Syncope

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Syncope is most common when we pass out due to a decrease in the blood flow to the brain. What is the mechanism?

At the bifurcation of the carotid arteries there is an area called the carotid sinus. There are small receptors called barorecptors embedded in the walls of the carotid artery here. These receptors are stimulated by stretch of the arterial walls and are linked to the brainstem via the ninth cranial nerve (glossopharyngeal). They synapse on the cardiodepressive center within the medulla called the **Nucleus Tractus Solitarius (NTS).** The NTS provide active inhibition of the preganglionic cells of the sympathetic nervous system at cord levels T1-L2 also known as the Intermediolateral cell column (IML). It is these neurons that not only increase heart rate but also increase central vasoconstriction resulting in transient increases in blood pressure.

When a person stands up cerebral blood pressure transiently falls as the blood within the carotid sinus arteries falls towards the feet. The baroreceptors at the carotid sinus are less stimulated as the artery collapses. The cardiodepressive center (Nucleus Tractis Solitaries) of the brainstem decreases its ability to inhibit the sympathetic nervous system. Sympathetic outflow increases resulting in rapid heart rate and increased blood pressure within the brain at such a rate that the patient maintains stability.

Therefore what we do know is that the (NTS**)** reaches threshold and fires vagal efferents that decrease atrial contraction and subsequent output to the carotid tree. Decreased cerebral blood flow is the cause of the syncope. If the NTS in the pontomedullary areas is closer to threshold due to its loss of activation then other afferent barrages may bring it to threshold at moments when it may not to be brought to threshold. For example when a patient stands up and they feel dizzy and faint. This may be as a consequence of the vestibular system bringing the NTS and causing syncope. There may also be a decrease in sympathetic activity due to cord compression in the dorsal lateral funiculus. Complete evaluation is always a must and the brainstem would be a place to pay extra special attention.

This mechanism also works in reverse. When a person sits or lies down in bed, the amount of blood within the carotid arteries increases as the blood flows towards the head. This will increase cerebral blood pressure if not countered by the effects of the baroreceptors. As the arteries bulge, the frequency of firing of the baroreceptors increases. This increases the activation of the NTS and the inhibition of the sympathetic cells within the cord. The result is a decrease in the heart rate and cerebral blood pressure.

Understanding clinical neurology I am able to use receptors that will increase the cardiodepressive center (Nucleus Tractis Solitaries) of the brainstem. Thus decreasing its ability to inhibit the sympathetic nervous system. Sympathetic outflow increases resulting in rapid heart rate and increased blood pressure within the brain at such a rate that the patient maintains stability.