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ПОСТПРАНДИАЛЬНАЯ ГИПОТЕНЗИЯ У ПОЖИЛЫХ ПАЦИЕНТОВ: ПАТОФИЗИОЛОГИЯ, ДИАГНОСТИКА И МЕРЫ ПРОФИЛАКТИКИ

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Postprandial Hypotension in Elderly Patients: Pathophysiology, Diagnosis and Prevention Measures

Резюме

Постпрандиальная гипотензия (ППГ) является важным, но недостаточно распознаваемым состоянием, возникающим в результате неадекватной компенсаторной реакции сердечно-сосудистой системы на индуцированное приемом пищи висцеральное скопление крови. ППГ признана важной клинической проблемой поскольку имеет высокую распространенность в популяции старшей возрастной группы и связана с развитием сердечно-сосудистых осложнений и гериатрических синдромов. Возможные патофизиологические механизмы ППГ: повышенный висцеральный кровоток; ослабление барорефлекторной функции из-за нарушений, связанных с возрастом или вегетативной дисфункцией; неадекватная активация симпатических нервов; нарушение регуляции вазоактивных кишечных пептидов; инсулин-опосредованная вазодилатация. Опрос о симптомах гипотонии после приема пищи и снижение систолического артериального давления (АД) на ≥20 мм рт. ст. через 15- 60 минут после еды имеет первостепенное значение для постановки диагноза ППГ. Одной из основных стратегий профилактики ППГ является снижение растяжения желудка (небольшие порции пищи и более частое питание), отдых лежа на спине после еды, употребление достаточного количества воды. Ходьба после приема пищи, по-видимому, также помогает восстановить АД после еды. Необходимо проявлять осторожность при назначении белковых добавок у пожилых людей, модифицировать диету путем замены высокопитательных подсластителей низкокалорийными (d-ксилоза, ксилит, эритрит, мальтоза, мальтодекстрин и тагатоза). Метформин или акарбоза модулируют сердечно-сосудистую реакцию у пациентов с сахарным диабетом, уменьшают постпрандиальную гипотензию. Таким образом, ППГ является достаточно распространенным и клинически значимым феноменом у пожилых больных. Повышение информированности врачей о патофизиологии и методах диагностики, профилактики позволит повысить эффективность и безопасность ведения гериатрических пациентов.

Ключевые слова: *постпрандиальная гипотензия, патофизиология, пожилой пациент, профилактика падений*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

Postprandial hypotension (PPH) is an important but underrecognized condition resulting from an inadequate compensatory cardiovascular response to meal-induced visceral blood pooling. PPG is recognized as an important clinical problem because it has a high prevalence in the older age group and is associated with the development of cardiovascular complications and geriatric syndromes. Possible pathophysiological mechanisms of PPG: increased visceral blood flow; weakening of baroreflex function due to disorders associated with age or autonomic dysfunction; inappropriate

activation of sympathetic nerves; dysregulation of vasoactive intestinal peptides; insulin-mediated vasodilation. Ask about symptoms of postprandial hypotension and a decrease in systolic blood pressure (BP) of ≥20 mm Hg. Art. 15-60 minutes after eating is of paramount importance for making a diagnosis of PPG. One of the main strategies for preventing PPG is to reduce gastric distension (small meals and more frequent meals), resting on your back after eating, and drinking enough water. Walking after eating also appears to help restore blood pressure after eating. Caution should be exercised when prescribing protein supplements in the elderly, modifying the diet by replacing high-nutrient sweeteners with low-calorie sweeteners (d-xylose, xylitol, erythritol, maltose, maltodextrin, and tagatose). Metformin or acarbose modulates the cardiovascular response in patients with diabetes mellitus and reduces postprandial hypotension. Thus, PPG is a fairly common and clinically significant phenomenon in elderly patients. Increasing the awareness of doctors about pathophysiology and methods of diagnosis and prevention will improve the efficiency and safety of managing geriatric patients.

Key words: *postprandial hypotension, pathophysiology, elderly patient, prevention of falls*

Conflict of interests
The authors declare no conflict of interests

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AH — arterial hypertension, BP — blood pressure, DBP — diastolic blood pressure, PPH — postprandial hypotension, SBP — systolic blood pressure, DM — diabetes mellitus, CVD — cardiovascular diseases, GLP-1 — glucagon-like peptide-1, GIP — glucose-dependent insulinotropic polypeptide

Introduction

Postprandial hypotension (PPH), a drop in systolic blood pressure (SBP) of at least 20 mm Hg after meals, is an important, but under-diagnosed condition, which is a result of an inadequate compensatory cardiovascular response to meal-induced visceral blood accumulation.

PPH is a recognised clinical issue due to its high incidence in elderly population [1, 2]. PPH affects 24–33 % of elderly people in care homes, 67 % of geriatric patients and about 50 % of people with unexplained syncope [3]. In inpatient patients, the incidence of PPH is 30.4 % [4]. A meta-analysis (2024) of the data from 3,021 subjects demonstrated that the PPH incidence in elderly people was 40.5 % [5]. PPH is most common in conditions associated with vegetative dysfunction. For example, in type 2 diabetes mellitus (DM) [6], the incidence of PPH is likely to be higher than that of orthostatic hypotension [7].

Some authors suggested a correlation between PPH and cardiovascular disease and mortality [8]. A prospective study in elderly population with AH showed that 83 % of patients admitted for PPH had cerebrovascular damages [9]. A prospective 36-month study demonstrated association between PPH and CVD development (adjusted risk factor: 11.18, 95 % confidence interval: 2.43–51.38, p = 0.002), which did not disappear even after consideration of other variables [4]. The maximum drop in postprandial blood pressure (BP) is an independent predictor of later falls, syncope, cardiovascular events (myocardial infarction and stroke), and general mortality [10, 11]. In a cohort study in 401 elderly patients with outpatient AH, 72.8 % of subjects had PPH, while falls after breakfast were the most powerful predictor of deaths in this cohort [10].

Methods of Literature Source Search

PubMed databases in Russian and English were used for a full-text search (automated search of documents, when a search is based not on document titles, but on their contents, both the entire contents and its part) with keywords (postprandial hypotension, elderly age, pathogenesis, risk factors of postprandial hypotension), with a 5-year period of time limitation (duplicate articles and non-full-text articles were excluded).

Pathophysiology of PPH

The pathophysiology of PPH is multifactorial and is understudied. PPH development points to an inadequate cardiovascular response to meals, which is a result of complex interactions between consumed nutrients and gastrointestinal tract. There are convincing evidences that gastrointestinal factors, such as meal composition, nutrient delivery rates to the small intestine (i.e. stomach emptying), nutrient absorption, are an integral part of the postprandial blood pressure response. Possible mechanisms of PPH (see Fig. 1):

1. Increased visceral blood flow
2. Reduced baroreflex function due to disorders associated with the age or vegetative dysfunction
3. Inadequate sympathetic nerve activation
4. Inadequate regulation of vasoactive intestinal peptides
5. Insulin-mediated vasodilation.

Healthy elderly people have more marked haemodynamic reactions to meals vs. healthy young people; higher, age-related noradrenaline levels are released, which causes a more marked haemodynamic reaction to meals, despite stable BP values (see Table 1).

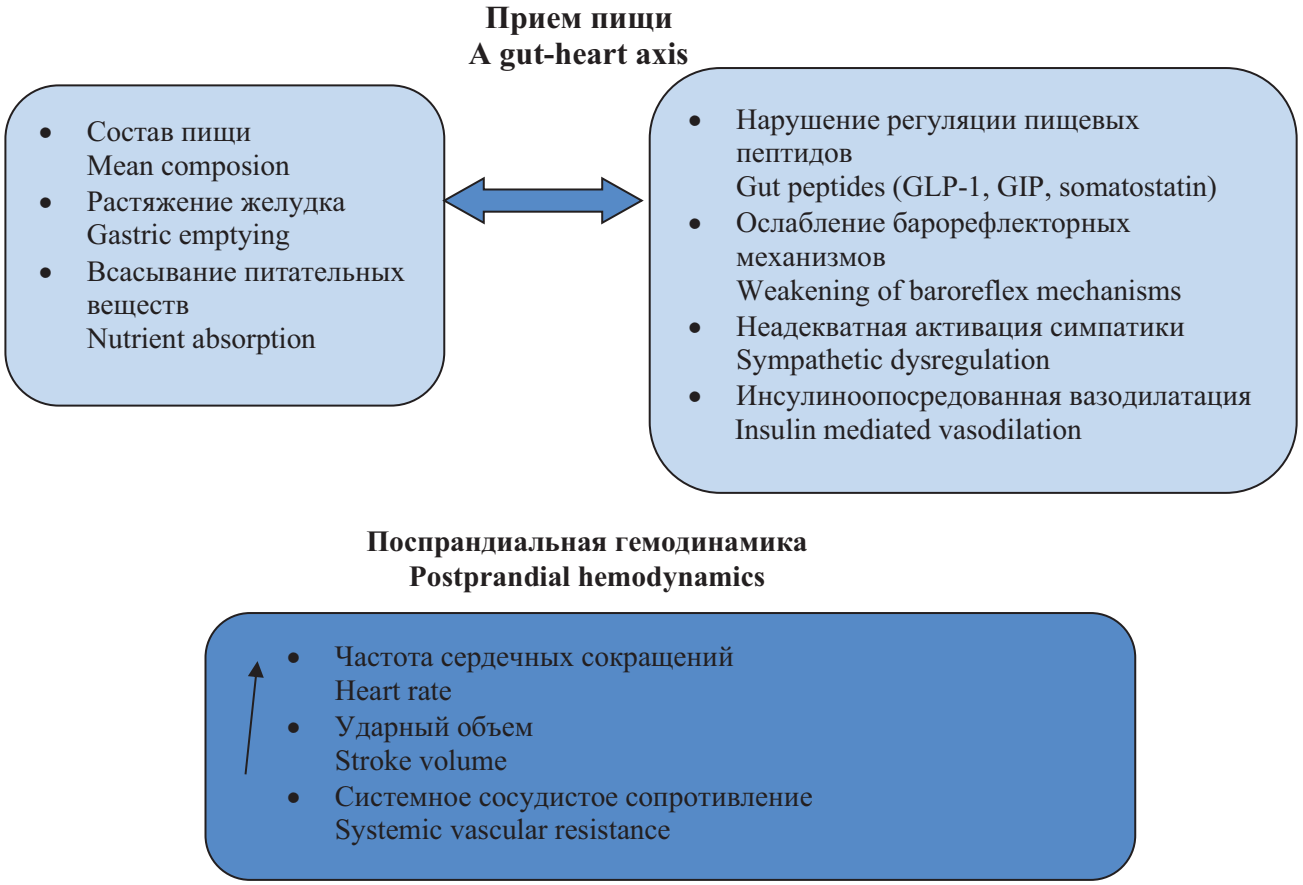

Figure 1. *Postprandial hemodynamic response (model)*
Abbreviations: GLP-1 — glucagon-like peptide 1, GIP — glucose-dependent insulinotropic polypeptide

Table 1. *Age-dependent physiological and pathophysiological postprandial reactions*

	Young healthy	Elderly healthy	PPG
Heart rate	↑	↑	↑↑
Arterial pressure	↔	↔ or ↓	↓↓
Norepinephrine in plasma	↔	↑	↔
Vascular response to norepinephrine	↑	↔ or ↓	↓↓

Abbreviations: PPG — postprandial hypotension, ↑ — increase, ↓ — decrease, ↔ — no change.

It has been demonstrated that carbohydrates, fats and proteins cause various haemodynamic reactions. For instance, in elderly people with AH, oral carbohydrates can reduce mean AH values faster than fats or proteins. In healthy elderly people, intraduodenal glucose administration reduced SBP and DBP more rapidly vs. low-calorie fat and protein, as well as led to a lower increase in flood flow in the superior mesenteric artery, which is an artificial indicator of visceral blood deposition as a response to intraduodenal protein [13]. Variable haemodynamic responses to macronutrients can be evidence of differences in neurohormonal profiles. For instance, fats vs. carbohydrates are a more potent stimulus for the secretion of two incretin hormones: glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP) [14].

The rate of nutrient delivery to the small intestine is strictly controlled by gastric emptying, which is very individual [15]. There are evidences that stomach emptying is a major factor affecting response of the blood pressure to meals. For example, postprandial drop in SBP is higher when the stomach is emptied relatively more rapidly, thus reflecting a stronger association between nutrients in small intestine and visceral blood accumulation [16]. However, the association between intestinal emptying and postprandial BP is not linear; in healthy elderly people, BP reduction increased with a higher rate of intraduodenal glucose infusion

from 1 to 2 kcal/min, but did not differ between 2 and 3 kcal/min [17].

Nutrient absorption in the small intestine also affects the postprandial dynamics. Interventions, which inhibit the rate of carbohydrate absorption in the small intestine, such as intake of alpha-glucosidase inhibitors, acarbose, are associated with reduced visceral deposition of blood and less pronounced drop in SBP in healthy elderly people [13]. It has been demonstrated that exposure to glucose in the duodenum results in a more marked SBP reduction and more intensive blood flow in the superior mesenteric artery vs. iliac artery, together with more rapid glucose absorption, higher GIP release and lower GLP-1 secretion [17].

After meals, the blood flow in the superior mesenteric artery doubles; and in healthy young people with preserved baroreflex function, an increase in the visceral blood flow is associated with an increase in the heart rate, peripheral vascular resistance, systolic discharge, and cardiac output. In patients with PPH, these compensatory responses are inadequate, postprandial drop in BP is more prominent, when stomach emptying is faster [18], while distended stomach lowers PPH both in young and elderly subjects [19]. Usually, a drop in the system blood flow as a result of visceral vasodilation is compensated by a combination of an increase in cardiac output resulting from higher heart rate and/or systolic discharge, and a higher systemic vascular resistance [8]. Cardiovascular responses to meals involve numerous neurohormonal mechanisms. Stomach distension after meals triggers a gastrovascular reflex including stimulation of noradrenaline secretion, which boosts sympathetic neural activity. This response is often subsided in elderly people, especially those with PPH. Modulation of secretion or signalling of these intestinal peptides can significantly affect the blood pressure response to meals, bringing about potentially new targets for PPH therapy [9].

PPH pathophysiology includes sympathetic dysfunction associated with vegetative neuropathy (e.g. in Parkinson disease, DM and heart failure) and causing reduced baroreceptor reflex. These patients are unable to increase the heart rate in response to abrupt reduction in BP when visceral blood flow increases as a result of postprandial vasodilatation of gastrointestinal vessels [20].

Intestinal peptides, especially GLP-1, GIP and somatostatin, can significantly impact postprandial haemodynamic responses [21]. GLP-1 stimulates insulin secretion, inhibits glucagon secretion and slows down stomach emptying. It has been shown that GLP-1 infusion mitigates BP drop after oral or intraduodenal glucose administration.

Genetic susceptibility to postprandial BP dysregulation remains understudied. Although some scientists demonstrated correlation between polymorphism of beta-adrenergic receptor genes and orthostatic BP dysregulation in patients with AH [22], additional studies are required to characterise any PPH-associated genetic susceptibility.

Diagnosis of PPH

Asking about symptoms of hypotension after meals is vital for correct diagnosis of PPH. Some patients may have asymptomatic PPH; however, the most common signs and symptoms of PPH are motive weakness, dizziness, delirium, syncope, falls, angina, nausea and vision disorders; also, patients may be unable to stand or walk after meals [22]. There is evidence of transient ischaemic attacks in elderly patients, who had significantly reduced postprandial BP, with the symptoms disappearing when BP values return to normal. Cerebral symptoms ~~depend on or~~ depend on the characteristics of cerebral hypoperfusion [3].

PPH is preferably diagnosed with outpatient BP monitoring. Baseline BP and heart rate before meal (after a 5-minute rest) are measured; BP and heart rate are then measured every 10 minutes for about two hours. Diagnostic reduction in BP (a drop in SBP by ≥ 20 mm Hg) is usually diagnosed 15 minutes after meals in 15 % of patients with PPH and in 30–60 minutes in 70 % of patients. During tests, there were no limitations in food or caloric value; however, it might be preferable to use low-carb test food because of the impact from insulin-induced reactive hypoglycaemia. Intrasubject reproducibility of PPH is quite high, therefore, a single test is enough to diagnose this condition. Diagnostic procedures performed in the morning can be more efficient [22].

Risk factors of PPH

1. It has been demonstrated that delayed stomach emptying with moderately distended stomach causes a 200 % increase in the sympathetic nervous system activity [23]. This activation of sympathetic signalling can be efficient in the maintenance of postprandial BP. Abundant meals are highly likely to cause a drop in BP as compared to a light meal.

2. Fluid volume deficit in elderly can make patients susceptible to PPH.

3. BP response to sweeteners is usually unchanged in healthy young people; however, in elderly people, glucose causes the highest drop in postprandial BP, whereas response to sucrose is less pronounced [24].

4. The strategy, which becomes more common in prevention or therapy of malnutrition, weight loss and sarcopenia in elderly, involves consumption of high-energy, protein-rich supplements [18]. Oral protein or supplements rich in serum protein can lower BP to the point, where some elderly people face the risk of fall. The hypotensive effect of proteins is likely to be mediated by amino acids produced during digestion; it can explain the latent period and onset of changes in BP and heart rate after protein load. Consumption of 70 g of a serum protein drink is associated with a significant drop in BP in healthy elderly males; the majority of elderly subjects

had lower systolic BP (SBP) by 20 mm Hg or more, and the highest drop was observed 2–3 hours after the drink [25]. It is unclear whether the hypotensive effect of serum protein drinks is dose-dependent in elderly people, and whether serum doses below 70 g cause a significant drop in BP [25].

PPH prevention

It is worth noting that currently there is no efficient and safe PPH management strategy [6, 26]. Nevertheless, several methods were proposed to reduce the PPH risk:

1. One of the main therapeutic strategies is elimination of distended stomach in order to delay digestion products from entering the small intestine. Smaller portions were associated with a drop in postprandial BP by 11–20 mm Hg [23]. Therefore, it is advisable to regulate food intake in patients with PPH by consuming smaller portions at smaller intervals.

2. Symptomatic patients should also rest after meals lying flat on their backs, because standing or sitting tend to have additional hypotensive effect [9].

3. Sufficient hydration also facilitates protective stomach distension and delayed emptying: 350–480 mL of water increases BP by 20 mm Hg in patients with vegetative insufficiency [13].

4. In elderly people, who consumed 60 mg of caffeine (in tea or coffee) five times a day, SBP was 4 mm Hg higher without any impact on the baseline systolic BP [13]

5. Walking is likely to help to restore BP after meals. The mean blood pressure increased by 18 ± 4 mm Hg during exercises after the meal, but dropped 10 minutes later to the pre-exercise level [24]. It means that exercises after meals can be useful in preventing PPH.

6. Caution may be required when prescribing protein supplements in elderly people, and their haemodynamics should be monitored. Measures (e.g. standing position) should be recommended to reduce harmful effects of excessively reduced BP after meals. Diet modifications — replacing highly-nutritious sweeteners (glucose, fructose and sucrose) with low-calorie ones (d-xylose, xylitol, erythritol, maltose, maltodextrin, and tagatose) and calorie-free sweeteners — can be a simple, yet efficient PPH therapy.

7. Metformin modulates cardiovascular response to intraduodenal glucose in patients with DM2 and reduces postprandial hypotension. Mechanisms, by which metformin attenuates hypotension resulting from oral glucose, needs to be identified [27].

8. It has been demonstrated that delayed stomach emptying, e.g. consumption of food fibre or acarbose, slows down SBP drop after consumption of carbohydrate-rich food in healthy elderly people and DM2 patients [28]. Data from a meta-analysis (Wang B., 2021) show that acarbose attenuates drops in postprandial systolic and diastolic BP and is therefore efficient in PPH prevention. Acarbose inhibits enzymes, which are required to digest carbohydrates, reduces the amount of carbohydrate products to the duodenum and potentially delays stomach emptying. Moreover, inhibition of enzymes required for carbohydrate digestion in the stomach reduces release of intestinal peptides, such as vasoactive intestinal peptide, which mediates visceral vasodilation [29].

Conclusion

In spite of the common medical idea, the phenomenon of BP drops after meals is a common event, especially in elderly and old people. PPH is a clinically significant event associated with the risk of cardiovascular complications and geriatric syndromes (including the risk of fall, osteoporosis, sarcopenia). Epidemiological data show the low rate of PPH diagnosis in clinical practice; at the same time, the condition can be easily diagnosed, given the widespread introduction of outpatient blood pressure measurements. Awareness-building among medical professionals about pathophysiology and methods for diagnosis and prevention can boost efficiency and safety of geriatric patient management. Up-to-date knowledge of prevention and account of respective individual features of the patient make it possible to significantly enhance clinical efficiency and safety of antihypertensive therapy and to improve the quality of life for geriatric patients.

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Антропова О.Н.: разработка дизайна и написание рукописи, редактирование статьи, поиск литературных источников, утверждение финального варианта рукописи

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Author Contribution:

All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication

Antropova O.N.: development of the design and writing of the manuscript, editing the article, search for literary sources, approval of the final version of the manuscript

Efremushkina A.A.: development of the concept, search for literary sources, editing the article, approval of the final version of the manuscript

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