Receptors and the autonomic nervous system

The balance between the sympathetic and parasympathetic nervous system keeps our automated body functions working properly

Oct 12, 2010

Article updated December 11, 2017

The autonomic nervous system receptors act as on/off buttons that control the various sympathetic and parasympathetic effects in the body. When these buttons are turned on or off, things happen in your body. If you learn about these receptors and their actions described below, you will be able to understand what a beta-blocker drug does or what to expect from an alpha-agonist medication or how cocaine can be bad for you.

All of the receptors that we will discuss have additional actions that are not listed below, but these extra actions are not essential to your understanding of these receptors as they apply to the prehospital practice of medicine.

Understanding what the ANS does will help you understand the body's normal response to stress or lack of stress

Sympathetic receptors

The types of sympathetic or adrenergic receptors are alpha, beta 1 and beta 2. Alpha-receptors are located on the arteries. When the alpha receptor is stimulated by epinephrine or norepinephrine, the arteries constrict. This increases the blood pressure and the blood flow returning to the heart. The blood vessels in skeletal muscles lack alpha-receptors because they need to stay open to utilize the increased blood pumped by the heart.

Fight or flight

Remember the fight or flight response? It would not make sense to take blood from other parts of the body and pump it to the muscles so we can run away or defend ourselves if the blood vessels in the skeletal muscles are also constricted and cannot benefit from the increased blood circulation providing extra oxygen and nutrients.

So what do you think happens if we block these alpha-receptors? Right, the arteries dilate. Thus an alpha-blocker medication causes vasodilation and can be used to treat hypertension.

Next are the beta receptors. Beta 1 receptors are located in the heart. When Beta 1 receptors are stimulated they increase the heart rate and increase the heart's strength of contraction or contractility.

The beta 2 receptors are located in the bronchioles of the lungs and the arteries of the skeletal muscles. When these receptors are stimulated, they increase the diameter of the bronchioles to let more air in and out during breathing and they dilate the vessels of the skeletal muscles so they can receive the increased blood flow produced by stimulating the alpha and beta 1 receptors.

So reflect for a moment: If norepinephrine or epinephrine is the neurotransmitter of the sympathetic nervous system and it interacts with all the receptors we just described, then we know that norepinephrine or epinephrine stimulates the alpha, beta 1 and beta 2 receptors and thus it is an alpha agonist, a beta 1 agonist and a beta 2 agonist.

When we administer epinephrine or adrenaline to a patient, we expect alpha, beta 1 and beta 2 agonist effects; we expect an:

- Increase in blood pressure
- Increased heart rate
- Increased cardiac contractility
- Dilation of the bronchioles in the lungs
- Dilation of the vessels in the skeletal muscles

We can also stimulate a single receptor site such as a beta 2 agonist medication like an albuterol inhaler that stimulates beta 2 receptors in the lungs then we can dilate the bronchioles in the patient with bronchospasm without causing excessive stimulation of the heart.

Or we can use a beta 1 antagonist medication more commonly called a beta blocker such as metoprolol (or other drugs ending in ‘olol') which blocks Beta 1 receptors thus decreasing heart rate and contractility which decreases blood pressure for the hypertensive patient and decreases the chance of a dysrhythmia after a heart attack by controlling the heart rate.

The sympathetic receptors can be over-stimulated by the non-therapeutic use of substances like cocaine and methamphetamines. Or the excessive use or overdose of sympathomimetic medication like pseudoephedrine or those used to treat attention deficit disorders.

Severe alcohol withdrawal may also induce sympathetic overdrive. Excessive stimulation of the sympathetic receptors can result in dangerously high blood pressure, tachycardia, dysrhythmias and hyperthermia, any one of which may cause organ damage with the real potential for organism death.

Parasympathetic receptors

Now let's switch to the parasympathetic or cholinergic receptors. These are easier since there are only two types, muscarinic receptors and nicotinic receptors. And I will make it even easier by getting rid of the nicotinic receptors after I tell you they are involved in muscle contraction and are affected by substances such as curare (used on those poison tipped arrows) that cause muscle paralysis by blocking these nicotinic receptors.

Medications such as succinlycholine are available to block the nicotinic receptors and induce paralysis necessary for certain medical procedures.

We are left with the one parasympathetic receptor you must learn, the muscarinic receptor. When this receptor is stimulated, it causes a decrease in the heart rate, a decrease in heart contractility and a decrease in the size of the bronchioles. When we are at rest, we can slow down and conserve energy.

The parasympathetic nervous system helps us do this. What would happen if we block the muscarinic receptors? That would cause the heart rate and contractility to increase, dilation of the bronchioles and less production of secretions in the body.

This is the exact effect of atropine, a drug we use to counteract too much parasympathetic activity such as from over-stimulation of the vagus nerve or the effects of certain chemical warfare nerve agents and organophosphate poisoning. Atropine is a parasympatholytic, we can also call it a parasympathetic antagonist or parasympathetic blocker or an anticholinergic medication.

All these terms mean the same; it means they block the action of acetylcholine at the parasympathetic receptors. The effect of blocking any receptor causes the opposite effect we would expect from stimulating the receptor.

Ipratroprium is another example of a parasympathetic blocker medication but this one is inhaled so most of the effect occurs in the lungs, and when we block parasympathetic receptors in the lungs we cause the bronchioles to dilate and decrease production of secretions like mucus. That makes ipratroprium useful in the patient with COPD who produces excessive pulmonary mucous and in combination with albuterol for any wheezing patient.

But remember that the primary rescue medication for bronchospasm is a beta 2 agoinist such as albuterol although ipratrorium is often added and is available as a combination inhaler with albuterol called Combivent.

It is important to remember that it is the balance between the sympathetic and parasympathetic nervous system that keeps our automated body functions in balance and working properly. Outside forces, including drugs, medications or poisons can change the functioning of the autonomic nervous system. And it is wise to keep in mind that all medications are potential toxins that have some beneficial side effects.

In summary, if you are familiar with the actions of the autonomic nervous system receptors then you can easily recall the therapeutic actions of many commonly used medications and their overdose presentation as well as certain poisons and frequently abused drugs.

About the author

Jim Upchurch MD, MA, NREMT practices in Montana and is board certified in Family Practice with added qualification in Geriatrics. He has a master's degree in education and human development and is licensed as a critical care paramedic. Dr. Upchurch is a 'Legacy' member of the American College of Emergency Physicians and one of the last legacy members to achieve Fellowship. Since 1985 his practice has focused on emergency medicine and EMS while providing the full spectrum of care required in a rural/frontier environment. He provides medical direction for BLS and ALS EMS systems, including critical care interfacility transport; and for the Incident Medical Specialist Program, USDA Forest Service, Northern Region. Dr. Upchurch has served as American Heart Association ACLS Regional and National faculty for Montana and represented Montana on the Council of State EMS Medical Directors of the National Association of State EMS Officials.