**Anemia**
Is a condition of reduced hemoglobin (there are less red blood cells than normal)
Etiology: depressed or defective RBC production resulting from disturbed marrow function (aplastic anemia or due to infection, malignancy, collagen disease, renal failure, or liver disease), a substrate deficiency (iron, Vit B12, folate, copper, Vit C, EFA’s and protein)
Signs and Symptoms: weakness, fatigue, light-headedness, breathlessness, palpitations, angina, headache, rapid pulse, pale skin, mucous membranes or nailbeds, sleep distruptions including daytime sleepiness, shallow sleep patterns with frequent waking.
Pathophysiology: low red blood cell count, reduction in normal hemoglobin, abnormality in the maturation and morphology of the RBC
Types of anemia:
1) *Microcytic Hypochromic Anemia* (iron deficiency anemia)- low iron in the diet, poor uptake of iron in the gut (from hypochlorhydria), oxylates and phytates in the diet also cause poor uptake of iron, excessive blood loss, can occur in cases of chronic infection as the body sequesters iron away from microbes (microbes need iron to grow) and so there is an inability to mobalize stores of iron for hemoglobin production and normal cell turnover, due to chemo or chemicals generated by cancer (never give a person with cancer an iron supplement as it can feed the tumour
\*heme iron (animal source) is the most absorbable form
\*ferrous sulphate (plant source) can be poorly absorbed
2) *Macrocytic Normochromic (*megaloblastic*) Anemia* (low levels of folic acid or vitamin B12) – affects sensory nerve function, associated with hypochlorydia and common in older individuals and vegans are a higher risk group
\* eating unwashed organic vegetables provide enough soil based organisms to provide B12
\* never take folic acid alone because it will correct the RBC, but the B12 deficiency will continue leading to neurological effects
\* Perinicious anemia is a specific form of megaloblastic anemia caused by an autoimmune disease of the stomach resulting in the inability to produce intrinsic factor
3) *Normocytic Normochromic Anemia* (hemolytic anemia)- hemolysis or rupture of RBC cells due to intrinsic defect, malaria or due to the body making antibodies against the cells and destroying them
\*Sickle cell anemia (may be an evolutionary protective factor against malaria) where people are predisposed to B12 deficiency: also improved by foods rich in thiocyanate: African yams, lima beans, steamed carrots, cabbage, alfalfa sprouts, fax seed, millet, sorghum.
\*Thalassemia- abnormalities in the hemoglobin caused by genetic alterations: presents with chronic anemia, an enlarged liver and spleen, hyperplasia of bone marrow, thinning of the bones and increased iron stores which can damage the liver
Recommendations: Text p.343-344

**Hypertension**
Excessive blood pressure above 140/90 mm Hg
Etiology:
*Primary Hypertension*: unknown, inherited heightened sensitivity to sympathetic nervous stimuli (any outflow of epinephrine causes the cardiovascular system to react with higher pressures than normal), sodium ingestion, emotional stress and obesity seem to aggravate the condition (on the other hand over restricting sodium will cause hypertension through the release of aldosterone which stimulates the kidneys to conserve sodium causing water reabsorption and retention.
*Secondary Hypertention* (caused by conditions outside of the cardiovascular system): renal disease, pheochromocytoma (tumor that secretes catecholamines), Cushing’s disease, primary aldosteronism, hyperthyroidism, diabetes, anatomical and vavular heart problems.
Signs and Symptoms: |
*Chronic Benign* (most common form): symptomless, palpitations, audible pulsation in the head, headaches, attacks of dizziness, death can also occur due to left ventricle failure or stroke.
*Malignant or Accelerated* (can cause heart failure, stroke, and retinal eye damage): edema, papilledema (swelling of the optic disc or nerve), hemorrhages, infarcts and exudates potentially resulting in blindness, epileptic-like fits and transient paralysisdue to cerebral edema
Pathophysiology: Being overworked, the heart muscle enlarges and the bulging ventricular muscles can cut off blood flow through the coronary arteries when the muscle needs oxygen and nutrients. High blood pressure very is associated with coronary arteries that are full of atheromatous plaques, can lead to angina, infarction (damage and death to the muscle fibers) and eventual failure of the heart. Congestive heart failure is when both the peripheral tissues of the lungs become full of fluids (happens when the pumping action of the heart is so compromised that a blood traffic jam occurs in both the vessels returning to the heart from the body and in those returning from the lungs that the pressure builds up in these vessels to the point that the fluid begins to leak out of them into the tissues (commonly presents in individuals having thick ankles, lower legs and productive coughing). Can also cause kidney disease, eye disease and microaneurysms in the brain (weakening in the vessel walls that can rupture and hemorrhage resulting in stroke. Causes mechanical damage to the arterial endothelial linings with lead to atheromatous plaques (these deposits together with smooth muscle hypertrophy and increasing sclerosis (hardening) of the vessel walls, increase the resistance making hypertension worse (viscious cycle)
Recommendations: Text p. 347-350

**Hypotension**Decrease in blood pressure below normal
Etiology: adrenal exhaustion, anemia, blood loss, dehydration, anaphalaxsis, diabetes, Parkinson’s, hypothyroidism, side effects of medication (diuretics, MAOIs, narcotic analgesics, tranquillizers, higher than needed doses of antihypertensive meds, excessive thinning of the blood from taking NSAIDS (eg. Aspirin) over a long period of time or from taking too much Vit. E.
Signs and Symptoms: low blood pressure, fall in blood pressure when one moves from a lying position to a standing one (plus being lightheaded when adrenal related).
Pathophysiology: In low blood pressure the baroreceptors that respond to gravity induced pressure changes work abnormally causing blood pressure to drop when one moves from a lying position to a standing one .Normally pooling of the blood in the lower part of the body is prevented by a surge in epinephrine in response to baroreceptor-mediated regulation. The epinephrine causes vascoconstriction that efficently returns blood to the heart.
Recommendations: Text p. 351

**Vericose Veins**
Enlarged and dilated superficial veins.
Etiology: poor nutrition, lack of exercise, obesity, pregnancy (uterus puts pressure on the vessels), immobalization, post perative or post partum complications, heart problems, exogenous estrogen therapy (HRT and the pill)
Signs and Symptoms: sometimes painful, ballooned, blue veins under the skin that may feel warm to the touch
Pathophysiology: vessel wall weakness casing stagnation, stasis in the veins can lead to clotting, thrombus (inflamed vein) and with the skin over the veins getting less nutrition ulcers can form
Recommendations: Text p. 352-353

**Atherosclerosis, Hyperlipidemia & Coronary Artery Disease**
A form of arteriosclerosis in which atheromas containing cholesterol, lipid material blood cells, fibrin, smooth muscle and calcium are formed within the degenerated and thickened intima and inner media (middle layer) of large and medium-sized arteries. Atheromas affect the compliance of the vessel which eventually become sclerotic or brittle.
Etiology: occurs in people as they grow older
Theory 1: Damage-Free radical damage, homocysteine or an infection like Chlamydia.
\*Reduce free radical exposure, increasing antioxidant intake, reducing blood pressure, convert homocysteine to cysteine using Vit B6, folic acid and by building up the immune system for prevention
Theory 2: Oxidized ( and damaged) cholesterol is a toxic excess that disintegrates into foam cells which appear as the artheroma found in the vessel filled with cholesterol.
\* Vitamin C is especially important in preventing the development of the foam cells that make up the artheroma.
Theory 3: Smooth muscles start to mutate due to free radical damage.
\* Decrease free radical exposure, increase antioxidants and strengthen the immune system so that abnormal smooth cells are destroyed.
Theory 4: Insufficient Vitamin C resulting in sticky lipoproteins which are more likely to deposit out to damaged vessels.
\* some say atherosclerosis is a form of scurvy because Vit. C keeps the vessels stable and resilient.
\* increase Vit. C to bowel tolerance while increasing lysine in the circulation so that they lipoprotein sticks to it rather than the lysine in the wall.
Signs and Symptoms: symptoms are not apparent until there is a 70% blockage of the coronaries
*Early warning symptoms:* numbness or heaviness in the arms and legs, cramps, tingling of fingers or lips, cramps in the calves when walking, swollen ankles late in the day, a dry persistent cough, breathlessness from mild exertion, constant urination at night, memory loss and chest pains on exertion, loss of pulses-especially behind the knee, on the inner aspect of the ankle and on top of the foot.
*Symptoms of heart attack:* loss of heart function, it only takes four minutes for the heart and brain tissue to start to die. Presents with a crushing or compressing pain in the chest. The pain extends down the left arm and up the left side of the neck (these symptoms are more common in men, while women tend to experience less specific pain in the epigastrium and chest.
Pathophysiology: the distribution of atheromas is patchy and the development occurs slowly over the years. The thickening causes narrowing of the lumen, decrease in vessel compliance and slow degradation of that tissue. A thrombus or embolus can also develop on top of the atheroma and completely block the passages to the heart or the brain (causing a stroke). Aneurysms can occur anywhere.
How to prevent artherosclerosis: see stars above and reduce free radicals:
*External Sources of Free Radicals:* polyunsaturated fatty acids that have been exposed to heat, light, oxygen, rancid oils, hydrogenated fats, petrochemicals found in food colouring for example, preservatives, exhaust fumes, cleaning fluids, alcohol, tobacco tar, chlorinated drinking water, heavy metals, radiation and refined sugar.
*Internal Sources of Free Radicals:* sluggish liver, constipation, excessive athleticism, stress, dysbiosis
Read about cholesterol in text on p. 357-359 and take your own notes on this as you will need to educate your clients on this topic over and over.
Recommendations: Text p. 360-364

**Signs and Symptoms of a poorly functioning liver**
Text p.220

**Liver Detoxification Mechanism**
Phase 1: Cytochrome P450 System
Nutrients that support Phase 1 Detox:
alpha-ketoglutaric acid, choline, EFA’s, lecithin, methionine, copper, iron, manganese, molybdenum, sulphur, magnesium, zinc, B-complex, Vit. A, E, C, bioflavanoidsand folic acid
Inhibited by: Prozac, benzodiazepines, antihistamines, cimetidine and naringenin (from grapefruit juice)
\* If this system is overloaded an amino acid is created called amino levulinic acid (ALA) which turns on the area of the brain that results in aggression and hostility.
Phase 2: Antioxidant System
Nutrients that support Phase 2 Detox:
cysteine, garlic, glucarate, glycine, glutathione, N-acetyl cysteine (NAC), taurine, high quality protein, folic acid, vit B complex, germanium, magnesium, manganese, molybdenum, selenium, sulphur, zinc, cruciferous vegetables,
\* People with poor detoxification systems tend to be more environmentally sensitive, sensitive to alcohol, have more allergies and tend to get sick more often.

**What compromises the liver**
Text p. 222-223

**Recommendations for optimizing liver function**
p.223- 225

**Hepatitis**
Inflammation of the liver.
Etiology: virus, bacteria, fungus, protozoa, a fluke, drugs, alcohol
Signs and Symptoms: jaundice, nausea, vomiting, intolerance of fatty food, enlarged tender liver and a fever. Urine is dark, stool is light.
Pathophysiology: There is diffuse liver cell destruction and demonstrationable dysfunction. The bile canaliculi (passages) are often obstructed, so that the outflow of bile is impeded and there is cholestasis (bile stasis) with jaundice.
Recommendations: Text p. 229-230

**Cirrhosis**
Cirrhosis is a late and irreversable stage of chronic hepatitis with liver scarring of the normal hepatic architecture and areas of ineffective regeneration.
Etiology: alcohol abuse, chemical damage, Hep B or C infection, congenital syphilis and biliary obstruction
Signs and Symptoms: fatigue, malaise, loss of appetite, early satiety, dyspepsia, altered bowel habits, easy bruising and bleeding, varying alterations in mental status, personality and behaviour may be noticed, itching can accompany the jaundice.
Pathophysiology: The liver architecture is no longer normal , so that the flow of blood and bile are distorted and often blocked, leading to obstructive jaundice and portal hypertension. Death as a result.
Recommendations: Text. 231-232

**Cholelithiasis (gallstones)**Presence or formation stones in the gall bladder
Etiology: imbalance in the composition of bile, cholesterol is high and lecithin (needed to break it down) and bile salts are low, stones develop especially where there is stasis in the bladder and low water intake which is the vital solvent of bile. 85% of gallstones are made predominantly of cholesterol and some calcium (found most frequently in women who are fair, fat, forty, fertile and flatulent). Deficiency of Vit. C seems to be associated with the development of gallstones as well as anything than increases cholesterol levels will increase the risk of gallstones. See text p. 233
Signs and Symptoms: cause abdominal discomfort, bloating, belching, and intolerance to many foods, especially fatty ones
Pathophysiology: if a large stone is released from the gallbladder into the bile duct, pain occurs and bile backs up causing inflammation in the gallbladder, liver or pancreas depending on where the stone lodges. Is often released with the bile needed to emulsify a fatty meal.
Recommendations: Text p.234-236