

# ORTHOSTATIC HYPOTENSION: DEFINITION, PATHOPHYSIOLOGY, CLASSIFICATION, PROGNOSTIC ASPECTS, DIAGNOSIS AND TREATMENT

Pardayeva Zilola Suvonkulovna

Assistant of Pathophysiology Department of the Samarkand State University

<https://doi.org/10.5281/zenodo.7870542>

**Abstract.** *Orthostatic hypotension (OG) is a form of orthostatic instability (orthostatic tolerance) – the inadequacy of the responses of the cardiovascular system in response to the transition to the vertical position. It was first described by American physicians Samuel Bradbury and Carey Eggleston in 1925 as a degenerative disease of the autonomic nervous system, characterized by the gradual development of postural ( orthostatic) hypotension with a fixed heart rate (HR), anhidrosis, nocturia, disorders of the function of intestinal and urinary sphincters, visual disturbances, impotence, pain in the neck and occipital region, relieving in a horizontal position with a characteristic increase in symptoms in the morning, after eating, physical exertion, in hot weather.*

**Keywords:** *anhidrosis, nocturia.*

Progress in the study of the problem led to the need to clarify and expand the previous definitions of OG, and in 2011 An updated conciliation document was published , approved by the American Society for the Study of the Autonomic Nervous System (ANS; American Autonomous Society), the European Federation of Societies for the Study of ANS (European Federation of Autonomous Societies), a group of researchers studying the ANS The World Federation of Neurology and the ANS Violations Study Group of the American Academy of Neurology (Autonomous Research Group of the World Federation of Neurology and the Autonomic Disorders section of the American Academy of Neurology). The document clarified and updated the definition, pathophysiology and clinical features of OG [1]. For the first time, orthostatic instability syndromes were classified into 3 categories: 1) OG; 2) nerve- mediated (reflex) syncope 3) postural (orthostatic) tachycardia syndrome. Today , interest in the problem is due to the data obtained on the adverse effects of OH on different organs and systems (cardiovascular system, brain , kidneys, endocrine system, etc.), which, of course, requires a unified approach to the diagnosis, prevention and treatment of OH.

Definition The diagnostic criteria for OG were determined by the 2011 Consensus. [1] as a steady decrease in systolic blood pressure (SBP) by  $\geq 20$  mmHg and/or diastolic blood pressure (DBP) by  $\geq 10$  mmHg for 3 minutes after moving to an upright position (standing position) from a prone position or head tilt at least  $60^\circ$  when performing a tilt test; for patients with arterial hypertension (AH) detected in the supine position - a decrease in SAD by  $\geq 30$  mmHg [1]. In 2018 The European Society of Cardiology (ESC) Working Group on the diagnosis and treatment of syncopal conditions, European Society of Cardiology) criteria OG was supplemented by a decrease in SAD $<90$  mmHg from the baseline, especially in patients with SAD $<110$  mmHg in the supine position [3].

OG may be accompanied by symptoms, or it may be asymptomatic. Characteristic symptoms of OG include dizziness, fainting and fainting. Loss of consciousness usually has a

gradual onset, but it can also occur unexpectedly. Additional symptoms include: general weakness, fatigue, cognitive decline, weakness in the lower extremities ("waddling" of the legs), blurred vision, "flies" in front of the eyes, headache, nausea, pain in the neck, extending to the suboccipital zone, the back surface of the neck and shoulders, orthostatic shortness of breath or chest pain (by type of angina pectoris). Pathophysiological mechanisms orthostatic hypotension Normally, during the transition from horizontal to vertical position, there is a gravitationally- mediated redistribution of blood volume, 10-15% of which is deposited in the veins of the lower extremities, as a result of which venous return to the heart, stroke volume, cardiac output and arterial pressure (BP) decrease. These hemodynamic changes provoke a compensatory reflex response – an active the activity of baroreceptors (aortic arch, carotid sinuses, heart, pulmonary vessels), which, in turn, causes reflex enhancement of sympathetic and inhibition of parasympathetic innervation. These changes cause constriction of resistive and capacitive vessels in the visceral, musculoskeletal and renal vascular beds. As a result, there is a slight decrease SAD, a slight increase in DAD against the background of a moderate increase in heart rate. Systemic vasoconstriction is a key mechanism for maintaining blood pressure in a vertical position, more significant than an increase in heart rate. Rapid short-term adaptation to orthostatic stress is carried out exclusively by ANS [4]. The normal adaptive response to the transition to an upright position lasts approximately 60 s. Longer hemodynamic changes in orthostasis are considered pathological . Neuroendocrine systems are also involved in the regulation of orthostasis , primarily the renin-angiotensin-aldosterone system. The violation of the mechanisms of adaptation to vertical position at any level leads to orthostatic instability - the inability of the body to maintain a vertical position [5,6]. Vegetative dysregulation is based on a violation of noradrenergic neurotransmission, in which postganglionic sympathetic neurons do not release sufficient amounts of noradrenaline. The decreased release of neurotransmitters leads to a violation of vasoconstriction, an inadequately low increase in heart rate and a decrease in the volume of circulating blood (BCC) - all this contributes to the development of hypotension [7-11]. Any disease in which peripheral vegetative fibers are affected can lead to the development of autonomous dysregulation. An important characteristic of autonomic activation disorders is the absence of a damping effect of baroreflex. Baroreflex plays a priority role in the regulation of blood pressure [12]. Baroreflexive failure occurs in people suffering from hypertension, especially malignant and resistant hypertension; those who have undergone trauma, radiation, surgical operations, in persons with cancer of the oropharyngeal region and in the elderly [13]. With baroreflexive inconsistency, not only the protective function is lost in relation to stimuli that cause excessive fluctuations in blood pressure, but also ordinary, everyday stressors lead to inadequate changes in blood pressure. This lability of the blood pressure control system is manifested by a pronounced decrease in blood pressure in the heat or in hot places, during physical exertion or eating. For example, after eating, blood pressure in healthy people decreases by 1 mmHg, whereas in patients with reflex failure - up to 40 mmHg [14,15]. On the contrary, a glass of water drunk in these patients can cause a rise in blood pressure to 40 mm Hg [15-17]. The antihypertensive drug clonidine with autonomic insufficiency can cause a periodontal increase in blood pressure due to hypersensitivity -adrenoreceptors and of baroreflexive insufficiency [16]. With a decrease in BCC or medically induced OH, the ANS is relatively intact. In these cases , in the pathogenesis of development The reduced BCC and hemodynamic effects of drugs are important. Pronounced tachycardia during the transition to an upright position is characteristic of the exhaust gas with a reduced BCC [13]. Taking

antihypertensive drugs, especially diuretics, is considered as one of the main provoking factors of OH, often reducing their dose or cancellation contributes to the disappearance of symptoms [18-20].

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