Neural Control of Blood Pressure

Blood pressure is controlled by a feedback mechanism involving receptors in vessel walls and neuroendocrine mechanisms. Changes in blood pressure are detected by baroreceptors, specialised nerve endings that detect stretch of vessel walls. The most important are the aortic arch baroreceptors, which signal via the vagus nerve, passing through the nodose ganglion; and the carotid sinus baroreceptors, which signal via the glossopharyngeal nerve that passes through the petrosal ganglion. These baroreceptors send signals to the central nervous system, and relate the extent of the stretch through variations in the frequency of action potentials. An increased blood pressure will cause greater stretch and cause the baroreceptors to increase the frequency of action potentials. The baroreceptor reflex, or baroreflex, can activate rapidly, within seconds to quickly adapt to changes in blood pressure.

The aortic arch and carotid sinus nerves synapse within the nucleus tractus solitarius (NTS) within the medulla oblongata. The NTS contains both parasympathetic and sympathetic divisions. The sympathetic division projects glutamatergic neurons to the caudal ventrolateral medulla (CVLM), which then sends GABAergic inhibitory neurons to the rostral ventrolateral medulla (RVLM). The RVLM then send glutamatergic neurons to the intermediolateral nucleus in the spinal cord which innervates noradrenergic receptors on target organs, namely the heart where cardiac output is increased; blood vessels to induce vasoconstriction and the adrenal medulla to stimulate catecholamine release. An increase in activity of the baroreceptors, indicating increased blood pressure, will cause greater inhibition of the RVLM and so will reduce the sympathetic outflow to the target organs and helping reduce the blood pressure.

The NTS also has parasympathetic activity, and projects to the nucleus ambiguus and the dorsal motor nucleus of the vagus. These areas then innervate the heart via muscarinic receptors to reduce heart rate.

The baroreceptors are activated at resting blood pressure levels, and so there is tonic activation. The parasympathetic division causes constant depression of the heart rate below the natural pacemaker rhythm. Meanwhile, there is also some tonic activity in the RVLM to innervate blood vessels walls to maintain a baseline level of vasoconstriction.

The blood pressure is also controlled by the renin-angiotensin system in the kidneys which themselves can detect an increase in blood pressure, however there is also a neural input. RVLM innervates the kidneys via the sympathetic renal nerve, releasing noradrenaline, which triggers release of renin from juxtaglomerular granular cells. Renin then converts angiotensinogen released from the liver into angiotensin-I, which then is converted to angiotensin-II by angiotensin converting enzyme. Angiotensin-II then acts on a variety of targets to increase the blood pressure. Such as within the kidney, where it increases retention of sodium, and water to increase blood volume, can promote vasoconstriction. Angiotensin-II can also increase sympathetic activity centrally by activating AT1 receptors in the RVLM.