

Most Common Chronic Diseases

Coronary Artery Disease/Atherosclerosis

Blockage of the coronary arteries that occurs when fat, cholesterol, and other substances build up in the walls of arteries and form hard structures called plaques.

Eventually, the plaques can make the artery narrow and less flexible, making it harder for blood to flow. If the coronary arteries become too narrow, blood flow to that part of the heart can slow down or stop.

Pieces of plaque can break off and move through the bloodstream (embolization). This is a common cause of heart attack and stroke.

Formation of blood clots are accelerated in smoking, diabetes, and hypertension. Preventing blood clotting is a common strategy in preventing and treating atherosclerosis.

As we know there is an inflammatory component to atherosclerosis, with the correlation of periodontal disease and increased risk of atherosclerosis. Plaques form in the walls of the artery due to the presence of accumulated white blood cells and are promoted by low density lipoprotein.

The cardiac emergencies of angina, myocardial infarction and sudden cardiac arrest are the consequence of this disease.

Hypertension

Condition where blood pressure is elevated. Blood pressure is a measurement of force of the contraction of the heart against the elasticity of the blood vessels.

Many factors can affect blood pressure, including:

- How much water and salt you have in your body
- The condition of your kidneys, nervous system, or blood vessels
- The levels of different body hormones
- You are more likely to be told your blood pressure is too high as you get older. This is because your blood vessels become stiffer as you age.

Essential/Primary Hypertension - No medical cause can be found.

95% of hypertension is essential. It is one of the most complex disorders. There is no identifiable cause but there are lots of risk factors. The two biggest are sedentary lifestyle and obesity. Genetics also play a large role.

High blood pressure that is caused by another medical condition or medication is called secondary hypertension.

Secondary Hypertension - Result of another specific condition, habit or medication.

Tumors such as adrenal gland tumor or pheochromocytoma

Drugs such as steroids or appetite suppressants

Kidney disease

- Adrenal gland tumor
- [Alcohol abuse](#)
- Anxiety and stress
- [Arteriosclerosis](#)
- Birth control pills
- [Coarctation of the aorta](#)
- Cocaine use
- [Cushing syndrome](#)
- [Diabetes](#)
- Kidney disease, including:
 - [Glomerulonephritis](#) (inflammation of kidneys)
 - [Kidney failure](#)
 - Renal artery stenosis
 - [Renal vascular obstruction](#) or narrowing
- Medications
 - Appetite suppressants
 - Certain cold medications
 - Corticosteroids
 - Migraine medications
- [Hemolytic-uremic syndrome](#)
- [Henoch-Schonlein purpura](#)
- Obesity
- Pain
- [Periarthritis nodosa](#)
- Pheochromocytoma
- Pregnancy (called gestational hypertension)
- [Primary hyperaldosteronism](#)
- Renal artery stenosis

- [Retroperitoneal fibrosis](#)
- [Wilms' tumor](#)

The goal of treatment is to reduce blood pressure so that you have a lower risk of complications.

There are many different medicines that can be used to treat high blood pressure, including:

- Alpha blockers
- Angiotensin-converting enzyme (ACE) inhibitors
- Angiotensin receptor blockers (ARBs)
- Beta-blockers
- Calcium channel blockers
- Central alpha agonists
- Diuretics
- Renin inhibitors, including aliskiren (Tekturna)
- Vasodilators

When blood pressure is too high and remains that way, arterial walls become weakened and more prone to atherosclerosis. High blood pressure can also cause arteries to bulge (aneurysm) or burst (hemorrhage).

Diabetes

High blood sugar/glucose levels as a result of not producing enough insulin or because the body's cells do not properly respond to the insulin that is produced.

Type I - Body's failure to produce insulin.

Type II - Body's resistance to insulin produced

As you know, several things happen when food is digested:

- 1 Glucose enters the bloodstream.
- 2 The pancreas makes insulin. The role of insulin is to move glucose from the bloodstream into muscle, fat, and liver cells, where it can be used as fuel. When this can't happen an increase in blood glucose occurs.

The classical symptoms of diabetes are [polyuria](#) (frequent urination), [polydipsia](#) (increased thirst) and [polyphagia](#)(increased hunger).^[11]

Prolonged high blood glucose causes glucose absorption, which leads to changes in the shape of the lenses of the eyes, resulting in vision changes;

People may also present with [diabetic ketoacidosis](#), a state of metabolic dysregulation characterized by the smell of [acetone](#); a rapid, deep breathing known as [Kussmaul breathing](#); nausea; vomiting and [abdominal pain](#); and an altered states of consciousness.

A rarer but equally severe possibility is [hyperosmolar nonketotic state](#), which is more common in type 2 diabetes and is mainly the result of dehydration.

A number of skin rashes can occur in diabetes that are collectively known as [diabetic dermadromes](#).

Having diabetes or pre-diabetes puts you at increased risk for heart disease and stroke.

Diabetes occurs in 7% of population and is rising. Blood sugar and Glycosolated Hemoglobin or HbA1c levels are used for monitoring. Normal blood sugar level is 80 to 140. Glycosolated Hb forms when glucose attaches to hemoglobin. The amount of HbA1c in blood is indicative of how well blood glucose has been controlled. Normal/Non-diabetic level is 4 to 6.2 In a diabetic the levels should be 7 or less.

Stroke

Rapid loss of brain function due to disturbance in blood supply to the brain caused by blocked or ruptured blood vessels. (Ischemic vs. hemorrhagic stroke)

Pulmonary Diseases:

Chronic Bronchitis

Chronic inflammation of the bronchi (medium sized airways) in the lungs. Defined as clinically persistent cough that produces sputum for at least three months in two years.

Symptoms are sputum producing cough, shortness of breath, occasionally chest pain, fever, fatigue or malaise.

Smoking is the most common cause.

Emphysema

Long term progressive lung disease that causes shortness of breath. In emphysema the lung tissue necessary to support the physical shape and function of the lungs is destroyed. Specifically alveolar septum desintegrates due to inflammation. The alveolar sacks rupture leading to decrease in alveolar surface area for gas diffusion. Upon expiration the small air sacs of the lungs (alveoli) collapse and the function of the lungs is diminished.

Asthma

Asthma is a condition in which the bronchi are reversibly narrowed. During asthma attacks, the smooth muscle cells in the bronchi constrict, the airways become inflamed and swollen, and breathing becomes difficult.

Patients with Chronic Bronchitis have trouble breathing leaning back. They are best treated in an upright position since they may become increasingly dyspneic if laid supine. It may be difficult to use a rubber dam, as some patients with these conditions are mouth breathers and do not tolerate the additional obstruction.

Patients with Chronic Bronchitis or Emphysema depend on low oxygen levels as a primary stimulant to respiration. If we give such a patient nitrous oxide and oxygen, this higher concentration of oxygen tends to depress the stimulus to breathing.

A further complicating factor has to do with nitrous oxide's tendency to diffuse into closed spaces. The lungs of COPD patients often have large, gas-filled sacks or blebs. If nitrous oxide diffuses into these spaces, it can cause them to enlarge, possibly to the point of rupture.

Most Common Prescription Drugs

Contraindications: Self Explanatory, don't use it.

Avoid/Use Alternatives: Don't use it in if you can find an alternative. And I guess you could use it, in consultation with patient's MD if you couldn't find an alternative, or alternatives fail.

Monitor/Modify Treatment: Use it, while actively monitoring how it affects patient and decide to continue or discontinue accordingly.

Caution: Use it, but caution the patient about its effects. Continue or discontinue accordingly.

SSRI (Selective Serotonin Re-uptake Inhibitors)

Blocks re-uptake of serotonin back into the presynaptic cell. This causes an increase in serotonin in the synaptic cleft between presynaptic neuron and postsynaptic neuron. These drugs are selective to neurons in the brain, so you don't see systemic effect from this increase level of neurotransmitter in the synaptic cleft in other parts of the body.

Common SSRI Drugs:
fluoxetine/Prozac
paroxetine/Paxil
sertraline/Zoloft

SNRI (Serotonin Norepinephrine Re-uptake Inhibitors)

Increases serotonin and norepinephrine in the synaptic cleft between presynaptic neuron and postsynaptic neuron.

Common SNRI Drugs:

venlafaxine/Effexor
duloxetine/Cymbalta

Caution (Level 4) with NSAID's and SSRI and SNRI drugs. In combination there can be an increase in bleeding with them, due to inhibition of serotonin uptake by the platelet.

Caution (Level 4) The drug tramadolhydrochloride/Ultram possesses agonist actions at the opioid receptor (mu receptor) and inhibits the re-uptake of norepinephrine and and serotonin. By itself, it can reduce an individual's seizure threshold. It can also potentiate the effects of other SSRI, MAOI, and Tricyclic antidepressants, with seizure being the most common adverse drug interaction of Ultram with these other drugs.

Propoxyphene/Darvocet can precipitate Serotonin Syndrome in individuals taking SNRI. Serotonin Syndrome occurs because of accumulation of high level of serotonin in your system. Symptoms are agitation, confusion, rapid heart rate. It can lead to seizures, arrhythmias and death.

Tricyclic Antidepressants

Also blocks re-uptake of serotonin and norepinephrine into the presynaptic cell. Not selective to the brain, so they have a wide general effect in the body. This means they affect autonomic nerves that control heart rate and blood pressure.

Common Tricyclic Antidepressants:
amitriptyline/Elavil
desipramine/Norpramin
clomipramine/Anafranil

MAOI (Monoamine Oxidase Inhibitors)

Enzyme called MAO degrades serotonin and norepinephrine in synaptic cleft. Once again, not selective to the brain, so they have a wide general effect in the body. Once again, this means they affect autonomic nerves that control heart rate and blood pressure.

Avoid/ Use Alternative (Level 2): The effects of epinephrine on the central nervous system will be increased with Tricyclic Antidepressants and MAO I drugs. Avoid administration of epinephrine in individuals taking them. Epi and Nor Epi are monoamines. These drugs inhibit their breakdown which leads to increased blood levels of epi.

Common MAOI Drugs:
Isocarboxazid/Marplan
Tranylcypromine/Parnate
Phenelzine/Nardil
Selegiline/Emsam

Mood Stabilizers

Lithium is the prototypical one. Increase in prescriptions as some depressions now being treated a bipolar disorder. Lithium affects flow of sodium through nerve and muscle cells. Sodium affects excitation of a cell, but exact mechanism of action how lithium influences the effects of sodium on a cell are not known.

Monitor/Modify Tx (Level 3) NSAID's can increase lithium levels in the body, leading to toxicity.

Smoking Cessation Medications

Varenicline tartrate/Chantix

A partial agonist selective for alpha 4 beta 2 nicotinic receptor subtypes. Translated this means it can occupy nicotine receptor and partially block the effect of nicotine on the nicotine receptors in the central nervous system.

Bupropion hydrochloride/ Wellbutrin or Zyban.

Antidepressant where the mechanism for smoking cessation is unknown; exact mechanism of action for depression unknown but it does inhibit neuronal uptake of norepinephrine and dopamine.

Black Box Warning: monitor for serious neuropsychiatric events incl. behavior change, hostility, agitation, depression, and suicidality as well as worsening of preexisting psychiatric illness which have occurred in pts taking varenicline and after discontinuation; some cases possibly complicated by nicotine withdrawal sx, but also reported in pts who continue to smoke while taking varenicline; weigh varenicline risks vs. benefits of smoking cessation.

Zyban may decrease efficacy of narcotic pain medicine. Caution (level 4) Increase risk of seizure in patients taking Penicillin class of antibiotics.

Hypertension Medications

ACE Inhibitors

Angiotensin/Renin cascade helps regulate BP. Angiotensin Converting Enzyme is a substance that increases salt and water retention in the body, thereby increasing blood pressure. Inhibiting this enzyme leads to a lowering of blood pressure. ACE inhibitors make blood vessels relax which decreases blood pressure and allows more oxygen rich blood to reach the heart. Therefore they are useful treatment after a heart attack. They allow more blood to reach to heart.

Common ACE Inhibitors:

captopril/Capoten
enalapril/Vasotec
lisinopril/Prinivil

Monitor/Modify Tx (Level 3) *NSAID's may decrease antihypertensive effectiveness of these drugs. (Inhibit renal prostaglandins, increase sodium and fluid retention)*

Alpha Blockers/Beta Blockers

Stimulation of alpha blockers cause constriction, in the case of the Alpha1 receptors constriction of coronary arteries. Stimulation of the Alpha2 receptors cause generalized constriction of blood vessels.

Beta 1 receptors in the heart increase heart rate and force of contraction when stimulated by epinephrine and norepinephrine. Beta 2 receptors relax smooth

muscle. Beta 2 receptors in the lungs inhibit mast cell release of histamine and cause bronchodilation. Beta 3 receptors in the fat cells enhance lipolysis.

Beta Blockers are primarily used to reduce the work load on the heart and help it beat more regularly.

Common Beta Blockers:

atenolol/Tenormin
propranolol/Inderal
metoprolol/Lopressor

propranolol/Inderal
carvedilol/Coreg

Caution (Level 4): NSAID's may decrease antihypertensive efficacy. (Inhibit renal prostaglandins, increase sodium and fluid retention)

Caution (Level 4) should be given when administering epinephrine with non-selective beta blocker, as there is an increase risk of severe hypertension (non-beta affects just the alpha affects that constrict blood vessels. With this hypertension the body can try to compensate with decrease in heart rate and hypotension.

Calcium Channel Blockers

Disrupt calcium conduction through the cells. Calcium effects the depolarization/activation of many excitable cells in the body: heart smooth muscle, and blood vessels. Calcium channel blockers decrease the blood pressure by affecting the action of these excitable cells.

Common Calcium Channel Blockers:

amlodipine/Norvasc
nifedipine/Procardia
felodipine/Plendil

Monitor/Modify Tx (Level 3) Erythromycins administered with these drugs can increase risk of hypotension. (Decreased hepatic metabolism)

Diuretics

Schematic of how a kidney works. Diuretics help kidneys eliminate excess salt and water from body's tissues and blood. Essentially decrease volume in blood vessels and thereby decrease pressure.

Loop diuretics - Furosemide/Lasix

Inhibit's body's ability to resorb sodium in ascending loop of nephron
Thiazide diuretics - Hydrochlorothiazide/Microzide
Inhibit sodium and chloride resorption in DCT
Potassium sparing diuretics - amiloride, spironolactone/Aldactone
Do not promote secretion of potassium in urine.

Caution (Level 4) with NSAID's – These inhibit renal prostaglandins, which lead to a decrease in diuretic effect of these drugs.

Oral Antiplatelets

Inhibits the action of the platelet becoming activated which leads to blood clotting. There are two drugs that do this clopidogrel and ticlopidine. Clopidogrel is sold under the trade name of Plavix, Clopilet and Clavix. Ticlopidine is sold as Ticlid.

Aggrenox is extended release aspirin with dipyridamole both of which decrease platelet activation leading which leads to blood clots. Coumadin, in contrast does not work on platelets, it decreases blood clotting by inhibiting the vitamin K dependent clotting factors, II, VII, IX, and X.

Should avoid aspirin (Level 2) with these drugs, should monitor (Level 2) use of NSAID's with these drugs.

Vasodilators

Works directly on smooth muscle of arteries to relax their walls so blood can move more easily through them. Nitroglycerin is the only one used in an outpatient setting. It is used to relax spasming cardiac arteries and thereby increasing blood flow

Insulin

Insulin is a hormone that has a profound effect on metabolism. It causes cells in the liver, muscle and fat to take up glucose from the blood and store it as glycogen in these cells, where it can be used as an energy source.

metformin/Glucophage and glyburide/Diabeta are both sulfonylurea drugs. These drugs stimulate the release of insulin from the pancreas.

Insulin is synthesized in the pancreas in the beta cells of the Islets of Langerhans

Types of insulin:

Short acting (R) - works in 5 to 15 minutes, active for 5 to 8 hours.

Intermediate acting (N) - works in 1 to 3 hours, active for 16 to 24 hours.

glarginine/Lantis - starts in 1 to 2 hours, active for 24 hours.

Protein Pump Inhibitors

The protein pump is the terminal stage in gastric acid secretion by the gastric parietal cell. Protein pump inhibitors decrease gastric acid secretion by inhibiting gastric acid secretion by the gastric parietal cells.

Common Protein Pump Inhibitors:

omeprazole/Prilosec
lansoprazole/Prevacid
esomeprazole/Nexium

H2 Receptor Antagonists

These agents block the action of histamine on the H2 receptor of gastric parietal cells. It is the action of histamine at this site which promotes the secretion of gastric acid.

Common H2 Receptor Antagonists:

cimetidine/Tagamet
Famotidine/Pepcid

Lipid Regulators/Statins

Lower cholesterol by inhibiting enzyme HMG-CoA reductase. This is the rate limiting enzyme in the synthesis of cholesterol. Inhibition of this enzyme results in a decrease of cholesterol synthesis and an increase in clearance of LDL from the bloodstream.

Common Lipid Regulators/Statins

atorvastatin/Liptor
lovastatin/Mevacor
rosuvastatin/Crestor