REM) sleep paralysis, resulting in apnea, hypoxia, and arousal. Suggestive symptoms include snoring, excessive daytime sleepiness (resulting from inadequate REM sleep), and morning headache of uncertain pathogenesis. These may be accompanied by neurocognitive and/or mood disturbances. Validated risk scores which predict the presence of OSA have been developed and include the Epworth Sleepiness Scale (Johns 1991) and the STOP-BANG (Chung et al. 2008) score. Diagnosis is confirmed during observed sleep through polysomnography, or alternatively through sleep oximetry which may be carried out in the patient's home. It is based on the frequency of recorded hypopnea (reduction in ventilation of at least 50% resulting in a fall in oxygen saturation by 4% or more) and apneas (cessation of breathing for >10 s). The number of episodes per hour of sleep is termed the Apnea-Hypopnea index (AHI). Mild OSA is defined as an AHI of 5–15, moderate 15–30, and severe >30. AHI increases exponentially with BMI (Newman et al. 2005), and subsequent poor sleep may contribute to increased appetite and weight gain (Spiegel et al. 2004), locking patients into a vicious cycle of increasing weight and worsening OSA. The presence of OSA is associated with

an increased risk of cardiovascular events and the development of pulmonary arterial hypertension. Early weight loss intervention is central to reducing the severity. For patients with OSA undergoing bariatric surgery, anesthetic and analgesic drugs may further reduce tone in the dilatory muscles of the upper airway, impair the arousal response to hypoxemia and reduce ventilatory drive, increasing the likelihood of respiratory failure in the perioperative period. Preoperative diagnosis with initiation and optimization of nonsurgical management (continuous positive airway pressure) is likely to reduce the risk of complications at the time of surgery.

A separate condition, but one that often coexists with OSA (particularly at the severe end of the OSA spectrum) is obesity hypoventilation syndrome (OHS). The restrictive effect of excess body tissue mass on ventilation necessitates a response from the respiratory centers of the central nervous system to increase minute-ventilation and maintain a normal partial pressure of carbon dioxide (pCO_2). In patients who develop OHS, this response is impaired, resulting in hypoventilation and hypercapnia (raised pCO_2). Hypercapnia may cause an increase in somnolence and cognitive impairment ("CO_2 narcosis"), flushing (due to CO_2-induced vasodilatation), muscle twitches/myoclonus, and palpitations. Hypoventilation also worsens hypoxia and thus pulmonary hypertension leading to more severe dyspnea and higher right heart pressures. Physical signs include plethora, subcutaneous edema, asterixis ("CO_2 retention flap"), right ventricular heave, split S2 with a loud pulmonary component, heart murmurs (initially the early diastolic murmur of pulmonary regurgitation, resulting in progressive volume overload, right ventricular dilatation, and then the pansystolic murmur of tricuspid regurgitation), and elevated JVP (initially with a prominent A-wave, then, when tricuspid regurgitation develops, a giant V-wave). Diagnosis is confirmed by the presence of daytime hypercapnia on arterial blood gas analysis and treatment is with weight loss and noninvasive ventilation (either by continuous or bilevel positive airway pressure, with or without supplemental oxygen).

Asthma is diagnosed with higher frequency in the obese population, although BMI does not appear to be clearly associated with either an increase in airway hyper-responsiveness or airway inflammation (Zammit et al. 2010). The symptoms may instead be due to the physical effect of increased abdominal pressure and chest wall tissue mass on lung compliance, lung volumes, and airway caliber and demonstrates the capacity of obesity to worsen lung function in the presence of any underlying lung pathology. As such, identification of pulmonary disease represents an indication for more urgent efforts at weight loss in the obese patient. The possible exception is in the case of end-stage obstructive airways disease in which elevated BMI may confer a survival advantage (Schols et al. 1998), an example of the obesity paradox – the counterintuitive association of obesity with a survival advantage in certain conditions that also include congestive cardiac failure, malignancy, and critical illness.

Summary
Basic respiratory assessment should include:

- Enquiry after symptoms of breathlessness and the circumstances of these
- Enquiry after symptoms of snoring or daytime sleepiness
- Enquiry after symptoms of CO_2 retention
- Questionnaire-based screen for OSA (e.g., Epworth or STOP-BANG questionnaire)
- Examination of respiratory and cardiovascular systems
- Arterial blood gas if OHS suspected
- Spirometry
- Polysomnography or overnight sleep oximetry if screening questionnaire for OSA high or if considering surgical intervention for weight

Musculoskeletal disease. Obesity has been consistently shown to be a strong risk factor for osteoarthritis (OA). For example, in the Framingham Study, the relative risk for the presence of osteoarthritis in women of the highest weight quintile was 2.07 compared to those of the lowest three quintiles (Felson et al. 1988). Obesity results in biomechanical strain through the joints of the lower limbs, hastening cartilage loss. Metabolic and systemic inflammatory changes in obesity have also been implicated: obesity increases the risk of small joint osteoarthritis as well as that of large joints (Yusuf et al. 2010). Obesity makes lower limb joint arthroplasty technically more challenging and results in more rapid wearing of joint prostheses, both of which contribute to poorer outcomes from arthroplasty in obese patients. BMI-based eligibility criteria for elective joint replacement surgery are becoming commonplace (The Royal College of Surgeons of England 2016). Weight loss results in improvement of symptoms of native joint osteoarthritis and more successful arthroplasty, leading to disabling osteoarthritis being a common indication for bariatric surgery. Obesity may also be a risk factor for the development of inflammatory arthritis (George and Baker 2016) which, due to widespread awareness of the strength of obesity as a risk factor for osteoarthritis, may be misdiagnosed as OA. Synovial inflammation is the predominant feature of inflammatory arthritides and is much less important (though often still present) in osteoarthritis. Its major symptom, painful joint stiffness after a period of disuse (e.g., in the morning), is thus much more pronounced and lasts for longer (an hour or more) in inflammatory arthritides than in OA (usually less than half an hour). Pain on use of the joint is more pronounced in OA.

Low back pain is amongst the commonest chronic symptoms in the general population and is even more prevalent in obese patients (Shiri et al. 2010). Pain may be related to pathology of the bony structures of the spine such as degenerative disc disease or spinal stenosis, or to that of the surrounding soft tissues. Nerve root

impingement in the exit foramina of the spine by intervertebral disc herniation may result in sciatica, the symptom of burning, or electric shock-like pain in the legs, and is reproducible on examination on a straight-leg-raise test. The etiology of low back pain is not always clear even after extensive investigation and imaging is best guided by orthopedic specialists. The clinical approach is usually conservative with exercise and chronic pain management.

Clinical assessment of orthopedic conditions in obese patients should be geared toward accurate diagnosis and an attempt to quantify its impact on function and quality of life to allow for goal-orientated interventions with shared decision-making by obesity and orthopedic or rheumatology teams and the patient, allowing for realistic predictions of the impact of bariatric or orthopedic surgical interventions on the morbidity associated with musculoskeletal disease.

Summary

Basic musculoskeletal assessment should include:

- Enquiry after symptoms of inflammatory and degenerative joint disease
- Medication history, especially steroid use as a potential driver of weight gain
- Determination of the extent of associated functional limitation
- Determination of any BMI or obesity-related barriers to accessing treatment

Neoplastic disease. Obesity has been estimated to account for up to 20% of cancer deaths (Calle et al. 2003), while significant and sustained weight loss is protective against cancer mortality (Sjöström et al. 2007); See ▶ Chap. 1, Epidemiology of Obesity). The authors of the second report on Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective by the World Cancer Research Fund and American Institute for Cancer Research (World Cancer Research Fund/American Institute for Cancer Research 2007) concluded that there exists convincing evidence to link adiposity to cancer of the esophagus (adenocarcinoma), pancreas, colorectum, breast (in post-menopausal women), endometrium, and kidney, as well as a probable link to gallbladder cancer. The symptom of recent and significant weight loss, common to all advanced malignancies, is not rare in the obesity clinic due to ongoing attempts to lose weight. However, weight loss that is surprising for the patient or seems excessive for the degree of effort should prompt further investigation.

The cardinal symptom of esophageal cancer is dysphagia, though odynophagia or retrosternal pain may also be present. A high index of suspicion is warranted for patients with a history of severe reflux or Barrett's esophagus and diagnosis is usually by endoscopy.

Pancreatic cancer is most often diagnosed late with the onset of painless obstructive jaundice (for tumors of the pancreatic head) or epigastric pain that may radiate to the back and be relieved by sitting forward (for tumors of the body and tail). Obstruction of the pancreatic duct may result in acute pancreatitis and destruction of the pancreatic parenchyma may result in new onset diabetes mellitus with insulin deficiency at diagnosis.

Colorectal cancer may present with unexplained iron deficiency anemia, change of bowel habit or rectal bleeding. Approximately half are rectal and palpable on digital examination.

Following public awareness campaigns, many women are used to performing self-examination of the breasts and may be able to report and locate any lumps they have noticed. Other symptoms include nipple discharge that may be blood stained, new nipple inversion, or skin changes. Breast cancer screening is routine from age 50 in the UK and attendance should be checked. Breast examination should be performed for those aged over 50 who have not attended screening and breast lumps are best investigated in a dedicated clinic, usually with ultrasound-guided fine-needle aspiration or biopsy of suspicious nodules.

Endometrial cancer is rare before the age of 45 years and the usual presentation is with postmenopausal bleeding which should prompt referral for transvaginal ultrasound and/or uteroscopy for diagnosis.

Renal cell cancer can present with painless hematuria, loin pain, or palpation of an asymptomatic abdominal mass. Unilateral left varicocele is a textbook but uncommon presentation.

In any obese patient with malignancy, caution should be employed before pursuing aggressive weight loss interventions due to the possibility of a survival advantage in obese patients. Studies have found a survival benefit for patients with overweight or obesity in several tumor types and including disseminated malignancies (Tsang et al. 2016), although the validity of this finding has been questioned (Lennon et al. 2016).

Summary

Basic cancer assessment should include:

- In women approaching menopause or postmenopausal:
  - Breast examination (unless they are up to date with an effective screening program)
  - Enquiry after symptoms of intermenstrual or postmenopausal PV bleeding
- Enquiry after symptoms of dysphagia
- Enquiry after a change of bowel habit
- Abdominal examination for epigastric or right-upper quadrant masses
- Urinalysis for blood

Neurological disease. Obesity is convincingly linked to the syndrome of idiopathic intracranial hypertension (IIH), also known as pseudotumor cerebri and previously known as benign intracranial hypertension, a name which fell out of favor due to the small but appreciable incidence of blindness in patients with IIH of approximately 2% per annum (Best et al. 2013). Ninety percent occurs in obese women of childbearing age and incidence in this population is approximately 20 per

100000 per annum. Patients report symptoms of high-pressure headache (worse in the mornings, on Valsalva maneuver or lying flat) and/or with visual symptoms including diplopia and blurred vision. Reduced visual acuity, a VIth nerve palsy, and papilledema (causing an enlarged blind spot) may be present on examination. Brain imaging may show subtle changes including an empty sella turcica (which may or may not result in hypopituitarism) and stenosis of the transverse cerebral venous sinus. Diagnosis is made on lumbar puncture in the lateral position with an opening pressure of $>25 cmH_2O$ and cerebrospinal fluid of normal composition. Weight loss of 5–10% of total body weight is effective at reducing intracranial pressure, symptoms, and papilledema (Wong et al. 2007). While waiting for this, pharmaceutical treatment is with carbonic anhydrase inhibitors (acetozolomide) or occasionally high-dose steroids if there are concerns about imminent visual loss. Therapeutic lumbar puncture to lower CSF pressure can be effective and CSF-shunting surgical procedures are required when other treatments have failed. Bariatric surgery-induced weight loss has been shown to be a highly effective treatment for the reduction of headache and papilledema (Manfield et al. 2017).

In sufferers of migraine headache, obesity has been linked to increased severity and frequency of episodes, perhaps through effects on inflammatory mediators and/or neurotransmitter disturbances, though whether migraine prevalence increases with BMI is uncertain (Ornello et al. 2015; Bond et al. 2011). Few data exist on the impact of weight loss on migraine, although one study in a pediatric population suggests that weight loss may reduce the frequency of migraine attacks (Hershey et al. 2009).

Summary
Basic neurological assessment should include

- Enquiry after symptoms of high pressure headache (particularly in younger, female patients)
- If headaches present, fundoscopy to assess for papilledema
- Enquiry after symptoms of nerve entrapment syndromes, particularly carpal tunnel syndrome, and any functional limitation associated with these
- Neurological examination

Psychiatric. In addition to the impact on physical health, obesity is also linked to psychiatric ill health. Depression and anxiety disorders are more prevalent in obese people, although typically studies have found this to be only a modestly strong association (Scott et al. 2008) which is greater in females than males. There is a high prevalence of obesity among people with psychotic disorders such as schizophrenia (Ventriglio et al. 2015), which is likely to be mediated through the impact of psychosis on lifestyle and the appetite stimulating effect of antipsychotic medication. Interestingly, there appears to be an inverse correlation between BMI and suicide risk in the general population (Bjørngaard et al. 2015) and a possible link between bariatric surgery and an increased suicide risk postoperatively (Tindle et al. 2010).

Overall, major psychiatric disorders are relatively common in obese patients and must be fully assessed and addressed prior to embarking on bariatric surgery or other radical weight loss interventions.

For a small minority of patients, their obesity is primarily driven by the presence of a definable disorder of over eating, most notably binge eating disorder (BED), bulimia nervosa (BN), and night eating syndrome (NES), although prevalence may be significantly higher in patients presenting for interventions like bariatric surgery (Niego et al. 2007). In the fifth revision of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-5), the key diagnostic criterion for both BED and BN is the combination of the recurrent eating of excessive amounts of food within a discrete time-frame with a sense of a lack of control over eating during the episode. BN is characterized by attempts to compensate for overeating and avoid weight gain (e.g., vomiting or excessive exercise), which may be successful or unsuccessful (and thus potentially leading to obesity). BED is characterized by feelings of distress regarding the binges, rapid eating, eating when not hungry or when feeling uncomfortably full, and eating in secret due to embarrassment. NES involves recurrent episodes of awakening from sleep to eat or excessive food consumption after the evening meal in a manner that is not consistent with a diagnosis of BED. Patients often find it difficult to discuss eating disorders in face-to-face consultation and questionnaire-based screening tools may be more revealing in the first instance, followed by more specific enquiry based on the patient's answers. Intervention for eating disorders is predominantly with psychological interventions such as cognitive behavioral therapy. Interestingly, some studies have shown very high response rates to placebo and also to weight loss programs that do not consider eating disorders (Stunkard and Allison 2003). Spontaneous remission is also common.

Summary

Basic psychiatric assessment should include:

- Assessment by a dietician, psychologist, or psychiatrist with expertise in eating disorders
- Screening for depression and anxiety (for example, by using a questionnaire such as the depression module from the PHQ questionnaire (Spitzer et al. 1999) (PHQ-9), or the Hospital Anxiety and Depression Scale (Zigmond and Snaith 1983))
- In patients with a history of psychotic illness, a full medication history with attempts to map it to weight changes
- In patients with suicidal ideation, assessment of safety by a psychiatrist prior to interventions such as bariatric surgery

Quantifying obesity severity – scoring systems. Obesity is a multifactorial, multifaceted, and complex disease and as such defining the severity of a patient's obesity is not straightforward. Nonetheless, attributing a severity rating to a

patient's obesity is useful to assess the urgency for intervention and follow its success over time. In the simplest terms, obesity is defined and then staged according to BMI in the World Health Organization classification: 25.0–29.9 kg/m^2 = overweight, 30.0–34.9 = class I obesity, 35.0–39.9 = class II obesity, and 40.0 or higher = class III obesity. This has the advantage of being easy to calculate, requiring few data and being translatable between populations. However, it ignores body composition such that even those with very little adiposity but high muscle mass may be classified as obese and does not allow for any description of comorbid status. To address these and other deficiencies, more sophisticated scoring systems have been developed.

Aylwin and Al-Zaman published The King's Obesity Staging Criteria in 2008 (Aylwin and Al-Zaman 2008). The impact of obesity is considered with respect to distinct domains of health and well-being with each domain staged according to defined criteria, from stage 0 (normal health), through stage 1 (at risk of disease) and stage 2 (established disease) to stage 3 (advanced disease). An updated version of the criteria (Whyte et al. 2014) is reproduced in Table 1. Serial scoring allows clinicians to assess the impact of interventions and has been used to show the benefit of bariatric surgery across all domains (Neff et al. 2014).

Sharma and Kushner introduced The Edmonton Clinical Staging System in 2009 (Sharma and Kushner 2009), which stages obesity according to the presence of comorbidity and limitations on function, from stage 0 with no impact, to stage 4 with potentially end-stage disease or severely disabling limitation on function. The full-staging criteria are reproduced in Table 2, with the suggested management from the original publication for patients at each stage. Edmonton stage has been shown to predict mortality in obese people. In one study, when applied to data from the National Health and Human Nutrition Surveys (NHANES) III, an Edmonton stage of 2 was associated with a mortality hazard ratio of 1.57 and a stage of 3 with a hazard ratio of 2.69 when compared to stages 0 and 1 (Padwal et al. 2011).

Summary

Whether or not obesity constitutes a disease continues to be debated, but there is no doubt that it is a major risk factor, and in many instances the primary risk factor, for a number of the biggest health issues in the modern world. These affect every body system and have a knock-on effect beyond the purely medical to the detriment of health-related quality of life, economic activity, and social inclusion. A holistic assessment of obesity must therefore cover all these aspects of health and well-being to enable an effective management strategy to be planned. Careful clinical history taking and examination can suggest the likely etiology of an individual's obesity. In some cases it is related to remediable external factors such as poor food choices, excess alcohol consumption, or appetite-stimulating medications, or to remediable internal factors such as eating disorders or Cushing's syndrome. More often than not, it is due to largely nonmodifiable

Table 1 Modified King's Obesity Staging Criteria (Whyte MB, Velusamy S, Aylwin SJ. Disease severity and staging of obesity: a rational approach to patient selection. Curr Atheroscler Rep. 2014;16(11):456, reproduced with permission)

	Stage 0	Stage 1	Stage 2	Stage 3
A – **A**irway	**Normal** No snoring Neck circumference <43 cm Epworth score <10	**Mild sleep apnea** Snoring Epworth score ≥10 AHI <15/h Neck circumference >43 cm Mild asthma	**Requires CPAP** Witnessed apnea AHI ≥15/h Uses CPAP (controlled) Severe asthma	**Cor pulmonale** Obesity hypoventilation syndrome Uncontrolled OSA
B – **B**ody Mass Index	**<35 kg/m^2**	**35–50 kg/m^2**	**50–60 kg/m^2**	**>60 kg/m^2**
C – **C**ardiovascular Risk	**<10%** CVD risk <10% over 10 years	**10–20%** CVD risk 10–20% over 10 years T2DM	**Heart disease** Stable IHD CCF NYHA class I–II >20% CVD risk over 10 years	**Heart failure** Severe angina CCF NYHA III–IV
D – **D**iabetes	**Normal** Fasting or random glucose <5.7 mmol/L Normal HbA1c	**Impaired fasting glycemia** Impaired fasting glucose Impaired glucose tolerance Previous gestational diabetes	**Type 2 diabetes** Diet, insulin or oral hypoglycemic agent controlled HbA1c <9%	**Uncontrolled type 2 diabetes** HbA1c ≥9% Advanced microvascular disease
E – **E**conomic Complications	**Normal** Obesity has no financial impact	**Financial impact** Increased travel costs Increased clothes costs	**Workplace disadvantage** Earnings limited by obesity Receiving benefits due to obesity	**Unemployed** Unemployed due to obesity Financial effect on third party (e.g., carer required to reduce income)
F – **F**unction	**Normal** No limitation	**Mildly impaired** Manages 1 flight of stairs Limitation on recreation	**Moderately impaired** Can manage <1 flight of stairs Third party assistance for ADLs or for dependents Limitation on work	**Severely impaired** Housebound Wheelchair user Registered disabled

(*continued*)

Table 1 (continued)

	Stage 0	Stage 1	Stage 2	Stage 3
G – **G**onadal and Reproductive Health	**Normal** Normal sexual and reproductive function Celibate (not seeking a physical relationship)	**PCOS/ erectile dysfunction** PCOS Low testosterone (men) Impaired sexual function/ erectile dysfunction	**Subfertility** Subfertility or unable to access IVF Marital/ relationship breakdown due to obesity Cessation of all sexual activity	
H – Mental **H**ealth Status	**Normal** Good mental and physical wellbeing	**Low mood** Low mood or poor QoL	**Mild-moderate depression** Takes treatment for depression	**Severe depression** Suicidal ideation Active self-harm Unmanaged substance misuse
I – Body **I**mage and Eating Behavior	**Normal** Minimal or no concern Normal eating pattern	**Does not like looking in mirror** Comfort eating Inappropriate eating cues Mild body image dysphoria	**Avoids social interaction or mirrors** Severe body image dysphoria Controlled eating disorder	**Eating disorder** Active eating disorder Social phobia
J – Gastro-esophageal **J**unction	**Normal** No symptoms of GORD	**GORD** GORD controlled on standard PPI therapy	**Esophagitis** Esophagitis on OGD within last 12 months Severe GORD symptoms Requires high-dose PPI	**Barrett's esophagus**
K – **K**idney	**Normal**	**Proteinuria**	**GFR <60 ml/ min**	**GFR <30 ml/min**
L – **L**iver	**Normal**	**Raised LFT/ NAFLD on ultrasound**	**NASH**	**Liver failure**

Abbreviations: *AHI*, apnea/hypopnea index; *CVD*, coronary vascular disease; *T2DM*, type 2 diabetes; *IHD*, ischemic heart disease; *CCF*, congestive cardiac failure; *NYHA*, New York Heart Association; *ADL*, activity of daily living; *IVF*, in vitro fertilization; *GORD*, gastroesophageal reflux disease; *PPI*, proton pump inhibitor; *OGD*, oesophagogastroduodenoscopy; *CPAP*, continuous positive airway pressure; *BMI*, body mass index; *PCOS*, polycystic ovary syndrome; *QoL*, quality of life; *GFR*, glomerular filtration rate; *LFT*, liver function test; *NAFLD*, nonalcoholic fatty liver disease; *NASH*, nonalcoholic steatohepatitis

Table 2 The Edmonton Clinical Staging System (Sharma AM, Kushner RF. A proposed clinical staging system for obesity. Int J Obes (Lond). 2009;33(3):289–95, reproduced with permission)

Stage	Description	Management
0	No apparent obesity-related risk factors (e.g., blood pressure, serum lipids, fasting glucose, etc., within normal range), no physical symptoms, no psychopathology, no functional limitations, and/or impairment of well being	Identification of factors contributing to increased body weight. Counseling to prevent further weight gain through lifestyle measures including healthy eating and increased physical activity
1	Presence of obesity-related subclinical risk factors (e.g., borderline hypertension, impaired fasting glucose, elevated liver enzymes, etc.), mild physical symptoms (e.g., dyspnea on moderate exertion, occasional aches and pains, fatigue, etc.), mild psychopathology, mild functional limitations, and/or mild impairment of well being	Investigation for other (non-weight related) contributors to risk factors. More intense lifestyle interventions, including diet and exercise to prevent further weight gain. Monitoring of risk factors and health status
2	Presence of established obesity-related chronic disease (e.g., hypertension, type 2 diabetes, sleep apnea, osteoarthritis, reflux disease, polycystic ovary syndrome, anxiety disorder, etc.), moderate limitations in activities of daily living, and/or well being	Initiation of obesity treatments including considerations of all behavioral, pharmacological, and surgical treatment options. Close monitoring and management of comorbidities as indicated
3	Established end-organ damage such as myocardial infarction, heart failure, diabetic complications, incapacitating osteoarthritis, significant psychopathology, significant functional limitations, and/or impairment of well being	More intensive obesity treatment including consideration of all behavioral, pharmacological, and surgical treatment options. Aggressive management of comorbidities as indicated
4	Severe (potentially end-stage) disabilities from obesity-related chronic diseases, severe disabling psychopathology, severe functional limitations, and/or severe impairment of well being	Aggressive obesity management as deemed feasible. Palliative measures including pain management, occupational therapy, and psychosocial support

influences on the balance between appetite and food intake on the one hand, and energy expenditure on the other. Imaging, laboratory tests, and other clinical investigations are necessary to screen for complications that are not easily detectable through history and examination. Proper assessment requires the skills of multiple disciplines including physicians, surgeons, psychiatrists, psychologists, and dieticians. A service structured to facilitate the effective multidisciplinary team working of these healthcare professions with the engagement of the patient and patient-support groups is essential to optimize the management of obesity in a patient-centered, individualized manner.

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