

American Society of Bariatric Physicians (ASBP)

ASBP Obesity Algorithm: Adult Adiposity Evaluation and Treatment 2013

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Disclaimer

The American Society of Bariatric Physicians® (ASBP) Obesity Algorithm was developed to assist health care professionals in medical decision making; however, it is in no way a substitute for a medical professional's independent judgment and should not be considered medical advice. Most of the content herein is based on the medical literature and the clinical experience of obesity medicine specialists. In areas of uncertainty, professional judgment of the authors was applied. The Obesity Algorithm is a working document that reflects the state of the field of obesity medicine at the time of publication. Because rapid changes in this area are expected, periodic revisions are inevitable. We encourage medical professionals to use this information in conjunction with, and not as a replacement for, their best clinical judgment. The presented recommendations may not be appropriate in all situations. Any decision by practitioners to apply these guidelines must be made in light of local resources and individual patient circumstances.

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ASBP Obesity Algorithm:

Adult Adiposity Evaluation and Treatment 2013

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Purpose

- *To provide clinicians an overview of principles important to the care of patients with increased body fat, based upon scientific evidence, supported by the medical literature, and derived from the clinical experiences of members of the American Society of Bariatric Physicians.*

ASBP Obesity Algorithm: Adult Adiposity Evaluation and Treatment 2013

Process

The ASBP Algorithm was derived from input by volunteer ASBP members consisting of:

- Academicians
- Clinicians
- Clinical trialists
- Researchers

The ASBP Algorithm did not receive industry funding, had no input from industry, and the authors received no payment for their contributions.

ASBP Obesity Algorithm: Adult Adiposity Evaluation and Treatment 2013

Intent of use

The ASBP Algorithm 2013 is intended to be a “living document” updated once a year (as needed), and intended as an educational tool to assist in the translation of medical science and the clinical experience of the authors towards assisting clinicians better manage their overweight and obese patients.

This algorithm is not intended to be interpreted as “rules” and/or directives regarding medical care of an individual patient.

While it is hoped many clinicians may find this algorithm helpful, the final decision regarding the optimal care of the overweight and obese patient is dependent upon the individual clinical presentation, and the judgment of the clinician who is tasked with directing a treatment plan that is in the best interest of the patient.

ASBP Obesity Algorithm: Adult Adiposity Evaluation and Treatment 2013

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Overall Management Goals

**Adult patient with
overweight or obesity**

**Improve
patient health**

**Improve
quality of life**

**Improve
body weight and
body composition**

Obesity classification: BMI

Obesity classification: % body fat

Obesity classification: waist circumference

**Increased body fat
(adiposity)**

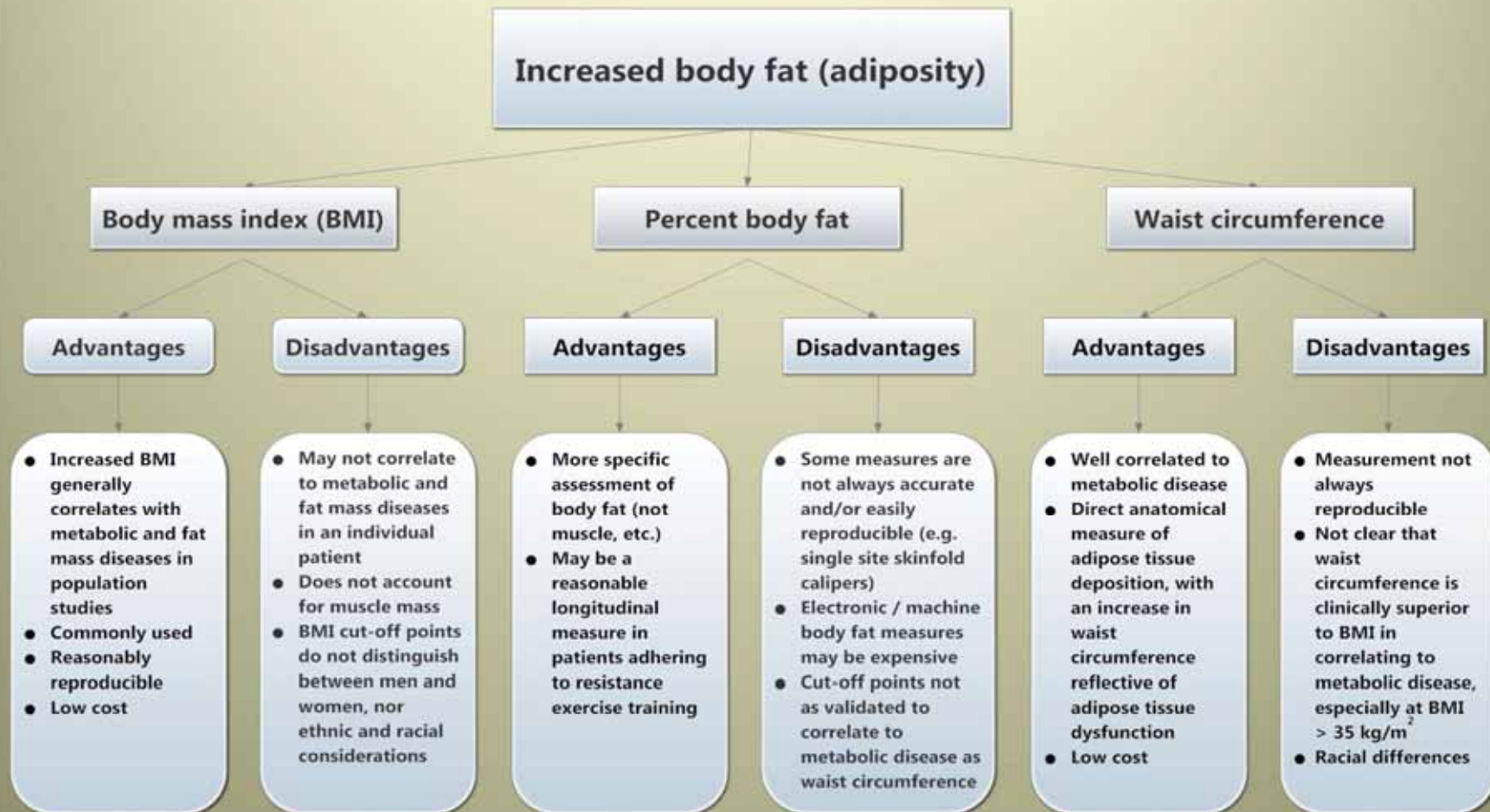
**Overweight and obesity classification:
Waist Circumference (WC)**

Men Abdominal Obesity
 ≥ 40 inches (≥ 102 cm)*

Women Abdominal Obesity
 ≥ 35 inches (≥ 88 cm)*

** Different WC abdominal obesity cut-off points may be appropriate for different races, such as ≥ 90 cm for Asian men and ≥ 80 cm for Asian women*

Obesity classifications



Obesity as a disease

Increased body fat both directly and indirectly promotes and/or causes adverse health consequences, and thus by definition, is a disease

General Definition of a disease

A disordered or incorrectly functioning organ, part, structure, or system of the body

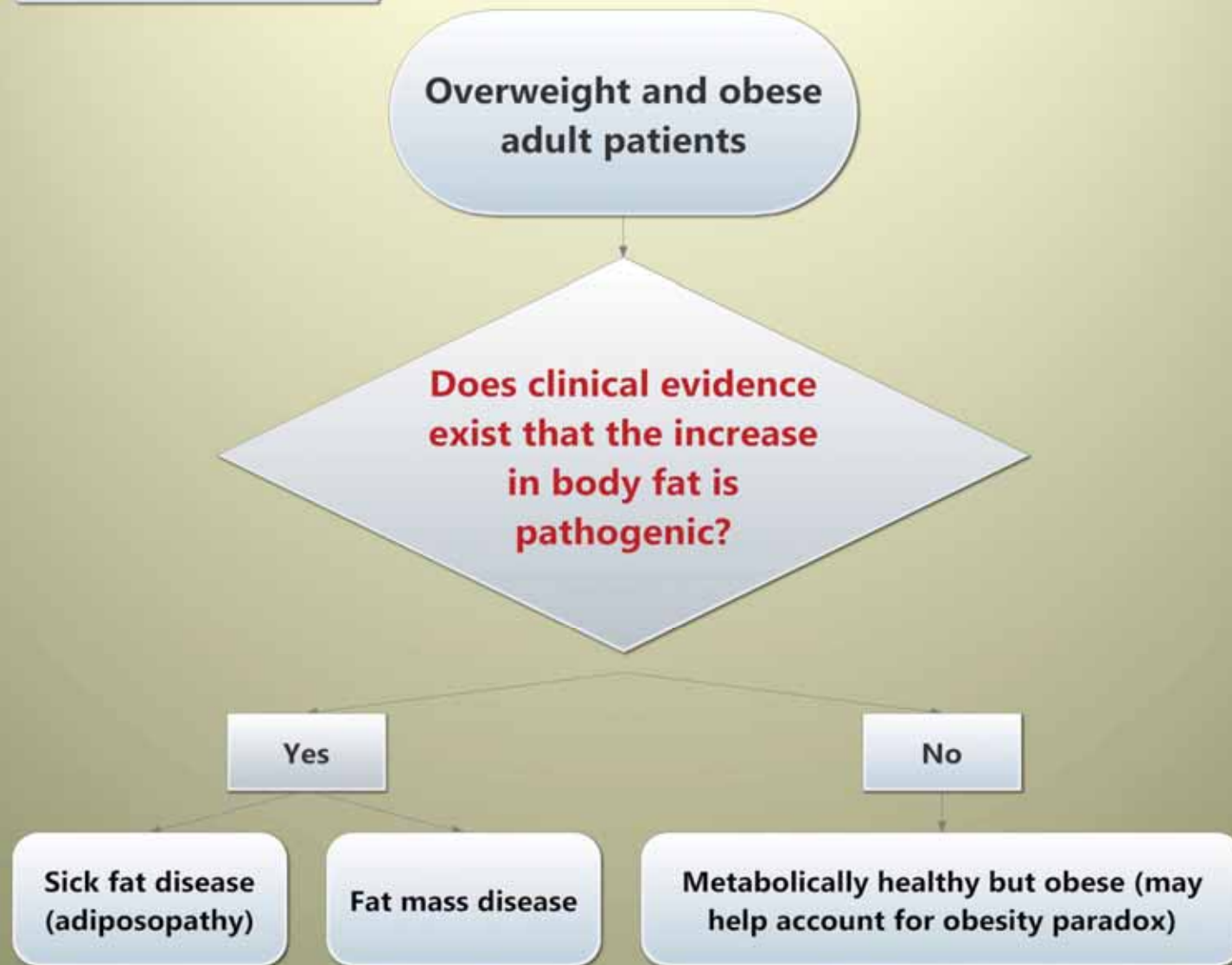
Results from the effect of genetic or developmental errors, infection, poisons, nutritional deficiency or imbalance, toxicity, or unfavorable environmental factors

Manifests as illness, sickness, or ailments

Obesity as a disease**Obesity is a disease when . . .**

- The patient has an abnormal increase in body fat as assessed by a reliable measure
- Increased body fat is caused by genetic or developmental errors, infections, hypothalamic injury, adverse reactions to medications, nutritional imbalance, and/or unfavorable environmental factors
- Pathogenic adipocyte and/or adipose tissue endocrine and immune function contribute to metabolic disease (adiposopathy or "sick fat" disease) and/or;
 - Pathogenic physical forces from excessive body fat cause damage to other body tissues (fat mass disease)

The adverse health consequences of increased body fat are not simply "co-morbidities" or "associated risk factors"

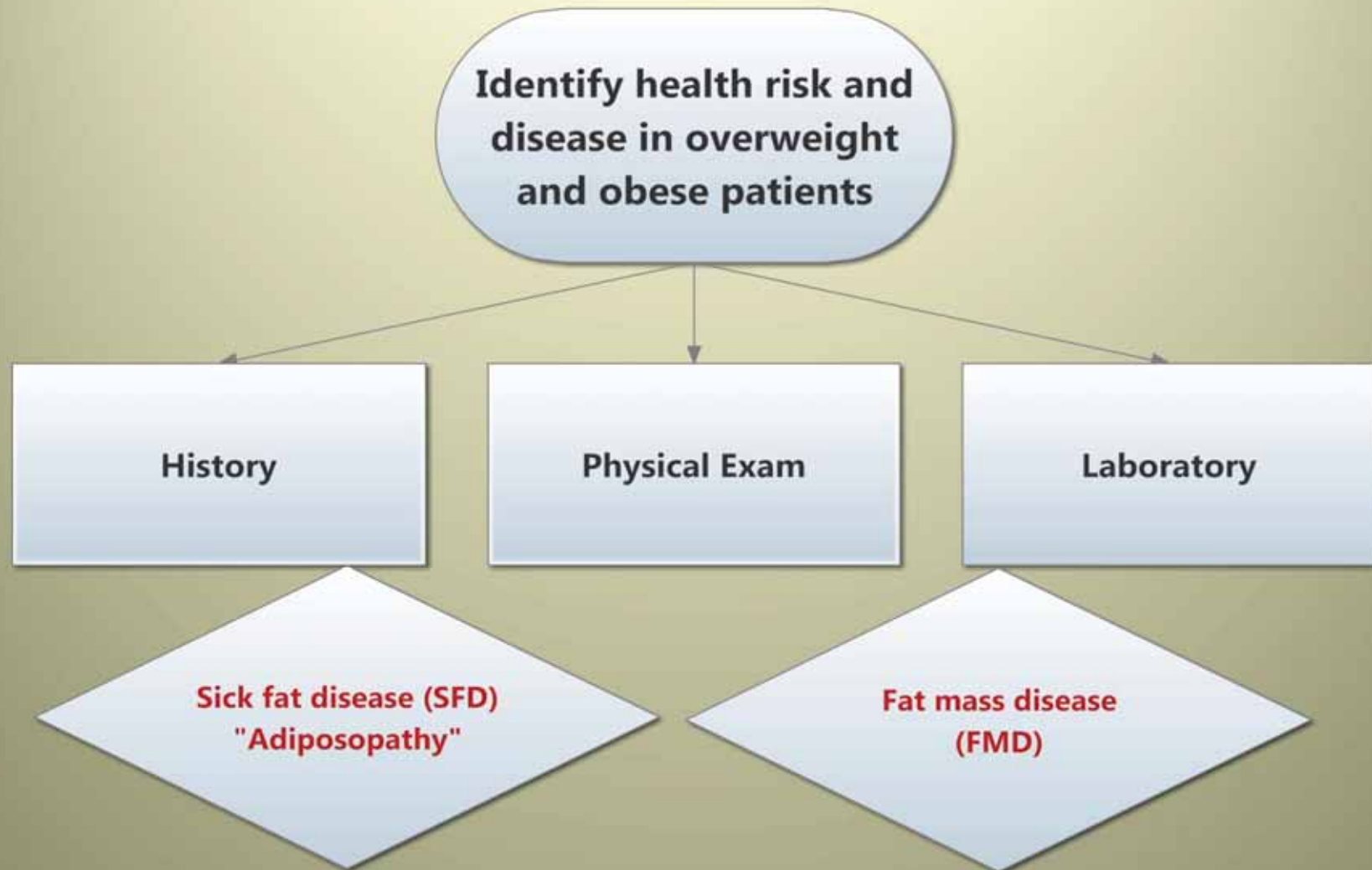
Obesity as a disease

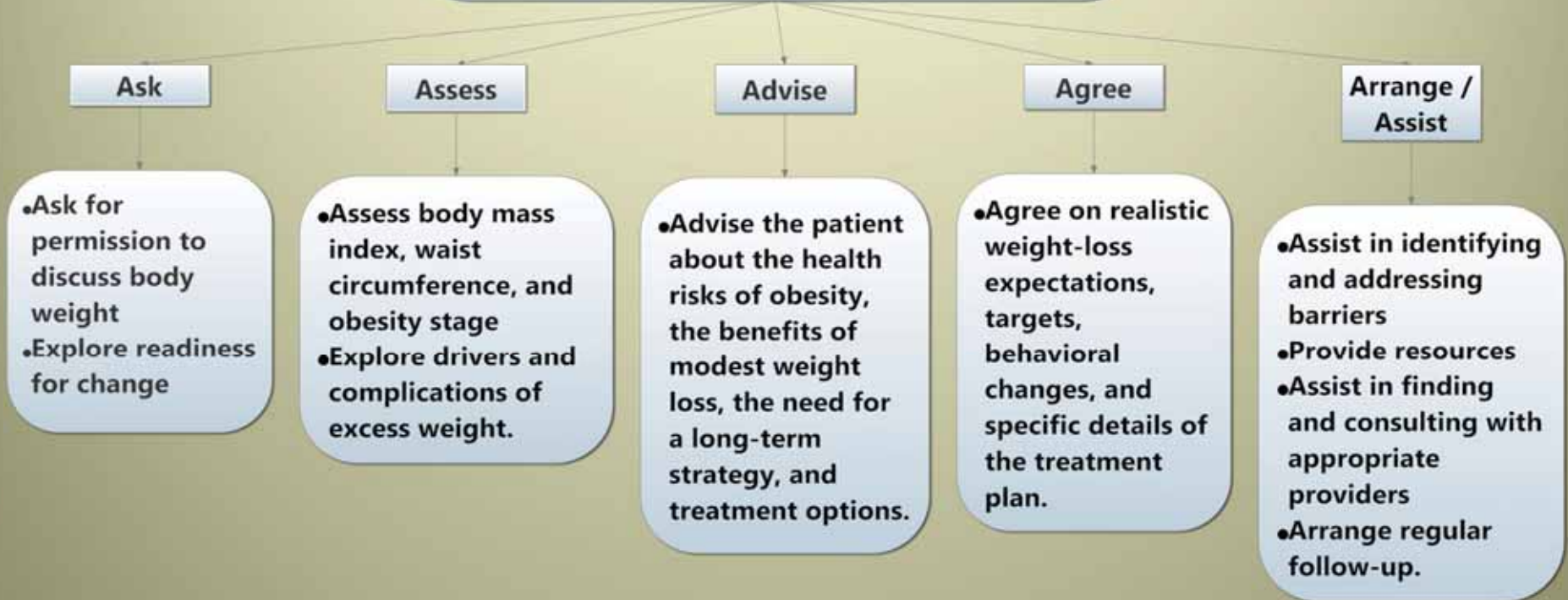
Obesity as a disease**Within subsets of overweight and obese patients:****Deranged endocrine and immune responses****Sick fat disease (SFD)
(Adiposopathy)**

- Elevated blood glucose
- Elevated blood pressure
- Dyslipidemia
- Other metabolic diseases

Abnormal and pathologic physical forces**Fat mass disease (FMD)**

- Stress on weight bearing joints
- Immobility
- Tissue compression (e.g. sleep apnea, gastrointestinal reflux, high blood pressure, etc.)
- Tissue friction (e.g. intertrigo, etc.)

Diagnose disease

Overall approach**5 A's of Obesity Management**

Diagnose fat mass disease (FMD)**Fat Mass Disease****Cardiovascular**

- Congestive Heart Failure & Cor Pulmonale
- Varicose Veins
- Thromboembolic events (e.g. pulmonary embolus, stroke, etc.)
- Hypertension (e.g. compression of kidney)

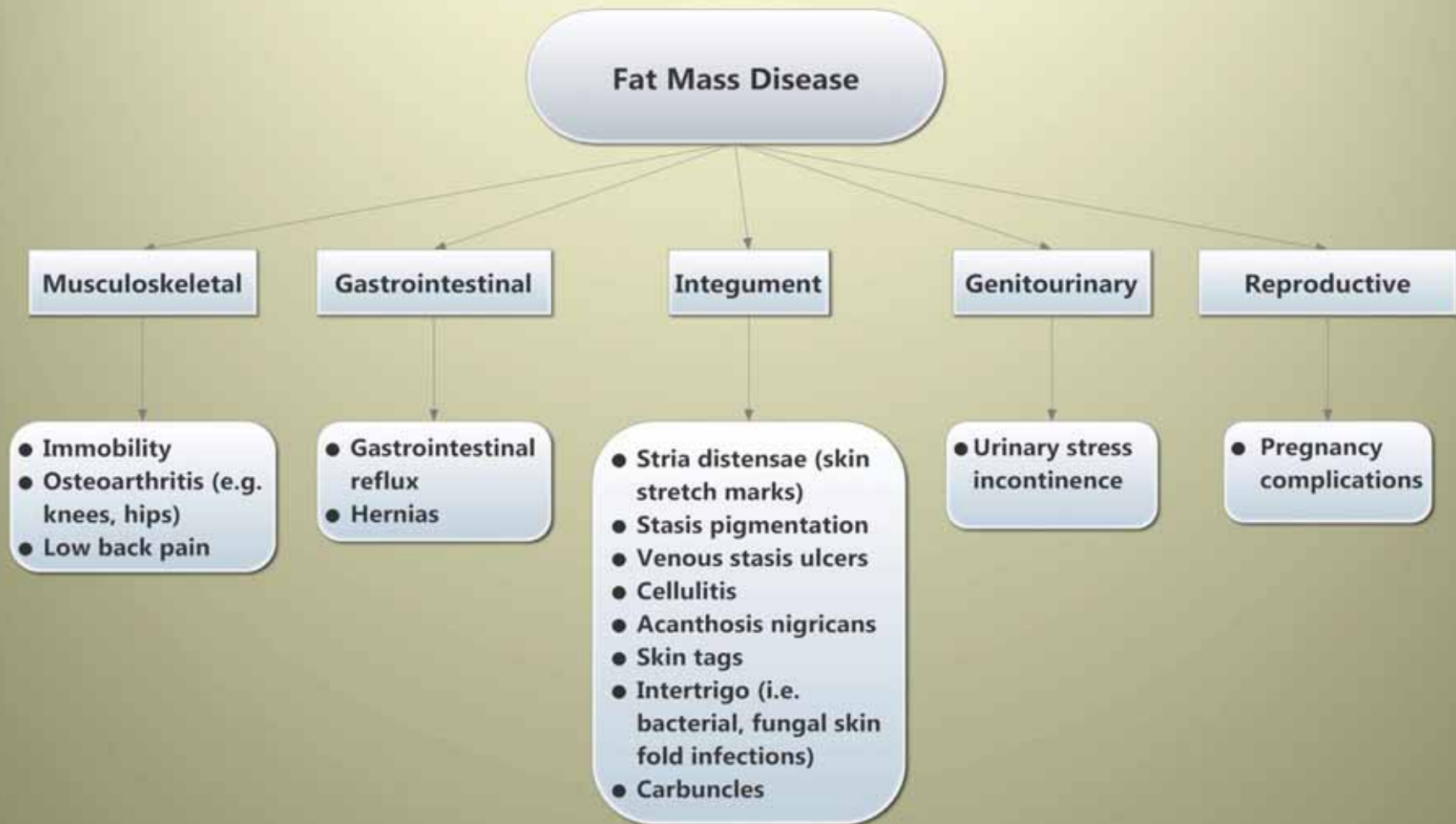
Pulmonary

- Dyspnea
- Obstructive Sleep Apnea
- Hypoventilation Syndrome
- Pickwickian Syndrome
- Asthma

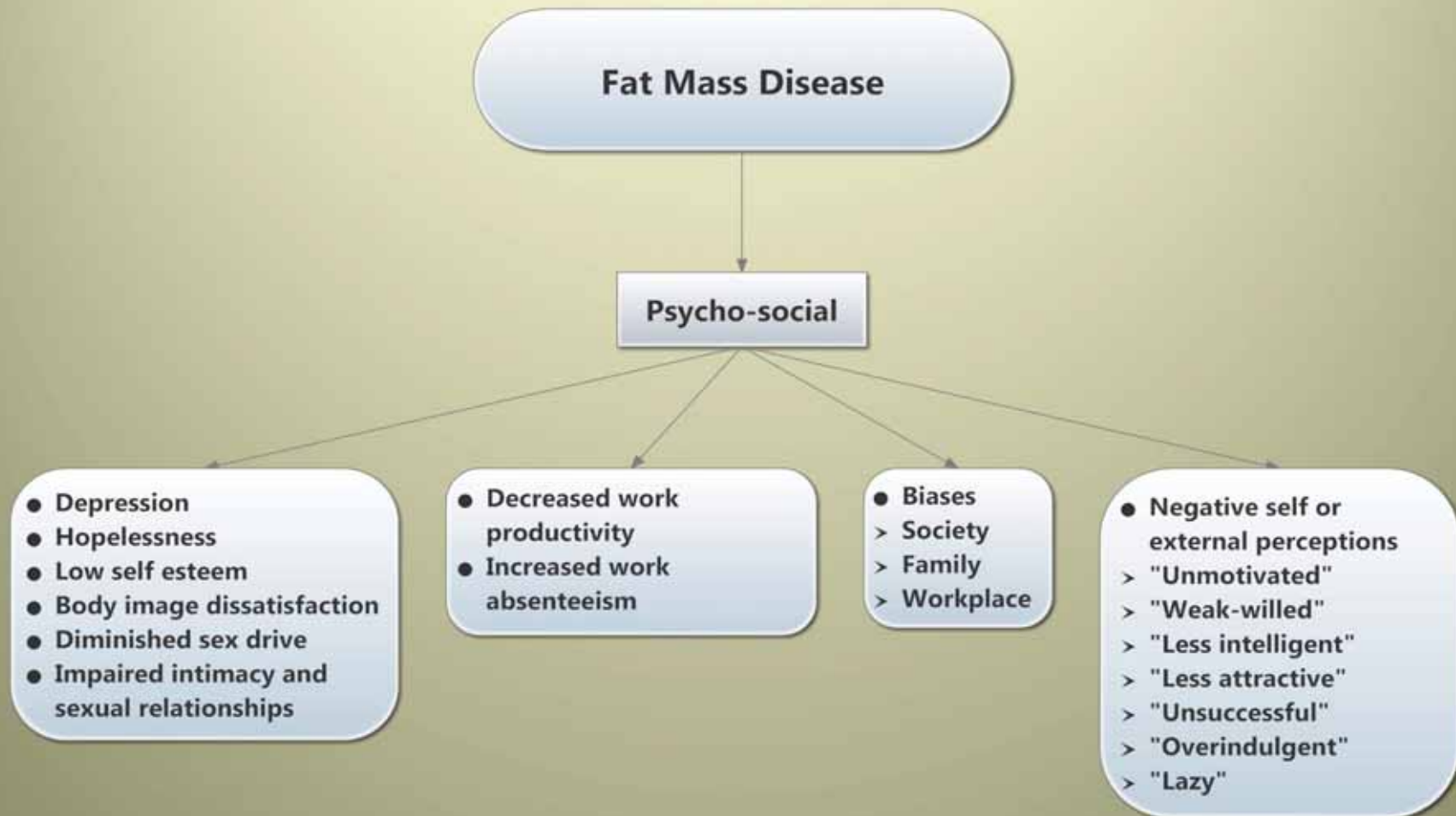
Neurologic

- Intracranial hypertension (pseudotumor cerebri) i.e. due to increased intra-abdominal pressure, sleep apnea, etc.
- Stroke (see "cardiovascular")
- Nerve entrapment (e.g. meralgia paresthetica, carpal tunnel syndrome, etc.)

Diagnose fat mass disease (FMD)
- continued -



Diagnose fat mass disease (FMD)
- continued -



Diagnose "sick fat disease" (SFD)**Adiposopathy****Clinical manifestations**

- High blood glucose (prediabetes mellitus, type 2 diabetes mellitus)
- High blood pressure
- Metabolic syndrome
- Adiposopathic dyslipidemia
 - > Increased triglyceride levels
 - > Decreased high density lipoprotein cholesterol levels
 - > Increased atherogenic particle number (increased apolipoprotein B)
 - > Increased small dense low density lipoprotein particles
 - > Increased triglyceride-rich lipoproteins
 - > Increased lipoprotein remnant lipoproteins

- Insulin resistance
- Hepatosteatorsis (fatty liver)
- Hyperuricemia and gout
- Cholelithiasis
- Nephrolithiasis
- Glomerulopathy
- Pro-thrombotic predisposition
- Neuropsychiatric diseases (such as worsening depression due to adiposopathic immune and endocrine responses)
- Asthma (due to adiposopathic immune and endocrine responses)
- Worsening of other inflammatory diseases (osteoarthritis, atherosclerosis, etc.)

Diagnose "sick fat disease" (SFD)**Adiposopathy****Clinical manifestations**

- Hyperandrogenemia in women
- Polycystic ovarian syndrome
- Menstrual disorders
- Infertility
- Hypoandrogenemia in men

- Cancer - strongest evidence
 - > Endometrial cancer
 - > Postmenopausal breast cancer
 - > Colon cancer
 - > Renal cell carcinoma
 - > Liver cancer
 - > Gallbladder cancer
 - > Esophageal cancer
 - > Pancreatic cancer
- Cancer - mounting evidence
 - > Cervical cancer
 - > Ovarian cancer
 - > Prostate cancer (prognosis, not necessarily increased risk)
 - > Stomach cancer

Diagnose "sick fat disease" (SFD)**Adiposopathy****Etiology / Causes**

- Positive caloric balance, especially with high carbohydrate dietary intake
- Sedentary lifestyle with inadequate level of physical activity
- Genetic predisposition
- Environmental contributors to increased body fat
- Extra-genetic contributors to worsening fat function (e.g. certain medications, viral infections, gut microbiota transmission of pro-inflammatory state, etc.)

Anatomic abnormalities

- Adipocyte hypertrophy
- Increased visceral, pericardial, perivascular, and other periorgan adiposity
- Growth of adipose tissue beyond vascular supply
- Increased adipose tissue immune cells
- "Ectopic" fat deposition in other body organs (liver, muscle, possibly pancreas, etc.)

Pathophysiology

- Impaired adipogenesis
- Adipocyte organelle dysfunction (endoplasmic reticulum, mitochondria, etc.)
- Increased circulating free fatty acids
- Pathogenic adipose tissue endocrine responses
- Pathogenic adipose tissue immune responses
- Pathogenic interactions with other body organs such as fatty liver, vasculopathies (endothelial dysfunction, atherosclerosis, hypercoagulation), etc.

History**History****Medical history**

- Age
- Gender
- Race
- Ethnicity
- Fat mass disease (e.g osteoarthritis, sleep apnea etc.)
- Adiposopathy (e.g. type 2 diabetes mellitus, high blood pressure, etc.)
- Eating disorders
- Other medical and surgical conditions
- Medication and food allergies
- Medications
- Cigarette smoking
- Alcohol intake
- Illicit drug use (marijuana, cocaine, etc.)

Family history

- Overweight family members
- Applicable familial medical diseases

Socioeconomic & Cultural history

- Economic status
- Social status
- Cultural background
- Occupation
- Family structure
- Parenting behavior
- Marital status
- Living situation
- Possible abuse (physical, mental, sexual, etc.)

History**Nutrition history****Meals and snacks**

- Timing
- Frequency
- Nutritional content
- Preparer of food
- Access to foods
- Location of home food consumption (e.g. eating area, television, computer, etc.)
- Location of away food consumption (e.g. workplace restaurants, fast food, etc.)

Behavior

- Previous nutritional attempts to change body composition, and if unsuccessful or unsustained, what were short and long-term barriers to achieving or maintaining fat weight loss
- Triggers (e.g. hunger, cravings, anxiety, boredom, reward, etc.)
- Nighttime eating
- Binge eating
- Emotional eating
- Family influences
- Community influences
- Readiness for change

Records

- Food and beverage diary, including type & amount (72 hour recall, keep food and beverage record for a week and return for evaluation, etc.)

History**Physical Activity History**

- Success and/or failure of previous physical activity / exercise efforts
- Reasons if no longer engaged in a routine physical activity / exercise regimen

- Current mobility and equipment needs
- Current physical activity / exercise status
- Current fitness level or endurance capacity
- Access to locations amenable to increased physical activity / exercise (e.g. gym, workplace exercise facilities, home setting (urban, rural) etc.
- Physical activity / exercise preferences
- Perceived barriers to increased physical activity

- Medical conditions that should be evaluated before prescribing an exercise program
 - Diseases of the heart, lung, musculoskeletal and other body systems
 - Metabolic diseases having potential risks with increased physical activity such as atherosclerotic coronary heart disease (worsening ischemia), diabetes mellitus (hypoglycemia), high blood pressure (increase blood pressure with resistance training), etc.

History**History****Routine preventive medical care**

Encourage patient to be up-to-date with sentinel and preventive physical exam procedures that the patient and prior clinicians may have avoided, which depending upon gender and age, may include:

- Breast exam (and mammogram as applicable)
- Pelvic exam
- Pap smear
- Testicular exam
- Rectal exam and stool for occult blood (sigmoidoscopy or colonoscopy as applicable)
- Immunizations

Physical Exam**Physical exam****Vital signs**

- Height with bare or stocking feet measured with a stadiometer
- Weight using calibrated scale and method consistent from visit to visit (e.g. light indoor clothing or gown, etc.)
- Body mass index (BMI)
- Standing waist circumference using superior iliac crest (optional for BMI > 35 kg/m²)
- Neck circumference
- Blood pressure using appropriate sized cuff
- Pulse

General physical exam

- Physical exam of general body systems
- Special emphasis on physical exam of the lung, heart, musculoskeletal system, and integument

Laboratory**Laboratory****Adiposity-relevant blood testing****General laboratory testing**

- Fasting blood glucose
- Hemoglobin A1c
- Fasting lipid levels
 - > Triglycerides
 - > Low density lipoprotein (LDL) cholesterol
 - > High density lipoprotein (HDL) cholesterol
 - > Non-HDL cholesterol
- Liver enzymes and other liver blood tests
 - > Aspartate aminotransferase (AST)
 - > Alanine aminotransferase (ALT)
 - > Alkaline phosphatase
 - > Total bilirubin
- Electrolytes (e.g. potassium, sodium, calcium, phosphorous, etc.)
- Renal blood testing (e.g. creatinine, blood urea nitrogen, etc.)
- Uric acid
- Thyroid stimulating hormone (TSH)
- Vitamin D levels

- Complete blood count
- Urinalysis
- Urine for microalbumin

Laboratory**Laboratory****Individualized body system testing**

- Resting electrocardiogram
- Cardiac stress testing
- Echocardiogram
- Sleep studies
- Imaging studies of the liver (e.g. ultrasound)
- Anaerobic threshold/VO₂ testing
- Resting Metabolic Rate (RMR)
- Coronary calcium scores

Body composition quantitative testing

- Dual energy X-ray absorptiometry (DXA)
- Bioelectric impedance analysis
- Near-infrared interactance
- Whole-Body Air-Displacement Plethysmography (BOD POD)
- Quantitative magnetic resonance (QMR)
- Underwater weighing
- Deuterium dilution
- Myotape measurements for wrist and neck size (for potential use in percent body fat equations)
- Myotape measurements for muscle mass
- Caliper percent body fat measurements (e.g. 3-site skinfold calculations)

Laboratory**Laboratory****Individualized blood testing**

- Overnight dexamethasone suppression test cortisol or 24 hour urine collection for cortisol if endogenous hypercortisolism is suspected
- Estradiol, follicle stimulating hormone, luteinizing hormone, and pregnancy test in women with oligomenorrhea or amenorrhea
- Testosterone and other androgen levels (e.g. dehydroepiandrosterone sulfate/DHEAS) for women with hirsutism or with polycystic ovarian syndrome
- Testosterone for men with impotence or physical findings of hypogonadism [and if low to a clinically significant degree, possibly prolactin, follicle stimulating hormone and luteinizing hormone]
- Apolipoprotein B and/or lipoprotein particle number, especially if triglyceride levels are elevated.
- Iron studies (iron, total iron binding capacity, ferritin)
- C-reactive protein

Emerging science testing

- Fasting insulin
- Leptin
- Adiponectin
- Leptin to adiponectin ratio
- Free fatty acids
- Immune markers
 - > Tumor necrosis factor
 - > Interleukin 1 and 6
- Infectious testing
 - > Gut microbiota
 - > Adenovirus assays
 - > Evaluation for other microbes



Treatment**Treatment of adult patients with
overweight or obesity as a disease**

- Treat adipocyte and adipose tissue dysfunction, which treats sick fat disease (SFD or adiposopathy)
- Treat excessive body fat, which treats fat mass disease (FMD)

**Treating diseases due to excessive body fat improves patient health,
quality of life, body weight and body composition**

Treatment

Identify and manage secondary and/or contributing causes of SFD and FMD

Conditions that may promote fat mass gain

- **Medical conditions**

- Hypothalamic damage
- Prader Willi syndrome
- Immobility
- Insulinoma
- Some cases of untreated hypothyroidism
- Hypercortisolism (Cushing's disease)
- Reduced sleep / sleep apnea

- **Psychological / behavioral conditions**

- Mental stress
- Depression
- Anxiety
- Post-traumatic stress syndrome
- Binge eating disorder
- Night eating disorder

Treatment**Identify and manage concomitant pharmacotherapy that might influence body weight****Illustrative examples of pharmacotherapies that may promote fat mass gain**

- Steroid hormones (e.g. glucocorticoids, estrogens, progestins, tamoxifen)
- Diabetes therapies (e.g. some insulins, sulfonylureas, thiazolidinediones)
- Some highly active antiretroviral protease inhibitors
- Some B-adrenergic blockers (most commonly described with nonselective B-blockers (e.g. propranolol))
- Some adrenergic blockers
- Some antihistamines (e.g. diphenhydramine)
- Some antidepressants [tricyclic antidepressants, irreversible monoamine oxidase (MAO) inhibitors, mirtazapine, and some selective serotonin reuptake inhibitors (e.g. paroxetine, antiserotonin agents (e.g. pizotifen)]
- Some antiseizure drugs (e.g. valproate, gabapentin, and carbamazepine),
- Some psychotropic drugs (e.g. clozapine, olanzapine, risperidone, quetiapine, thioridazine, divalproex, and chlorpromazine and lithium)
- Some chemotherapies

Illustrative examples of pharmacotherapies that may promote fat mass loss

- Anti-diabetes mellitus agents
 - > Metformin
 - > Glucagon-like peptide-1 (GLP-1) agonists
 - > Sodium glucose co-transporter 2 (SGLT2) inhibitors
- Neurologic agents
 - > Topiramate
 - > Bupropion

Treatment**Nutritional Therapy**

- Energy consumption intended to cause negative caloric balance and fat weight loss
- Low calorie diet is often described as 800 - 1500 kcal / day
- Very low calorie diet is often described as < 800 kcal / day

Restricted dietary carbohydrate**Restricted dietary fat****Very low calorie diets**

Treatment**Carbohydrate-restriction
("low carb diet")**

Often defined as approximately 50 - 150 grams of carbohydrate per day, but sometimes less

Weight loss

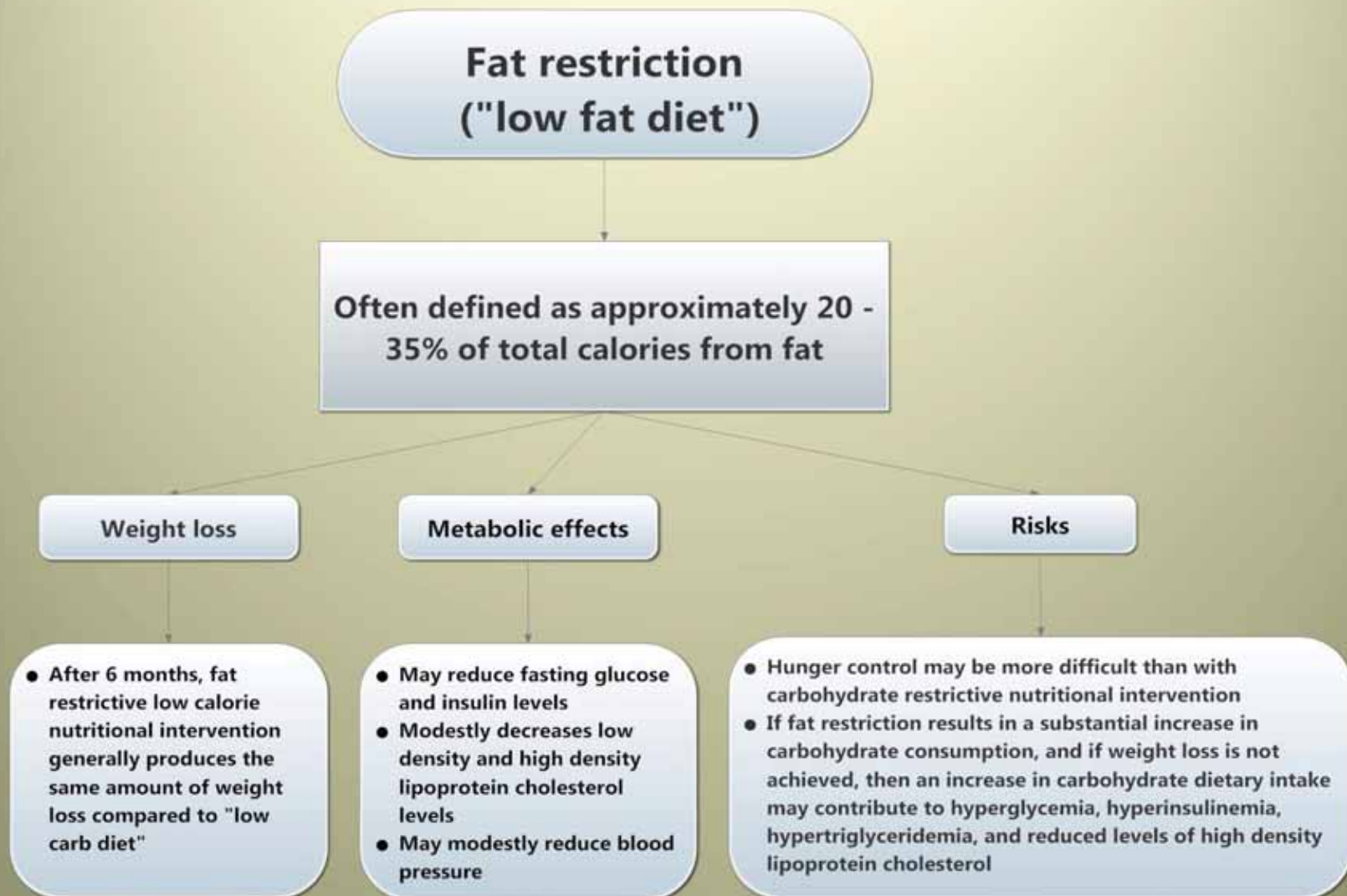
- May produce modestly greater weight loss compared to fat restricted dietary intake for the first 6 months, wherein afterwards, the net weight loss may be similar to other calorie restricted nutritional interventions

Metabolic effects

- Reduces fasting glucose and insulin levels
- Reduces triglyceride levels
- Modestly increases high density lipoprotein cholesterol levels
- May modestly increase low density lipoprotein cholesterol levels
- The metabolic effects noted above may occur with or without weight loss
- May modestly reduce blood pressure
- In patients with epilepsy, ketogenic diets may reduce seizures
- Ketogenic diet may possibly improve diabetes mellitus complications (e.g. nephropathy)

Risks

- May produce carbohydrate cravings within the first few days of implementation, which may be mitigated by artificial sweeteners or adding low glycemic index foods

Treatment

Treatment**Very low calorie diets**

Often defined as 400 - 800 kcal/day,
commonly implemented by use of
commercially prepared formulas

Weight loss

- Produces more rapid weight loss than standard carbohydrate and/or fat restricted dietary intake

Metabolic effects

- Reduces fasting glucose and insulin levels
- Reduces triglyceride levels
- May modestly increase high density lipoprotein cholesterol levels
- May modestly decrease low density lipoprotein cholesterol
- Reduces blood pressure

Risks

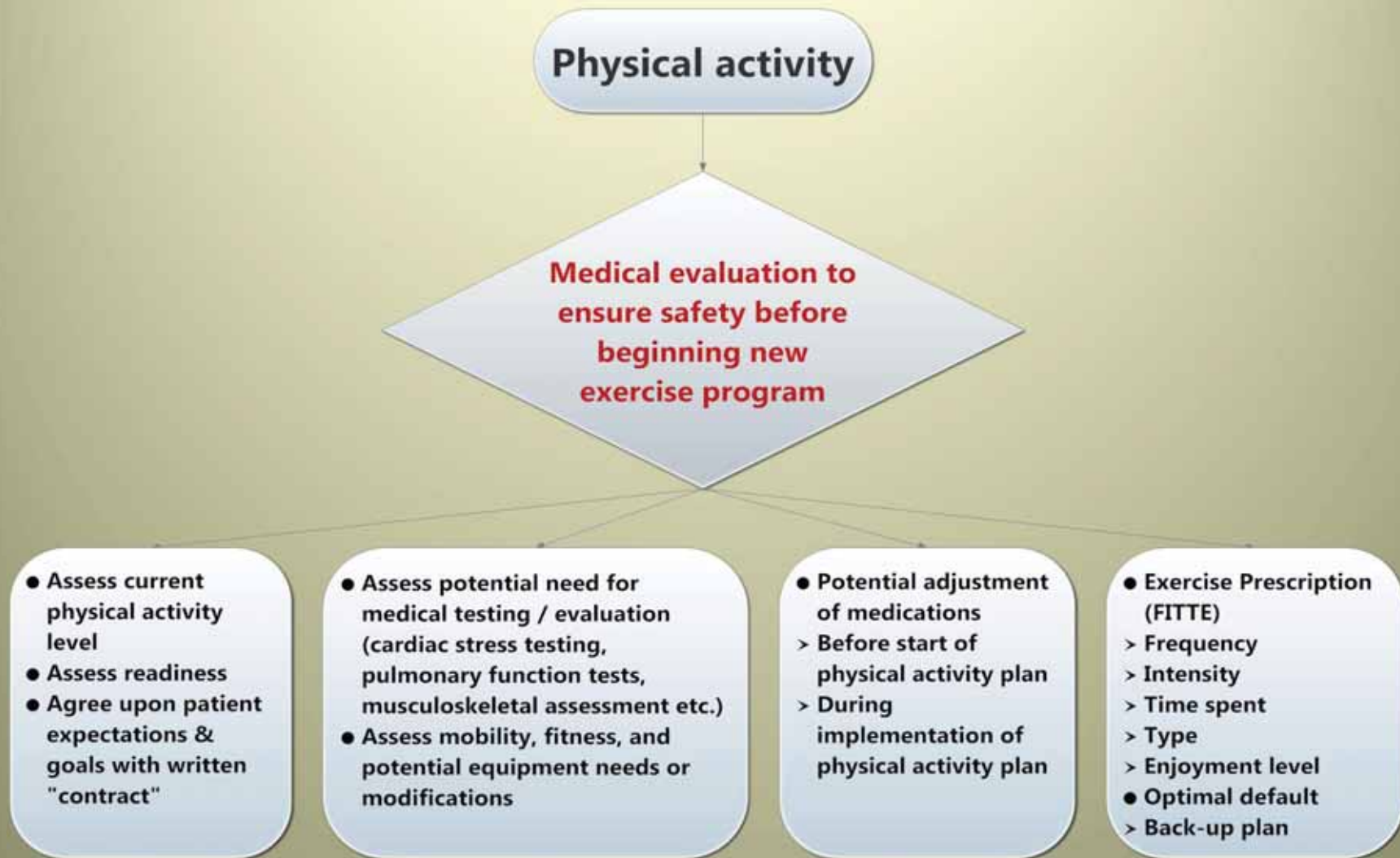
- Fatigue, nausea, constipation, diarrhea, hair loss, and brittle nails
- Cold intolerance
- Dysmenorrhea
- Gallstones
- Kidney stones
- Gout
- Insufficient mineral intake may predispose to:
 - > Palpitations and cardiac dysrhythmias
 - > Muscle cramps
 - > Possible increased risk of osteoporosis
 - > Tooth decay

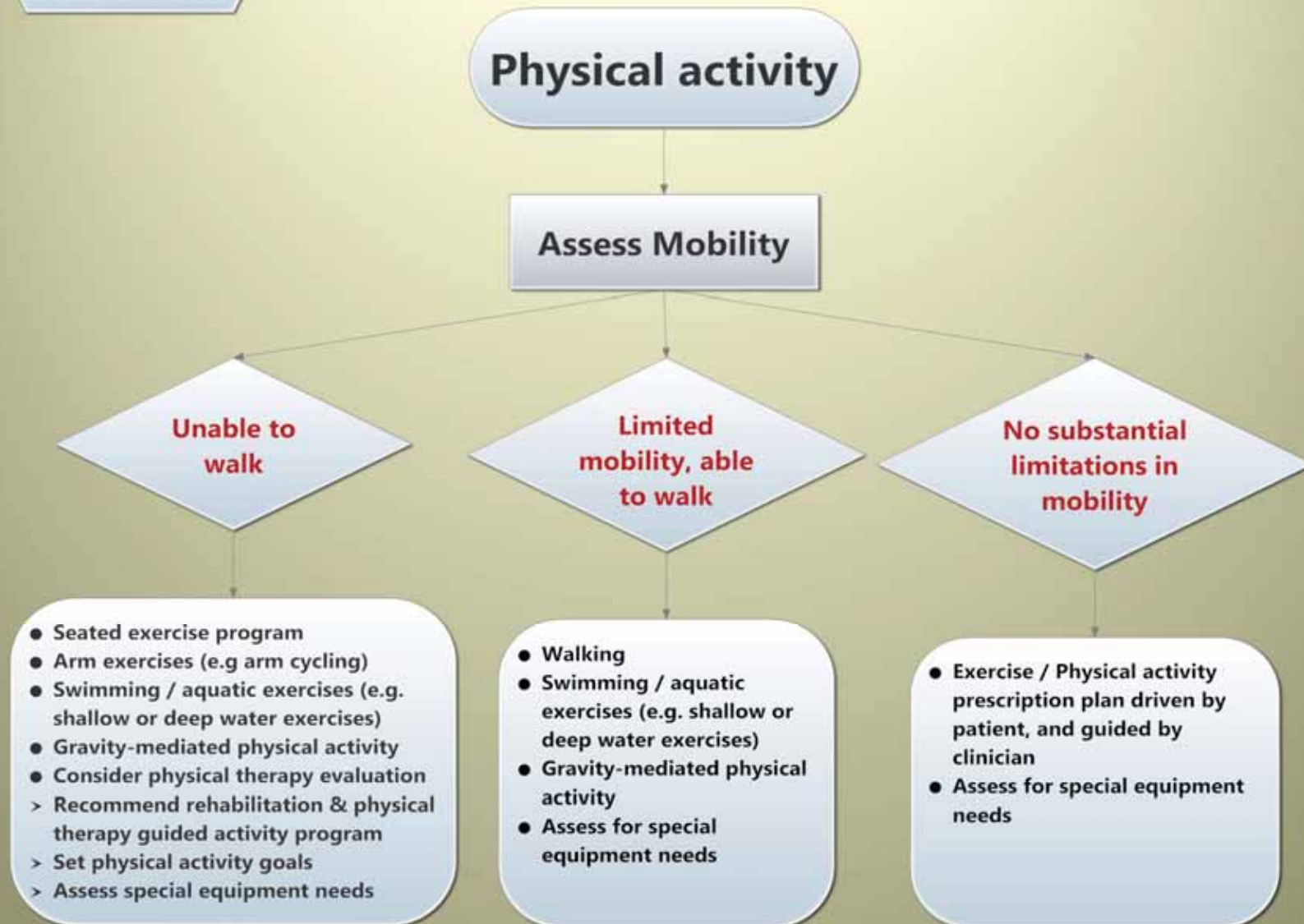
Treatment**Physical activity****Adiposopathy (sick fat disease)**

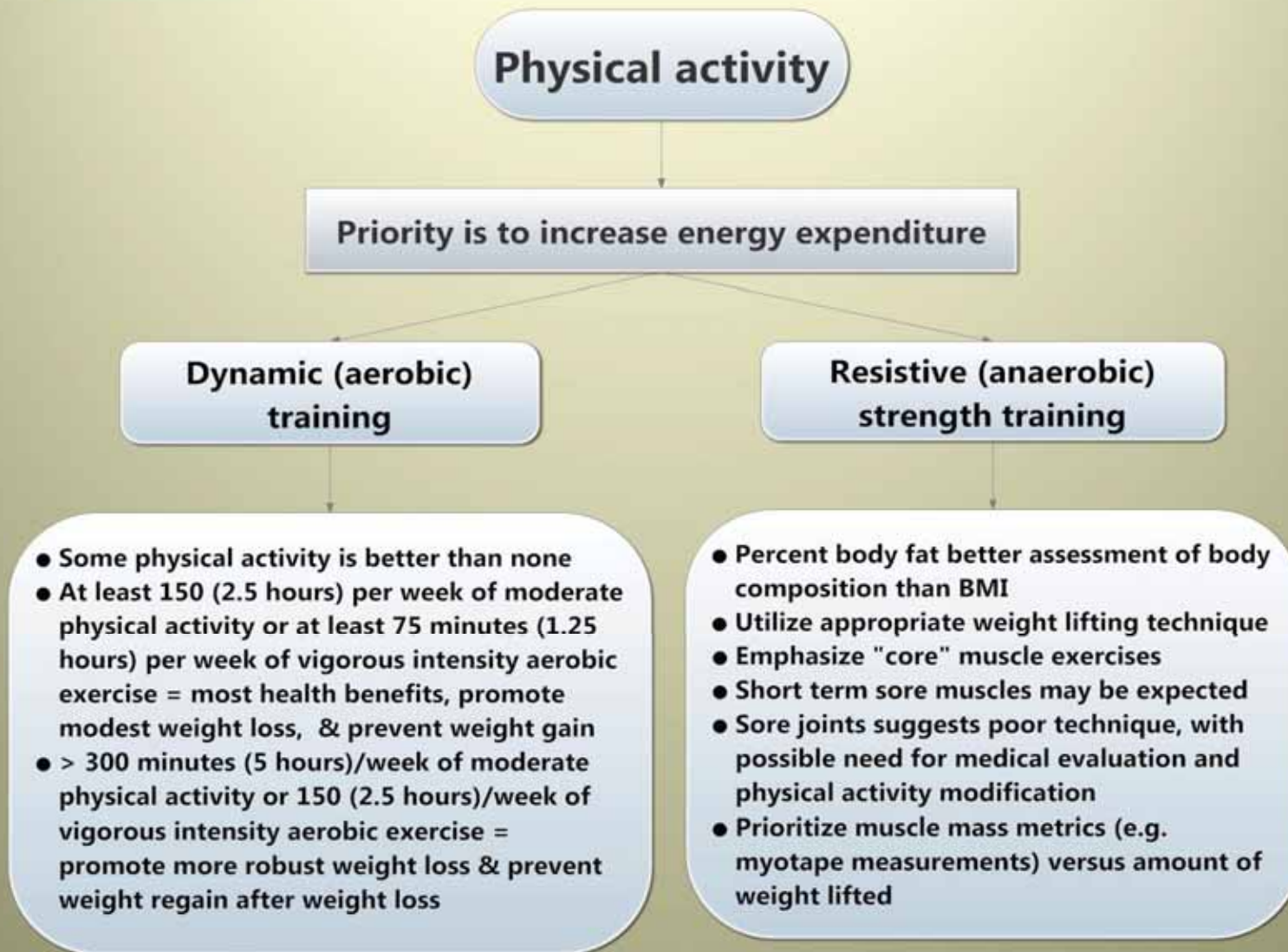
- Assist with weight maintenance
- Assist with weight loss
- Improve body composition
- Possibly improve adipocyte function ("train" fat cells)
 - Improve insulin sensitivity
 - Increase mitochondrial biogenesis
 - Increase browning of fat cells

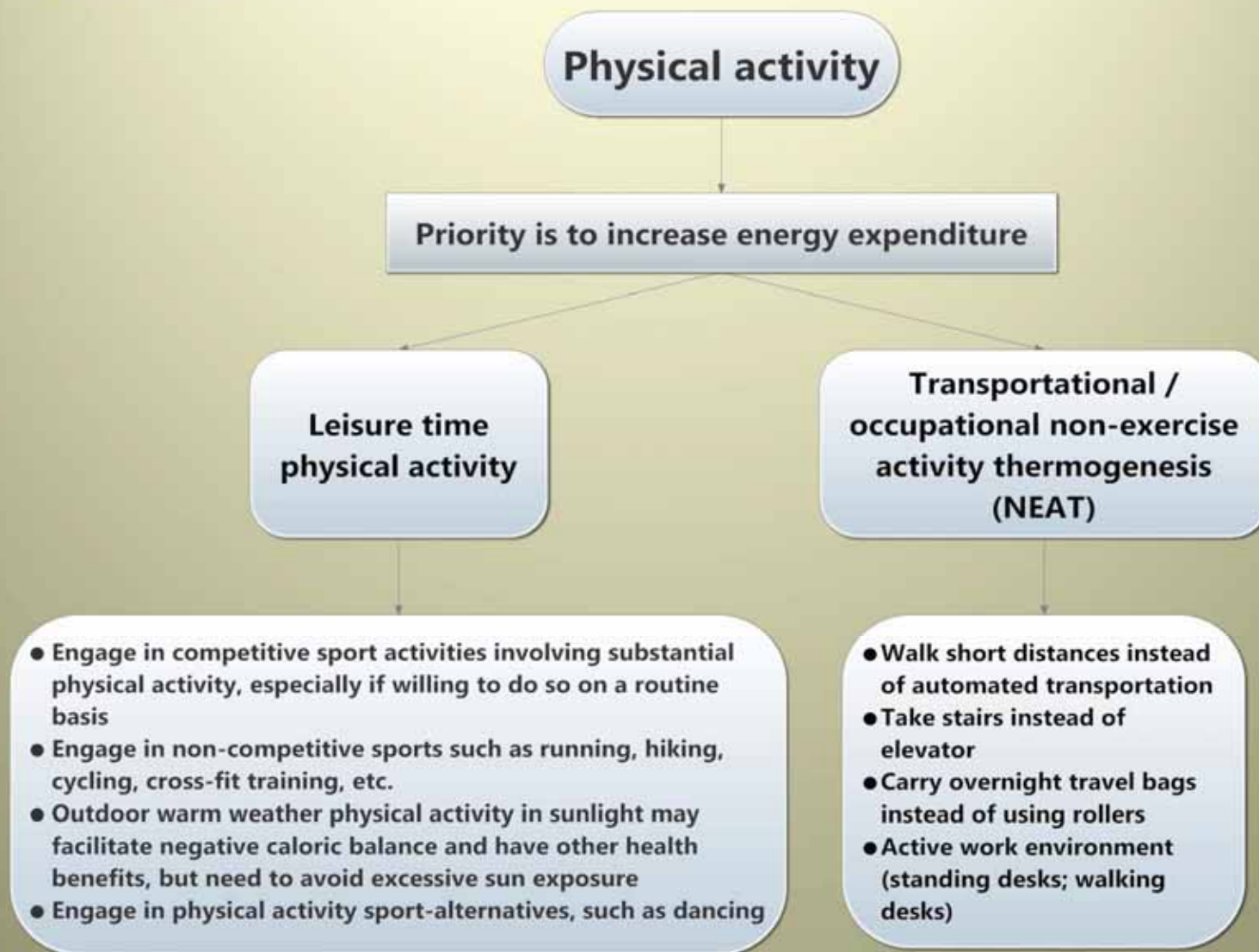
Non-adipose health parameters

- Improve metabolic health
- Improve musculoskeletal health
- Improve cardiovascular health
- Improve pulmonary health
- Improve mental health
- Improve sexual health

Treatment

Treatment

Treatment

Treatment

Treatment**Physical activity****Physical activity accounting records**

- Daily activity logs
- Pedometer / accelerometer logs
- Dynamic training metrics (miles run, laps swam, etc.)
- Resistance training metrics (muscle circumference measurements, reps, sets, etc.)
- Percent body fat measurements

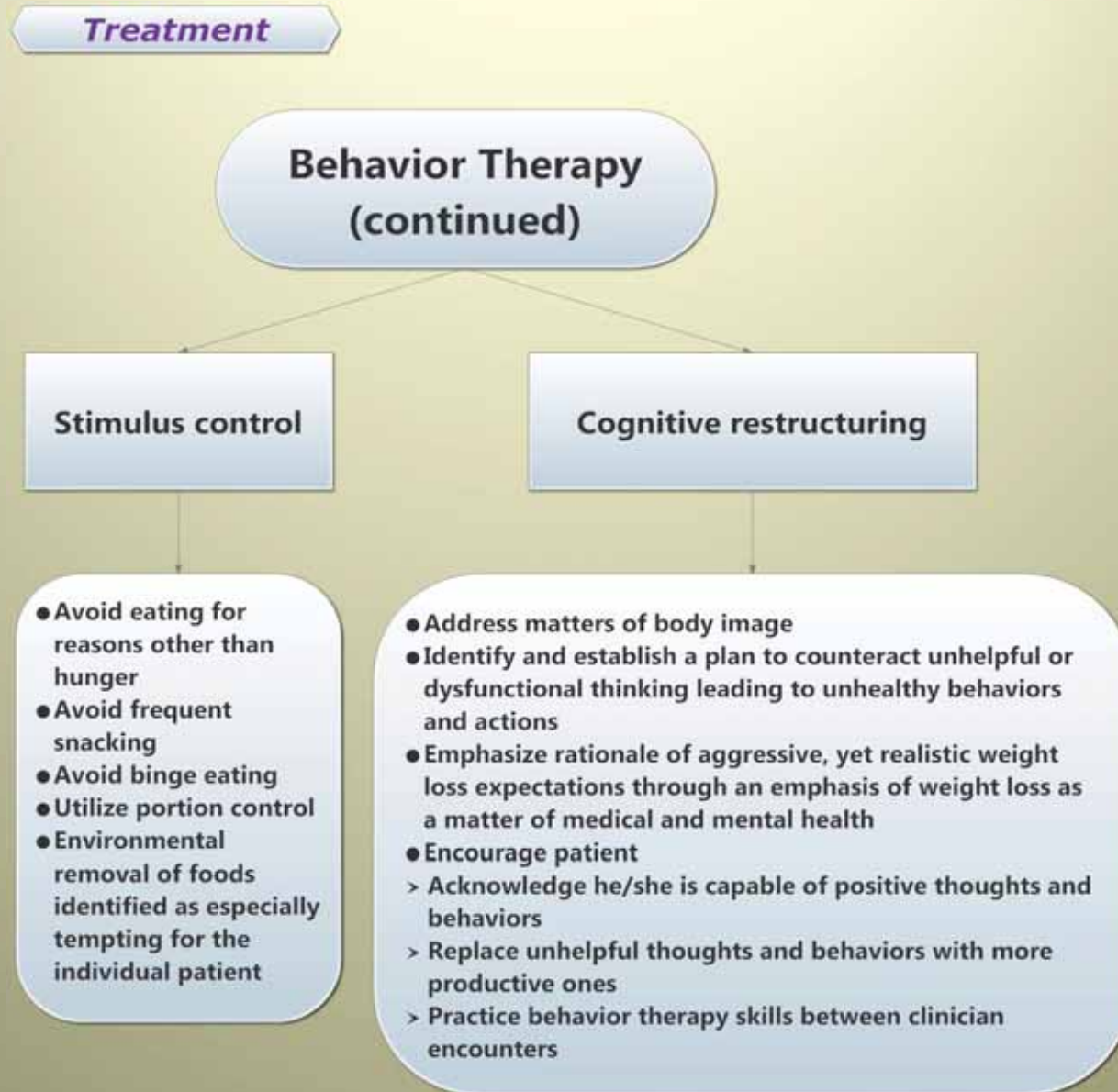
Treatment**Behavior Therapy**

Frequent encounters with medical professional or other resources free from provider bias

Education

- Physician
- Dietitian
- Nurse educator
- Physical activity professional (trainer, physiologist, etc.)
- Mental health professional
- Web-based programs
- Mobile access (text messages, applications, etc.)
- Multidisciplinary approach
 - > Clinicians with professional expertise
 - > Patient with self expertise

- Medical health
- Mental health
- Nutrition
- Physical activity
- Establish healthy sleep habits
- Establish healthy eating habits (e.g. reduce speed of eating, drink water between meals, choose and have available healthy snacks, etc.)



Treatment**Behavior Therapy
(continued)****Goal setting**

- Patients are given step-by-step instructions to accomplish goals (e.g. nutrition and physical activity prescriptions)

Self monitoring

- Daily or weekly body weights
- Other routine self anthropometric measurements (calipers for percent body fat, tape measure for waist circumference, myotape for muscle mass, etc.)
- Food diaries (including online services or mobile applications)
- Physical activity logs
- Pedometer / accelerometer measures



Treatment**Weight Management Pharmacotherapy**

Adjunct to nutritional, physical activity, and behavioral therapies

Objectives:

- **Treat disease**
 - **Adiposopathy or sick fat disease (SFD)**
 - **Fat mass disease (FMD)**
- **Facilitate management of eating behavior**
- **Slow progression of weight gain / regain**
- **Improve the health, quality of life, and body weight of the obese and/or overweight patient**

Treatment**Pathologic metabolic and/or fat mass consequences of increased body fat**

5 - 10 % weight loss may improve adipocyte and adipose tissue metabolic and immune function

5 - 10 % weight loss may improve metabolic disease

5 - 10 % weight loss may improve abnormal and pathologic physical and mechanical forces

5 - 10 % weight loss may improve fat mass diseases

Treatment**Pharmacotherapy**

Examples of weight management agents approved 1999 or before

- Phentermine
- Diethylpropion
- Phendimetrazine
- Benzphetamine
- Orlistat

Examples of weight management agents approved 2012 and beyond

- Lorcaserin
- Phentermine HCl / topiramate extended-release

Treatment**Pharmacotherapy****Sympathomimetic amines**

- Examples: Phentermine, diethylpropion, phendimetrazine, benzphetamine
- Increases satiety
- Drug Enforcement Agency Schedule weight management agents
 - > DEA IV for phentermine and diethylpropion
 - > DEA III for phendimetrazine and benzphetamine
- Potential adverse experiences include palpitation, tachycardia, increased blood pressure, overstimulation, tremor, dizziness, insomnia, dysphoria, headache, dryness of mouth, dysgeusia, diarrhea, constipation
- Pregnancy category X

Gastrointestinal lipase inhibitors

- Example: Orlistat
- Impairs gastrointestinal energy absorption
- Potential adverse experiences include oily discharge from the rectum, flatus with discharge, increased defecation, fecal incontinence, may increase risk of cholelithiasis, may increase risk of urinary oxalate, rare postmarketing reports of severe liver injury, may decrease fat-soluble vitamin absorption (e.g. vitamins A, D, E, K, and beta carotene)
- Pregnancy category X

Treatment**Weight Management Pharmacotherapy****Currently Approved Pharmacotherapy Principles****Approved weight management pharmacotherapy indications:**

- Obese patients (e.g. BMI $\geq 30 \text{ kg/m}^2$)*
- Overweight patients (e.g. BMI $\geq 27 \text{ kg/m}^2$) with presence of adiposity complication (e.g. type 2 diabetes mellitus, hypertension, dyslipidemia)*

If no clinical improvement after 12 weeks with one weight management pharmacotherapy, then consider alternative weight management pharmacotherapy, or increasing weight management pharmacotherapy dose (if applicable).

*** While BMI (body mass index) is the only measure listed in the prescribing information for weight management pharmacotherapy, BMI may have limitations; in some circumstances, obesity and overweight are more accurately assessed by other measures**

Treatment**Lorcaserin****Indications & use**

- Serotonin (5-hydroxytryptamine) 2c receptor agonist weight management agent
- Drug Enforcement Agency Schedule IV drug
- Dose = 10 mg twice a day

Potential drug interactions

- The safety of lorcaserin coadministration with other serotonergic or antidopaminergic agents is not yet established, which includes selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, monoamine oxidase inhibitors, triptans, bupropion, dextromethorphan, St. John's Wort

Pharmacokinetics

- Lorcaserin is metabolized in the liver with metabolites excreted in the urine.

Treatment**Lorcaserin****Potential Adverse experiences**

- Headache
- Dizziness
- Fatigue
- Nausea
- Dry mouth
- Constipation
- Cough
- Reduced heart rate
- Hyperprolactinemia

Contraindications

- If signs or symptoms of valvular heart disease develop, then discontinuation of lorcaserin should be considered during evaluation for valvulopathy
- Use with caution with use of hazardous machinery because of the potential for cognitive impairment with disturbances in attention or memory
- Use with caution among patients with psychiatric disorders, including euphoria and dissociation
- Use with caution among patients with psychiatric disorders and predisposed to depression who should be monitored for depression or suicidal thoughts, with discontinuation of lorcaserin if symptoms develop
- Weight-loss lorcaserin may produce hypoglycemia in patients treated for diabetes mellitus
- Use with caution in men with history of priapism or predisposition to priapism
- Contraindicated during pregnancy or nursing mothers (Pregnancy category X)

Treatment**Phentermine HCl / topiramate extended-release**

Completion of Risk Evaluation and Mitigation Strategy (REMS) program to inform prescribers and women patients about the increased risk of congenital malformations (especially orofacial clefts) in infants exposed to phentermine HCl / topiramate ER during the first trimester of pregnancy

Indications & use

- Drug Enforcement Agency Scheduled IV
- Phentermine is a shorter acting sympathomimetic amine approved as monotherapy as a weight management drug
- Topiramate is a longer-acting neurostabilizer approved as monotherapy for seizure disorders and migraine headache prevention
- Doses = Once daily in the morning with or without food
 - > Starting dose = 3.75-mg/23-mg (phentermine 3.75 mg/topiramate 23 mg extended-release); then after 14 days intervals, and as clinically indicated, escalate doses to:
 - > Recommended dose = 7.5 mg/46 mg
 - > Titration dose = 11.25 mg/69 mg
 - > Top dose = 15 mg/92 mg

Potential drug interactions

- PHEN/TPM ER may alter the exposure to oral contraceptives, causing irregular menstrual bleeding, but not an increased risk of pregnancy.
- Oral contraceptives should not be discontinued if spotting occurs.
- PHEN/TPM ER may potentiate central nervous system depressants such as alcohol; thus, patients should avoid concomitant alcohol
- PHEN/TPM ER may potentiate hypokalemia of non-potassium sparing diuretics

Pharmacokinetics

- Phentermine is metabolized by the liver, with most excreted by the kidney.
- Topiramate is excreted mainly by the kidney

Treatment

Phentermine HCl / topiramate extended-release

Completion of Risk Evaluation and Mitigation Strategy (REMS) program to inform prescribers and women patients about the increased risk of congenital malformations (especially orofacial clefts) in infants exposed to phentermine HCl / topiramate ER during the first trimester of pregnancy

Potential Adverse experiences

In clinical trials, adverse reactions occurring greater than or equal to 5% include:

- Paresthesia
- Dizziness
- Dysgeusia (taste distortion/perversion)
- Insomnia
- Constipation
- Dry mouth

Laboratory abnormalities may include:

- Metabolic acidosis
- Elevated creatinine
- Lowering of glucose levels

Contraindications

PHEN/TPM ER is contraindicated:

- Glaucoma
- Hyperthyroidism
- During or within 14 days of taking monoamine oxidase inhibitors
- Women of reproductive potential should have negative pregnancy test before treatment and monthly thereafter, and use effective contraception while on PHEN/TPM ER
- Pregnancy or nursing (Pregnancy category x)

PHEN/TPM ER should be discontinued in patients with:

- Unacceptable increases in adrenergic responses, such as increase in heart rate especially those with cardiac and/or cerebrovascular disease
- Suicidal behavior and ideation
- Acute myopia and secondary angle closure glaucoma
- Unacceptable mood and sleep disorders
- Cognitive impairment
- Pregnancy or nursing

Early Treatment / Prevention**44 year old woman with overweight / obesity:**

- Prediabetes mellitus
- Prehypertension
- Mild dyslipidemia
- Discomfort to weight bearing joints
- Mild snoring
- Low self esteem due to increased body weight

Optimal treatment strategy: Decide to engage in early, pro-active interventions intended to prevent onset of sick fat disease (diabetes mellitus, dyslipidemia, hypertension) and prevent fat mass disease

- Optimize nutritional therapy and physical activity
- Initiate behavioral therapy
- Consider weight management pharmacotherapy
- Consider bariatric surgery

- Prevent onset of metabolic disease:
 - > Diabetes mellitus
 - > Dyslipidemia
 - > Hypertension
- Prevent fat mass diseases:
 - > Osteoarthritis
 - > Sleep apnea
 - > Depression

Delayed Treatment**44 year old woman with overweight / obesity:**

- Prediabetes mellitus
- Prehypertension
- Mild dyslipidemia
- Pain to weight bearing joints
- Mild snoring
- Low self esteem due to increased body weight

Suboptimal treatment strategy: Wait for the onset of diabetes mellitus, dyslipidemia, hypertension, osteoarthritis, sleep apnea, and depression

Once adverse health consequences are blatantly apparent:

- Optimize nutritional therapy and physical activity
- Initiate behavioral therapy
- Consider weight management pharmacotherapy
- Consider bariatric surgery

Follow:

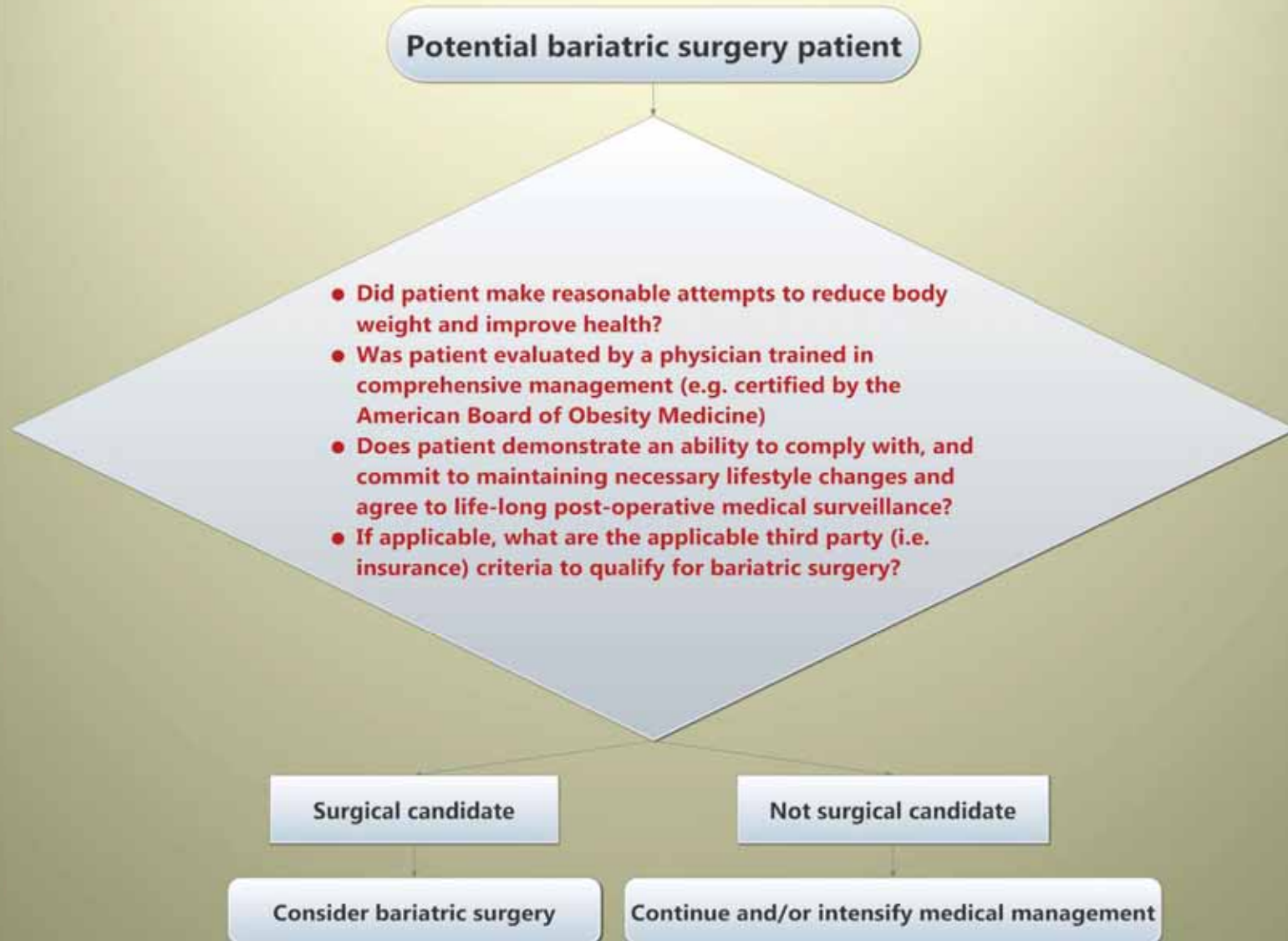
- Diabetes mellitus evaluation and treatment guidelines such as the American Diabetes Association Clinical Practice Recommendations
- Lipid evaluation and treatment guidelines such as the National Cholesterol Education Program, Adult Treatment Panel, or the American Diabetes Association / American College of Cardiology Consensus Report
- Blood pressure guidelines such as the Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
- Follow other disease-specific guidelines

Delayed Treatment**44 year old woman with overweight / obesity:**

- Prediabetes mellitus
- Prehypertension
- Mild dyslipidemia
- Pain to weight bearing joints
- Mild snoring
- Low self esteem due to increased body weight

If the decision was made to wait for the onset of diabetes mellitus, dyslipidemia, hypertension, osteoarthritis, sleep apnea, and depression (continued):

- Utilize diabetes mellitus therapies most likely to improve adipose tissue function
- In patients with fat mass disease, utilize diabetes mellitus therapies having neutral or body weight loss effects, such as metformin, glucagon-like peptide-1 (GLP-1) agonists, sodium glucose cotransporter-2 (SGLT2) inhibitors, etc.)
- Utilize lipid therapies most likely to reduce atherosclerotic coronary heart disease risk and least likely to increase body weight
- Utilize blood pressure therapy most likely to reduce cardiovascular disease risk, which may also provide other health benefits (e.g. diuretics, angiotensin converting enzyme inhibitors, etc.)
- Utilize non-steroidal anti-inflammatory agents to treat osteoarthritis
- Treat sleep apnea
- Utilize anti-depressant medications least likely to promote further weight gain
- Administer additional pharmaceuticals and/or treatment modalities as indicated

Treatment

Treatment**Possible bariatric surgery candidate**

- What is body mass index (BMI in kg/m^2)
- Does clinical evidence exist of adverse health consequences (AHC) due to excessive body fat (SFD and/or FMD)?

BMI ≥ 30 with one or more AHC**BMI ≥ 40 with or without AHC**

Treatment**Bariatric surgery pre-op evaluation**

- Medical evaluation by physician specializing in the care in overweight and/or obese patients
- Surgical consultation by bariatric surgery specialist
- Cardiology, Pulmonary, Gastroenterology, other specialty consultation as indicated
- Mental health professional
- Nutritional support (such as through a dietitian)
- Educational support (such as through pre-operative seminar)

Treatment**Laparoscopic adjustable gastric banding**

A surgical procedure where an adjustable band is placed around the upper stomach creating a small pouch. The band diameter is adjustable through introduction of saline via a subcutaneous port that can be accessed from the upper abdomen.

General

- Outpatient procedure
- Recovery usually one week
- Contraindications
 - > Poor surgical candidate
 - > Severe psychiatric disorder
 - > Intolerance to general anesthesia
 - > Pregnancy
 - > Drug or alcohol addiction
 - > Untreated esophagitis

Potential acute complications:

- Band too tight with gastrointestinal obstructive symptoms (e.g. dysphagia)
- Leakage of gastric contents into abdomen
- Hemorrhage
- Gastrointestinal bleeding
- Infection
- Cardiac dysrhythmias
- Atelectasis and pneumonia
- Deep vein thrombosis
- Death

Potential chronic complications

- Weight regain or no weight loss
- Band slippage, erosion, ulceration, port infection, disconnection and displacement
- Esophageal dilation
- Rare nutrient deficiencies if persistent vomiting or marked and sustained decrease in nutritional intake
- Depression

Treatment

Sleeve gastrectomy

A surgical procedure wherein the stomach is reduced to about 25% of its original size by the surgical removal of a large portion of the stomach along the greater curvature resulting in a narrower sleeve or tube like structure

General

- Hospital stay 1 - 2 days
- Recovery 1 - 2 weeks
- Contraindications
 - > Poor surgical candidate
 - > Severe psychiatric disorder
 - > Intolerance to general anesthesia
 - > Pregnancy
 - > Drug or alcohol addiction
 - > Untreated or severe esophagitis
 - > Barrett's esophagus
 - > Severe gastroparesis
 - > Achalasia
 - > Previous gastrectomy
 - > Previous gastric bypass
- Sometimes used as a staged approach to gastric by-pass

Potential acute complications

- Gastrointestinal obstruction
- Hemorrhage
- Gastrointestinal bleeding
- Anastomotic staple line leaks
- Infection
- Cardiac dysrhythmias
- Atelectasis and pneumonia
- Deep vein thrombosis
- Pulmonary emboli
- Rhabdomyolysis
- Dehydration
- Death

Potential chronic complications

- Weight regain or lack of long-term weight loss
- Marginal ulcers
- Esophageal dilation
- Dumping syndrome with reactive hypoglycemia
- Small bowel obstruction caused by internal hernias or adhesions
- Luminal stenoses (stomal narrowing)
- Anastomotic staple line leak
- Fistula formation
- Gallstones
- Calcium deficiency
- Secondary hyperparathyroidism
- Iron deficiency
- Protein malnutrition
- Other nutritional and mineral deficiencies (e.g. deficiencies of vitamins A, C, D, E, B, and K, folate, zinc, magnesium, thiamine, etc.)
- Anemia (often related to mineral and nutrition deficiencies)
- Metabolic acidosis
- Bacterial overgrowth
- Kidney stones (oxalosis)
- Neuropathies (resulting from nutritional deficiencies)
- Osteoporosis (often caused by calcium deficiency and chronically elevated parathyroid hormone levels)
- Depression

Treatment

Gastric bypass

A surgical procedure wherein the stomach is divided into a large residual section and a smaller section (pouch) that is attached to a limb of the small intestine at variable distances from the first part of the small intestine, largely bypassing the stomach and part of the duodenum.

General

- Hospitalization 2 - 4 days
- Recovery 2 - 4 weeks
- Contraindications
 - > Poor surgical candidate
 - > Severe psychiatric disorder
 - > Intolerance to general anesthesia
 - > Pregnancy
 - > Drug or alcohol addiction
 - > Untreated esophagitis
 - > Unwillingness or an inability for appropriate long-term follow-up

Potential acute complications:

- Gastrointestinal obstruction
- Hemorrhage
- Gastrointestinal bleeding
- Anastomotic leaks
- Infection
- Cardiac dysrhythmias
- Atelectasis and pneumonia
- Deep vein thrombosis
- Pulmonary emboli
- Rhabdomyolysis
- Dehydration
- Death

Potential chronic complications:

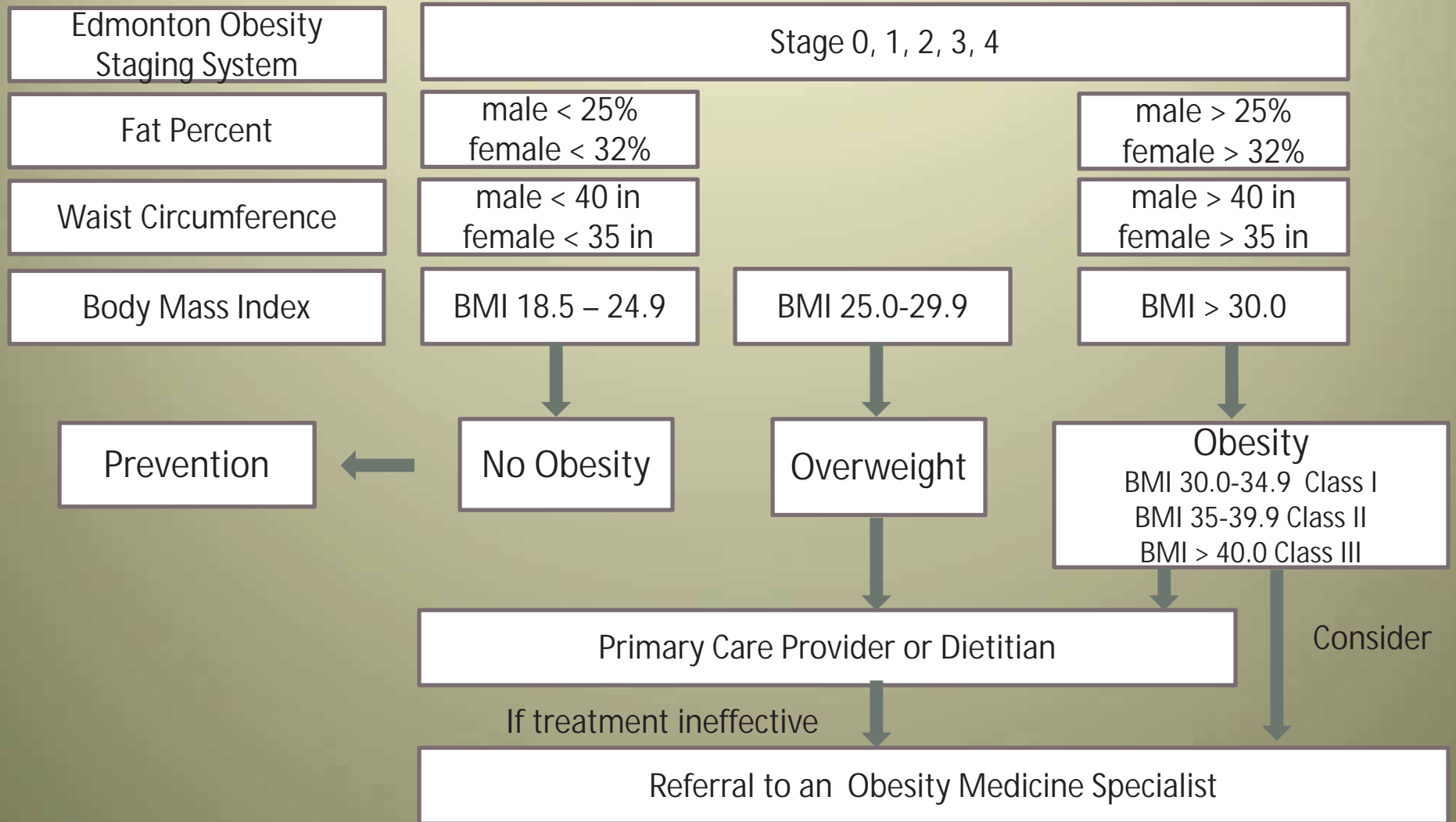
- Weight regain
- Marginal ulcers
- Esophageal dilation
- Dumping syndrome with reactive hypoglycemia
- Small bowel obstruction caused by internal hernias or adhesions
- Anastomotic stenoses (stomal narrowing)
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- Calcium deficiency
- Secondary hyperparathyroidism
- Iron deficiency
- Protein malnutrition
- Other nutritional and mineral deficiencies (e.g. deficiencies of vitamins A, C, D, E, B, and K, folate, zinc, magnesium, thiamine, etc.)
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- Bacterial overgrowth
- Kidney stones (oxalosis)
- Neuropathies (resulting from nutritional deficiencies)
- Osteoporosis (often caused by calcium deficiency and chronically elevated parathyroid hormone levels)
- Depression

ASBP Obesity Algorithm: Executive Summary

Assess for the Presence of Obesity, Adiposopathy, Fat Mass Disease

Obesity may be assessed using several criteria: presence of adiposity-related disease, fat percent, waist circumference or body mass index. Thresholds vary based on ethnicity.

Criteria



ASBP Obesity Algorithm: Executive Summary

Edmonton Obesity Staging System

STAGE 0: No apparent risk factors (e.g., blood pressure, serum lipid and fasting glucose levels within normal range), physical symptoms, psychopathology, functional limitations and/or impairment of well-being related to obesity

STAGE 1: Presence of obesity-related subclinical risk factors (e.g., borderline hypertension, impaired fasting glucose levels, elevated levels of liver enzymes), mild physical symptoms (e.g. dyspnea on moderate exertion, occasional aches and pains, fatigue), mild psychopathology, mild functional limitations and/or mild impairment of well-being

STAGE 2: Presence of established obesity-related chronic disease (e.g., hypertension, type 2 diabetes, sleep apnea, osteoarthritis), moderate limitations in activities of daily living and/or well-being)

STAGE 3: Established end-organ damage such as myocardial infarction, heart failure, stroke, significant psychopathology, significant functional limitations and/or impairment of well-being

STAGE 4: Severe (potentially end-stage) disabilities from obesity-related chronic diseases, severe disabling psychopathology, severe functional limitations and/or severe impairment of well-being

[Sharma AM, Kushner RF. A proposed clinical staging system for obesity. Int J Obesity 2009;33:289-295.]

Fat Percent

Body Fat Percent can be calculated using bioimpedance, near infrared reactance, DEXA scan or whole-body-air-displacement plethysmography.

Waist Circumference

Waist circumference can be measured by tape measure around the abdomen at the level of the anterior superior iliac crests, parallel to the floor. Tape should be snug against skin without compressing.

Body Mass Index

Body Mass Index = (weight in kg) / (height in m)² OR
703 x (weight in pounds) / (height in inches)²

Obesity Medicine Specialists, certified by the American Board of Obesity Medicine, dedicate a portion or all of their practice to the treatment of obesity. They perform a medical evaluation (history, physical, laboratory, body composition) and provide medical supervision for lifestyle change (nutrition, activity, behavior change), medications, or very low calorie diets. Obesity is a chronic medical disease, and may require lifelong treatment.

ASBP Obesity Algorithm: Executive Summary

Obesity Medicine Specialist Evaluation may include:

History

Weight history, past medical history, family history, social history, screening for weight promoting medications, food intake, activity, review of systems

Physical Examination

Height, weight, blood pressure, body composition analysis, waist measurement, complete physical examination

Laboratory Tests

Complete blood count, electrolytes, liver function, kidney function, fasting lipid profile, thyroid tests, hemoglobin A1c, uric acid, vitamin D

Diagnostic Testing

EKG, Echocardiogram, exercise stress test, sleep study, barium swallow or esophagoduodenoscopy

Individualized Treatment Plan may include:

Diet

Caloric restriction, carbohydrate restriction, food journaling
Very Low Calorie Diet (VLCD) programs

Activity

Give exercise prescription, use pedometers, limit TV and computer time, goal of 150 minutes/week of moderate intensity physical activity

Counseling

Eliminate provider bias/stigma, identify self-sabotage, develop strong support, stress management, sleep optimization, other psychological support

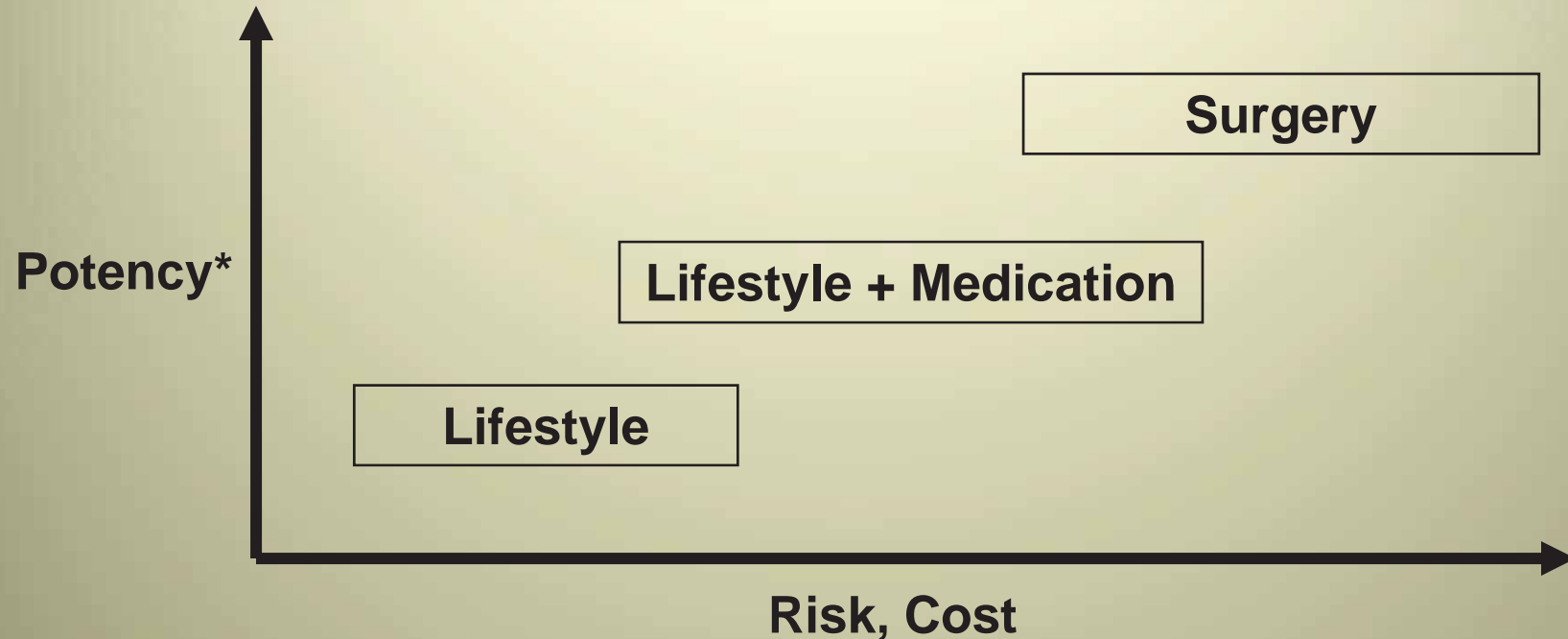
Pharmacotherapy

Use medications as part of a comprehensive program

If ineffective, consider referral to a Metabolic and Bariatric Surgeon.

Optimal pre- and post-operative bariatric surgery care includes an Obesity Medicine Specialist.

Current Treatment Options for Obesity



Lifestyle: Includes nutrition, exercise, behavioral programs

Lifestyle + Medication: May include Lifestyle, VLCDs w supplements, and weight loss medications

Surgery: (in order of lowest risk/cost and potency): Gastric Banding < Gastric Sleeve < Gastric Bypass (Roux-en-Y)

* Potency includes many factors such as the amount, rate and sustainability of weight loss, and long-term resolution of adiposopathy and fat mass disease. Potency varies greatly for each individual: long-term adherence to a lifestyle program can be as potent as gastric bypass surgery.

References

Internet Website References:

- [200] Seger JC, Horn DB, Westman EC, Lindquist R, Scinta W, Richardson LA, Primack C, Bryman DA, McCarthy W, Hendricks E, Sabowitz BN, Schmidt S, Bays HE. American Society of Bariatric Physician Obesity Algorithm: Adult Adiposity Evaluation and Treatment 2013. www.obesityalgorithm.org
- [201] http://www.nhlbi.nih.gov/guidelines/obesity/prctgd_c.pdf National Heart, Lung, And Blood Institute, North American Association For The Study Of Obesity: The Practical Guide, Identification, Evaluation, And Treatment Of Overweight And Obesity In Adults. (Accessed July 26, 2013)
- [202] http://www.acefitness.org/acefit/healthy_living_tools_content.aspx?id=2 American Counsel on Exercise Percent Body Fat (Accessed July 1, 2013)
- [203] http://www.idf.org/webdata/docs/MetSyndrome_FINAL.pdf The International Diabetes Federation consensus worldwide definition of the metabolic syndrome. (Accessed July 27, 2013)
- [204] <http://dictionary.reference.com> Dictionary.com – Definition of Disease. (Accessed July 1, 2013)
- [205] https://www.acponline.org/eBizATPRO/images/ProductImages/books/sample%20chapters/Obesity_Ch05.pdf. Mastbaum LI, Gumbiner B. Chapter 5: Medical Assessment and Treatment of the Obese Patient. (Accessed July 26, 2013)

References

Internet Website References (continued):

- [206] <http://www.acsm.org/docs/brochures/pre-participation-physical-examinations.pdf> American College of Sports Medicine Information on Pre-Participation Physical Examinations
- [207] <http://www.health.gov/paguidelines/guidelines/pdf/paguide.pdf> US Department of Health and Human Services. 2008 Physical Activity Guidelines for Americans (Accessed August 20, 2013)
- [208] http://www.belviq.com/pdf/Belviq_Prescribing_information.pdf Belviq Prescribing Information (Accessed July 28, 2013)
- [209] <http://www.vivus.com/docs/QsymiaPI.pdf> Qsymia Prescribing Information (Accessed July 28, 2013)
- [210] http://care.diabetesjournals.org/content/36/Supplement_1 American Diabetes Association Clinical Practice Guidelines 2013 (Accessed July 28, 2013)
- [211] <http://www.nhlbi.nih.gov/about/ncep/> National Heart and Lung Blood Institute, National Cholesterol Education Program (Accessed July 28, 2013)
- [212] <http://www.nhlbi.nih.gov/guidelines/hypertension/> Blood Pressure in Adults: Systematic Evidence Review from the Joint National Committee (JNC) (Accessed July 28, 2013)

References

Journal References:

1. Carroll JF, Chiapa AL, Rodriquez M, Phelps DR, Cardarelli KM, Vishwanatha JK, Bae S, Cardarelli R: Visceral fat, waist circumference, and BMI: impact of race/ethnicity. *Obesity (Silver Spring)* 2008 16:600-607.
2. Wang Z, Ma J, Si D: Optimal cut-off values and population means of waist circumference in different populations. *Nutr Res Rev* 2010 23:191-199.
3. Kushner RF, Blatner DJ: Risk assessment of the overweight and obese patient. *J Am Diet Assoc* 2005 105:S53-62.
4. Allende-Vigo MZ: Pathophysiologic mechanisms linking adipose tissue and cardiometabolic risk. *Endocr Pract* 2010 16:692-698.
5. Bays H, Abate N, Chandalia M: Adiposopathy: sick fat causes high blood sugar, high blood pressure and dyslipidemia. *Future Cardiol* 2005 1:39-59.
6. Bays HE, Gonzalez-Campoy JM, Henry RR, Bergman DA, Kitabchi AE, Schorr AB, Rodbard HW: Is adiposopathy (sick fat) an endocrine disease? *Int J Clin Pract* 2008 62:1474-1483.
7. Bays HE: "Sick fat," metabolic disease, and atherosclerosis. *Am J Med* 2009 122:S26-37.
8. Bays HE, LaFerrere B, Dixon J, Aronne L, Gonzalez-Campoy JM, Apovian C, Wolfe BM: Adiposopathy and bariatric surgery: is 'sick fat' a surgical disease? *Int J Clin Pract* 2009 63:1285-1300.
9. Bays HE: Adiposopathy is "sick fat" a cardiovascular disease? *J Am Coll Cardiol* 2011 57:2461-2473.
10. Bays HE: Adiposopathy, diabetes mellitus, and primary prevention of atherosclerotic coronary artery disease: treating "sick fat" through improving fat function with antidiabetes therapies. *Am J Cardiol* 2012 110:4B-12B.
11. Vallis M, Piccinini-Vallis H, Sharma AM, Freedhoff Y: Clinical review: modified 5 As: minimal intervention for obesity counseling in primary care. *Can Fam Physician* 2013 59:27-31.
12. Alexander SC, Cox ME, Boling Turer CL, Lyna P, Ostbye T, Tulskey JA, Dolor RJ, Pollak KI: Do the five A's work when physicians counsel about weight loss? *Fam Med* 2011 43:179-184.

References

Journal References (continued):

13. Kushner RF, Roth JL: Assessment of the obese patient. *Endocrinol Metab Clin North Am* 2003 32:915-933.
14. Bays HE: Current and investigational antiobesity agents and obesity therapeutic treatment targets. *Obes Res* 2004 12:1197-1211.
15. Bays HE, Toth PP, Kris-Etherton PM, Abate N, Aronne LJ, Brown WV, Gonzalez-Campoy JM, Jones SR, Kumar R, La Forge R, Samuel VT: Obesity, adiposity, and dyslipidemia: A consensus statement from the National Lipid Association. *J Clin Lipidol* 2013 7:304-383.
16. Lim SS, Norman RJ, Davies MJ, Moran LJ: The effect of obesity on polycystic ovary syndrome: a systematic review and meta-analysis. *Obes Rev* 2013 14:95-109.
17. Bays HE, Gonzalez-Campoy JM, Schorr AB: What men should know about metabolic syndrome, adiposopathy and 'sick fat'. *Int J Clin Pract* 2010 64:1735-1739.
18. Hursting SD, Dunlap SM: Obesity, metabolic dysregulation, and cancer: a growing concern and an inflammatory (and microenvironmental) issue. *Ann N Y Acad Sci* 2012 1271:82-87.
19. Bays HE, Gonzalez-Campoy JM, Bray GA, Kitabchi AE, Bergman DA, Schorr AB, Rodbard HW, Henry RR: Pathogenic potential of adipose tissue and metabolic consequences of adipocyte hypertrophy and increased visceral adiposity. *Expert Rev Cardiovasc Ther* 2008 6:343-368.
20. Lam YY, Mitchell AJ, Holmes AJ, Denyer GS, Gummesson A, Caterson ID, Hunt NH, Storlien LH: Role of the gut in visceral fat inflammation and metabolic disorders. *Obesity (Silver Spring)* 2011 19:2113-2120.
21. Wells JC: Ethnic variability in adiposity, thrifty phenotypes and cardiometabolic risk: addressing the full range of ethnicity, including those of mixed ethnicity. *Obes Rev* 2012 13 Suppl 2:14-29.
22. Cuypers KF, Loos RJ, Kvaloy K, Kulle B, Romundstad P, Holmen TL: Obesity-susceptibility loci and their influence on adiposity-related traits in transition from adolescence to adulthood--the HUNT study. *PLoS One* 2012 7:e46912.
23. Wilson BJ, Carroll JC, Allanson J, Little J, Etchegary H, Avard D, Potter BK, Castle D, Grimshaw JM, Chakraborty P: Family history tools in primary care: does one size fit all? *Public Health Genomics* 2012 15:181-188.
24. Wells JC: The evolution of human adiposity and obesity: where did it all go wrong? *Dis Model Mech* 2012 5:595-607.

References

Journal References (continued):

25. Kanter R, Caballero B: Global gender disparities in obesity: a review. *Adv Nutr* 2012 3:491-498.
26. Jaworowska A, Blackham T, Davies IG, Stevenson L: Nutritional challenges and health implications of takeaway and fast food. *Nutr Rev* 2013 71:310-318.
27. Moore CJ, Cunningham SA: Social position, psychological stress, and obesity: a systematic review. *J Acad Nutr Diet* 2012 112:518-526.
28. Beechy L, Galpern J, Petrone A, Das SK: Assessment tools in obesity - psychological measures, diet, activity, and body composition. *Physiol Behav* 2012 107:154-171.
29. Horn DB, O'Neill JR, Pfeiffer KA, Dowda M, Pate RR: Predictors of physical activity in the transition after high school among young women. *J Phys Act Health* 2008 5:275-285.
30. Vanhees L, De Sutter J, Gelada SN, Doyle F, Prescott E, Cornelissen V, Kouidi E, Dugmore D, Vanuzzo D, Borjesson M, Doherty P: Importance of characteristics and modalities of physical activity and exercise in defining the benefits to cardiovascular health within the general population: recommendations from the EACPR (Part I). *Eur J Prev Cardiol* 2012 19:670-686.
31. Vanhees L, Geladas N, Hansen D, Kouidi E, Niebauer J, Reiner Z, Cornelissen V, Adamopoulos S, Prescott E, Borjesson M, Bjarnason-Wehrens B, Bjornstad HH, Cohen-Solal A, Conraads V, Corrado D, De Sutter J, Doherty P, Doyle F, Dugmore D, Ellingsen O, Fagard R, Giada F, Gielen S, Hager A, Halle M, Heidbuchel H, Jegier A, Mazic S, McGee H, Mellwig KP, Mendes M, Mezzani A, Pattyn N, Pelliccia A, Piepoli M, Rauch B, Schmidt-Trucksass A, Takken T, van Buuren F, Vanuzzo D: Importance of characteristics and modalities of physical activity and exercise in the management of cardiovascular health in individuals with cardiovascular risk factors: recommendations from the EACPR. Part II. *Eur J Prev Cardiol* 2012 19:1005-1033.
32. Steelman GM, Westman EC: *Obesity: Evaluation and Treatment Essentials*. New York: Informa Healthcare 2010

References

Journal References (continued):

33. Beam JR, Szymanski DJ: Validity of 2 skinfold calipers in estimating percent body fat of college-aged men and women. *J Strength Cond Res* 2010 24:3448-3456.
34. Muller MJ, Bosy-Westphal A, Lagerpusch M, Heymsfield SB: Use of balance methods for assessment of short-term changes in body composition. *Obesity (Silver Spring)* 2012 20:701-707.
35. Ginde SR, Geliebter A, Rubiano F, Silva AM, Wang J, Heshka S, Heymsfield SB: Air displacement plethysmography: validation in overweight and obese subjects. *Obes Res* 2005 13:1232-1237.
36. Corona G, Rastrelli G, Monami M, Saad F, Luconi M, Lucchese M, Facchiano E, Sforza A, Forti G, Mannucci E, Maggi M: Body weight loss reverts obesity-associated hypogonadotropic hypogonadism: a systematic review and meta-analysis. *Eur J Endocrinol* 2013 168:829-843.
37. Shen J, Obin MS, Zhao L: The gut microbiota, obesity and insulin resistance. *Mol Aspects Med* 2013 34:39-58.
38. Dhurandhar NV: A framework for identification of infections that contribute to human obesity. *Lancet Infect Dis* 2011 11:963-969.
39. Hochberg I, Hochberg Z: Expanding the definition of hypothalamic obesity. *Obes Rev* 2010 11:709-721.
40. Allison KC, Grilo CM, Masheb RM, Stunkard AJ: High self-reported rates of neglect and emotional abuse, by persons with binge eating disorder and night eating syndrome. *Behav Res Ther* 2007 45:2874-2883.
41. Thaler JP, Guyenet SJ, Dorfman MD, Wisse BE, Schwartz MW: Hypothalamic inflammation: marker or mechanism of obesity pathogenesis? *Diabetes* 2013 62:2629-2634.
42. St-Onge MP: The role of sleep duration in the regulation of energy balance: effects on energy intakes and expenditure. *J Clin Sleep Med* 2013 9:73-80.
43. Hasnain M, Vieweg WV, Hollett B: Weight gain and glucose dysregulation with second-generation antipsychotics and antidepressants: a review for primary care physicians. *Postgrad Med* 2012 124:154-167.

References

Journal References (continued):

44. Astrup A, Caterson I, Zelissen P, Guy-Grand B, Carruba M, Levy B, Sun X, Fitchet M: Topiramate: long-term maintenance of weight loss induced by a low-calorie diet in obese subjects. *Obes Res* 2004 12:1658-1669.
45. Astrup A, Carraro R, Finer N, Harper A, Kunesova M, Lean ME, Niskanen L, Rasmussen MF, Rissanen A, Rossner S, Savolainen MJ, Van Gaal L: Safety, tolerability and sustained weight loss over 2 years with the once-daily human GLP-1 analog, liraglutide. *Int J Obes (Lond)* 2012 36:843-854.
46. Bays H: From victim to ally: the kidney as an emerging target for the treatment of diabetes mellitus. *Curr Med Res Opin* 2009 25:671-681.
47. Gonzalez-Campoy JM, St. Jeor ST, Castorino K, Ebrahim A, Hurley D, Jovanovic L, Mechanick JI, Petak SM, Yu YH, Harris KA, Kris-Etherton P, Kushner R, Molini-Blandford M, Nguyen QT, Plodkowski R, Sarwer DB, Thomas KT: Clinical Practice Guidelines for healthy eating for the prevention and treatment of metabolic and endocrine diseases in adults: Cosponsored by American Association of Clinical Endocrinologists and The Obesity Society: American Association of Clinical Endocrinologists Task force on Healthy Eating Clinical Practice Guideline. *Endocr Pract* 2013 (In Press)
48. Clifton PM: Dietary treatment for obesity. *Nat Clin Pract Gastroenterol Hepatol* 2008 5:672-681.
49. Brown T, Avenell A, Edmunds LD, Moore H, Whittaker V, Avery L, Summerbell C: Systematic review of long-term lifestyle interventions to prevent weight gain and morbidity in adults. *Obes Rev* 2009 10:627-638.
50. Tsai AG, Wadden TA: Systematic review: an evaluation of major commercial weight loss programs in the United States. *Ann Intern Med* 2005 142:56-66.
51. Westman EC, Feinman RD, Mavropoulos JC, Vernon MC, Volek JS, Wortman JA, Yancy WS, Phinney SD: Low-carbohydrate nutrition and metabolism. *Am J Clin Nutr* 2007 86:276-284.
52. Yancy WS, Jr., Olsen MK, Guyton JR, Bakst RP, Westman EC: A low-carbohydrate, ketogenic diet versus a low-fat diet to treat obesity and hyperlipidemia: a randomized, controlled trial. *Ann Intern Med* 2004 140:769-777.

References

Journal References (continued):

53. Volek JS, Phinney SD, Forsythe CE, Quann EE, Wood RJ, Puglisi MJ, Kraemer WJ, Bibus DM, Fernandez ML, Feinman RD: Carbohydrate restriction has a more favorable impact on the metabolic syndrome than a low fat diet. *Lipids* 2009 44:297-309.
54. Foster GD, Wyatt HR, Hill JO, Makris AP, Rosenbaum DL, Brill C, Stein RI, Mohammed BS, Miller B, Rader DJ, Zemel B, Wadden TA, Tenhave T, Newcomb CW, Klein S: Weight and metabolic outcomes after 2 years on a low-carbohydrate versus low-fat diet: a randomized trial. *Ann Intern Med* 2010 153:147-157.
55. Shai I, Spence JD, Schwarzfuchs D, Henkin Y, Parraga G, Rudich A, Fenster A, Mallett C, Liel-Cohen N, Tirosh A, Bolotin A, Thiery J, Fiedler GM, Bluher M, Stumvoll M, Stampfer MJ: Dietary intervention to reverse carotid atherosclerosis. *Circulation* 2010 121:1200-1208.
56. Tirosh A, Golan R, Harman-Boehm I, Henkin Y, Schwarzfuchs D, Rudich A, Kovsan J, Fiedler GM, Bluher M, Stumvoll M, Thiery J, Stampfer MJ, Shai I: Renal function following three distinct weight loss dietary strategies during 2 years of a randomized controlled trial. *Diabetes Care* 2013 36:2225-2232.
57. Westman EC, Yancy WS, Jr., Mavropoulos JC, Marquart M, McDuffie JR: The effect of a low-carbohydrate, ketogenic diet versus a low-glycemic index diet on glycemic control in type 2 diabetes mellitus. *Nutr Metab (Lond)* 2008 5:36.
58. Lutas A, Yellen G: The ketogenic diet: metabolic influences on brain excitability and epilepsy. *Trends Neurosci* 2013 36:32-40.
59. Mobbs CV, Mastaitis J, Isoda F, Poplawski M: Treatment of diabetes and diabetic complications with a ketogenic diet. *J Child Neurol* 2013 28:1009-1014.
60. Schwingshackl L, Hoffmann G: Long-term effects of low-fat diets either low or high in protein on cardiovascular and metabolic risk factors: a systematic review and meta-analysis. *Nutr J* 2013 12:48.
61. Meckling KA, O'Sullivan C, Saari D: Comparison of a low-fat diet to a low-carbohydrate diet on weight loss, body composition, and risk factors for diabetes and cardiovascular disease in free-living, overweight men and women. *J Clin Endocrinol Metab* 2004 89:2717-2723.
62. Mulholland Y, Nicokavoura E, Broom J, Rolland C: Very-low-energy diets and morbidity: a systematic review of longer-term evidence. *Br J Nutr* 2012 108:832-851.

References

Journal References (continued):

63. Johansson K, Sundstrom J, Marcus C, Hemmingsson E, Neovius M: Risk of symptomatic gallstones and cholecystectomy after a very-low-calorie diet or low-calorie diet in a commercial weight loss program: 1-year matched cohort study. *Int J Obes (Lond)* 2013 May 22 [Epub ahead of print]
64. Warburton DE, Nicol CW, Bredin SS: Health benefits of physical activity: the evidence. *CMAJ* 2006 174:801-809.
65. Jakicic JM, Davis KK: Obesity and physical activity. *Psychiatr Clin North Am* 2011 34:829-840.
66. Standford KI, Middelbeek R, Townsend KL, Lee MY, Ding AN, Markan KR, Hellbach K, YFanti C, Nielsen S, Akerstrom T, Hirshman MF, Pedersen BK, Tseng YH, LJ G: Exercise Training Alters Subcutaneous White Adipose Tissue (scWAT) in Mice and Humans. American Diabetes Association President's Oral Session; 73 Scientific Sessions American Diabetes Association. June 21, 2013. Chicago Illinois USA 2013 17-OR/0017:
67. Meriwether RA, Lee JA, Lafleur AS, Wiseman P: Physical activity counseling. *Am Fam Physician* 2008 77:1129-1136.
68. Vincent HK, Raiser SN, Vincent KR: The aging musculoskeletal system and obesity-related considerations with exercise. *Ageing Res Rev* 2012 11:361-373.
69. Parr EB, Coffey VG, Hawley JA: 'Sarcobesity': a metabolic conundrum. *Maturitas* 2013 74:109-113.
70. Strasser B: Physical activity in obesity and metabolic syndrome. *Ann N Y Acad Sci* 2013 1281:141-159.
71. Garland T, Jr., Schutz H, Chappell MA, Keeney BK, Meek TH, Copes LE, Acosta W, Drenowatz C, Maciel RC, van Dijk G, Kotz CM, Eisenmann JC: The biological control of voluntary exercise, spontaneous physical activity and daily energy expenditure in relation to obesity: human and rodent perspectives. *J Exp Biol* 2011 214:206-229.
72. Ng SW, Popkin BM: Time use and physical activity: a shift away from movement across the globe. *Obes Rev* 2012 13:659-680.
73. Van Camp CM, Hayes LB: Assessing and increasing physical activity. *J Appl Behav Anal* 2012 45:871-875.
74. Butte NF, Ekelund U, Westerterp KR: Assessing physical activity using wearable monitors: measures of physical activity. *Med Sci Sports Exerc* 2012 44:S5-12.
75. Richardson LA: Bariatric society is here to help. *J Fam Pract* 2010 59:488.
76. Jacob JJ, Isaac R: Behavioral therapy for management of obesity. *Indian J Endocrinol Metab* 2012 16:28-32.

References

Journal References (continued):

77. Van Dorsten B, Lindley EM: Cognitive and behavioral approaches in the treatment of obesity. *Med Clin North Am* 2011 95:971-988.
78. Karasu SR: Psychotherapy-lite: obesity and the role of the mental health practitioner. *Am J Psychother* 2013 67:3-22.
79. Rutledge T, Groesz LM, Linke SE, Woods G, Herbst KL: Behavioural weight management for the primary care provider. *Obes Rev* 2011 12:e290-297.
80. Jeffery RW, Bjornson-Benson WM, Rosenthal BS, Lindquist RA, Johnson SL: Behavioral treatment of obesity with monetary contracting: two-year follow-up. *Addict Behav* 1984 9:311-313.
81. Brambila-Macias J, Shankar B, Capacci S, Mazzocchi M, Perez-Cueto FJ, Verbeke W, Traill WB: Policy interventions to promote healthy eating: a review of what works, what does not, and what is promising. *Food Nutr Bull* 2011 32:365-375.
82. Bray GA: Why do we need drugs to treat the patient with obesity? *Obesity (Silver Spring)* 2013 21:893-899.
83. Varady KA, Tussing L, Bhutani S, Braunschweig CL: Degree of weight loss required to improve adipokine concentrations and decrease fat cell size in severely obese women. *Metabolism* 2009 58:1096-1101.
84. Ratner RE: An update on the Diabetes Prevention Program. *Endocr Pract* 2006 12 Suppl 1:20-24.
85. Hendricks EJ, Greenway FL, Westman EC, Gupta AK: Blood pressure and heart rate effects, weight loss and maintenance during long-term phentermine pharmacotherapy for obesity. *Obesity (Silver Spring)* 2011 19:2351-2360.
86. Cercato C, Roizenblatt VA, Leanca CC, Segal A, Lopes Filho AP, Mancini MC, Halpern A: A randomized double-blind placebo-controlled study of the long-term efficacy and safety of diethylpropion in the treatment of obese subjects. *Int J Obes (Lond)* 2009 33:857-865.
87. Le Riche WH, Van Belle G: Study of phendimetrazine bitartrate as an appetite suppressant in relation to dosage, weight loss and side effects. *Can Med Assoc J* 1962 87:29-31.
88. Bays HE: Lorcaserin and adiposopathy: 5-HT_{2c} agonism as a treatment for 'sick fat' and metabolic disease. *Expert Rev Cardiovasc Ther* 2009 7:1429-1445.

References

Journal References (continued):

89. Bays HE: Lorcaserin: drug profile and illustrative model of the regulatory challenges of weight-loss drug development. *Expert Rev Cardiovasc Ther* 2011 9:265-277.
90. Bays H: Phentermine, topiramate and their combination for the treatment of adiposopathy ('sick fat') and metabolic disease. *Expert Rev Cardiovasc Ther* 2010 8:1777-1801.
91. Bays HE, Gadde KM: Phentermine/topiramate for weight reduction and treatment of adverse metabolic consequences in obesity. *Drugs Today (Barc)* 2011 47:903-914.
92. Landsberg L, Aronne LJ, Beilin LJ, Burke V, Igel LI, Lloyd-Jones D, Sowers J: Obesity-related hypertension: pathogenesis, cardiovascular risk, and treatment--a position paper of the The Obesity Society and The American Society of Hypertension. *Obesity (Silver Spring)* 2013 21:8-24.
93. Neff KJ, Olbers T, le Roux CW: Bariatric surgery: the challenges with candidate selection, individualizing treatment and clinical outcomes. *BMC Med* 2013 11:8.
94. Dixon JB: Referral for a bariatric surgical consultation: it is time to set a standard of care. *Obes Surg* 2009 19:641-644.
95. Mechanick JI, Youdim A, Jones DB, Garvey WT, Hurley DL, McMahon MM, Heinberg LJ, Kushner R, Adams TD, Shikora S, Dixon JB, Brethauer S: Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient--2013 update: cosponsored by American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery. *Obesity (Silver Spring)* 2013 21 Suppl 1:S1-27.
96. Appachi S, Kashyap SR: 'Adiposopathy' and cardiovascular disease: the benefits of bariatric surgery. *Curr Opin Cardiol* 2013 28:540-546.
97. Folli F, Sabowitz BN, Schwesinger W, Fanti P, Guardado-Mendoza R, Muscogiuri G: Bariatric surgery and bone disease: from clinical perspective to molecular insights. *Int J Obes (Lond)* 2012 36:1373-1379.

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