Review article

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Exaggerated Blood Pressure Response to Physical Load among Youths

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Hypertension and impaired blood pressure regulation may manifest in childhood, making the hemodynamic response to physical load a potential indicator of increased cardiovascular risk. This review outlines the physiological basis, defining criteria, prevalence, risk factors, pathophysiological mechanisms, and clinical significance of exaggerated blood pressure response to physical load (EBPR) in youths. No universally accepted pediatric definition exists, while differences in testing protocols, peak intensity, measurement methods, and percentile thresholds limit comparability between studies. The magnitude of the response is influenced by age, sex, pubertal status, height, body composition, cardiorespiratory fitness, and familial predisposition. Its pathophysiological basis likely involves interconnected endothelial dysfunction, sympathetic hyperactivity, activation of the renin–angiotensin–aldosterone system (RAAS), oxidative stress, insulin resistance, and arterial stiffness. EBPR is associated with masked hypertension, future sustained hypertension, and early indicators of target-organ damage, although its independent prognostic value in youths remains uncertain. Therefore, EBPR should not be considered an independent diagnosis but interpreted within the overall cardiometabolic risk profile. This means taking into account test quality and repeated blood pressure measurements. Ambulatory blood pressure monitoring (ABPM) should be considered when additional indications are present. Isolated EBPR does not provide a sufficient basis for pharmacological treatment or restriction of sports participation. Further clarification of its clinical

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and prognostic significance requires standardized testing protocols, age-specific reference values, and longitudinal studies.

Key words*: masked hypertension, endothelial dysfunction, body composition, cardiometabolic risk.*

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PREKOMERNI PORAST ARTERIJSKOG KRVNOG PRITISKA TOKOM FIZIČKOG OPTEREĆENJA

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Arterijska hipertenzija i rani poremećaji regulacije krvnog pritiska mogu se ispoljiti već u detinjstvu, zbog čega se hemodinamski odgovor na fizičko opterećenje razmatra kao mogući pokazatelj povećanog kardiovaskularnog rizika. Cilj ovog preglednog rada bio je da se prikažu fiziološke osnove, kriterijumi definisanja, učestalost, faktori rizika, patofiziološki mehanizmi i klinički značaj prekomernog porasta arterijskog krvnog pritiska tokom fizičkog opterećenja (EBPR) kod dece i adolescenata. Dosadašnji rezultati istraživanja pokazuju da ne postoji univerzalno prihvaćena pedijatrijska definicija, dok razlike u protokolima testiranja, dostignutom intenzitetu, načinu merenja i primenjenim percentilnim pragovima ograničavaju uporedivost istraživanja. Na izraženost odgovora utiču uzrast, pol, pubertetski status, telesna visina, telesni sastav, kardiorespiratorna kondicija i porodična predispozicija. U patofiziološkoj osnovi najverovatnije se nalaze međusobno povezana endotelna disfunkcija, simpatička hiperaktivnost, aktivacija sistema renin–angiotenzin–aldosteron, oksidativni stres, insulinska rezistencija i arterijska krutost. EBPR može biti udružen sa maskiranom hipertenzijom, rizikom od kasnije perzistentne hipertenzije i ranim pokazateljima oštećenja ciljnih organa, ali njegova nezavisna prognostička vrednost kod mladih još nije pouzdano utvrđena. Zbog toga ovaj nalaz ne treba tumačiti kao samostalnu dijagnozu, već u okviru ukupnog

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kardiometaboličkog rizika, uz proveru kvaliteta testa, ponovljena merenja i, kada postoje dodatne indikacije, ambulantno praćenje krvnog pritiska. Izolovani nalaz EBPR ne predstavlja dovoljan osnov za uvođenje farmakološkog lečenja, niti ograničavanje sportskih aktivnosti. Dalji napredak u razumevanju kliničkog i prognostičkog značaja EBPR zahteva standardizaciju protokola testiranja, utvrđivanje uzrasno prilagođenih referentnih vrednosti i sprovođenje longitudinalnih istraživanja.

Ključne reči: *maskirana hipertenzija, endotelna disfunkcija, telesni sastav, kardiometabolički rizik.*

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Introduction

Hypertension is no longer a disease of adulthood, since the onset of functional and structural changes can be traced back to childhood. As a result, in addition to measuring resting blood pressure and 24-hour ambulatory blood pressure monitoring (ABPM), the hemodynamic response to physical load has also received considerable attention. In healthy individuals, dynamic physical activity causes a gradual increase in systolic pressure, while diastolic pressure remains relatively stable or decreases slightly. A disproportionate increase in blood pressure is known as an exaggerated blood pressure response to load (EBPR). Systematic reviews of longitudinal studies indicate that hypertension in childhood and adolescence is associated with the greater likelihood of hypertension and adverse cardiovascular outcomes in adulthood (1-3). EBPR is regarded as a possible early indicator of impaired vascular regulation and could be associated with masked or later persistent hypertension. Among adults it is associated with hypertrophy of the left ventricle, arterial stiffness, and increased cardiovascular risk (2,3). Although evidence in youths (children and adolescents) remains limited, available data suggest that certain pathophysiological processes may already be present during childhood and adolescence Data compiled for youths (children and adolescents) are more limited but indicate that certain pathophysiological processes can manifest as early as the period of growth and development (1,4). The rate of systolic blood pressure increase, the relationship between blood pressure and total work performed, and blood pressure values during submaximal exercise and recovery may also be clinically relevant. Still, EBPR is not a diagnosis, nor does its presence necessarily indicate any progression towards hypertension, which is why it should be analyzed in the context of the developmental characteristics of youths and other risk factors.

The hemodynamic response to physical load

At the onset of physical activity, there is an increase in the activity of the sympathetic, and a decrease in the activity of the parasympathetic nervous system. As a result, heart rate, contractility, stroke volume, and cardiac output increase (5,6). At the same time, local vasodilatation occurs in the skeletal muscles, mediated by nitric-oxide (NO) and metabolic vasoactive substances, which in turn decreases the overall peripheral resistance. As a result, systolic pressure rises progressively, while diastolic remains unchanged or slightly elevated. In turn, the baroreflex physiologically adapts to a higher operating blood pressure, enabling suitable perfusion of the active

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muscles (5,6). Basic mechanisms among youths are similar to those of adults, but the increase of cardiac output is more conditioned by the rise in heart rate due to reduced stroke volume. More elastic arteries, lower peripheral resistance, and preserved endothelial function contribute to a more moderate elevation in blood pressure. Therefore, analyzing any results must take into consideration age, gender, height, peak achieved intensity of the physical load, and pubertal status (7,8). Type of physical load and type of muscle contractions also determine the hemodynamic pattern of response. During dynamic contractions, which occur during aerobic work, the increase in cardiac output is accompanied by vasodilatation in the active muscles, while static contractions and exercise loads with a more pronounced strength component can lead to a more significant increase in systolic and diastolic pressure due to the mechanical compression of blood vessels and increased peripheral resistance (7-9). For a clinical interpretation, the peak achieved intensity of the load, confirmation that maximal exercise capacity was achieved, the amount of the muscle mass engaged, body position, and blood pressure measurement techniques are all significant (9).

The criteria for and limitations of defining EBPR

A universally accepted definition of EBPR for youths does not exist. The existing literature mostly refers to a maximum systolic pressure above the 95th or 97th percentile for age and gender, an increase in blood pressure greater than expected for the peak achieved intensity of physical load, increase in blood pressure per unit of work or the relationship between increase in blood pressure and oxygen consumption (1). An absolute threshold is insufficient, since the same value can be physiological in the case of older adolescents and elevated among younger youths (1,10). A comparison of existing studies is additionally complicated by different protocols on the treadmill and bicycle ergometer, methods and measurement intervals, as well as different levels of exercise intensity achieved. Values achieved on the treadmill can be the result of the engagement of a larger muscle mass. As a result, standardized testing protocols and pediatric nomograms adjusted for age, gender, body height, and load are needed. Assessment based only on peak blood pressure overlooks the dynamics of response during a test. Clinical significance can also be achieved by the rate of systolic pressure increase, the relationship between blood pressure and total work performed, values in submaximal phases, and recovery (10). A submaximal response could better reflect hemodynamic load during typical physical activity, while a slow recovery can indicate prolonged sympathetic activation. However, even for these indicators there are still no sufficiently reliable baseline values for youths (1,10).

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Blood pressure measurement during testing must also be standardized. Auscultatory blood pressure measurement can be affected by noise, vibration, and movement of the extremities, while automatic devices are not equally validated for all ages and exercise intensity (11,12). The size of the cuff, the measurement site, and rounding off values can have a significant impact on the classification of a youth whose score is close to the percentile threshold value. Future standardization should include not only the criteria for EBPR, but also equipment, stages of measurement, conditions prior to testing, and the manner in which the values obtained are expressed (11,12). Previous physical activity, anxiety, sleep, environmental temperature, intake of stimulants (various forms of caffeine and energy drinks), and level of dehydration could all affect the response to physical load. Motivation and understanding of the protocol determine the intensity achieved and test duration, especially among youths. Consequently, results which deviate from those expected should primarily be assessed in relation to the quality of the testing procedure, and if the result can alter the clinical decision, it should be verified by means of repeat testing in comparable conditions. Without data on within-subject variability, it is difficult to distinguish between a stable phenotype and a transitory response (1).

Frequency and factors associated with EBPR among youths

The actual frequency of EBPR among youths has not been determined accurately due to varying definitions, protocols, and characteristics of the samples. The percentage of healthy children and adolescents with values which in certain studies were classified as high or excessive was approximately 5–14%; however, the assessments obtained cannot be compared directly, and the frequency is greater among youths with cardiovascular risk factors. EBPR can be an isolated result, but for a subgroup of the youth population it could represent an early measurable sign of impaired blood pressure regulation (1,7,13-15).

Maximum systolic blood pressure rises with age, body height, muscle mass, and cardiac output, so percentile ranges are more suitable than unique baselines. Gender differences become more pronounced in puberty: boys on average achieve higher values due to higher stroke volume and cardiac output, while estrogen in girls can have a positive effect on endothelial function. These differences cannot be viewed separately from pubertal maturation and change in body composition, since an increase in lean body mass and cardiac volume can lead to elevated physiological systolic pressure, while an increase in body fat can indicate an unfavorable metabolic profile. As a result, normative values should distinguish between the effects of growth, biological maturity, and adiposity

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instead of being based solely on chronological age (7,8). The descendants of individuals with hypertension can have a pronounced sympathetic response, lower vasodilatory reserve or adverse vascular structural alterations even prior to elevated resting blood pressure. However, a family anamnesis also includes shared dietary patterns, physical activity patterns, and exposure to stress. As a result, it should be interpreted as an important unifying indicator of risk, and not as proof of a solely genetic mechanism (1).

In the case of young athletes, physiologically elevated values of systolic blood pressure occur due to increased cardiac output and should not be confused with a disproportionately higher response which cannot be explained by achieved exercise intensity, physical characteristics, and cardiorespiratory fitness levels. That is why the results of ergometric tests should be interpreted in the context of fitness (physical fitness), symptoms, and other clinical findings (14,16,17). Low fitness levels can affect interpretation: with the same absolute load, a youth with lower fitness levels will exercise at a higher relative intensity, which naturally causes a more pronounced hemodynamic reaction. A comparison based on peak blood pressure achieved without normalizing for work capacity can therefore overestimate the risk. The relationship between blood pressure and load, i.e. oxygen consumption, theoretically provides a more precise assessment, but requires a standardized cardiopulmonary test and age-appropriate baseline data (10,16).

The pathophysiological basis of EBPR

EBPR most likely emerges through the interaction of impaired vasodilatation, exaggerated cardiac and sympathetic responses, and unfavorable vascular anatomy. The relative contribution of these mechanisms differs among individuals, which can in part explain the heterogeneity of clinical manifestations (18,19). The endothelium is key to the adaptation of vascular tonus to increased blood flow. Shear stress during load activates the endothelial NO-synthase and increases NO production, which in turn relaxes smooth muscle cells and decreases peripheral resistance. Once this mechanism is weakened, vasodilatation does not compensate for the increase in cardiac output. Reactive oxygen species additionally inactivate NO and activate inflammatory pathways, so functional abnormality over time can be associated with the proliferation of smooth muscle cells and the remodeling of the vascular wall. An excessive sympathetic reaction simultaneously increases cardiac output and limits the physiological reduction of peripheral resistance. Activation of the Renin-Angiotensin-Aldosterone System (RAAS), in particular the activity of angiotensin II, increases vasoconstriction, the secretion of aldosterone, and oxidative stress. This creates a feedback loop in

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which the sympathetic activation, RAAS, endothelial dysfunction, and oxidative stress mutually sustain an unfavorable hemodynamic response (20,21).

Insulin resistance and chronic low-grade inflammation can additionally be associated with metabolic and vascular risk. Hyperinsulinemia promotes the retention of sodium and sympathetic activity, while pro-inflammatory cytokines disrupt the signal pathways dependent on NO (20). The outcome is not simply a greater peripheral resistance during load but also a gradual decrease in vascular compliance. The mechanisms described can explain why EBPR is more frequently being detected among youths with central obesity and metabolic disorders, but do not prove it is a consequence of the same pathophysiological process in each youth (19). In the case of one youth, the deciding factor could be an exaggerated cardiac response, while in another limited peripheral vasodilatation, and in a third a combination of adiposity, poor physical fitness, and elevated baseline blood pressure values. This heterogeneity explains why baseline assessment has limited specificity and why the approach to assessment and monitoring cannot be the same for all youths. Phenotyping based on heart rate, physical fitness, body composition, ABPM, and indicators of vascular function could identify clinically different subgroups; however, such approaches are still not ready for routine implementation (16,19,22).

Increased major artery stiffness reduces the ability to absorb the pulsatile energy and speeds up the return of the reflected pulse wave, whereby the central aortic pressure and left ventricular load increase. This can be a two-way relationship: stiffness could contribute to a more exaggerated blood pressure response, while the repeat episodic elevations in blood pressure could promote vascular remodeling. Yet, it is still unclear whether EBPR is an independent functional abnormality or a manifestation of an already initiated subclinical vascular dysfunction, nor has it been confirmed that it represents consecutive phases of the same pathophysiological process (1,19).

The prognostic significance of EBPR and the limitations of the available evidence

The prognostic significance of EBPR must be assessed along with baseline blood pressure values, obesity, family anamnesis, physical fitness, insulin resistance, and other risk factors. Future studies which involve follow-up, beginning in childhood and lasting into adulthood, should determine the extent to which EBPR provides additional prognostic information compared to the already available clinical indicators. It is particularly important to make a distinction between prediction and causality. The association between EBPR and subsequent hypertension can indicate a shared underlying basis, such as hereditary predisposition, adiposity, or early vascular aging, and not

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necessarily an independent adverse effect of episodes of elevated blood pressure (23). In one of the largest longitudinal studies of cardiovascular risk, greater exposure to elevated blood pressure during the course of one's life was associated with a more exaggerated blood pressure response to physical load in adulthood. This finding supports the existence of a developmental continuum of blood pressure regulation but does not prove that EBPR in childhood independently causes hypertension later in life (23).

The association between body composition and EBPR

Obesity is one of the most consistently described factors associated with EBPR. Adipose tissue functions as an endocrine organ: adipokines and pro-inflammatory mediators promote sympathetic activation, oxidative stress, insulin resistance, and endothelial dysfunction. As a result, youths with a higher body mass index (BMI) more often achieve higher blood pressure values as a response to physical load (1,16). However, BMI does not distinguish between adipose and lean tissue, does not indicate the distribution of fat tissue, and might not accurately reflect cardiometabolic risk. Body composition analysis may therefore help identify individuals at higher cardiometabolic risk among youths with similar BMI values; however, the optimal assessment methods and clinically relevant cutoff values remain to be determined. Total body fat is associated with increased production of leptin, tumor necrosis factor α, and interleukin-6, while visceral fat tissue, compared to subcutaneous fat tissue, is strongly associated with an unfavorable adipokine profile, chronic low-grade inflammation, dyslipidemia, insulin resistance, and an activated RAAS. These mechanisms can simultaneously increase sympathetic tonus, circulating blood volume, vascular resistance, reduce NO bioavailability and promote arterial stiffness. Increased body mass also affects the blood pressure response by an increase in metabolic demand during movement. That is why higher blood pressure values during a standard protocol can in part be a consequence of greater absolute work, and not solely vascular dysfunction (1,16,19). A combination of waist circumference, percentage of body fat, indicators of visceral fat, and BMI values can provide more detailed information than any single measure. Future longitudinal studies should determine whether changes in fat and lean mass, as well as improvement in cardiorespiratory fitness, are associated with a more favorable blood pressure response to load, irrespective of changes to the BMI. The results of a two-year follow-up study of adolescents additionally point to the fact that adiposity mediates the longitudinal association of cardiorespiratory fitness and subsequent blood pressure

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values. These results indicate that physical fitness and body composition cannot be considered mutually independent determinants of hemodynamic risk (24).

EBPR, masked hypertension, and target-organ damage

EBPR is associated with masked hypertension, wherein the blood pressure measured in the doctor's office is normal, while it is elevated during everyday activities or during ABPM. Physical load in that sense can detect a disorder which is not visible at rest, probably due to partially shared mechanisms, including sympathetic hyperactivity, endothelial dysfunction, and increased arterial stiffness. In certain studies, a more exaggerated blood pressure response to load is associated with increased left ventricular mass and unfavorable vascular anatomy, which can be early indicators of increased hemodynamic load (25,26). Still, these findings were not consistently confirmed and can be influenced by obesity, baseline blood pressure values, and physical fitness. Therefore, in the case of youths, it cannot be concluded that EBPR directly leads to structural and functional changes, or that the hypothesized continuum from EBPR, via masked and persistent hypertension, to target-organ damage has sufficiently been confirmed by longitudinal studies. A systematic review and meta-analysis dating from 2023 indicated that masked hypertension in the general pediatric population was found in approximately 10% of the participants and is associated with a higher left ventricular mass index, more frequent left ventricular hypertrophy, and increased pulse wave velocity (27).

Clinical interpretation of EBPR and recommendations for further research

EBPR is still not considered an independent diagnostic criterion for hypertension but could assist in providing a more precise assessment of cardiovascular risk. Its clinical significance is greater among obese youths, youths with metabolic disorders, a positive family anamnesis, or borderline resting blood pressure values. In such cases, repeated office blood pressure measurements and assessment of the overall cardiometabolic profile are warranted, while ABPM may be considered as part of further evaluation. Assessing subclinical target-organ damage, including the echocardiographic evaluation of the left ventricle and an evaluation of vascular parameters, is not

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carried out routinely merely based on the EBPR, but in accordance with additional clinical indications and overall risk assessment (19,26,28).

Pharmacological treatment in most youths is not introduced solely due to EBPR. Further actions depend on the overall clinical findings, while in obese children or youths with other risk factors, normalization of body mass, a balanced diet, regular blood pressure monitoring, and moderate physical activity are prioritized. Isolated EBPR is not a verified indicator for limiting recreational or competitive sport; the decision whether or not to limit exertion is based on symptoms, confirmed cases of hypertension, structural changes, and other high-risk indicators. Imposing unjustified restrictions on physical activity can be counter-productive; thus, an exercise program should be individualized based on clinical findings, nutritional status, physical fitness, and the presence of symptoms. A recently published meta-analysis has shown that structured exercise programs can lower systolic and diastolic blood pressure in obese youths, even though the interventions and the results obtained were considerably heterogeneous (29).

A clinical decision must be based on a graded assessment of risk, and not the results of a single ergometric test. Mild and isolated exceedance of the proposed baseline primarily requires an assessment of the conditions and precision of the measurement process, as well as further monitoring. Contrary to that, a repeat or disproportionately high response, especially if conjoined with increased ABPM values or signs of target-organ remodeling has greater clinical significance. This kind of approach decreases the possibility of unjustifiably classifying healthy children as belonging to a high-risk group, but also the risk of overlooking the early phases of disorders among youths who require more intense monitoring.

Conclusion

EBPR among youths does not represent a unique and independent clinical phenotype, but a hemodynamic pattern whose meaning depends on developmental characteristics, peak achieved intensity of the physical load, cardiorespiratory fitness, body composition, and the accompanying cardiometabolic risk. Its potential significance lies in the possibility of indicating impaired regulation not evident during resting blood pressure measurement; however, the available evidence still does not allow a more reliable assessment of its independent prognostic value for subsequent hypertension and target-organ damage. Until then, it would be most appropriate to implement a selective approach, based on the circumstances in which the results were obtained, so that its significance would not be overlooked, but also that the normal physiological reaction would not

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unjustifiably be interpreted as pathological. Further progress will primarily depend on the standardization of testing and longitudinal studies which will determine whether EBPR provides additional prognostic value compared to resting blood pressure measurement, ABPM, and previously established risk factors.

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