



Assessment of Cardiac Functions in Coronary Artery Disease

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Abstract

Background. One of the most common cardiovascular diseases is coronary artery disease, which occurs when there is a buildup of plaque in the arteries that supply blood to the heart. CAD, or coronary artery disease, is a type of cardiovascular disease characterized by the narrowing or blockage of the coronary arteries that supply oxygen-rich blood to the heart. This narrowing or blockage is typically caused by the accumulation of cholesterol and other materials, which form a buildup called plaque on the inner walls of the arteries. The prevalence of risk variables in healthy people demonstrates the likelihood of CAD soon. Echocardiography is the most commonly used method to assess cardiac function through different modes and modalities. This is due to many factors including easy access, non-invasiveness, and the huge progress resulted in better, more accurate results. The relationship between left ventricular function and the degree of coronary artery disease in patients with and without a history of myocardial infarction was examined in 96 patients who reported chest pain. Coronary artery disease alters diastolic function to varying degrees. Following myocardial infarction and acute ischemia, certain anomalies are generated. In these two diseases, the etiology of diastolic anomalies is discussed. The left ventricle's filling pressures rise during acute ischemia. Pulmonary oedema could develop. Less of a rise in filling pressures results with silent ischemia. With varying contributions from altered myocardial relaxation, increased muscular stiffness, acute pericardial constriction, ventricular interaction, and acute chamber dilatation, the diastolic pressure-volume ratio is moved upward. Because of the factors that change the pressure-volume relationship, the left ventricle's ability to fill during ischemia is altered. Given the decreased left ventricular compliance, the abrupt rise in left atrial pressure may raise filling rates relatively unexpectedly

Keywords: Cardiac Functions, Coronary Artery Disease

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Introduction

Individual with the left dominance are at high risk of developing coronary artery diseases (CAD). Therefore, dominance is a major determinant in acquired CAD. Although right dominance circulation is commonest, CAD is more frequent on left dominant circulation (1-3).

Dominance also showed a role in left anterior descending artery stenosis. It was observed that in left dominance, the LAD usually wraps around the apex of the heart, supplying the major portion of the myocardium. As such, lesions in LAD would have more profound clinical importance in a left dominant heart than in a right dominant one. There is a decrease in incidence of left dominance and codominance with advancing age, this is hypothesized to be due to poor prognosis associated these variants (4-8). It is conceivable that the type of coronary artery dominance also has an effect on the occurrence and outcome of non obstructive CAD (9).

Cardiac functions in coronary artery disease

Cardiovascular diseases are a leading cause of death worldwide. They are responsible for approximately 17.9 million deaths each year, according to the World Health Organization. One of the most common cardiovascular diseases is coronary artery disease, which occurs when there is a buildup of plaque in the arteries that supply blood to the heart. CAD, or coronary artery disease, is a type of cardiovascular disease characterized by the narrowing or blockage of the coronary arteries that supply oxygen-rich blood to the heart. This narrowing or blockage is typically caused by the accumulation of cholesterol and other materials, which form a buildup called plaque on the inner walls of the arteries. The prevalence of risk variables in healthy people demonstrates the likelihood of CAD soon. Chromosome 9p21.3 has been linked to the early development of CAD, according to genome-wide association studies. Diabetes mellitus, high blood pressure, smoking, hyperlipidemia, obesity, homocystinuria, and psychological stress are all risk factors for coronary artery disease (CAD). Through significant studies and trials, the elimination and management of CAD have been established. Some of the few therapeutic medications used to treat symptomatic angina associated with CAD include antiplatelet medicines, nitrates, β -blockers, calcium antagonists, and ranolazine (10).

The reduced blood flow to the heart due to CAD can lead to various cardiac abnormalities. This includes arrhythmias, cardiac function impairment, etc. Cardiac function is the heart's capacity to meet the body's metabolic needs. Life is dependent on the elimination of cellular waste products and the flow of oxygenated blood. Filling volume (preload), vascular resistance (afterload), and myocardial contractility all have an impact on cardiac function. Heart catheterization allows for the precise measurement of heart function. Accurate measurements can be made of vascular resistance and hemodynamic parameters. The majority of professionals view this as the gold standard and consider it one of the most crucial instruments for assessing heart function. Exercise testing and echocardiography are two more techniques for evaluating heart function. The static or dynamic modes of magnetic resonance imaging (MRI) can be utilized to get data about the structure and operation of the heart (11).

Echocardiography is the most commonly used method to assess cardiac function through different modes and modalities. This is due to many factors including easy access, non-invasiveness, and the huge progress resulted in better, more accurate results.

A common tool for managing and assessing the risk of numerous cardiovascular disorders is the left ventricular (LV) ejection fraction (LVEF), which is a straightforward indicator of overall systolic performance. However, this parameter is constrained by pathophysiological entities where the ratio of stroke volume to LV cavity size is conserved as well as by technical difficulties. For the measurement of myocardial function, the assessment of global longitudinal strain (GLS) from speckle-tracking analysis of 2-dimensional echocardiography has emerged as a therapeutically viable substitute for LVEF. According to data acquired over the previous ten years, GLS is more prognostically informative and sensitive to left ventricular dysfunction (LVD) than LVEF. The technology has been approved, is widely used, and is replicable within a reasonable range (12).

The relationship between left ventricular function and the degree of coronary artery disease in patients with and without a history of myocardial infarction was examined in 96 patients who reported chest pain. For a precise delineation of the coronary anatomy, coronary arteriography was done in order to acquire cineangiograms (60 frames/sec) and large roll film angiograms (2 to 6 frames/sec). A typical history, a rise and decrease in serum glutamic oxaloacetic transaminase levels, and evolutionary S-T segment variations linked to Q waves of at least 0.03 seconds were required for the diagnosis of myocardial infarction. Measurements of left ventricular end-diastolic pressure, volume, ejection fraction, mass, and compliance were used to evaluate left ventricular function. Only 15 individuals had normal results, and the remaining 81 were divided into groups based on the number of diseased arteries and the presence or absence of myocardial infarction. Age or heart rate did not differ across groups. Patients with myocardial infarction in each vascular disease group had higher left ventricular end-diastolic volumes and worse ejection fractions. Ejection fraction decreased in patients with three vessel disease even if myocardial infarction had not yet occurred, and it

decreased even more when it did. Patients with three vessel disease, whether or not they had a myocardial infarction, had a higher left ventricular mass. These results suggest that in order to accurately assess cardiac function in patients with ischemic heart disease, a previous history of myocardial infarction must also be taken into account, along with anatomic abnormalities of the coronary arteries. A prior MI significantly alters left ventricular performance, and the ejection fraction is a more accurate indicator of left ventricular function than left ventricular end-diastolic pressure or volume (13).

After a myocardial infarction, the pathophysiology of left ventricular remodelling has been thoroughly documented. Briefly, repeated or chronic myocardial ischemia causes cardiomyocytes to remodel maladaptively and extend extracellular matrix, which results in cavity enlargement and systolic dysfunction (14).

An increase in LVEDP of 0.8 mmHg for every 0.05-1.1 increase in coronary segmental stenosis score was found in a foreign clinical study that looked at the relationship between the degree of coronary stenosis and left ventricular diastole. This finding suggests that myocardial ischemia, which results from inadequate coronary blood supply, lowers left ventricular compliance. Myocardial ischemia caused by the severity of coronary artery disease contributed to diastolic insufficiency. Coronary artery disease, together with the ischemia and hypoxia alterations it causes in the small arteries, can reduce diastolic function, and that the severity of the coronary artery disease is inversely correlated with the diastolic function. Additionally, myocardial atherosclerosis may lessen the compliance of the coronary arteries and so constrain the heart's ability to contract and go into diastole. A loss in diastolic function might hypothetically come from atherosclerosis of the coronary arteries, which can lessen the flexibility of the vessels and have a constricting effect on the heart. The LAD and associated small artery lesions were mostly responsible for the change in LV diastolic performance. Additionally, the diastolic performance is worse in the absence of outside assistance the more extensive the LAD lesion is. As the stent opened the conduit and the coronary vessels adapted to the stent, allowing vascular compliance to return to normal after a year, the patient's left ventricular function essentially restored to normal (15).

Lv et al. (16) confirmed that coronary atherosclerosis can cause a decrease in the compliance of the coronary arteries travelling on the surface of the heart and the small vessels distributed between the myocardia. It was also discovered that as the degree of coronary stenosis increased, LV diastolic function gradually decreased. They revealed a direct correlation between the severity of the LV diastolic dysfunction decrease and the degree of LAD vascular damage and LAD stent insertion.

The LIFE study researchers investigated the effect of CAD in Patients with hypertension who have left ventricular hypertrophy on left ventricular systolic geometry and function. The study hypothesized that there is a direct connection between clinical signs of coronary artery disease (CAD) and changes in LV structure and function that raise their risk. 963 hypertensive patients (mean age 66.7 years, 41% women) with electrocardiographic LV hypertrophy had their echocardiograms recorded. These patients were divided into 149 with and 814 without CAD, as determined by clinical (previous myocardial infarction or angina pectoris) or electrocardiographic evidence. LV interior dimensions were greater in CAD patients, increased LV mass, lower ejection fraction, higher circumferential end-systolic wall stress (cESS), and higher total peripheral resistances. The study concluded that persons with structural and functional abnormalities are identified as being at high risk for cardiovascular events when they have clinical evidence of CAD in hypertensive patients and electrocardiographic evidence of LV hypertrophy. In hypertensive patients with CAD, the noninvasive LV mass, cESS, and ejection time index, which mirrors myocardial oxygen demand per beat, is particularly high (17).

In the INDYCE survey, It was assessed the LVEF in French outpatients with stable CAD. In conclusion About 30% of patients with stable CAD have moderate or severe left ventricular systolic dysfunction, even when LVEF is generally intact. In some people, it may be completely asymptomatic and go unnoticed despite the fact that it should alter the prognosis and course of treatment. They showed that stable CAD patients require ongoing systolic ventricular function evaluation.

Cardiovascular magnetic resonance imaging was performed on a total of 282 Type 2 Diabetes patients, including 104 patients with CAD (T2DM (CAD +)) and 178 without [T2DM (CAD -)] as well as 83 healthy

controls with similar ages and sexes. Measurements were made of the LV's structure, function, global strains (including peak systolic and diastolic strain rates in the radial, circumferential, and longitudinal directions), and late gadolinium enhancement (LGE) characteristics. Based on the median Gensini score (60), which was generated to evaluate the severity of CAD, T2DM (CAD +) patients were split into two subgroups. The causes of decreased LV function were investigated using multivariable linear regression analysis.

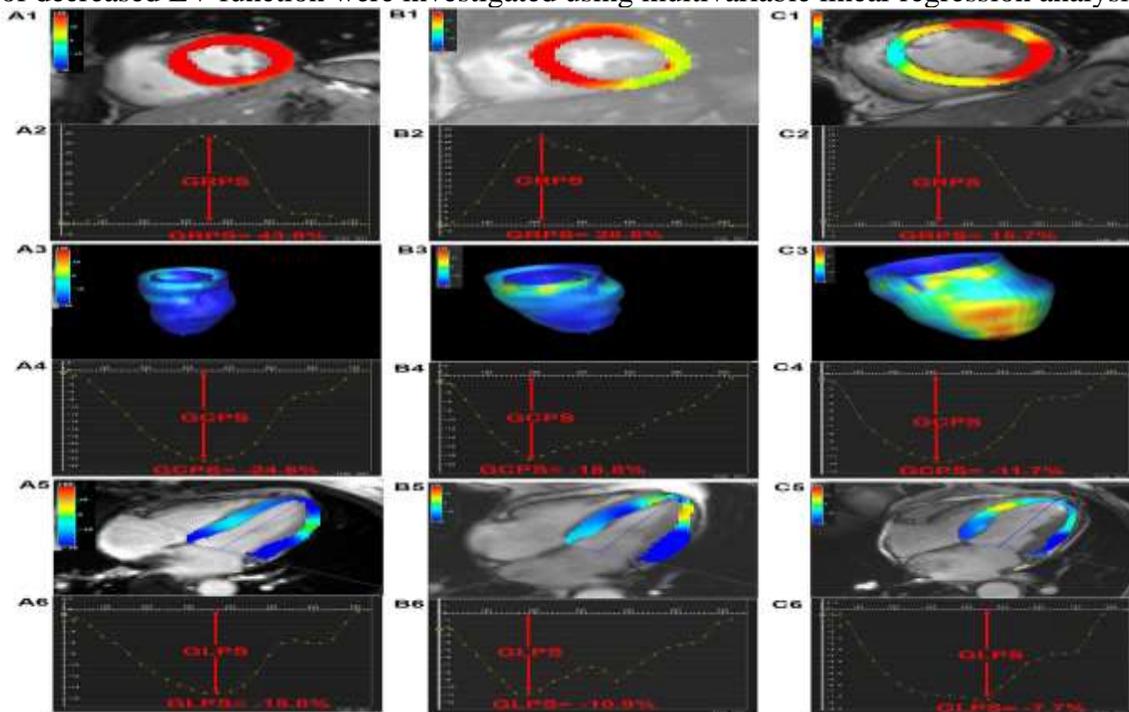


Figure (1): Representative CMR pseudo-color scans of a normal control, a patient with T2DM (CAD), and a patient with T2DM (CAD +) at end-systole and on the entire PS curve. A1-C1 pseudo-color images of LV radial PS in short axis; A3-C3 three-dimensional pseudo-color maps of LV circumference; A5, B5, C5 pseudo-color images of LV longitudinal PS in four-chamber long axis; A2-C2 LV global PS curve in radial direction; A4, B4, C4 LV global PS curve in circumferential direction; and A6, B6, C6 LV global PS curve in longitudinal direction. PS peak strain, the left ventricle, type 2 diabetes mellitus, coronary artery disease, and CMR (cardio magnetic resonance).

In conclusion, CAD negatively impacted LV systolic and diastolic function in those with T2DM. Reduced contractile and diastolic performance was linked to the Gensini score, which measures the severity of CAD in T2DM (CAD +) patients. The exploration of the underlying processes by which the coexistence of these two stimuli (CAD and DM) influences the myocardium and how these effects could be avoided in the future should be promoted (18).

Coronary artery disease alters diastolic function to varying degrees. Following myocardial infarction and acute ischemia, certain anomalies are generated. In these two diseases, the etiology of diastolic anomalies is discussed. The left ventricle's filling pressures rise during acute ischemia. Pulmonary oedema could develop. Less of a rise in filling pressures results with silent ischemia. With varying contributions from altered myocardial relaxation, increased muscular stiffness, acute pericardial constriction, ventricular interaction, and acute chamber dilatation, the diastolic pressure-volume ratio is moved upward. Because of the factors that change the pressure-volume relationship, the left ventricle's ability to fill during ischemia is altered. Given the decreased left ventricular compliance, the abrupt rise in left atrial pressure may raise filling rates relatively unexpectedly. Filling pressures may increase as a result of myocardial fibrosis after an infarction, although the extent of the increase is tightly related to the intravascular volume status. A loss of chamber compliance caused by an increase in muscle stiffness is reflected in changes in the diastolic pressure-volume relation. This permanent increase in muscle stiffness, which is essential to the diastolic irregularities in chronic coronary artery disease, is brought on by increased extracellular matrix, notably collagen (19).

Eleven patients with coronary artery disease were examined for left ventricular diastolic function. During a period of angina that followed a period of fast atrial pacing, single plane ventriculography (30 degrees right anterior oblique projection) was done. A catheter with a micromanometer tip simultaneously measured the left ventricular pressure. End systolic volume increased and ejection fraction decreased in angina, while end diastolic volume did not alter significantly. Although the diastolic pressure-volume ratio and the lowest and end diastolic pressures of the left ventricle both increased, the peak left ventricular pressure remained unaltered. The left ventricular pressure fall's time constant was extended. At rest, the left ventricle received more than 50% of the stroke volume during the early diastole phase. As a result, a substantially bigger percentage of the stroke volume entered the ventricle during late diastole because this proportion was significantly decreased during angina. Angina pectoris significantly impairs left ventricular function in early diastole, and it is proposed that loss of elastic recoil and dissipation of this restoring force by asynchronous onset of relaxation and abnormal shape changes are significant contributors to this disturbance of function. In CAD, the right ventricle frequently takes part. The many right ventricular involvements can be seen using the imaging modalities that are currently available, providing fresh insights into pathogenesis, medical treatment, and management (20).

200 people with recent chest pain who had been initially diagnosed with acute ST-segment elevation myocardial infarction (MI) participated in an observational cross-sectional study. An emphasis on the tricuspid annular plane systolic excursion (TAPSE) score was given during the echocardiography procedure. Major adverse cardiac events (MACE), also known as all-cause mortality, cardiovascular mortality, recurrent MI, heart failure, or stroke in patients with acute myocardial infarction (AMI), are among the in-hospital clinical outcomes. Patients developed AWTMI in 68% of cases and IWTMI in 32%. According to the TAPSE score, patients with AWTMI had higher right ventricular dysfunctional alterations than those with IWTMI. MACE was more common in AWTMI (27.1%) than IWTMI (12.5%) (right ventricular dysfunction was present in 41.9% vs. 18.75%, respectively). In terms of mortality (17.64% in AWTMI vs. 6.25% in IWTMI), the outcome of patients with AWTMI was worse than that of patients with IWTMI. Patients with AMI with a TAPSE score of 18 mm, which indicates right ventricular dysfunction, had a greater rate of MACE, respectively. It can be inferred from this study that AWTMI causes more right ventricular dysfunction than IWTMI does. Additionally, compared to patients with AMI without right ventricular dysfunction, individuals with AMI who also had concomitant right ventricular dysfunction had worse outcomes in terms of a lengthier hospital stay, a higher incidence of MACE, and a higher fatality rate (21).

Showkat et al. (22) demonstrated that the presence of RV dysfunction as measured by echocardiography (RVEDD, or RV end diastolic diameter), TAPSE, FAC, E/E', and RV MPI, or myocardial performance index, by TDI (tissue Doppler imaging) was common in acute MI (STEMI/NSTEMI), regardless of the location of the infarction, and was more prevalent in AWTMI than IWTMI. This study showed that, regardless of the site of infarction, patients with RV dysfunction had a significant rate of morbidity and mortality in acute MI, more so in STEMI than NSTEMI. To evaluate the state of right ventricular function in patients having an invasive surgery who have NSTEMI, 150 patients eligible for reperfusion therapy by invasive percutaneous coronary intervention were admitted with a first NSTEMI. Two groups of these patients were created: group A, which included those with normal RV function, and group B, which included those with impaired RV function as determined by the tricuspid annular plane systolic excursion (TAPSE) threshold value of 17 mm. All patients had angioplasty procedures and were monitored for three months while still in the hospital. 95 individuals (61.3%) in the research sample experienced RV dysfunction. After three months, TAPSE significantly improved when compared to TAPSE at baseline. When compared to baseline, those with poor RV function had improved TAPSE scores after three months. The independent predictors of RV dysfunction according to multivariate analysis were RVEDD > 26 mm, RVFAC 35%, RAA > 20 cm², and TAPSE 17 mm. When utilising the criteria of TAPSE 17 mm, RV dysfunction in NSTEMI is not uncommon. TAPSE monitoring of RV function revealed a marked improvement over baseline following 3 months of effective PCI (23).

To assess the predictive significance of RV diastolic dysfunction in patients with inferior STEMI and RVI, **El Amrawy et al. (24)** enrolled sixty patients with inferior STEMI and RV infarction, who underwent primary

PCI. Patients who had a history of clinical problems that might have impacted RV function were not included. The RV systolic and diastolic functions were evaluated with echocardiography within twenty-four hours of the PCI, with a focus on tricuspid inflow velocities (E velocity, A velocity, and E/A ratio) by pulsed wave (PW) doppler and tricuspid annular velocities by tissue doppler index (TDI) (E', A', and E/E' ratio). Within a three-month follow-up period, clinical characteristics and MACE, such as cardiogenic shock, arrhythmia, stroke, reinfarction, and mortality, were examined in all of our patients. 10% of the participants in the study were female, and their average age was 51.58 ± 10.11 years. Five patients experienced MACE, four of which happened while they were in the hospital during the first 48 hours (death, cardiogenic shock and pulmonary edoema, anterior STEMI and cardiogenic shock, recurrent inferior STEMI, and arrhythmia and stroke). All of the MACE patients had high filling pressures because their E/E' ratios were greater than 6. As 25% of patients with E' velocity ≤ 6 experienced MACE compared to 2.3% of patients with E' velocity > 6 , with a p value of 0.015, E' velocity ≤ 6 cm/sec was linked to an elevated risk of MACE. The study came to the conclusion that Tricuspid annular velocities measured by TDI are crucial for assessing RV diastolic dysfunction. In patients with inferior STEMI with RV infarction, E/E' and E' velocity have predictive value; E/E' > 6 and E' velocity ≤ 6 cm/sec were related with a higher MACE rate in these patients (24).

To evaluate right ventricular functions using echocardiography in individuals with proximal right coronary artery (RCA), CTO and to correlate those results with clinical and angiographic factors, with no additional substantial left coronary system lesions, **Missiri and Guindy (25)** looked at 60 consecutive patients with CTO of the proximal RCA. Echocardiography was performed in order to measure left ventricular (LV) dimensions, LVEF, RV end diastolic area (RVEDA), RV end systolic area (RVESA), RV fractional area change (RVFAC), tricuspid annular plane systolic excursion (TAPSE), Doppler myocardial performance index (MPI), trans-tricuspid E and A waves, E/A ratio, S', e' and a'. Coronary collaterals were evaluated based on their grade. With TAPSE and RVFAC, Doppler MPI displayed a negative correlation. E/e' ratio and trans-tricuspid E/A ratio had a positive connection. More fully established (grade 3) coronary collaterals (83.8 vs. 17.4%) were found in patients who had no signs of MI. Patients with evidence of MI had lower RVFAC, lower TAPSE and higher MPI, higher trans-tricuspid E/A ratio, and E/e' ratio. TAPSE and RVFAC were lower and MPI was higher in patients with poorly formed collaterals. RV functions are significantly impaired in patients with proximal RCA CTOs and indications of inferior wall MI, in addition to having poorly established coronary collaterals. The RV functions are improved in patients with proximal RCA CTOs and well-developed coronary collaterals.

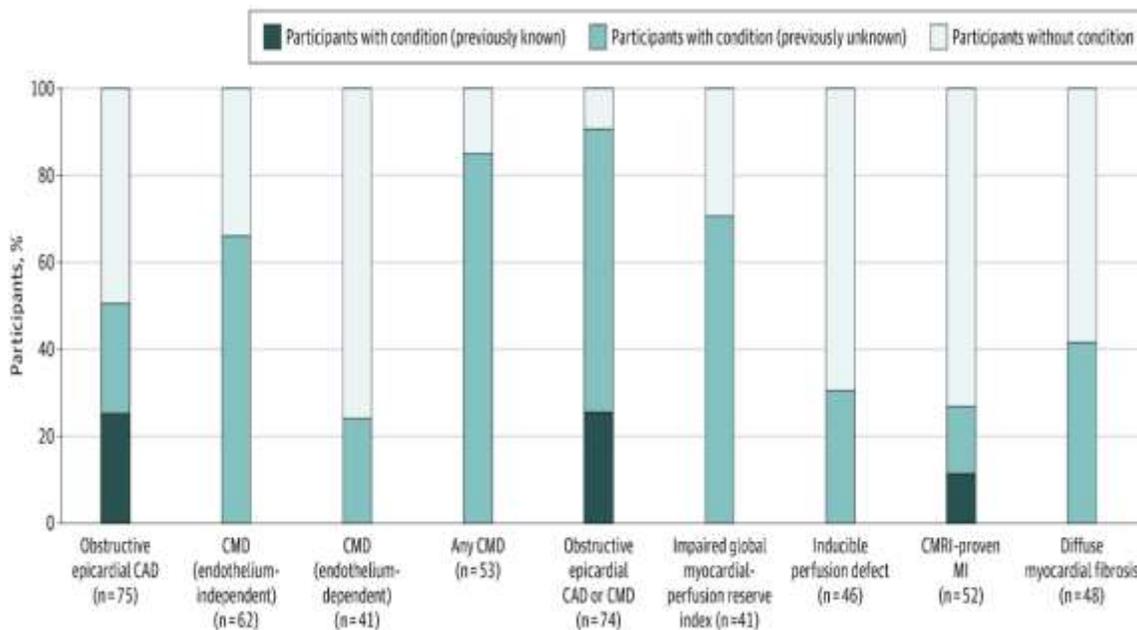


Figure (2)

To determine the prevalence of CAD and CMD in HFpEF hospital patients, **Rush et al. (26)** evaluated a total of 106 consecutive patients hospitalized with HFpEF. Data analysis took place between March 4 and September 6, 2019. Prior to coronary vasoreactivity testing, participants had coronary angiography with guidewire-based measurements of coronary flow reserve, index of microvascular resistance, and fractional flow reserve. Extracellular volume was measured using late gadolinium enhancement during cardiac magnetic resonance imaging. The myocardial-perfusion reserve index was used to qualitatively and semi-quantitatively evaluate myocardial perfusion. 91% of the patients with HFpEF in this cohort research had epicardial CAD, CMD, or both present. 81% of people with CMD had no obstructive CAD. Hospitalised individuals with HFpEF tend to frequently have obstructive epicardial CAD and CMD, which may be therapeutic targets. **(26)**

In the treatment of coronary heart disease (CHD), a cardiac rehabilitation and prevention programme (CRPP) is a well-known nonpharmacological technique. However, the impact of a CRPP on the heart's systolic performance is debatable, and there is no information on the diastolic function in CHD. To address these problems, a randomised, controlled trial was carried out. Patients (n = 269) who had recently undergone percutaneous coronary intervention (PCI) (n = 76) or experienced an acute myocardial infarction (n = 193) were randomly assigned to receive either CRPP (2-hour twice-weekly exercise programme for 8 weeks) or standard treatment (control group). Phases 1 (baseline), 2 (post-exercise training), and 3 (8-month follow up) involved serial treadmill exercise testing and at-rest echocardiography. Only in phase 3 (65% vs 88%, $p = 7.6$) did the prevalence of left ventricular (LV) aberrant relaxation pattern (ARP) indicating diastolic dysfunction increase in the control group. In the CRPP group, particularly in individuals with recent acute myocardial infarction or ARP, significant improvement of individual LV diastolic measures towards less severe delayed relaxation was also seen. In the CRPP group compared to the control group, the increase in exercise capacity came more quickly and significantly, and it was also significantly connected with LV diastolic indicators in individuals with ARP. The rate-pressure product and LV systolic function were unaffected by exercise training. Without impacting systolic function, CRPP in CHD patients stopped the evolution of resting LV diastolic dysfunction. The increase in exercise capacity in people with ARP was predicted by the improvement in diastolic function **(27)**.

Zhang et al. (28) investigated the role of exercise after percutaneous coronary intervention (PCI). Randomized trials comparing exercise to no exercise after PCI in patients with coronary heart disease (CHD) were conducted. To compare the curative effect of exercise programme with exclusive exercise programme after PCI, left ventricular end diastolic diameter (LVEDD), left ventricular ejection fraction (LVEF), 6-minute walking distance (6MWD), cardiac death, myocardial infarction, coronary angioplasty, coronary artery bypass surgery (CABG), and angina pectoris or restenosis per randomized patients were all examined. Analysis was done on ten randomised controlled trials with 1274 participants (636 in the exercise group and 638 in the control group). They revealed that exercise following PCI may be advantageous for those with CHD by increasing LVEF, lowering risk of cardiac mortality, myocardial infarction, coronary angioplasty, angina pectoris, and restenosis, and improving LVEF. Exercise had no discernible impact on LVEDD, 6MWD, or CABG in CHD patients after PCI. It is still difficult to implement a revascularization approach in individuals with acute myocardial infarction (AMI) with multivessel coronary disease. Complete percutaneous revascularization during index hospitalization is one of the possible treatments. This approach might improve left ventricular ejection fraction (LVEF). Records of 171 patients whose CCR was carried out in stages during first admission and who had been diagnosed with AMI and multi-vessel coronary artery disease (CAD) on index angiography were evaluated. Clinical information was gathered through hospital discharge letters and in-hospital medical records. One day prior to release, a cardiac ultrasound (CU) was done, with the LVEF in particular being assessed. Complete revascularization (CR) performed at the initial stage in patients with AMI and multi-vessel disease is linked to an increase in LVEF and a decrease in the MACE rate. Poorer LVEF patients have worse clinical outcomes and have a greater rate of MACE **(29)**.

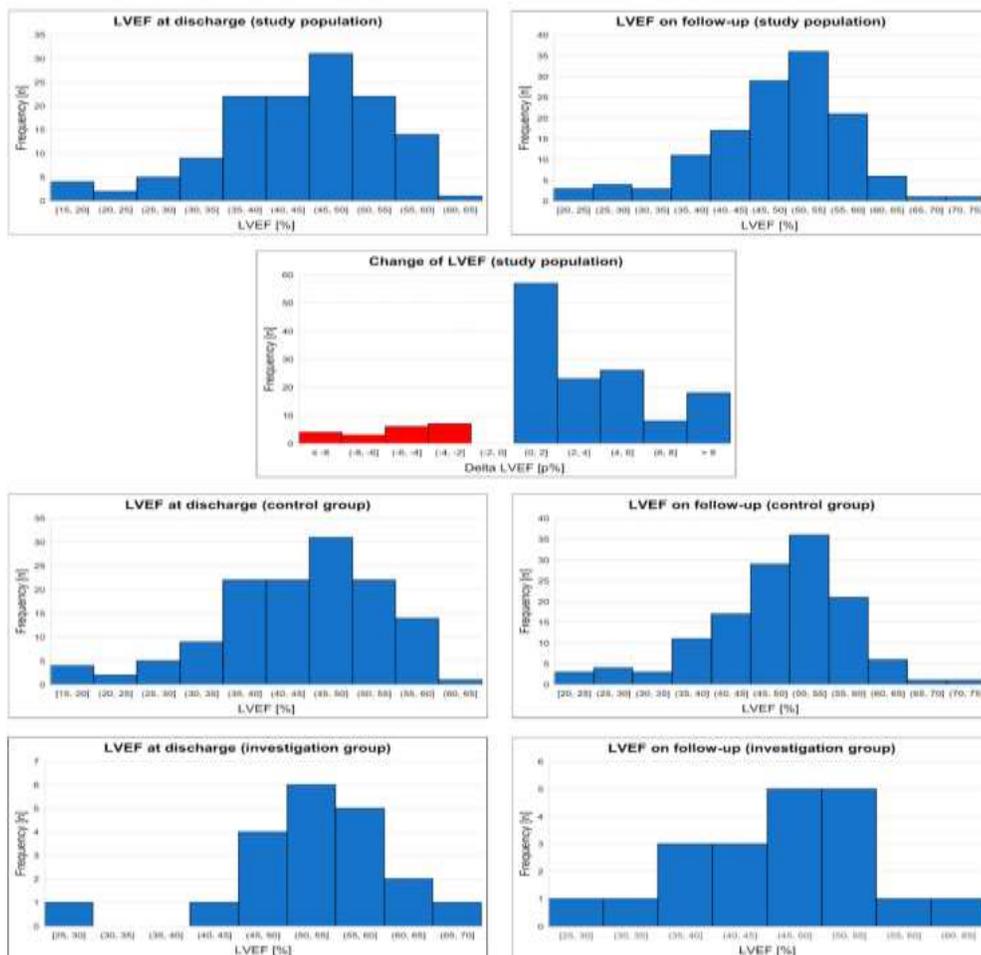


Figure (3)

These studies and trials highlight the significance of determining dominance in coronary artery anatomy within multiple aspects of treating cardiac pathology. The increased risk seems to correlate with left heart dominant circulation, but more research has to be done to determine if different strategies need to be implemented to ensure better outcomes.

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