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# THE ROLE OF MYOCARDIAL HYPERTROPHY AND VENTRICULAR CONTRACTION INDEX IN HEART FAILURE

**Yorkulova Guljakhan Rakhmatjon kizi**
2nd year student of the Faculty of Pediatrics, Samarkand State Medical University, Samarkand, Uzbekistan
e-mail: yorqulovagulakhon959@gmail.com
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**ABSTRACT**

*Left ventricular hypertrophy (LVH) and concomitant ejection fraction are the most common myocardial structural abnormalities associated with heart failure. LVH is controlled by neurohumoral activation, increased mechanical load, that is, increased pressure on the heart, and arterial hypertension, chronic kidney disease, diabetes, and other cytokines. In this article, we discuss experimental and clinical evidence linking LVH to diastolic dysfunction and establishing it as one diagnostic marker for heart failure. The mechanisms leading to diastolic dysfunction in LVH are not yet fully understood, but we can consider changes in the extracellular matrix, vascular dysfunction, as well as changes in the mechano-elastic properties of cardiomyocytes. In this study, we investigated increased Ca2+ exchange and impaired relaxation in cardiomyocytes from hypertrophied hearts. Diastolic dysfunction may appear independently of LVH in the clinical picture of patients with complex joint diseases. This may explain why approaches to reduce LVH have not been effective in improving symptoms and prognosis. In addition, we mentioned above about the ejection fraction, that is, it is the volume of oxygen-rich blood that is released into our body with each heartbeat. The Frank-Starling law states that left ventricular volume increases due to increased systolic contraction due to myocyte stretching, leading to LVH. As a result of the research, we found out that Frank-Starling's laws are a description of cardiac hemodynamics and that it is related to the elongation and contraction of myocytes.*

## Introduction

First of all, we need to know that heart failure is not a diagnosis. I mean, heart failure is caused by something, and we need to find out why. For example:

(i) Ischemic heart disease - in which the arteries that supply blood to the heart become blocked with fatty substances (atherosclerosis), which leads to a heart attack.
(ii) High blood pressure can put extra pressure on the heart, which can lead to heart failure over time.
(iii) Arrhythmia – heart rhythm problems (atrial fibrillation)
(iv) Cardiomyopathy - factors affecting the heart muscle
(v) Congenital heart disease - conditions affecting the normal functioning of the heart
(vi) Left ventricular hypertrophy is caused by an increase in the volume of the ventricle, which can compress a part of the esophagus.

We know from hemodynamics that the more Ca2+ in the body, the faster the heart contracts. Let's briefly touch on hemodynamics to make it easier for you to understand. Hemodynamics, that is, blood volume (stroke volume) is affected by 3 factors. These are:

1. The amount of blood entering the heart (preload)
2. Heart contraction (contractility)
3. Afterload

Of the ones mentioned above, we will pay more attention to heart contraction, that is, contractility. Because the topic of our today's scientific article is related to this. Currently, nanotechnology is developing day by day in our country, and through this we can see the mechanism of Ca2+ inside the myocides. In this case, the enzyme cotecholamine activates the enzyme protein kinase A, which affects the protein phospholamban, in turn, this protein opens the athephase in the wall of the reticular sarcoplasm, and from it comes the familiar Ca2+. As Ca2+ increases within the myocyte, contraction increases. Decreased contractility leads to heart failure.

**Main part**

**Clinical presentation of left ventricular hypertrophy**

Most patients with symptomatic heart failure have changes in left ventricular (LV) geometry, such as hypertensive heart disease, including myocardial hypertrophy. Myocardial hypertrophy is defined as an increase in ventricular myocardium mass. As a result of research, we have found that, in clinical practice and in animal studies, left ventricular hypertrophy (LVH) is often detected by measuring the thickness of the septal barrier and left ventricular posterior wall, and is associated with a normal or dilated left ventricular cavity. We found that it can be. There are 22 traditional ways to diagnose left ventricular hypertrophy in the clinic, and we will get acquainted with one of them below. We receive echocardiography (ECG) analysis results and:

i. We select the longest S wave (S wave) in the lower part of the isoline in V1 and V2
ii. We select the longest R wave (R wave) at the top of the isoline in V5 and V6
iii. We add the sum of our results. The norm should be R+S=3.5 mV

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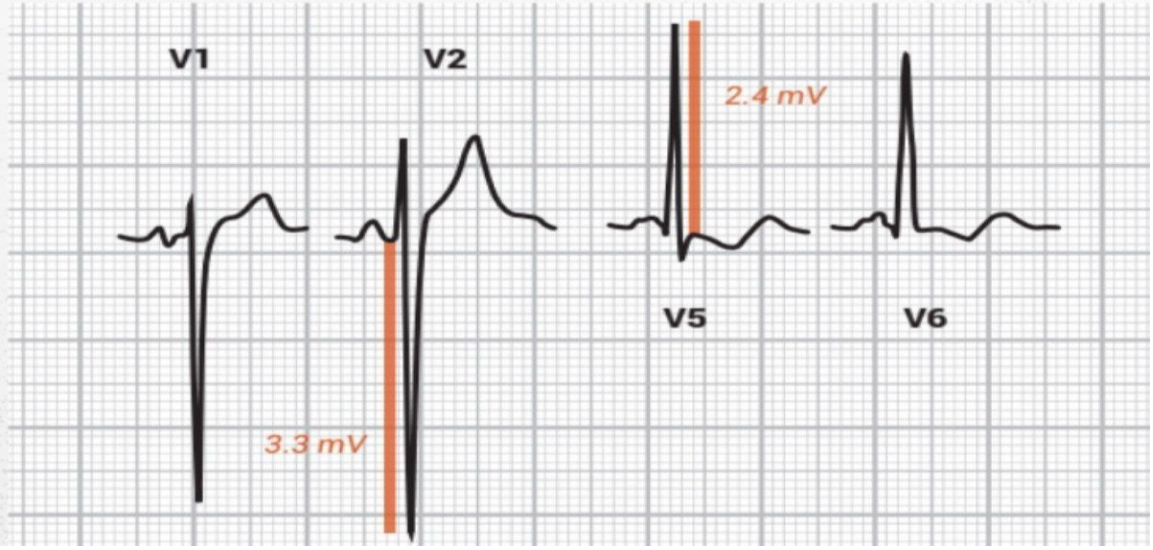


If the analysis results are higher than 3.5 mV, then we can diagnose the patient with left ventricular hypertrophy. Left ventricular mass is considered the most common parameter of LVH in clinical studies. As a result of our research, in echocardiography, we observed an increase in left ventricular mass in people who died with heart failure. It is also the same in animals, for example, in mice.

**Causes and consequences of left ventricular hypertrophy**

The left ventricle is the main chamber of our heart. It is responsible for pumping oxygen-rich blood into the aorta (the largest artery in the body). If the heart has to work too hard to pump blood, the muscles of the walls of the left ventricle thicken, and this thickening is called hypertrophy. Research results have shown that approximately 15% to 20% of the population is affected, that is, 1 in 5 people are affected by this disease (LVH). The most common cause is high blood pressure. Because in high blood pressure, the heart works harder than usual. The more the heart contracts, the greater the volume of blood. We can find it based on the following formula: **SV/EDV=EF** more than 55% is pathology.

**Myocardial dysfunction associated with pathological left ventricular hypertrophy**

In fact, left ventricular hypertrophy (LVH) is one of the most common cardiac abnormalities in heart failure. As a result of research, it was found that in most patients, arterial hypertension is the reason for hospitalization with heart failure. However, the underlying pathomechanisms that may link left ventricular hypertrophy to diastolic dysfunction and heart failure remain relevant. However, the functional effect of the left ventricle has been widely studied in hypertrophic cardiomyopathy. As a result of research, it has become clear to us that patients with heart failure have more pronounced concentric hypertrophy than patients with hypertensive heart disease without heart failure. Notably, patients with LVH performed worst during exercise, which is associated with reduced contraction reserve as well as chronotropic changes. We found significantly weak inverse correlations between exercise capacity. And we compared this process based on the table. In this table, we consider the prevalence percentage of arterial hypertension and left ventricular hypertrophy in heart failure patients with preserved ejection fraction.

**1-table**

| Learning | Arterial hypertension | LVH |
|---|---|---|
| Relax | 85% | 48% |
| Topcat substudy | 91% | 47% |
| Leather - es | 64% | 52% |
| Paramount | 92% | * |

Relax is a means of exercise in heart failure; topcat substudy – treatment of heart failure with preserved heart function with aldosterone; leather - candesartan in congestive heart failure; Paramount – ECG study to evaluate reduction in mortality and morbidity.

**Alteration of normal contractility in left ventricular hypertrophy and heart failure**

Our previous studies have shown that patients with LV hypertrophy and EF may have subtle systolic dysfunction that is not reflected by EF. In recent years, left ventricular (LV) deformation during systole has been identified using tissue markers in multiple echocardiographic waves. Among them, 3 types of contraction are distinguished: longitudinal, radial and circular. An increase in radial stress can hide the loss of contractile function along the longitudinal axis of the heart (longitudinal contraction). According to the above-mentioned PARAMOUNT study, impaired longitudinal contractility in heart failure patients was considered a marker of systolic dysfunction despite preserved EF, independent of other signs of diastolic dysfunction. However, several scientists have found that a decrease in global longitudinal tension in hypertensive patients is associated with diastolic dysfunction, but not with LVH. In summary, in experimental and clinical studies, LVH is associated with global dysfunction and heart failure, which is the basis for including LVH as a diagnostic marker in the clinical algorithm for heart failure.

**Clinical management of left ventricular hypertrophy and its effects**

From our studies, we observed that in various models of heart failure, interfering with left ventricular hypertrophic signaling pathways significantly reduces left ventricular hypertrophy and improves diastolic function, often independent of changes in blood pressure. These observations, while mediating diastolic dysfunction and ameliorating LVH as a therapeutic target, later revolute heart failure in most of these models, preventing the transfer of these findings to the multifactorial state of clinical heart failure. It is important to remember that diastolic function is a function of pressure after pressure, so the treatment effect reflects, at least in part, a decrease in arterial resistance and does not improve left ventricular compliance per se. The results of our research confirmed the regression of LVH with standard antihypertensive therapy such as angiotensin receptor blockers (ARBs), angiotensin-converting enzyme inhibitors (ACEI), Ca2+ antagonists, as well as new approaches. The relationship between a decrease in LVH and an improvement in diastolic function has not been well established in clinical studies. According to preliminary data, teratorol or sotalol beta-blockers independently improve diastolic function in LVH. Improvements in LVH diastolic function with current antihypertensive therapy have been reported in smaller uncontrolled studies over the past two decades. The multifactorial origin of diastolic dysfunction in the clinical setting may also explain the weak correlation between LVH regression and improvement in diastolic function. This is further proof that a better

understanding of the cellular mechanisms of LVH and heart failure is necessary for the improvement of therapeutic treatment.

**Effects of exercise on left ventricular hypertrophy and heart failure**

We know that exercise is difficult in patients with vascular dysfunction and heart failure, but we nevertheless observed an increase in left ventricular pressure during exercise. We have reviewed several research trials that have been conducted in this regard. Although they showed a significant improvement in exercise capacity and quality of life, they did not show improvements in cardiac systolic or diastolic function or LVH. Therefore, future research is urgently needed to further refine the effects of exercise on cardiac function.

**Conclusion**

In conclusion, experimental and clinical studies demonstrate maladaptive left ventricular hypertrophy (LVH), i.e., spontaneous diastolic dysfunction in the presence of pathological stimuli. and thus may contribute to the heart failure phenotype. As a result of research, it became clear to us that the mechanisms are diverse, probably specific to the etiology, vascular dysfunction and potential reduction of blood vessels, changes in the composition of the extracellular matrix, including changes in the internal active and passive contractile properties of cardiac myocytes. change is observed. In the multifactorial clinical conditions of heart failure, diastolic dysfunction is observed even in the absence of LVH. That is, as a result of our research, we found that the decrease in LVH is not associated with the improvement of diastolic function. Thus, the current clinical evidence cannot be viewed as a marker for even short-term improvement in heart failure and does not support regression of LVH.

## References:

1. Dunn. FG. Systolic and diastolic function after regression of left ventricular hypertrophy in hypertension. [Pub.med] [Google schools]
2. Bernardo BC, Weeks KL, Pretorius L, McMullen JR. Molecular distinction between physiological and pathological cardiac hypertrophy: experimental findings and therapeutic strategies. [PubMed] [Google Scholar]
3. Bisping E, Wakula P, Poteser M, Heinzel FR. Targeting cardiac hypertrophy: towards a causal heart failure therapy. J Cardiovasc Pharmacol. 2014;64:293–305. [PubMed] [Google Scholar]
4. Caselli S, Di Paolo FM, Pisicchio C, Pandian NG, Pelliccia A. Patterns of left ventricular diastolic function in Olympic athletes. J Am Soc Echocardiogr. 2015;28:236–244. [PubMed] [Google Scholar]
5. Hamdani N, Bishu KG, von Frieling-Salewsky M, Redfield MM, Linke WA. Deranged myofilament phosphorylation and function in experimental heart failure with preserved ejection fraction. Cardiovasc Res. 2013;97:464–471. [PubMed] [Google Scholar]
6. Hayashida W, Donkkier J, Van MH, Charlier AA, Pouleur H. Diastolic properties in canine hypertensive left ventricular hypertrophy: effects of angiotensin converting enzyme inhibition and angiotensin II type-1 receptor blockade. Cardiovasc Res. 1997;33:54–62. [PubMed] [Google Scholar]

7. Ibrahim MM, Zaghloul SS, Helmi SM. Effect of regression of left ventricular hypertrophy following sotalol therapy on diastolic function in hypertensive patients. J Hypertens Suppl. 1987;5:S411–S414. [PubMed] [Google Scholar]
8. Kruger M, Linke WA. The giant protein titin: a regulatory node that integrates myocyte signaling pathways. J Biol Chem. 2011;286:9905–9912. [PMC free article] [PubMed] [Google Scholar]
9. LeWinter MM, Granzier H. Cardiac titin: a multifunctional giant. Circulation. 2010;121:2137–2145. [PMC free article] [PubMed] [Google Scholar]