

THE IMPACT OF AGE-ASSOCIATED CHANGES IN MICROCIRCULATION ON CARDIOVASCULAR RISK FORMATION AND MYOCARDIAL FUNCTIONAL STATUS

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Abstract

The microcirculatory network plays a key role in ensuring adequate tissue perfusion and maintaining metabolic homeostasis of the cardiovascular system. Age-associated changes in microcirculation are considered one of the early and underestimated factors in the formation of cardiovascular risk, preceding the development of clinically overt forms of cardiovascular disease. This paper analyzes contemporary concepts of the structure and function of the microcirculatory network, examines mechanisms of age-related microcirculatory impairment

and their impact on myocardial functional status, and discusses diagnostic and preventive approaches aimed at correcting microcirculatory disorders in older adults.

Keywords: microcirculation, cardiovascular aging, capillary network, myocardial perfusion, endothelial dysfunction, cardiogerontology.

Introduction

Traditionally, cardiovascular risk assessment in clinical practice has been based on the analysis of macrohemodynamic parameters such as arterial blood pressure, cardiac output indices, and the condition of large arteries. However, in recent years, increasing attention has been paid to the role of the microcirculatory network as a key component ensuring adequate blood supply to tissues and organs.

Microcirculation includes a system of arterioles, capillaries, and venules where the primary exchange of oxygen, nutrients, and metabolites occurs. Microcirculatory disturbances may develop long before the appearance of clinical signs of cardiovascular disease and can play an important role in the pathogenesis of age-associated cardiovascular disorders.

1. Structural and Functional Organization of the Microcirculatory Network

The microcirculatory network represents a complex and highly organized system that adapts to changing metabolic demands of tissues. Regulation of microcirculation is carried out at the level of the endothelium, arteriolar smooth muscle cells, as well as through neurohumoral mechanisms.

Capillaries are the main structural element of microcirculation, providing diffusion of oxygen and metabolic substrates. The density of the capillary network and its functional state determine the efficiency of tissue perfusion and the resistance of organs to ischemic stress.

2. Age-Associated Changes in Microcirculation

2.1. Structural Changes of the Capillary Network During Aging

One of the most characteristic manifestations of age-associated changes in microcirculation is the reduction of the capillary network (capillary rarefaction), which is observed in skeletal muscle, myocardium, skin, and the brain. Capillary rarefaction leads to a decrease in the total exchange surface area and an increase in diffusion distance between capillaries and cells, thereby impairing the delivery of oxygen and metabolic substrates.

Experimental and clinical studies have shown that with aging, the density of functioning capillaries decreases, while some vessels remain anatomically preserved but lose the ability to provide adequate perfusion. This condition is described as functional capillary

insufficiency, in which the microcirculatory network cannot be fully recruited even under increased metabolic demand of tissues.

An important structural change is the thickening of the capillary basement membrane, associated with the accumulation of collagen, glycosylated proteins, and advanced glycation end products. These changes impair the diffusion of oxygen and biologically active molecules and disrupt interactions between endothelial cells and pericytes. Dysfunction of pericytes, which play a key role in stabilizing the capillary wall, further contributes to reduced microcirculatory adaptability.

In addition, aging is accompanied by heterogeneity of the microcirculatory network, in which some capillaries experience chronic hypoperfusion, while other regions remain relatively preserved. Such heterogeneity of blood flow enhances local ischemia and contributes to the development of focal metabolic disturbances, especially in tissues with high energy demand, such as the myocardium.

2.2. Functional Impairments of Microcirculation in Older Adults

Functional impairments of microcirculation during aging are closely related to alterations in mechanisms regulating vascular tone and to impaired endothelium-dependent vasodilation. One of the key factors is reduced bioavailability of nitric oxide, resulting both from decreased synthesis and increased inactivation by reactive oxygen species.

Age-related enhancement of oxidative stress leads to an imbalance between vasodilatory and vasoconstrictive factors, as a result of which the microcirculatory network loses its ability to respond adequately to local metabolic signals. This is particularly evident during physical exertion or ischemia, when older patients exhibit a pronounced limitation of reactive hyperemia.

An additional factor is impaired autoregulation of microcirculation, in which vessels lose the ability to maintain stable blood flow in response to changes in perfusion pressure. This results in increased tissue sensitivity to fluctuations in systemic arterial pressure and contributes to episodes of transient hypoperfusion.

Age-related changes in blood rheological properties also play a significant role, including increased viscosity and reduced erythrocyte deformability. These factors exacerbate microcirculatory disturbances, especially under conditions of reduced capillary reserve.

Taken together, structural and functional alterations of microcirculation form a state of chronic low-grade tissue hypoxia, which may persist for a long time without clinical symptoms but has a substantial impact on the progression of cardiovascular aging.

3. Microcirculation and Myocardial Functional Status: Evidence from Clinical and Experimental Studies

The role of microcirculation in maintaining normal myocardial function has been confirmed by numerous experimental and clinical studies. Unlike large coronary arteries, the myocardial microcirculatory network is directly responsible for blood flow distribution at the level of cardiomyocytes, and its dysfunction may lead to ischemia even in the absence of significant stenoses of epicardial vessels.

A number of studies using positron emission tomography and magnetic resonance perfusion imaging have demonstrated that older patients with preserved patency of the coronary arteries often exhibit a reduction in coronary microvascular reserve. This condition is associated with impaired diastolic function and decreased exercise tolerance.

A classic example is the description of coronary microvascular dysfunction syndrome in patients with heart failure with preserved ejection fraction. In such cases, microcirculatory disturbances are regarded as a key pathogenetic mechanism linking age-related vascular changes with functional myocardial insufficiency.

Experimental models of aging have shown that a reduction in myocardial capillary density is accompanied by impaired mitochondrial function of cardiomyocytes and decreased efficiency of oxidative phosphorylation. These changes lead to a reduction in cardiac energy reserve and increased vulnerability to ischemic stress.

Clinical observations also confirm that myocardial microcirculatory dysfunction is closely associated with the development of diastolic dysfunction. Increased resistance of the microcirculatory network elevates left ventricular filling pressure and promotes myocardial remodeling, creating conditions for the progression of heart failure.

Thus, data from contemporary studies confirm that microcirculation is not a secondary but a central component in the pathogenesis of age-associated myocardial functional impairment, and its condition largely determines the clinical outcome of cardiovascular aging.

4. Pathophysiological Mechanisms of Microcirculatory Disorders During Aging

(expanded version)

Age-associated microcirculatory disorders develop as a result of the interaction of multiple mechanisms affecting the endothelial layer, cellular elements of the vascular wall, hemorheology, and neurohumoral regulation. It is important to consider that the microcirculatory network exhibits marked heterogeneity: the severity of age-related changes may vary significantly depending on the organ involved (myocardium, brain, skin, kidneys), tissue metabolic demands, and the presence of concomitant risk factors such as arterial hypertension, diabetes mellitus, dyslipidemia, and chronic kidney disease. Below are the key pathophysiological mechanisms most consistently described in contemporary scientific literature and of major clinical relevance.

4.1. Endothelial Dysfunction of the Microcirculatory Compartment

The endothelium of the microcirculatory network serves as a “local regulator” of blood flow. At the level of arterioles and precapillary sphincters, endothelial function determines the ability of tissues to rapidly increase perfusion in response to rising metabolic demand (physical activity, stress, ischemia) and to maintain an adequate microcirculatory balance at rest. With aging, endothelial regulation becomes impaired, manifesting as reduced endothelium-dependent vasodilation, altered vascular permeability, and activation of proinflammatory and prothrombotic signaling pathways.

A key mechanism is considered to be reduced synthesis and/or bioavailability of nitric oxide (NO). Age-related decline in endothelial nitric oxide synthase (eNOS) activity, together with NO inactivation by reactive oxygen species, results in the predominance of vasoconstrictive influences and a reduction in microcirculatory reserve. As a consequence, tissues tolerate hypoperfusion episodes less effectively, and reactive hyperemia becomes less pronounced.

Simultaneously, production of vasoconstrictors (such as endothelin-1) increases, and vascular sensitivity to sympathetic influences is enhanced. These changes are particularly significant in older patients with arterial hypertension, in whom microcirculation initially functions under conditions of elevated vascular resistance.

Example (clinical situation):

In an elderly patient without significant stenoses of major coronary arteries, complaints of exertional dyspnea and rapid fatigability may persist. In such cases, one possible explanation is a reduction in coronary microvascular reserve due to endothelial dysfunction of arterioles. This dysfunction impairs myocardial blood flow distribution, especially during exertion, and contributes to the development of diastolic dysfunction.

Endothelial dysfunction within the microcirculatory compartment is also accompanied by impairment of the endothelial barrier function. Increased vascular permeability promotes extravasation of fluid and proteins, which may enhance interstitial edema. In the myocardium, interstitial edema and remodeling of the extracellular matrix are factors that impair myocardial relaxation and increase myocardial stiffness, directly contributing to diastolic dysfunction.

4.2. Role of Inflammation and Oxidative Stress

Aging is characterized by the phenomenon of chronic low-grade systemic inflammation, often referred to as *inflammaging*. This condition is accompanied by increased activity of proinflammatory cytokines and altered immune regulation, exerting a direct impact on the microcirculatory network.

Oxidative stress represents a critical mediator linking inflammation and endothelial dysfunction. Reactive oxygen species not only reduce NO bioavailability but also trigger cascades leading to damage of cellular membranes, proteins, and DNA in endothelial cells. This impairs the ability of blood vessels to adapt to hemodynamic and metabolic changes.

In addition, inflammation promotes the expression of adhesion molecules on the endothelial surface, facilitating leukocyte adhesion and migration into the vascular wall. Local inflammation sustains microvascular remodeling, contributes to thickening of arteriolar walls, and reduces vascular reactivity.

Example (pathogenetic chain):

An elderly patient with abdominal obesity and insulin resistance often exhibits signs of systemic inflammation. Even in the absence of clinically overt atherosclerosis, microcirculation may be impaired due to the combined effects of inflammatory endothelial injury and oxidative stress. Clinically, this manifests as reduced peripheral perfusion, cold extremities, delayed healing of minor injuries, and, on the cardiac level, decreased exercise tolerance.

Special attention should be paid to the impact of oxidative stress on erythrocytes and platelets. Damage to erythrocyte membranes reduces their deformability, increases blood viscosity, and impairs capillary passage. Platelet activation elevates the risk of microthrombosis within the microcirculatory bed, potentially leading to focal tissue hypoperfusion.

4.3. Impaired Capillary Recruitment and Microcirculatory Reserve

Microcirculation possesses a unique ability to increase the number of functioning capillaries in response to rising tissue oxygen demand. This mechanism is referred to as capillary recruitment. With aging, the capacity for capillary recruitment declines, limiting microcirculatory reserve and resulting in a mismatch between oxygen delivery and tissue requirements.

Age-related reduction in capillary recruitment is associated with several factors: endothelial dysfunction, thickening of the basement membrane, reduced sensitivity of arterioles to metabolic signals, and functional rarefaction (where some capillaries remain anatomically preserved but do not participate in perfusion).

Example (exercise test in real clinical practice):

In an elderly patient, moderate physical exertion may rapidly induce dyspnea, while macrohemodynamic parameters (e.g., ejection fraction) remain within normal limits. One possible explanation is insufficient microcirculatory reserve: during exertion, capillaries are not recruited adequately, tissue perfusion does not increase appropriately, and both myocardium and skeletal muscles shift toward a more energy-deficient state.

Under conditions of reduced capillary recruitment, anaerobic metabolism becomes more prominent, lactate accumulation increases, and fatigue develops earlier. These processes may be particularly critical for the myocardium, especially in the presence of concomitant arterial stiffness, when coronary perfusion is already subject to additional hemodynamic stress.

4.4. Hemorheological Factors and Microvascular Thrombosis

Although microcirculatory disturbances are often described primarily in terms of vascular wall pathology, the properties of blood itself play a substantial role, including viscosity, erythrocyte deformability, platelet aggregation, and plasma protein composition.

With aging, blood viscosity may increase due to alterations in the ratio of cellular elements to plasma, elevated fibrinogen levels, and increased concentrations of acute-phase proteins. Even moderate increases in blood viscosity significantly impair capillary blood flow and promote the development of localized hypoperfusion.

Reduced erythrocyte deformability hinders their passage through narrow capillaries, exacerbating tissue hypoxia. In combination with age-related endothelial dysfunction, this creates conditions favorable for microthrombosis and the formation of “silent” ischemic zones within tissues.

Example (microvascular ischemia):

In elderly patients, episodes of transient myocardial ischemia may occur in the absence of significant stenoses of major coronary arteries. In such cases, the microcirculatory component may involve microvascular thrombosis and increased blood viscosity, leading to impaired perfusion at the level of capillaries and arterioles.

4.5. Neurohumoral Regulation and the Influence of Sympathetic Activity

With aging, the balance of autonomic regulation of vascular tone shifts: basal sympathetic activity increases, while baroreflex sensitivity declines. This leads to greater variability of arterial blood pressure and fluctuations in microcirculatory perfusion.

Excessive sympathetic activity promotes arteriolar vasoconstriction, reduces microcirculatory blood flow, and impairs tissue trophism. This mechanism is particularly relevant in elderly patients with anxiety disorders, sleep disturbances, and chronic stress, as psychoemotional factors may amplify vascular dysfunction.

Example (orthostatic intolerance):

In some elderly patients, standing up may provoke dizziness and weakness. In addition to systemic factors, microcirculatory mechanisms include insufficient autoregulation and excessive vasoconstriction, resulting in transient hypoperfusion of the brain and peripheral tissues.

4.6. Organ-Specificity of Microcirculatory Changes: The Myocardium as a “Sensitive Target”

Age-related microcirculatory disturbances are of particular significance in the myocardium, as the heart has high metabolic demands and a limited reserve for hypoxic adaptation. Even

minor impairments of capillary perfusion may induce metabolic stress in cardiomyocytes, promote accumulation of interstitial fibrosis, and aggravate diastolic dysfunction.

Myocardial microcirculatory dysfunction may also contribute to a reduction in coronary reserve and increase the likelihood of exertional ischemia. In combination with age-related arterial stiffness—which impairs diastolic coronary perfusion—microcirculatory alterations form a multilevel mechanism of declining cardiac function in older adults.

Example (HFpEF):

Heart failure with preserved ejection fraction in elderly patients is often accompanied by reduced myocardial microcirculatory reserve, pronounced endothelial dysfunction, and inflammatory activity. In this context, microcirculation becomes a key link connecting systemic age-related vascular changes with the clinical manifestations of heart failure.

Thus, microcirculatory disorders during aging result from a complex interaction of endothelial dysfunction, inflammation and oxidative stress, reduced capillary recruitment, hemorheological factors, and alterations in neurohumoral regulation. At the myocardial level, these mechanisms manifest as diminished microcirculatory and coronary reserve, impaired energetic support of cardiomyocytes, and progression of diastolic dysfunction. Elucidation of these mechanisms is essential for the development of preventive strategies aimed at preserving microcirculatory health and slowing cardiovascular aging.

5. Diagnostic Approaches to the Assessment of Microcirculation and Their Clinical Significance

Assessment of the microcirculatory network represents one of the most challenging tasks in clinical cardiology and vascular medicine, as microcirculation is not accessible to direct visualization by standard methods used to evaluate large vessels. At the same time, microcirculatory disorders often underlie early functional changes that precede the development of clinically overt cardiovascular disease. Contemporary diagnostic approaches are aimed at indirect or direct assessment of microcirculatory blood flow, vascular reactivity, and functional reserve of the microcirculatory bed.

5.1. Laser Doppler Flowmetry

Laser Doppler flowmetry (LDF) is one of the most widely used noninvasive methods for assessing microcirculation in both clinical and experimental studies. The method is based on recording the Doppler shift of laser radiation reflected from moving formed elements of blood, which allows estimation of the relative level of tissue perfusion.

One of the key advantages of LDF is the ability to perform functional tests, including reactive hyperemia testing, thermal stimulation, and pharmacological interventions. Analysis of microcirculatory responses to these stimuli makes it possible to assess the state of endothelial regulation, neurogenic mechanisms, and the myogenic component of vascular tone.

In the context of aging, LDF demonstrates characteristic changes, including a reduction in the amplitude of endothelium-dependent oscillations, decreased microcirculatory reserve, and increased heterogeneity of blood flow. These features correlate with age, the level of arterial stiffness, and the presence of concomitant risk factors.

Clinical example:

In elderly patients without pronounced macrovascular pathology, LDF combined with an occlusion test often reveals delayed and attenuated recovery of perfusion, indicating endothelial dysfunction of the microcirculatory compartment. Such changes may be present even in individuals with normal arterial blood pressure values and in the absence of clinical symptoms.

Limitations of the method include high sensitivity to external factors (skin temperature, emotional state), limited depth of assessment, and the need for strict standardization of examination conditions.

5.2. Videocapillaroscopy and Microcirculatory Visualization

Videocapillaroscopy allows direct visualization of the capillary network, most commonly at the level of the nailfold. The method enables assessment of capillary density, morphology, blood flow characteristics, and the presence of structural abnormalities.

With aging, characteristic changes detected by capillaroscopy include a reduction in the density of functioning capillaries, elongation and deformation of capillary loops, and slowing of blood flow. These features reflect both structural and functional remodeling of the microcirculatory bed.

Example of clinical application:

In elderly patients with chronic heart failure, videocapillaroscopy may reveal pronounced capillary rarefaction and congestive alterations in blood flow, which correlate with the severity of functional class and reduced exercise tolerance.

Despite its informative value, the method remains limited in routine clinical practice due to labor-intensive analysis, the need for specialized equipment, and the absence of universally accepted diagnostic criteria.

5.3. Assessment of Coronary Microcirculation

More sophisticated instrumental methods are used to assess myocardial microcirculation, including positron emission tomography (PET), magnetic resonance imaging with perfusion protocols, and invasive techniques for measuring coronary microcirculatory reserve.

PET studies allow quantitative evaluation of myocardial blood flow and coronary reserve, enabling detection of microcirculatory dysfunction even in the absence of stenoses of epicardial coronary arteries. With aging, a decline in coronary microcirculatory reserve is

observed, which is associated with diastolic dysfunction and an increased risk of heart failure with preserved ejection fraction.

Example from clinical studies:

In elderly patients with dyspnea of unclear origin and preserved ejection fraction, reduced coronary microcirculatory reserve assessed by PET often proves to be a key factor explaining clinical symptoms in the absence of obstructive coronary artery disease.

Limitations of these methods include high cost, limited availability, and invasiveness (for certain techniques), which complicate their widespread use in screening programs.

5.4. Biochemical Markers of Microcirculatory and Endothelial Dysfunction

In recent years, biochemical markers reflecting the state of the endothelium and microcirculatory bed have been actively investigated. These include levels of endothelin-1, asymmetric dimethylarginine, inflammatory markers (C-reactive protein, interleukins), and indicators of oxidative stress.

In elderly patients, elevated levels of these markers often correlate with impaired microcirculation and an unfavorable cardiovascular prognosis. However, substantial interindividual variability and the influence of concomitant diseases limit their use as independent diagnostic tools.

Clinical context:

Elevated levels of asymmetric dimethylarginine in elderly patients may indicate reduced activity of NO-dependent mechanisms and can be considered an additional marker of endothelial dysfunction, particularly when combined with instrumental evidence of microcirculatory impairment.

5.5. Integrated Approach and Assessment of Microcirculatory Reserve

The most promising approach is an integrated assessment of microcirculation that combines instrumental, functional, and laboratory methods. Such an approach allows more accurate characterization of microcirculatory reserve and identification of early age-associated changes.

Integration of microcirculatory data with indicators of arterial stiffness, diastolic function, and systemic inflammation forms the basis for personalized assessment of cardiovascular risk in older adults.

Example of integrative diagnostics:

An elderly patient with normal ejection fraction, moderate arterial stiffness, and reduced microcirculatory reserve according to LDF and echocardiography may be classified as having a high risk of developing heart failure, despite the absence of classical signs of ischemic heart disease.

Thus, diagnosis of microcirculatory disorders in aging requires a multilevel and interdisciplinary approach. Contemporary methods allow detection of early functional microcirculatory changes with important prognostic significance. Incorporation of microcirculatory assessment into diagnostic algorithms for elderly patients may substantially improve the effectiveness of cardiovascular disease prevention and lead to better clinical outcomes.

6. Prevention and Correction of Microcirculatory Disorders During Aging

Prevention and correction of microcirculatory disorders during aging represent one of the most promising yet simultaneously challenging tasks of modern cardiology and gerontology. Unlike macrovascular lesions, microcirculatory disorders rarely respond to isolated correction and require a comprehensive approach aimed at influencing the endothelium, the vascular wall, the rheological properties of blood, and systemic risk factors. A key principle in this context is early intervention, since microcirculatory alterations often precede clinically overt cardiovascular pathology.

6.1. Non-Pharmacological Strategies for the Correction of Microcirculation

6.1.1. Physical Activity and Training-Induced Adaptation of the Microcirculatory Network

Regular physical activity is one of the most effective non-pharmacological methods for improving microcirculation in older adults. Aerobic exercise of moderate intensity stimulates endothelium-dependent vasodilation, increases nitric oxide bioavailability, and promotes capillary recruitment.

Studies demonstrate that even in individuals over the age of 65, regular aerobic training leads to an increase in the density of functioning capillaries and improvement of microcirculatory reserve. These changes are accompanied by enhanced myocardial perfusion and increased exercise tolerance.

Clinical example:

In older patients with early manifestations of diastolic dysfunction, the inclusion of a program of regular walking or cycling training results in improved microcirculatory parameters and reduced severity of dyspnea, even in the absence of significant changes in macrohemodynamic indices.

6.1.2. Dietary Factors and Metabolic Support of the Endothelium

Dietary patterns exert a substantial influence on the state of the microcirculatory network. Diets rich in antioxidants, polyunsaturated fatty acids, and plant polyphenols contribute to the

reduction of oxidative stress and inflammatory activity, thereby improving endothelial function.

Of particular interest is the impact of nutrients that enhance nitric oxide bioavailability, including L-arginine and nitrate-containing foods. Although clinical data remain heterogeneous, several studies have reported improved microcirculatory responses in older patients who adhere to such dietary recommendations over prolonged periods.

6.1.3. Control of Psychoemotional Stress and Autonomic Balance

Age-associated enhancement of sympathetic nervous system activity exerts an adverse effect on microcirculation by promoting vasoconstriction and reducing tissue perfusion. In this regard, non-pharmacological interventions aimed at normalizing autonomic balance—including breathing techniques, cognitive-behavioral interventions, and sleep optimization—may exert an indirect yet clinically meaningful effect on microcirculatory function.

6.2. Pharmacological Approaches to the Correction of Microcirculatory Disorders

6.2.1. Antihypertensive Therapy and Microcirculation

Blood pressure control is a fundamental component of microcirculatory disorder prevention. Medications targeting the renin–angiotensin–aldosterone system not only reduce systemic blood pressure but also exert beneficial effects on the endothelium and microcirculation.

Angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers reduce inflammatory activity, decrease oxidative stress, and improve endothelium-dependent vasodilation. In older adults, their use is associated with improved microcirculatory blood flow and attenuation of progressive vascular remodeling.

6.2.2. Lipid-Lowering Therapy and Endothelial Function

In addition to their lipid-lowering effects, statins possess pleiotropic properties, including improvement of endothelial function and reduction of inflammatory activity. In older patients, statin therapy may contribute to enhanced microcirculatory responses, particularly when combined with correction of other cardiovascular risk factors.

It is important to note that the effects of statins on microcirculation do not always directly correlate with lipid levels, underscoring their role in the comprehensive prevention of cardiovascular aging.

6.2.3. Antiplatelet and Hemorheological Therapy

Age-related microcirculatory changes are often accompanied by increased platelet aggregation and impaired blood rheological properties. In selected clinical situations, the use of antiplatelet agents may reduce the risk of microthrombosis and improve tissue perfusion.

However, in older adults, antiplatelet therapy requires caution due to an increased risk of bleeding. Decisions regarding its use should be individualized, taking into account overall cardiovascular risk and the presence of comorbid conditions.

6.2.4. Emerging Pharmacological Directions

Contemporary research actively investigates pharmacological agents targeting molecular mechanisms of aging, including anti-inflammatory therapies, modulators of mitochondrial function, and agents influencing endothelial signaling pathways. Although most of these approaches remain at the stage of clinical investigation, they represent substantial interest for the future treatment of microcirculatory disorders.

6.3. Individualization of Preventive Strategies in Older Adults

One of the key principles in correcting microcirculatory disorders during aging is the individualization of therapy. The biological age of the cardiovascular system, the patient's functional status, and the presence of comorbidities determine the selection and intensity of interventions.

Example of a personalized approach:

In an older patient with moderate arterial stiffness, subclinical diastolic dysfunction, and reduced microcirculatory reserve, a combination of moderate aerobic physical activity, optimization of antihypertensive therapy, and lifestyle modification is advisable. Such an approach may slow the progression of age-associated changes and delay the development of clinically overt heart failure.

Prevention and correction of microcirculatory disorders during aging require a comprehensive and multilevel approach aimed at eliminating endothelial dysfunction, reducing inflammatory activity, improving blood rheological properties, and restoring microcirculatory reserve. The integration of non-pharmacological and pharmacological strategies tailored to individual patient characteristics offers promising opportunities for effective deceleration of cardiovascular aging and improvement of clinical outcomes.

Conclusion

7.1. Synthesis of Key Pathophysiological Concepts

The analysis presented confirms that microcirculatory disorders are not a secondary phenomenon but rather a central pathogenetic component of cardiovascular aging. Age-associated endothelial alterations, low-grade inflammation, oxidative stress, reduced capillary

recruitment, and deterioration of hemorheological properties of blood form a multilevel cascade leading to chronic tissue hypoperfusion. At the myocardial level, these processes manifest as a reduction in microcirculatory and coronary reserve, impaired energetic support of cardiomyocytes, and progression of diastolic dysfunction.

An important conclusion is the concept of **ventriculo–microcirculatory coupling**, in which age-related changes in the vascular and cardiac components mutually reinforce one another. Increased arterial stiffness and microcirculatory dysfunction create unfavorable conditions for coronary perfusion during diastole, which is of particular significance in older age.

7.2. Clinical Implications for Diagnosis and Risk Stratification

The findings of this review indicate the need to expand diagnostic algorithms in older patients beyond the assessment of macrovascular hemodynamics. Integration of microcirculatory assessment methods (laser Doppler flowmetry, capillaroscopy, perfusion imaging techniques), indicators of arterial stiffness, and parameters of diastolic function allows more accurate evaluation of the biological age of the cardiovascular system and identification of high-risk groups at the preclinical stage.

Assessment of microcirculatory reserve is of particular clinical value in patients with preserved ejection fraction and nonspecific symptoms (dyspnea, early fatigue), when conventional diagnostic methods fail to detect significant pathology. In such cases, microcirculatory dysfunction may serve as a key to explaining symptoms and guiding management strategies.

7.3. Practical Implications for Prevention and Management of Older Patients

Comprehensive correction of microcirculatory disorders should be regarded as a strategy for both primary and secondary prevention of cardiovascular diseases in older age. Effective interventions include a combination of non-pharmacological measures (aerobic physical activity, dietary approaches, control of psychoemotional stress) and pharmacological therapy targeting endothelial function, inflammation, and arterial stiffness.

A key principle is the individualization of preventive strategies based on the patient's functional status and biological rather than chronological age. Such an approach allows avoidance of excessive therapy while simultaneously enabling timely intervention in the pathogenetic mechanisms of aging.

7.4. Perspectives for Scientific Research and Interdisciplinary Approaches

The data obtained underscore the need for further research aimed at:

- standardization of methods for microcirculatory assessment in clinical practice;

- development of integrative risk models incorporating microcirculatory parameters;
- investigation of molecular and cellular mechanisms of endothelial aging;
- evaluation of the effectiveness of targeted interventions aimed at restoring microcirculatory reserve.

Cardiogerontology and vascular biology are emerging as key interdisciplinary fields capable of integrating clinical observations with fundamental research on aging. The development of personalized preventive strategies based on microcirculatory status may substantially transform approaches to the management of aging populations.

7.5. Final Statement

Microcirculatory disorders during aging should be considered an early, systemic, and potentially modifiable factor of cardiovascular risk. Their timely diagnosis and correction open opportunities for slowing cardiovascular aging, improving myocardial functional status, and enhancing the quality of life of older patients. Incorporation of the microcirculatory component into the clinical reasoning of cardiologists represents an important step toward more precise and effective medicine of the future.

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