OBESITY

TUCOM
Dep. of Medicine
3 rd year
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OBESITY

Learning objectives:

1. Make the definition and classification of obesity according to body mass index (BMI) formula.
2. Recognize the types of obesity according to various body fat distribution patterns.
3. Review the pathogenesis of obesity.
4. Understand the causes of obesity.
5. Clarify the complications of obesity.
6. Explain the clinical assessment of obese patient.
7. List the important investigations of obesity.
8. Explain the management of obesity.

Obesity

Define as: A nutritional disorder in which excess body fat has been accumulated to the extent that it may have an adverse effect on health.

Body Mass Index (BMI) is ≥ 30 kg/m^2 .

body mass index (BMI) = weight/$height^2$ (in kg/m^2)

Quantifying obesity with body mass index (weight/height2)

BMI (kg/m2)	Classification	Risk of obesity comorbidity
<18.5	Underweight	
18.5–24.9	Reference range	Negligible
25.0–29.9	Overweight	Mildly increased
≥ 30.0	Obese	
30.0–34.9	Class I	Moderate
35.0–39.9	Class II	Severe
≥ 40.0	Class III	Very severe

The types of obesity according to body fat distribution:

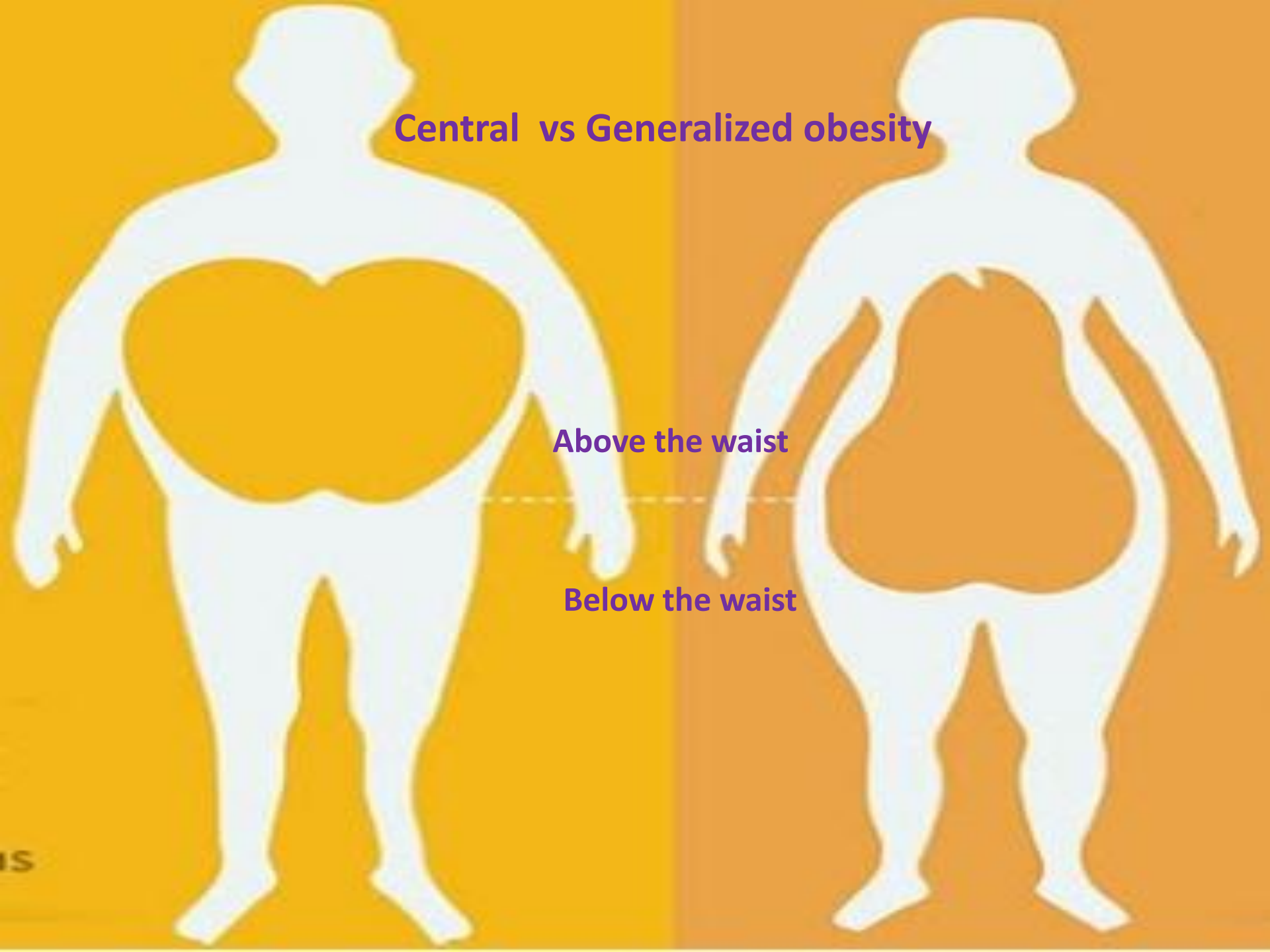
- **1- Intra-abdominal fat accumulation causes 'central' ('abdominal', 'visceral', 'android' or 'apple-shaped') obesity.** BMI) is ≥ 30 kg/m^2
- Waist circumference: > 102 cm in men > 88 cm in women
- waist-to-hip circumference ratio (WHR) greater than 1.0 in men or greater than 0.8 in women, indicative of central obesity.
- This form is more common in men and is more closely associated with type 2 diabetes mellitus (due to insulin resistance), the metabolic syndrome, cardiovascular disease, hypertension, dyslipidemia, and hyperuricemia.

2- Generalized fat accumulation ('gynoid' or 'pear-shaped') obesity. Moe common in female in which fat accumulates in the hips and gluteal and femoral regions, has milder metabolic complications.

Quantifying obesity with BMI and waist circumference for risk of type 2 diabetes and cardiovascular disease

		Waist circumference[2]		
BMI (weight in kg/height in m^2)	Classification[1]	Men <94 cm Women <80 cm	Men 94–102 cm Women 80–88 cm	Men >102 cm Women >88 cm
18.5–24.9	Reference range	Negligible	Mildly increased	Moderate
25.0–29.9	Overweight	Negligible	Moderate	Severe
>30.0	**Obese**			
30.0–34.9	Class I	Moderate	Severe	Very severe
35.0–39.9	Class II	–	Very severe	Very severe
>40.0	Class III	–	Very severe	Very severe

Central vs Generalized obesity



Metabolic syndrome or 'insulin resistance syndrome': it is a cluster of conditions characterized by:

- Central obesity
- Insulin resistance, and type 2 diabetes mellitus
- Hypertension
- Dyslipidaemia (characterized by elevated levels of low-density lipoprotein (LDL) cholesterol and triglycerides, and a low level of high-density lipoprotein (HDL) cholesterol),
- Non-alcoholic fatty liver disease and, in women, polycystic ovarian syndrome.

The prevalence of obesity has increased threefold within the last 20 years and continues to rise.

- **Obesity is regarded as a pandemic, with potentially disastrous consequences for human health.**
- **Over 25% of adults in the UK were obese (i.e. BMI ≥ 30 kg/m2) in 2015, compared with 7% in 1980 and 16% in 1995.**
- **Affected people of Arabic countries, especially those in higher-income and oil-producing countries, due to rapid urbanization and improved living conditions (changes in food consumption, socioeconomic status and physical activity).**

Pathogenesis of obesity

- **Leptin: is a hormone that is produced in fat cells, mostly in subcutaneous fat , it is plasma level increase when there is increased fat mass. It is a potent satiety factor that acts on the hypothalamus to:**

- **Reduce the production of neuropeptide Y, which is a stimulator of food intake.**
- **Decrease food intake**
- **Increase energy expenditure**
- **Modulate glucose and fat metabolism**

Low leptin level signal starvation and stimulate feeding. Congenital leptin deficiency lead to hyperphagia and severe obesity.

Thus common forms of human obesity actually appear to be leptin resistant.

- **Other important factors**: intra-abdominal fat draining into the portal vein and thence directly to the liver. Thus many factors that are released from adipose tissue (including free fatty acids; ‘adipokines’, such as tumour necrosis factor alpha, adiponectin and resistin) may be at higher concentration. In the liver and muscle, and hence induce insulin resistance and promote type 2 diabetes.

Aetiology

Obesity result from discrepancy between energy consumption and expenditure. It is caused by long term positive energy balance.

Aetiology

1-Role of genetic and environment:

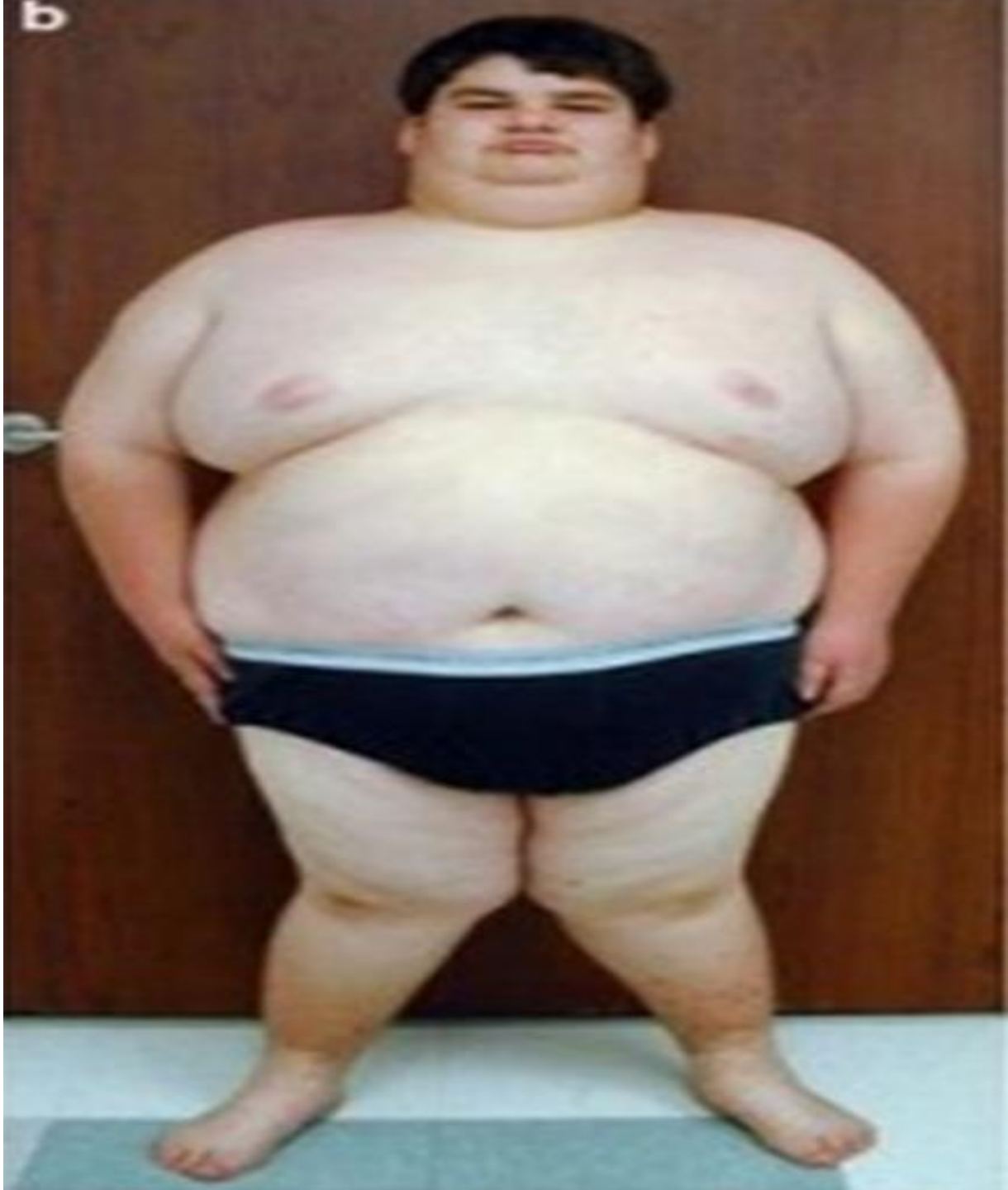
Obesity= Gene + availability of palatable food + sedentary lifestyle.

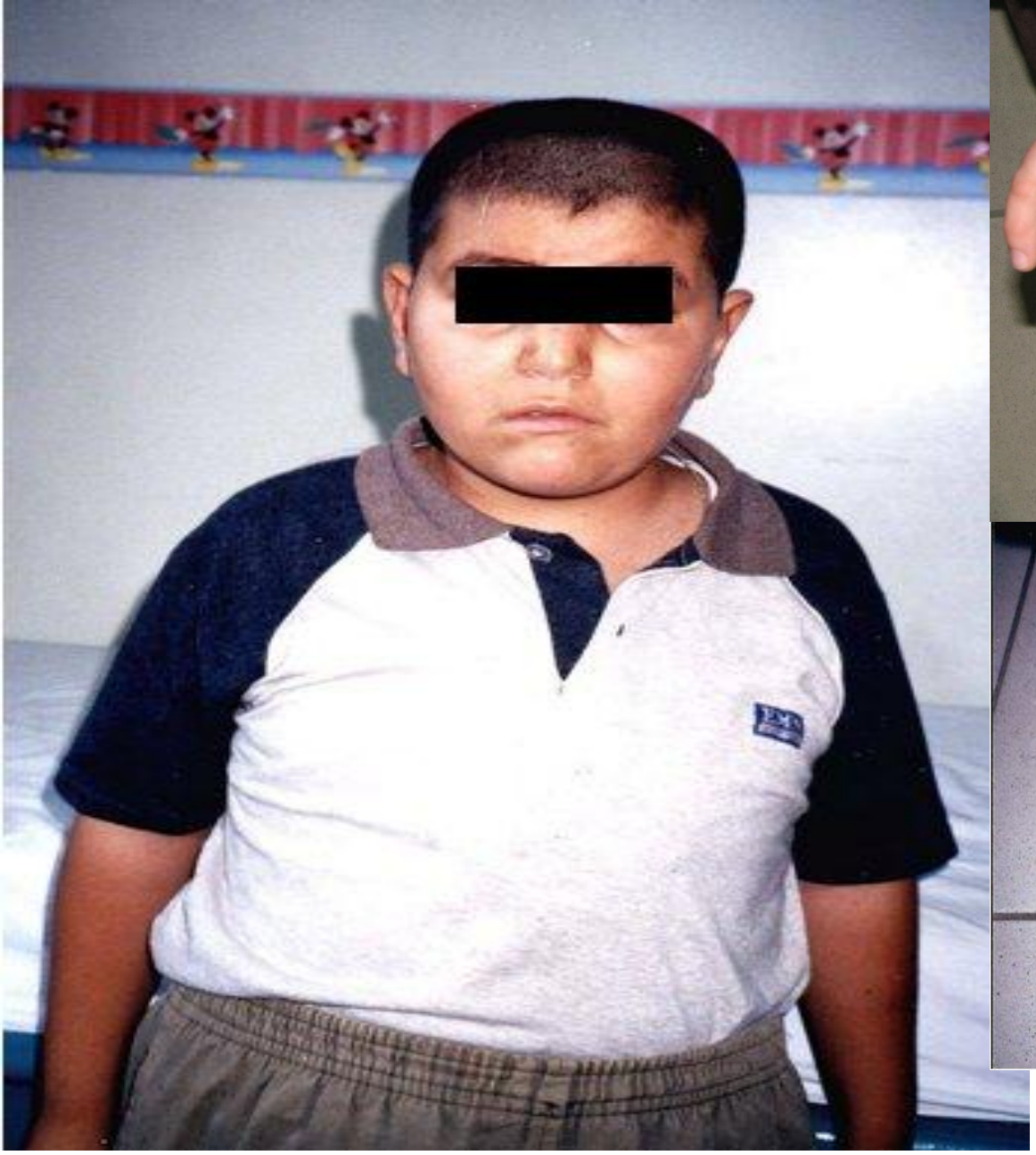
a- Polygenic disorder: Majority of human obesity is related to the combination of polygenic susceptibility traits and environmental conditions.

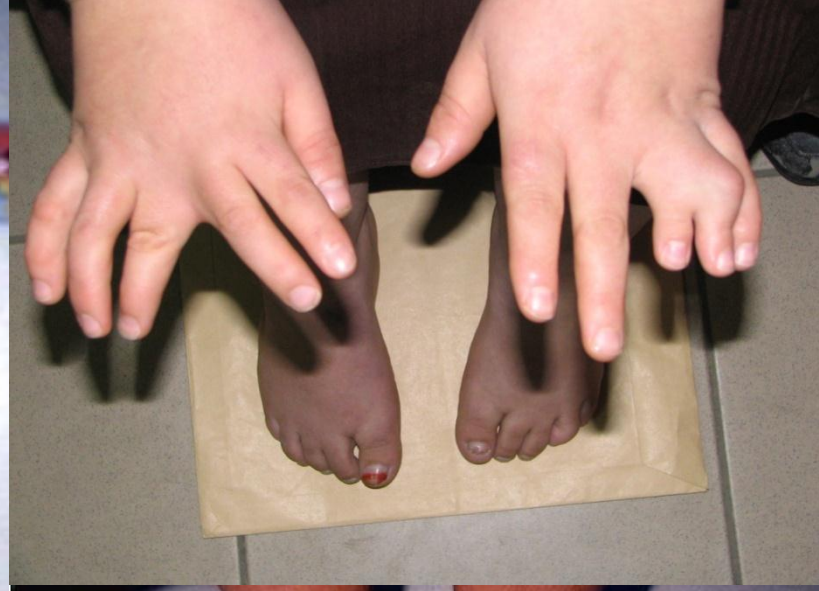
b- Single gene disorders: Rare, cause severe childhood obesity: Prader-Willi and Lawrence-Moon-Biedl syndromes.

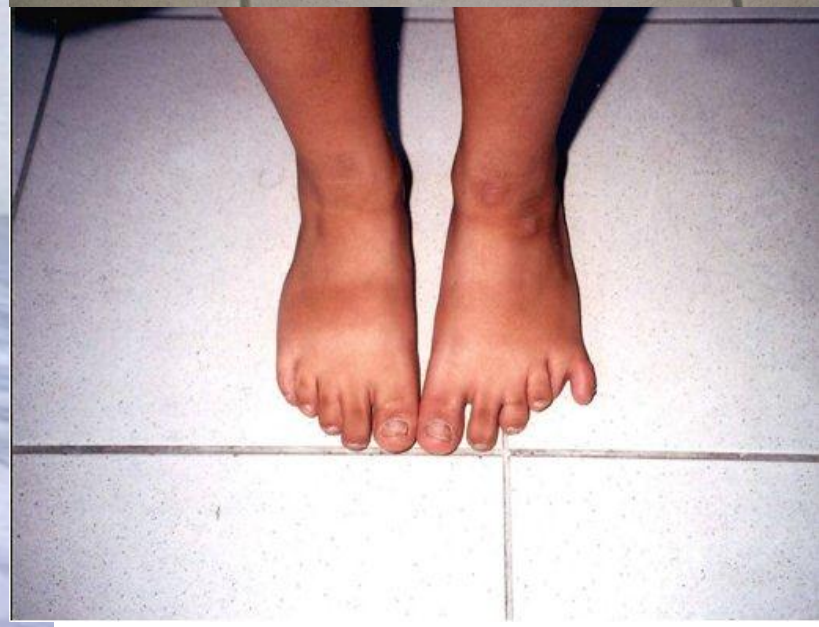
Prader- Willi Syndrome:

Short stature, poor motor skills, weight gain and underdeveloped sex organs.









Lawrence-Moon-Biedl syndromes: Short stature, obesity, polydactyly, retinal disorders, and hypogonadism.

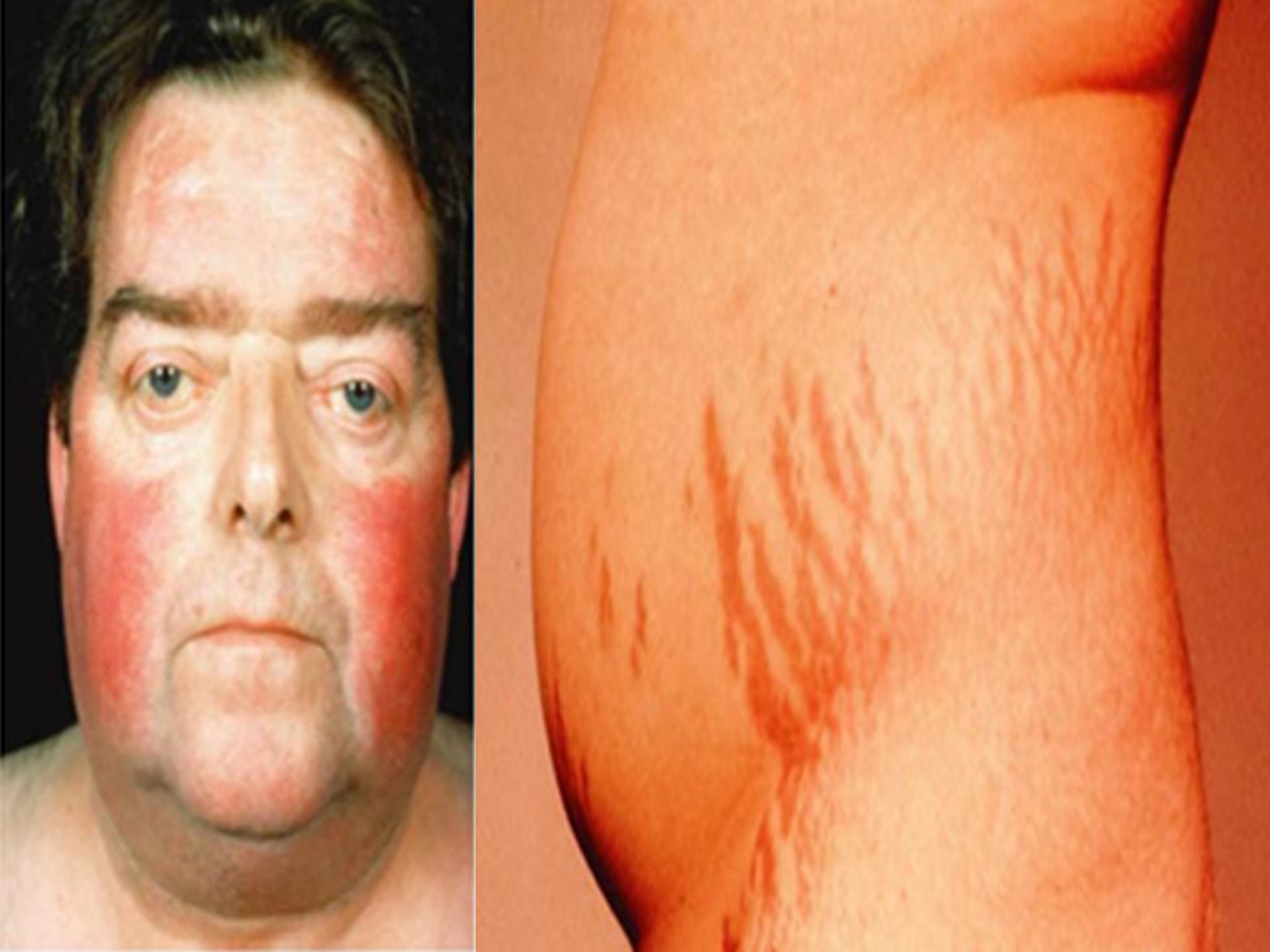
2- Reversible causes of obesity:

A- Endocrine factors

- Hypothyroidism
- Hypothalamic tumours or injury
- Cushing's syndrome
- Insulinoma

B- Drug treatments

- Antipsychotics (e.g. olanzapine)
- Tricyclic antidepressants
- Corticosteroids
- Sulphonylureas, thiazolidinediones and insulin
- Sodium valproate
- Oestrogen-containing contraceptive pill
- β-blockers
- Pizotifen

Complications of obesity

- Metabolic syndrome
- Type 2 diabetes (T2DM)
- Hypertension
- Dyslipidemia
- Coronary heart disease
- Congestive heart failure
- Atrial fibrillation
- Osteoarthritis
- Stroke
- Gall bladder disease
- Fatty liver and nonalcoholic steatohepatitis
- Sleep apnea
- Asthma
- Gastroesophageal reflux (GERD)
- Some cancers (endometrial, breast, and colon)
- Gynecologic disorders (abnormal menses, infertility, polycystic ovarian syndrome)

Clinical assessment

1- History: dietary history: food consumption, binge eating, nocturnal eating, alcohol consumption. Any symptoms related to weight gain e.g. poor sleep or snoring or dyspepsia. Taking relevant drugs

- **Underlying disorder such as hypothyroidism or Cushing's syndrome.**
- **Complications of obesity**
- **Family history of obesity, DM, HT, or ischemic heart disease.**
- **Impact of obesity on the patient's life and work. Is his work active or sedentary. Is there regular exercise? Are there any psychological problems as depression?**

2- Examination and measurements:

- **BMI =kg/m^2**

  Example: adult Wt 70 kg and Ht 1.75 m the BMI is =70/1.75^2 = 22.9

- **Waist circumference:**

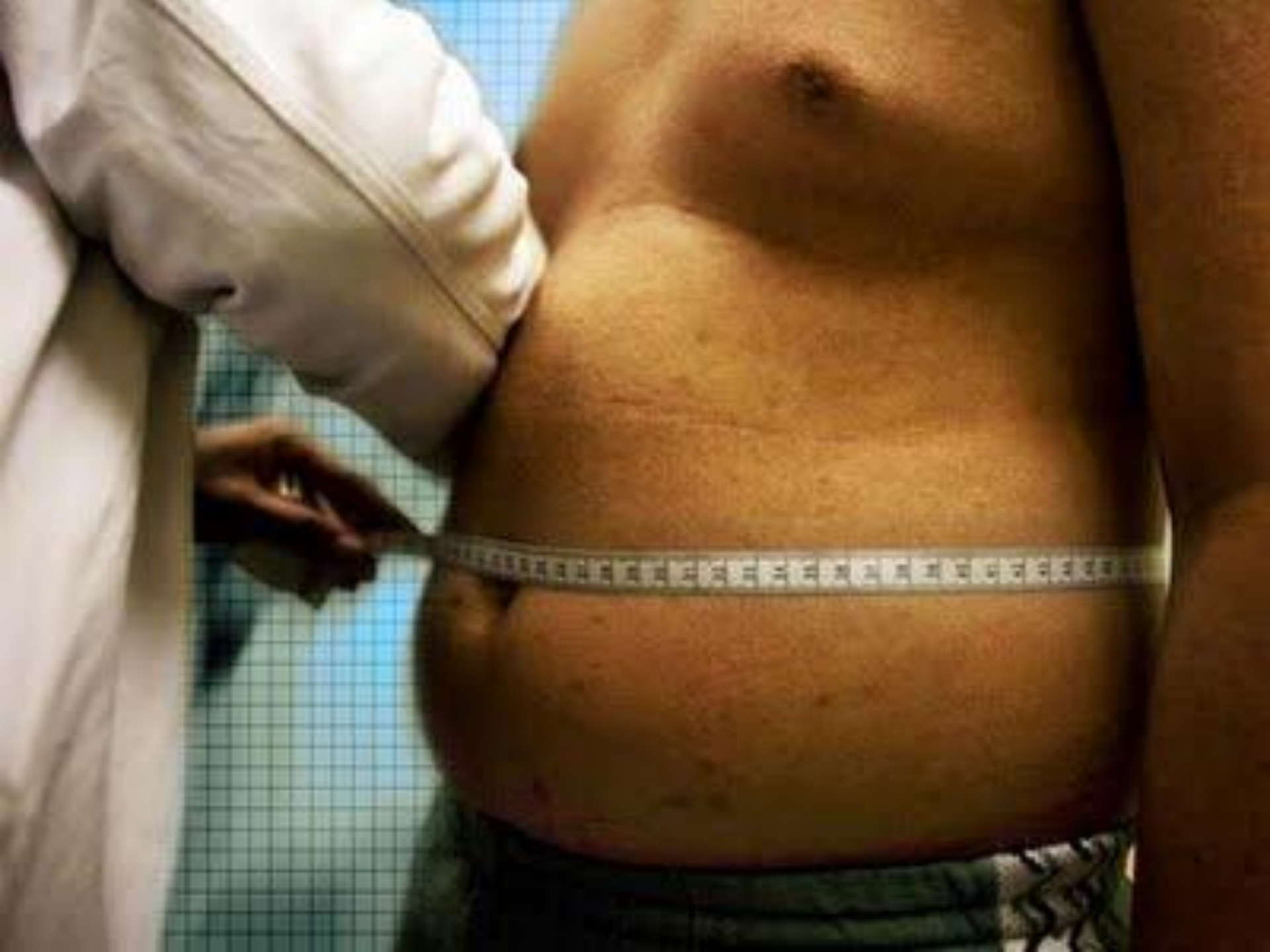
  > 102 cm in men = obesity

  > 88 cm in women = obesity

- **Waist-to-hip circumference ratio:**

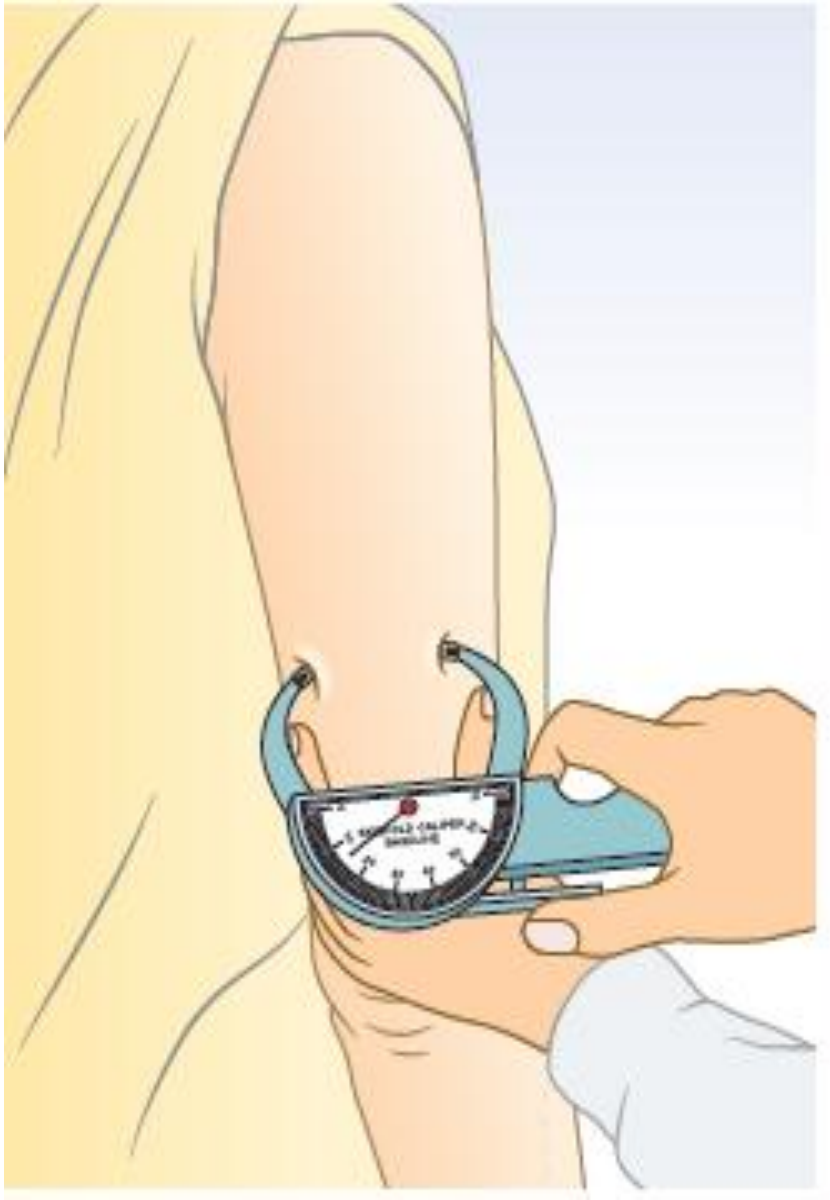
  > 1 in men = android obesity

  > 0.8 in women = android obesity



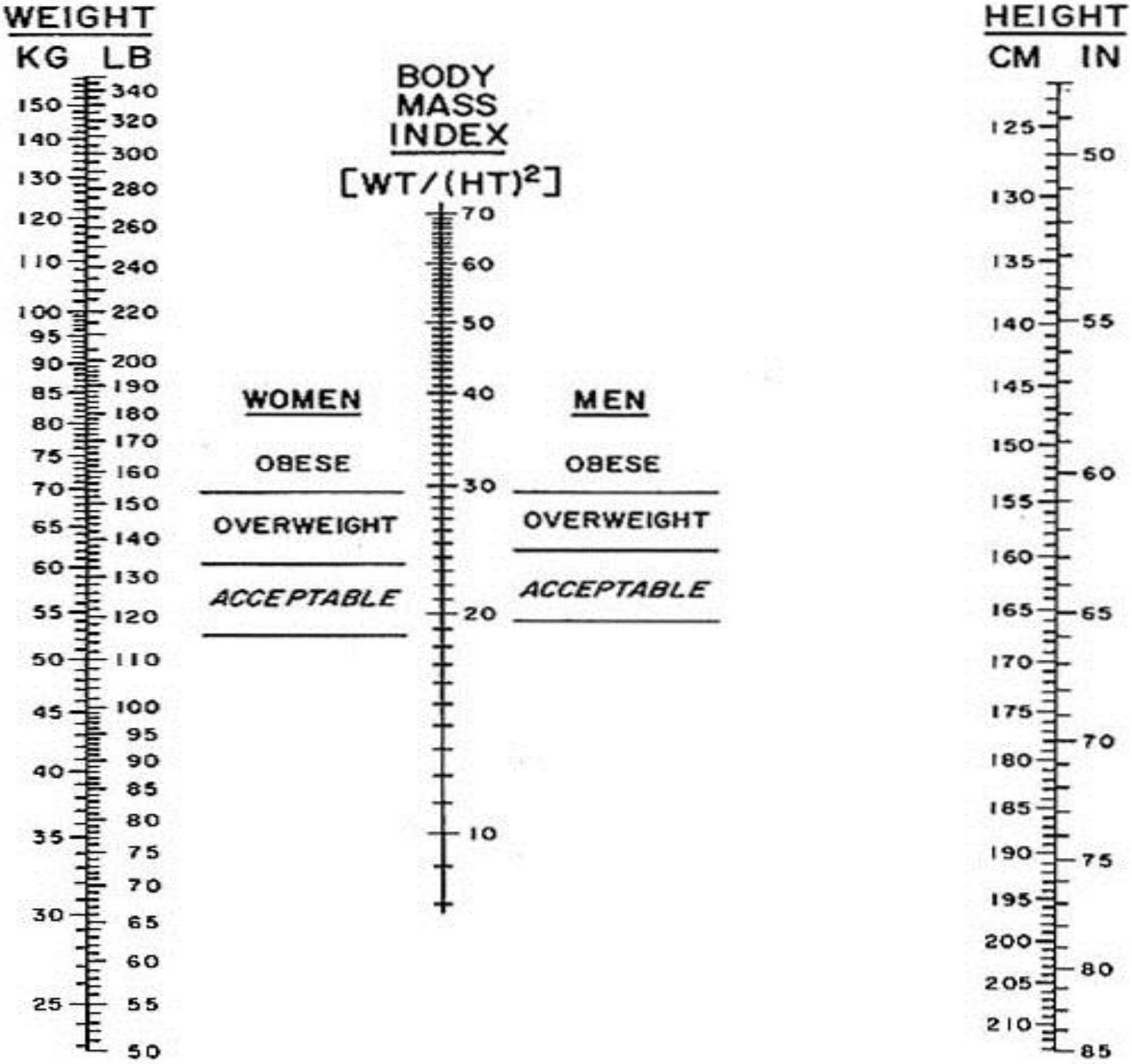

Female
Waist
Hip
Male
Waist
Hip

- **Anthropometry:** skin fold thickness by using caliper.
- **Blood pressure:** measured with a large cuff.
- **Search of complications.**
- **Look for signs of hypothyroidism or Cushing's syndrome.**



Triceps skin fold thickness. Lean patients 6–12 mm, obese patients 40–50 mm.

Nomogram for determining body mass index. To use this nomogram, place a ruler or other straight edge between the body weight (without clothes) in kilograms or pounds located on the left-hand line and the height (without shoes) in centimeters or inches located on the right-hand line.



Investigations

- **Blood glucose and a serum lipid profile:** for associated type 2 diabetes and dyslipidaemia
- **Serum transaminases:** elevated in patients with non-alcoholic fatty liver disease
- **Thyroid function tests:** for underlying hypothyroidism
- **Overnight dexamethasone suppression test or 24-hour urine free cortisol:** if Cushing's syndrome is suspected
- **ECG:** signs of left ventricular hypertrophy or ischemic heart disease.

Management

1-Lifestyle advice:

A-avoid of the 'obesogenic' environment.

Obesogenic environment: 1-Increasing energy intake: ↑ Portion sizes, ↑ Snacking and loss of regular meals, ↑ Energy-dense food (mainly fat). 2-Decreasing energy expenditure: ↑ Car ownership, ↓ Walking to school/work, ↑ Automation, ↓ manual activities , ↓ Sports in schools, ↑ Time spent on video games and watching TV, ↑ Central heating.

B- Changes in eating behavior: avoidance of snacking, and take regular meals to encourage satiety. Adequate hydration with meals helps to limit calorie intake by causing gastric distension.

C- Maximize their physical activity: e.g. walking rather than driving.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Note:

- The goal is to lose 0.5 kg/week.
- There is no role for starvation diets, which risk profound loss of muscle mass and the development of arrhythmias (and even sudden death) secondary to elevated free fatty acids, ketosis and deranged electrolytes.
- There is no role for diuretics, or thyroxin without biochemical evidence of hypothyroidism.

2- Drugs:

A- Orlistat: Orlistat inhibits pancreatic and gastric lipases and thereby decreases the hydrolysis of ingested triglycerides, reducing dietary fat absorption by approximately 30%. Dose of 120 mg is taken with each of the three main meals of the day.

Side-effects: loose stools, oily spotting, faecal urgency, flatus and malabsorption of fat and fat soluble vitamins.

B- The combination of low-dose phentermine and topiramate extended release has been approved in the USA; this results in weight loss of approximately 6% greater than placebo and benefits lipids and glucose concentrations.

Side effect: topiramate is teratogenicity and phentermine have and cardiovascular effects.

C- Lorcaserin: is a 5-HT2c inhibitor is also approved in the USA; it is moderately effective and has a relatively low rate of adverse effects.

D- The combination of the opioid antagonist naltrexone and the noradrenaline (norepinephrine)/ dopamine re-uptake inhibitor bupropion is also effective. The main adverse effects are dry mouth and constipation.

E- A higher dose of the injectable glucagon-like peptide-1 (GLP-1) receptor agonist liraglutide (3 mg) is also approved for use and has been shown to reduce the risk of diabetes in patients with pre-diabetes.

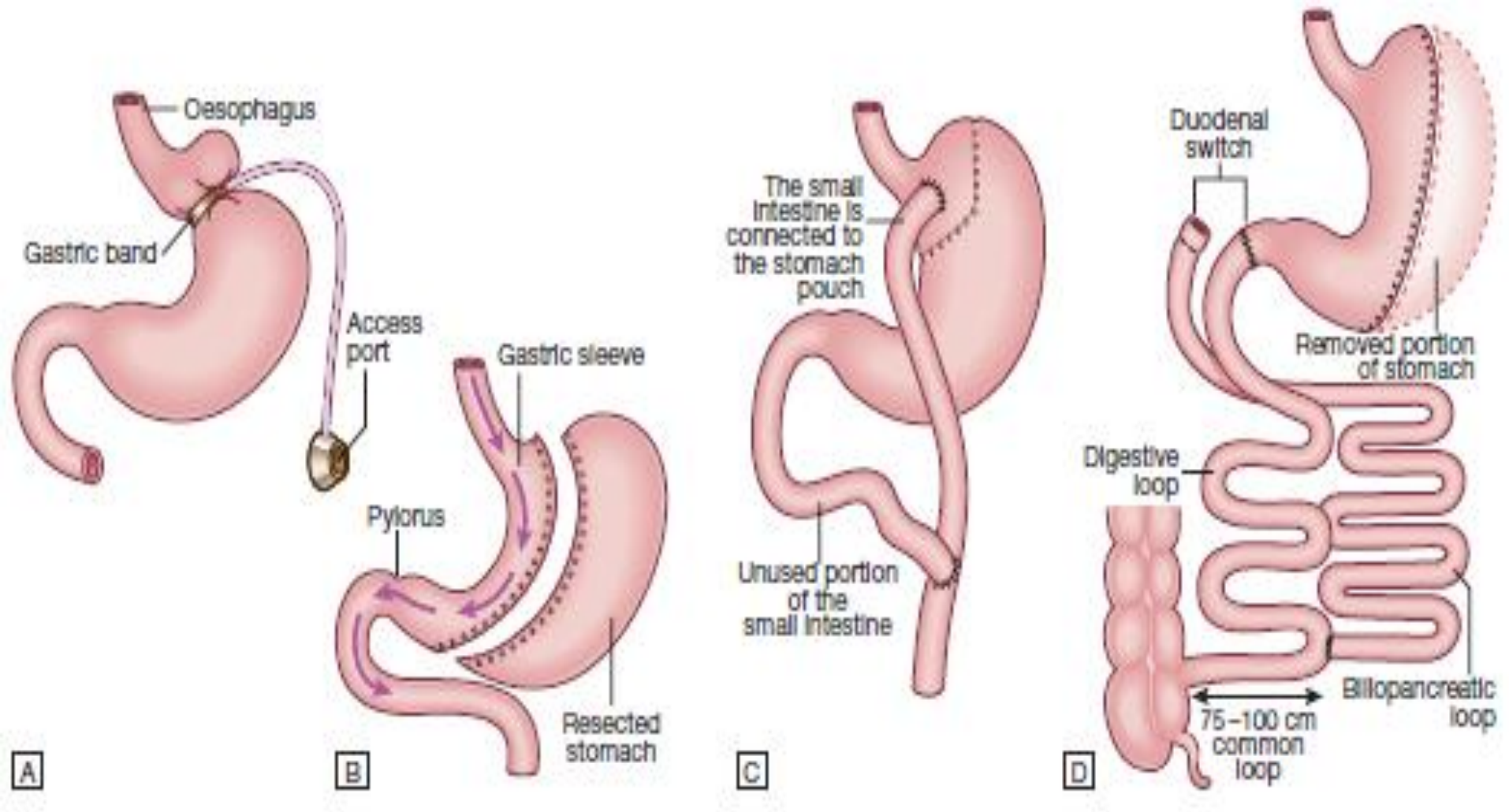
Drug therapy is usually reserved for patients with high risk of complications from obesity.

3-Surgery: 'Bariatric' surgery to reduce the size of the stomach is by far the most effective long-term treatment for obesity.

In whom have very high risks of complications of obesity and drug therapy has been ineffective.

4-Treatment of additional risk factors:

Smoking, excess alcohol consumption, diabetes mellitus, hyperlipidemia and hypertension.



Bariatric surgical procedures. A- Laparoscopic banding, with the option of a reservoir band and subcutaneous access to restrict the stomach further after compensatory expansion has occurred. B- Sleeve gastrectomy. C- Roux-en-Y gastric bypass. D- Biliopancreatic diversion with duodenal switch.

THANKS