

Treatments for Dysautonomias

Successful treatment of *dysautonomias* usually requires an individualized program, which can change over time.

You should understand that since the underlying mechanisms often are not understood well, treatment is likely to involve some trial and error.

Non-Drug Treatments

Several non-drug treatments are used for different types of *dysautonomias*. The reasons for a treatment depend on the particular *dysautonomia*. Sometimes, the responses of a patient to a treatment help the doctor determine the diagnosis. Patients with *dysautonomias* can feel differently from day to day, even without any clear reason why.

This means that if a treatment is tried, it may take several days to decide on whether the treatment has helped or not.

Elevation of the Head of the Bed

In patients who have a fall in blood pressure every time they stand up (*orthostatic hypotension*), elevation of the head of the bed at night, by a variety of ways, improves the ability to tolerate standing up in the morning.

Salt Intake

High salt intake tends to increase the volume of fluid in the body. A small percent of this volume is in the bloodstream. Doctors usually recommend a high salt diet for patients with an inability to tolerate prolonged standing (*chronic orthostatic intolerance*) or a fall in blood pressure during standing (*orthostatic hypotension*). Normally when a person takes in a high salt diet, the kidneys increase the amount of salt in the urine, and this limits the increase in *blood volume*. Drugs that promote retention of *sodium* by the kidneys, such as *Florinef*TM, are usually required for high salt intake to increase body fluid volume effectively.

Meals

Eating a big meal leads to shunting of blood toward the gut. In people with dizziness or lightheadedness when they stand up (*orthostatic intolerance*) or with *orthostatic hypotension*, it is usually advisable to take frequent small meals.

Reducing the amounts of sugars or other carbohydrates in meals may help manage symptoms.

Compression Hose

Compression hose or other compression garments tend to decrease the amount of pooling of blood in veins when a person stands. This can decrease leakage of fluid from the veins into the tissues and decrease swelling of the feet. In patients with veins that fill up or leak excessively during standing, compression garments can improve toleration of prolonged standing. In patients with a fall in blood pressure during standing (orthostatic hypotension), the problem may be less with the veins than with the arteries and arterioles, the blood vessels that carry oxygen-rich blood under high pressure to the organs and limbs. Wearing compression garments therefore may be disappointing in the management of orthostatic hypotension.

Coffee

Some patients with dysautonomias feel better drinking caffeinated coffee frequently. Others feel jittery or anxious and avoid caffeinated coffee. Still others notice no effect one way or the other.

Temperature

Patients with *dysautonomias* often have an inability to tolerate extremes of external temperature. When exposed to the heat, patients with failure of the *sympathetic nervous system* may not sweat adequately to maintain the core temperature by evaporation of the sweat. Patients with *chronic orthostatic intolerance*, such as from *postural tachycardia syndrome (POTS)*, can have heat intolerance because of loss of blood volume by sweating or shunting of blood away from the brain. When exposed to cold, patients with *sympathetic nervous system* failure may not constrict blood vessels adequately in the skin, so that the body temperature falls (*hypothermia*).

Exercise

Patients with *dysautonomias* sometimes benefit from an exercise training program. Often, however, the training does not decrease the sense of fatigue.

As a person exercises, the blood vessels carrying oxygen-rich blood to the exercising muscle (*arteries and arterioles*) tend to relax, due at least partly to the accumulation of byproducts of metabolism. The *sympathetic nervous system* normally counters this tendency, by increasing the tone of the blood vessel walls. The blood flow to the exercising muscle therefore is in a dynamic state of balance. Activation of *sympathetic nerves* to the heart during exercise increases the force and rate of the heartbeat, and the total amount of blood pumped by the heart in one minute (*cardiac output*) increases. Meanwhile, like squeezing a tube of toothpaste, pumping of muscle during exercise increases the movement of blood from the limbs back to the heart. The increased metabolic activity tends to increase body temperature, and sweating, which is stimulated importantly by *sympathetic nerves* to sweat glands, increases the loss of heat by evaporation, helping maintain an appropriate body temperature.

If a patient had failure of the *sympathetic nervous system*, excessive production of byproducts of metabolism, or a form of heart disease where there were an inability to increase the force or rate of the heartbeat, then the blood pressure would fall during exercise, producing a sense of fatigue or exhaustion.

After exercise, when muscle pumping ceases, the blood can begin to pool in the legs or abdomen, while the rate of *sympathetic nerve traffic* falls to the resting rate. If the decline in *sympathetic nerve traffic* did not balance the decline in production of byproducts of metabolism, then

the blood pressure would fall after exercise. At the same time, loss of body fluid via evaporative sweating would decrease the blood volume. Patients with a *dysautonomia* therefore can feel bad not only during exercise but also after exercise. It is important to stay hydrated and to avoid activities like eating a large meal after exercise, because this can divert already limited blood volume to the gut.

Perhaps surprisingly, even vigorously healthy, muscular, lean people can have a susceptibility to faint (*neurocardiogenic syncope*), and it is unclear if exercise training in general helps them. On the other hand, some patients can improve by isometric calf muscle training, where the patient learns to tense calf muscles when standing. This tends to decrease the amount of pooling of blood in the legs.

Pacemakers and Sinus Node Ablation

Insertion of a pacemaker in the heart can help patients with *neurocardiogenic syncope* or *POTS*. This is an area of active research and some controversy. In some patients with *neurocardiogenic syncope*, having a pacemaker inserted may not be a cure, because the low pulse rate at the time of fainting might not cause and might even be the result of low blood flow to the brain. On the other hand, a sudden absence of electrical activity in the heart produces loss of consciousness within seconds, and in this setting a pacemaker could be curative.

Some patients who have a very fast pulse rate undergo destruction of the *sinus node* pacemaker cells in the heart (*sinus node ablation*). The doctor must be sure that the fast pulse rate results from a problem with the heart and does not result from a compensation by the *sympathetic nervous system* for another problem, such as low blood volume, because eliminating the compensation could make the patient worse rather than better.

Neurosurgery

Some patients with *chronic orthostatic intolerance* have a type of change in the brainstem called *Chiari malformation*. This is an anatomic abnormality where part of the brainstem falls below the hole in the skull between the brain and spinal cord. Neurosurgery can correct the malformation, but the *orthostatic intolerance* does not necessarily disappear. This is a controversial topic, and we recommend that patients seek a second opinion before agreeing to this procedure.

Constipation or Urinary Retention

Patients with failure of the *parasympathetic nervous system* can have problems with constipation and retention of urine in the bladder. The constipation is treated non-specifically, with stool softeners, bulk laxatives, and if needed milk of magnesia, magnesium citrate, senna, or cascara. Urinary retention can be associated with urinary urgency and incontinence. Drugs that stimulate *receptors* for *acetylcholine*, such as *urecholine*, might be tried.

Often patients with *autonomic failure* must learn to self-catheterize to empty the bladder, by inserting a plastic or rubber tube into the urethra and then into the bladder, in order to obtain relief.

Water Drinking

A relatively recently described tactic to increase blood pressure in patients with *autonomic failure* is to drink 16 ounces of water or other fluid. Why water drinking should increase blood pressure in patients with *autonomic failure*, when doing so does not affect the blood pressure of healthy people, remains unclear.

Patients with *chronic orthostatic intolerance*, *neurocardiogenic syncope*, or *POTS* often keep a water container with them and sip from it repeatedly during the day. This habit might indicate a tendency to dehydration and low blood volume, but the meaning of what we call the “water bottle sign” remains unproven.

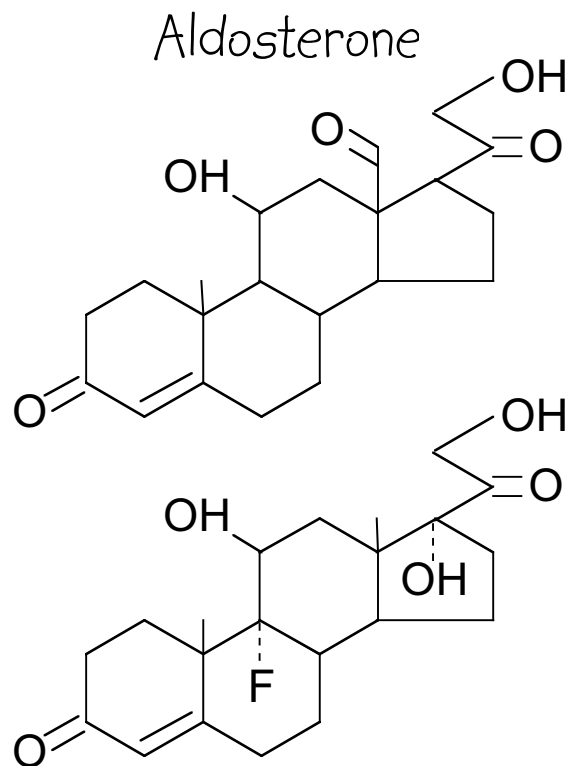
Drug Treatments

Several drug treatments are used for *dysautonomias*. Some of them are powerful or can produce bad effects. Patients should take medications only under the supervision of a doctor with expertise and experience in the treatment of *dysautonomias*.

Fludrocortisone (Florinef™)

Florinef™ is a man-made type of drug called a *salt-retaining steroid*, or *mineralocorticoid*. *Florinef™* closely resembles the body's main *salt-retaining steroid*, which is *aldosterone*.

Florinef™ must be taken with a high-salt diet to work. *Florinef™* forces the kidneys to retain *sodium*, in exchange for *potassium*. Water follows the sodium, and so *Florinef™* is thought to increase the blood volume. The patient gains “fluid weight,” and blood pressure increases. Because of the tendency of *Florinef™* to waste *potassium*, *Florinef™* can cause a fall in the serum *potassium* level, which if severe can be dangerous. Patients taking *Florinef™* should have periodic checks of their serum *potassium* level, and if it is low take a *potassium* supplement.



Florinef™ forces the body to retain salt.

Florinef™ given to patients with *chronic autonomic failure* can cause or worsen high blood pressure when the patient is lying down. Sometimes the doctor faces a difficult dilemma, balancing the long-term increased risk

of stroke, heart failure, or kidney failure from high blood pressure against the immediate risk of fainting or falling from *orthostatic hypotension*.

Beta-Adrenoceptor Blockers

The main chemical messenger of the *sympathetic nervous system* is *norepinephrine (noradrenaline)* and of the *adrenomedullary hormonal system* is *epinephrine (adrenaline)*. *Norepinephrine* and *epinephrine* produce their effects by binding to specific *receptors* on the target cells, such as heart muscle cells. There are two types of *receptors* for *norepinephrine* and *epinephrine*, called *alpha-adrenoceptors* and *beta-adrenoceptors*.

Epinephrine (adrenaline), which stimulates both *alpha-adrenoceptors* and *beta-adrenoceptors*, produces *vasoconstriction* in most parts of the body, such as the skin, due to stimulation of *alpha-adrenoceptors*, but with the important exception of the skeletal muscle, where the blood vessels relax, due to stimulation of *beta-adrenoceptors*. Because of the relaxation of the blood vessels in skeletal muscle, stimulation of *beta-adrenoceptors* tends to decrease the *total peripheral resistance*. Stimulation of *beta-adrenoceptors* also produces powerful effects on the heart, increasing the force and rate of the heartbeat. Because of the effects on the heart, the amount of blood pumped by the heart per minute (*cardiac output*) increases, and this increases the blood pressure when the heart is contracting, the *systolic blood pressure*.

There are at three types of *beta-adrenoceptors*, called beta-1, beta-2, and beta-3. *Beta-1 adrenoceptors* and *beta-2 adrenoceptors* are present in the human heart, and stimulation of these receptors produces about the same effects. In contrast, *beta-2 adrenoceptors* are much more abundant in skeletal muscle blood vessels than are *beta-1 adrenoceptors*. This difference may be relevant to the treatment of *neurocardiogenic syncope*, as explained below.

Beta-adrenoceptor blockers decrease the pulse rate, the force of heart contraction, and the *systolic blood pressure*. In patients with rapid pulse rates, associated with a sense of pounding or irregular beating of the heart (*palpitations*) or chest pain, *beta-adrenoceptor blockers* decrease the heart rate and can help relieve the pain and prevent abnormal heartbeats or heart rhythms; however, in patients where the chest pain results from stimulation of *alpha-adrenoceptors* in the *coronary arteries*, *beta-adrenoceptor blockers* may not help the pain. These drugs also are commonly used to treat long-term high blood pressure (*hypertension*). Because of decreased *systolic blood pressure* and heart rate, the rate of consumption of oxygen by the heart decreases, and this can help patients with *coronary artery disease*.

Non-Selective

Selective

Propranolol
(Inderal™)

Atenolol
(Tenormin™)

Nadolol
(Corgard™)

Metoprolol
(Toprol™)

Timolol
(Blocadren™)

Betaxolol
(Kerlone™)

Here are some beta-blockers. All beta-blockers decrease the rate and force of the heartbeat.

In patients with *postural tachycardia syndrome (POTS)*, the value of treatment with *beta-adrenoceptor blockers* may depend on whether the rapid pulse rate when the patient stands up reflected a primary or compensatory response. If the rapid pulse rate were a compensation for another problem, such as low blood volume due to bleeding, then blocking that compensation would not help the patient. But if the rapid pulse rate were the result

of an inappropriate, excessive rate of *sympathetic nerve traffic* to the heart, then blocking the effects of the excessive nerve traffic would help the patient.

There are two types of *beta-adrenoceptor blockers*, selective and non-selective. Selective *beta-adrenoceptor blockers* block beta-1 adrenoceptors, and non-selective *beta-adrenoceptor blockers* block both *beta-1 adrenoceptors* and *beta-2 adrenoceptors*. A potentially important difference between these drugs is that non-selective *beta-adrenoceptor blockers* block the *beta-2 adrenoceptors* in blood vessel walls of skeletal muscle, whereas *beta-1 adrenoceptor blockers* do not. In patients with *neurocardiogenic syncope* and high levels of *epinephrine* in the bloodstream, the *epinephrine* might stimulate *beta-2 adrenoceptors* on blood vessels in skeletal muscle. This would relax the blood vessels, decreasing the resistance to blood flow. This in turn could shunt blood away from the brain and towards the limbs, contributing to lightheadedness or loss of consciousness. In such patients, *non-selective beta-adrenoceptor blockers* might be preferable to selective blockers.

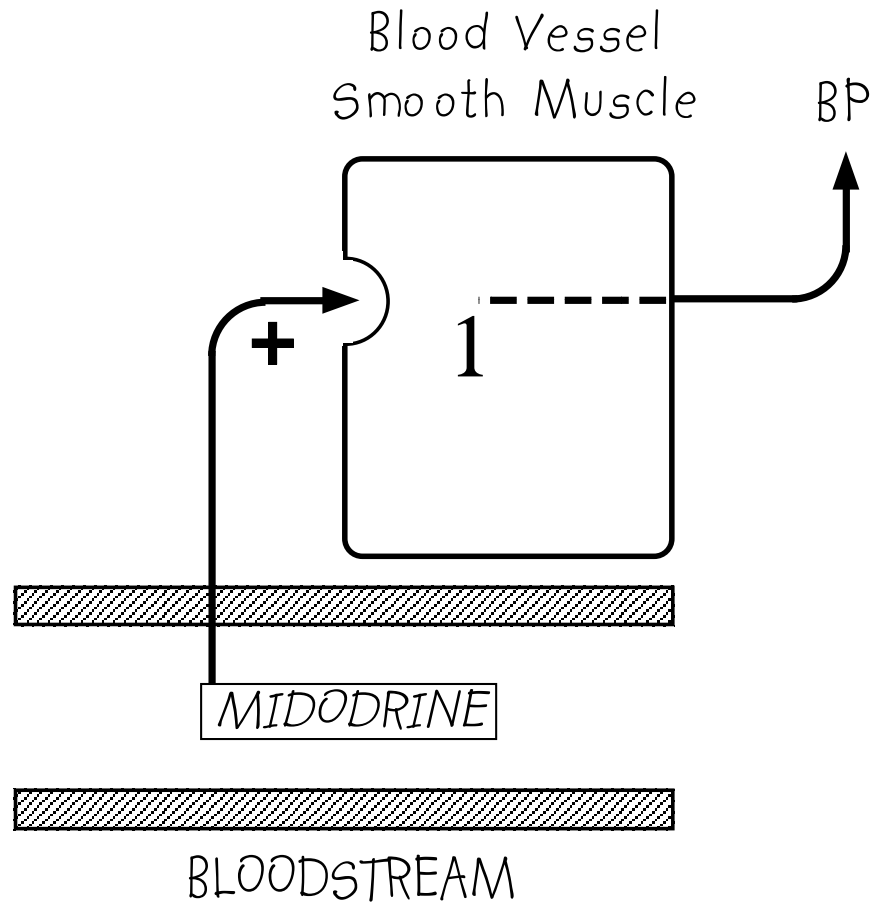
Midodrine (Proamatine™)

Midodrine (Proamatine™) is a relatively new drug that tightens blood vessels throughout the body. That is, it is a *vasoconstrictor*. *Midodrine* works by stimulating *alpha-adrenoceptors* in blood vessel walls.

Midodrine (Proamatine™) is used to treat conditions where there is a failure to tighten blood vessels appropriately, such as when a patient stands up. When a person stands up, the *sympathetic nervous system* is normally activated reflexively, the chemical messenger *norepinephrine* is released from the *sympathetic nerve terminals* in blood vessel walls, the *norepinephrine* binds to *alpha-adrenoceptors* in the blood vessel walls, and the stimulation of the *alpha-adrenoceptors* causes the blood vessels to constrict (*vasoconstriction*), increasing the blood pressure.

Midodrine acts like an artificial form of norepinephrine, by stimulating *alpha-adrenoceptors* directly. In some patients with a fall in blood pressure when they stand up (*orthostatic hypotension*), the cause is a loss of *sympathetic nerve terminals*, so that there is little or no *norepinephrine* to release. In this situation, the *alpha-adrenoceptors* on the cells in blood vessel walls accumulate on the cell surface, and the blood vessels become supersensitive (*denervation supersensitivity*). In these patients, *midodrine* can be very effective in raising the blood pressure.

In using *midodrine* to treat elderly men with *orthostatic hypotension*, the doctor should be aware that stimulation of *alpha-adrenoceptors* can worsen symptoms of prostate problems, such as urinary retention, urgency, and decreased urinary stream. *Alpha-1 adrenoceptor blockers* are effective in treating *benign prostatic hypertrophy (BPH)*, and *alpha-1 adrenoceptors blockers* interfere with *midodrine*.



Midodrine works like artificial norepinephrine, increasing blood pressure (BP) by stimulating alpha-adrenoceptors in blood vessel walls.

Clonidine

There are two types of *alpha-adrenoceptors*, called alpha-1 and alpha-2. Stimulation of either type of *receptor* in blood vessel walls causes the vessels to constrict (*vasoconstriction*).

Clonidine stimulates *alpha-2 adrenoceptors*. Stimulation of *alpha-2 adrenoceptors* in the brain decreases the rate of *sympathetic nerve traffic*. Stimulation of *alpha-2 adrenoceptors* on *sympathetic nerve terminals* decreases the amount of release of the chemical messenger, *norepinephrine*, from the terminals. Therefore, even though *clonidine* stimulates a type of *alpha-adrenoceptor*, *clonidine* normally decreases blood pressure.

Clonidine works both in the brain and outside the brain. It decreases the blood pressure and often causes drowsiness.

There are several uses of *clonidine* in the diagnosis and treatment of *dysautonomias*. In the *clonidine suppression test*, discussed in the section about tests for *dysautonomias*, *clonidine* is used to separate high blood pressure due to increased *sympathetic nervous system* activity from high blood pressure due to a tumor that produces *norepinephrine* and *epinephrine*, called *pheochromocytoma*. In patients with long-term high blood pressure (*hypertension*) due to excessive release of

norepinephrine from *sympathetic nerve terminals* (*hypernoradrenergic hypertension*), *clonidine* can be very effective in lowering the blood pressure. *Clonidine* is also effective in treating withdrawal from some addictive drugs.

Clonidine usually causes drowsiness and often causes a dry mouth. The sedation from *clonidine* can limit its use.

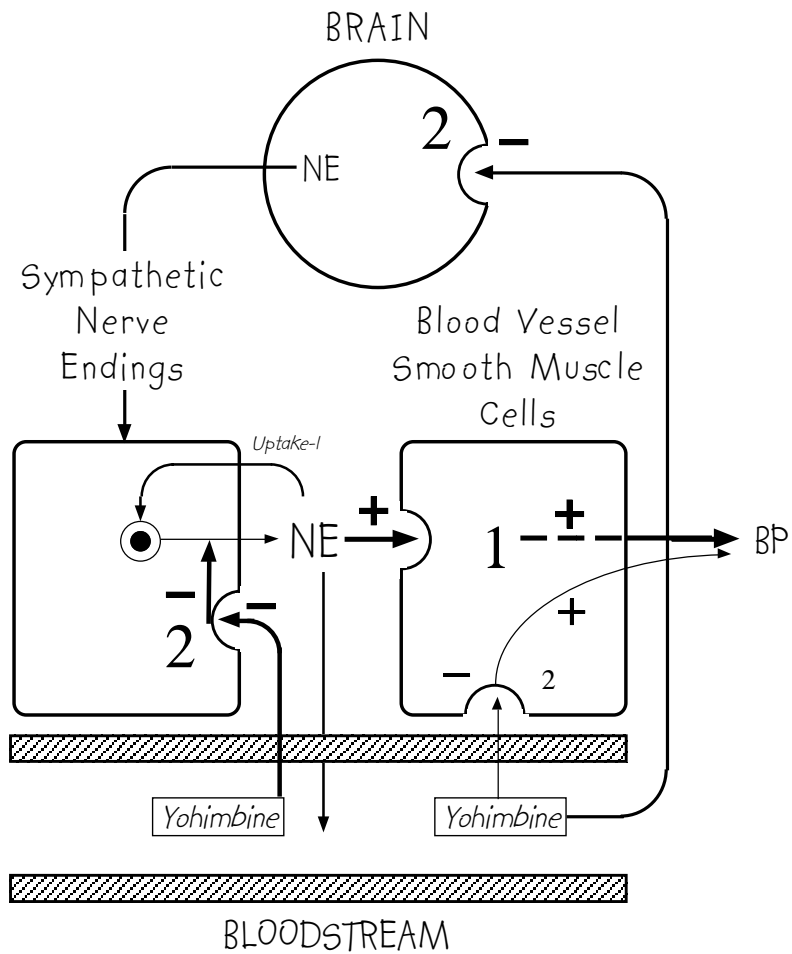
Yohimbine

When *alpha-2 adrenoceptors* in the brain are blocked, this increases *sympathetic nerve traffic* and increases the amount of *norepinephrine* release for a given amount of *sympathetic nerve traffic*.

Yohimbine blocks *alpha-2 adrenoceptors* in the brain and on *sympathetic nerve terminals*, and so it releases *norepinephrine* from the terminals. The released *norepinephrine* binds to *alpha-1 adrenoceptors* in blood vessel walls. This causes the blood pressure to increase.

Even though *yohimbine* blocks *alpha-2 adrenoceptors* in blood vessel walls, the drug releases so much *norepinephrine*, and there are so many *alpha-1 adrenoceptors* in blood vessel walls, that normally *yohimbine* increases the *plasma norepinephrine level* and increases the *blood pressure*.

In patients with *chronic autonomic failure* and an inability to regulate *sympathetic nerve traffic* to intact



Yohimbine works both in the brain and outside the brain. The drug increases blood pressure and the state of alertness.

terminals, such as in the *Shy-Drager syndrome*, *yohimbine* releases *norepinephrine* from the terminals and effectively increases the blood pressure. In patients with *neurocardiogenic syncope*, *yohimbine* may prevent episodes of fainting.

Yohimbine can cause trembling, paleness of the skin, goosebumps, hair standing out, an increase in salivation, or emotional changes.

Oral *yohimbine* is approved as a prescription drug to treat impotence from *erectile dysfunction* in men. *Yohimbine*, in the form of *yohimbe bark*, can be purchased in health food stores.

Intravenous Saline

Inability to tolerate prolonged standing can result from low blood volume, excessive pooling of blood in the veins of the legs during standing, or exit of fluid from the blood vessels into the tissues during standing (*extravasation*). In these situations, infusion of physiological saline solution can temporarily improve the ability to tolerate standing up. This is also useful for diagnostic purposes. Some patients with *chronic orthostatic intolerance* benefit from intravenous saline infusion given repeatedly by way of a permanent intravenous catheter.

Saline infusion temporarily increases blood volume.

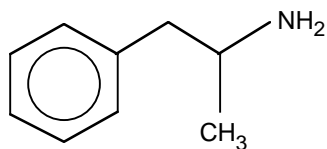
Amphetamines

Amphetamines are chemicals that resemble the drug, *dextro-amphetamine*, or *d-amphetamine*.

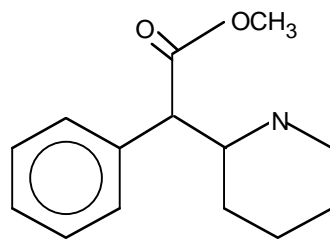
Amphetamines are in a class of drugs called *indirectly acting sympathomimetic amines*. They produce their effects at least partly by increasing delivery of *norepinephrine* to its *receptors*, both in the brain and outside the brain.

By way of effects in the brain, *amphetamines* increase the state of arousal and attention, prevent or reverse fatigue, decrease appetite, and at high doses increase the rate and depth of breathing. By way of effects on the *sympathetic nervous system*, they increase blood pressure.

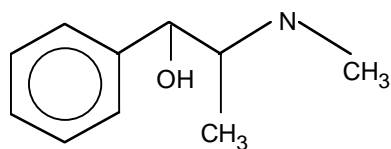
Pseudoephedrine (*Sudafed*TM) is structurally a mirror image (*stereoisomer*) of *ephedrine*. This difference changes the properties of the drug, producing much less central nervous system stimulation. By releasing *norepinephrine* from *sympathetic nerve terminals* in the mucous membranes of the nasal airways, *pseudoephedrine* tightens blood vessels, making them less leaky and thereby relieving nasal congestion.



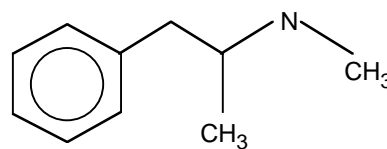
AMPHETAMINE



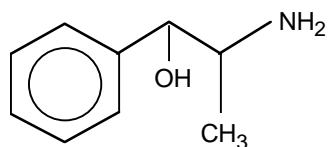
METHYLPHENIDATE



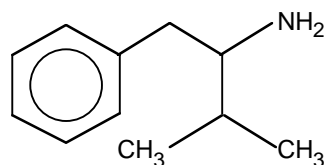
EPHEDRINE



METHAMPHETAMINE



PHENYLPROPANOLAMINE



PHENTERMINE

Amphetamines work both inside and outside the brain. They increase attention, decrease appetite, interfere with sleep, and often increase the blood pressure.

Methylphenidate (Ritalin™), another *sympathomimetic amine*, is used commonly to treat attention deficit-hyperactivity disorder.

Phenylpropanolamine (PPE) until relatively recently was used in over-the-counter diet pills, until the discovery of serious adverse effects such as severe high blood pressure and stroke.

Phentermine prescribed with *fenfluramine* (“*Phen-Fen*”) was an effective combination to decrease weight, until serious adverse effects of this combination came to light.

In treating patients with *dysautonomias*, *amphetamines* should be used sparingly, because of the potential for tolerance and dependence. In patients with *sympathetic neurocirculatory failure* from abnormal regulation of *sympathetic nerve traffic* to intact *sympathetic nerve terminals*, this type of drug releases *norepinephrine* from the terminals and increases the blood pressure. Some patients with *chronic orthostatic intolerance*, such as *neurocardiogenic syncope*, can improve.

Selective Serotonin Reuptake Inhibitors (SSRIs)

SSRIs inhibit a key process that is required for inactivating and recycling the chemical messenger, *serotonin*. The process is reuptake of released *serotonin* back into the nerve terminals that store it. *SSRIs* are widely used to treat depression, anxiety, and other

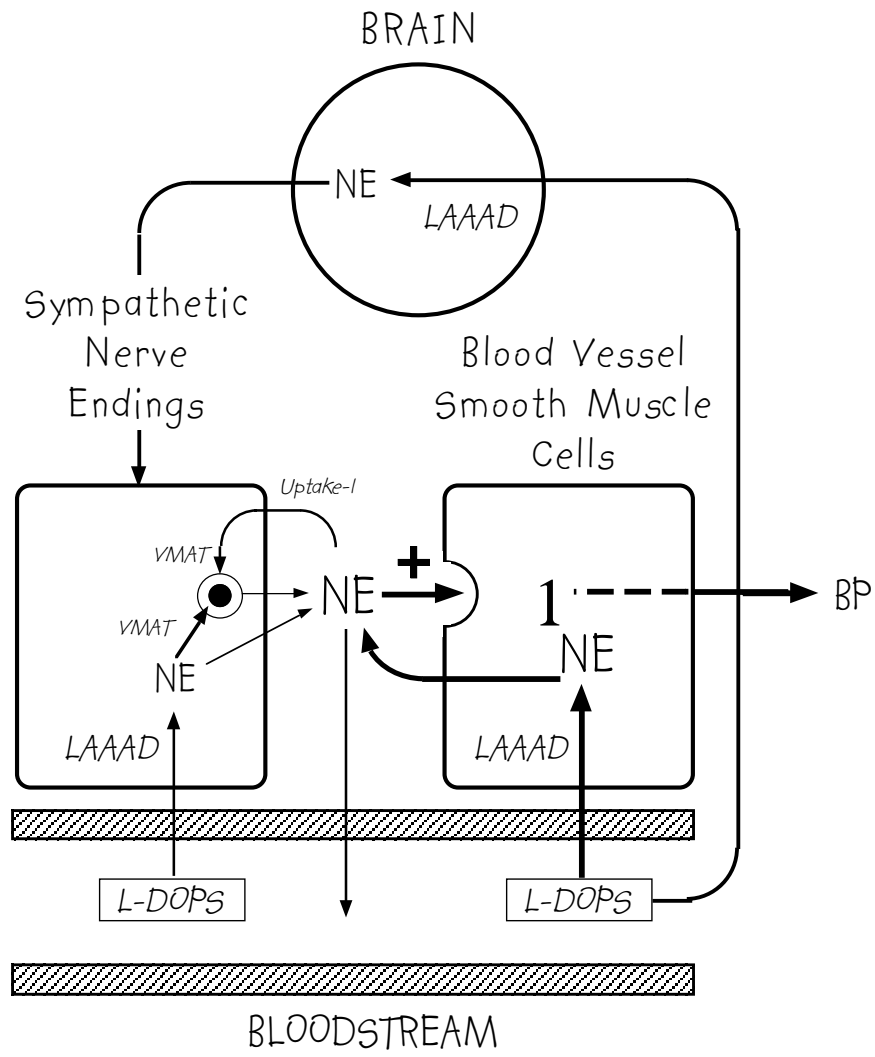
psychiatric or emotional problems. They are also used to treat some forms of *dysautonomias*.

Procrit™ (Erythropoietin)

Procrit™ (Erythropoietin) is a particular hormone that is used as a drug. *Erythropoietin* in the body is released into the bloodstream by the kidneys and acts on the bone marrow to increase the production of red blood cells. *Procrit™* therefore is helpful to treat low red blood cell counts (*anemia*), such as in kidney failure. Anemic patients look pale and feel tired. By mechanisms that remain incompletely understood, *Procrit™* tends to increase the *blood pressure*. Some doctors prescribe *Procrit™* to treat low *blood pressure* in patients with *chronic fatigue syndrome* who have a low red blood cell count.

L-Dihydroxyphenylserine (L-DOPS)

L-Dihydroxyphenylserine (L-DOPS) is a type of chemical called an *amino acid*. It is very closely related chemically to *L-dihydroxyphenylalanine (Levodopa, L-DOPA)*, which is an effective drug to treat *Parkinson's disease*. *L-DOPA* works by being converted in the brain to the *catecholamine, dopamine*. *L-DOPS* works by being converted to the closely related *catecholamine, norepinephrine*. Since norepinephrine is the chemical messenger of the *sympathetic nervous system*, *L-DOPS* can provide *norepinephrine* even in the absence of *sympathetic nerve terminals*.



L-DOPS is converted to norepinephrine both inside and outside the brain.

L-DOPS could increase delivery of *norepinephrine* to its *receptors* by at least three mechanisms. One is from uptake of *L-DOPS* into cells such as in blood vessel walls, followed by conversion of *L-DOPS* to *norepinephrine* that is speeded up by the enzyme, *L-aromatic-amino-acid decarboxylase (LAAAD)*. The *norepinephrine* exits the cells and binds to its *receptors* on the cell membrane. A second mechanism is from uptake of *L-DOPS* into *sympathetic nerve terminals*, again followed by conversion of *L-DOPS* to *norepinephrine*. The *norepinephrine* is taken up into *storage vesicles* and released in response to *sympathetic nerve traffic*. The *norepinephrine* exits the nerve terminals and binds to its *receptors* on cells in blood vessel walls. A third mechanism is from *L-DOPS* entering the brain, followed by conversion of *L-DOPS* to *norepinephrine*. The *norepinephrine* stimulates an increase in the rate of *sympathetic nerve traffic*, resulting in release of *norepinephrine* from the *sympathetic nerve terminals*. By all three mechanisms, *L-DOPS* administration would lead to stimulation of *alpha-adrenoceptors* in blood vessel walls, causing the vessels to constrict and increasing the blood pressure.

L-DOPS is currently an investigational drug. It has great promise to treat a fall in blood pressure when the patient stands up (*orthostatic hypotension*) or prevent fainting. A potential problem with using *L-DOPS* to treat *orthostatic hypotension* in patients with *Parkinson's disease* is that the patients often are treated at the same time with *Sinemet*[™]. *Sinemet*[™] is a combination of *L-DOPA* and *carbidopa*. The *carbidopa* interferes with the

conversion of *L-DOPA* to *dopamine*. Since *carbidopa* does not enter the brain, the combination results in increased delivery of *DOPA* to the brain and increased production of *dopamine*. *Carbidopa* also interferes with the conversion of *L-DOPS* to *norepinephrine*. This would be expected to prevent or blunt the hoped-for increase in blood pressure by *L-DOPS* treatment.

Bethanecol (Urecholine™)

Bethanecol is a drug that stimulates *receptors* for *acetylcholine*, the chemical messenger of the *parasympathetic nervous system*.

Urecholine™ increases production of saliva, increases gut activity, and increases urinary bladder tone.

Bethanecol increases the muscle tone of the bladder, digestive motions of the gut, and salivation. It may be useful to treat urinary retention or constipation in patients with *chronic autonomic failure*, but no formal study of this has been reported yet.

<i>Drug</i>	<i>Goal of Treatment</i>
Florinef™ =Fludrocortisone	Increase blood volume Increase blood pressure
Proamatine™ =Midodrine	Tighten blood vessels Increase blood pressure Prevent fainting
Beta-Blocker	Decrease heart rate Decrease blood pressure Decrease adrenaline effects Prevent fainting
Procrit™ (=erythropoietin)	Increase blood count Increase blood pressure
Amphetamines	Tighten blood vessels Increase alertness
Desmopressin	Tighten blood vessels
"NSAID"	Tighten blood vessels
Octreotide	Tighten blood vessels in gut
SSRI	Improve mood, allay anxiety
"Tricyclic"	Improve mood
Xanax™ (=Alprazolam)	Increase sense of calmness Improve sleep
Catapres™ (=Clonidine)	Decrease blood pressure Improve sleep
Urecholine™ (=Bethanecol)	Increase salivation Improve gut action Improve urination
Yohimbine	Increase blood pressure

Different centers use different drugs from a long "menu" to treat dysautonomias.