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Bezold–Jarisch reflex

The **Bezold–Jarisch reflex** (also called the **Jarisch-Bezold reflex** or **Von Bezold-Jarisch**[1]) involves a variety of cardiovascular and neurological processes which cause hypopnea (excessively shallow breathing or an abnormally low respiratory rate) and bradycardia (abnormally low resting heart rate).[2]

Physiology

Prolonged upright posture results in some degree of pooling of blood in the lower extremities that can lead to diminished intracardiac volume. This phenomenon is exacerbated if the individual is dehydrated. The resultant arterial hypotension is sensed in the carotid sinus baroreceptors, and afferent nerve fibers from these receptors trigger autonomic signals that increase cardiac rate and contractility. However, pressure receptors in the wall and trabeculae of the underfilled left ventricle may then sense stimuli, activating high-pressure C-fiber afferent nerves from these receptors. They may respond by sending signals that trigger paradoxical bradycardia and decreased contractility, resulting in additional and relatively sudden arterial hypotension. The bradycardia reaction to acetic acid veratril in the cardiac pacemaker region was first described by von Bezold. Jarisch identified the reaction as chemoreceptor reflex via the vagus nerve, relayed in the solitary nucleus.

The Bezold–Jarisch reflex is responsible for the sinus bradycardia that commonly occurs within the first 60 minutes following an acute myocardial infarction,[3] and explains the occurrence of atrio-ventricular (AV) node block in the context of acute posterior or inferior myocardial infarction.[4] Bradycardia in this setting may be treated with atropine.The Bezold–Jarisch reflex has been suggested as a possible cause of profound bradycardia and circulatory collapse after spinal anesthesia.[5] Also, it is one of the complications of interscalene brachial plexus block.[6] The reflex occurs with several biologically active chemicals, like nicotine and capsaicin,[7] when reaching sensitive areas. Veratrum alkaloids, bradykinin, atrial natriuretic peptides, prostanoids, nitrovasodilators, angiotensin II type 1 receptor (AT1) antagonists and serotonin agonists may also elicit the reflex.[8][9]

Reflex (vasodepressor) syncope or vasovagal syncope originates with activation of specific areas in the cerebral cortex. Indeed, stimulation of areas in the anterior cingulate gyrus can trigger a faint. Although the exact trigger is not known, in some cases reflex syncope has been attributed to activation of the Bezold–Jarisch reflex. This reflex—originally described as the cardiorespiratory response to the intravenous injection of veratrum alkaloids—causes bradycardia, hypotension, and apnea. In experimental animals, stimulation of arterial baroreceptors or ventricular baroreceptors by many chemicals—veratrum alkaloids, nicotine, capsaicin, histamine, serotonin, snake and insect venoms—can also trigger the Bezold–Jarisch reflex. In people, coronary injection of contrast material or of thrombolytic agents can cause reflex syncope, presumably by stimulating ventricular receptors eliciting the Bezold–Jarisch reflex. It is possible that these chemical stimuli activate the same stretch-sensitive Transient Receptor Potential (TRP) channels of arterial baroreceptors that are usually activated by high blood pressure. In humans, triggers clearly distinct from those known to initiate a

Bezold–Jarisch reflex can also elicit reflex syncope. Whatever the actual trigger, vagal afferents carry signals to higher central nervous system centers, which act through autonomic nuclei in the medulla to cause a massive stimulation of the parasympathetic system and abolition of sympathetic tone.

History

The reflex is named after Albert von Bezold and Adolf Jarisch Junior.[10] The significance of the discovery is the first recognition of chemical (non-mechanical) reflexes.

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